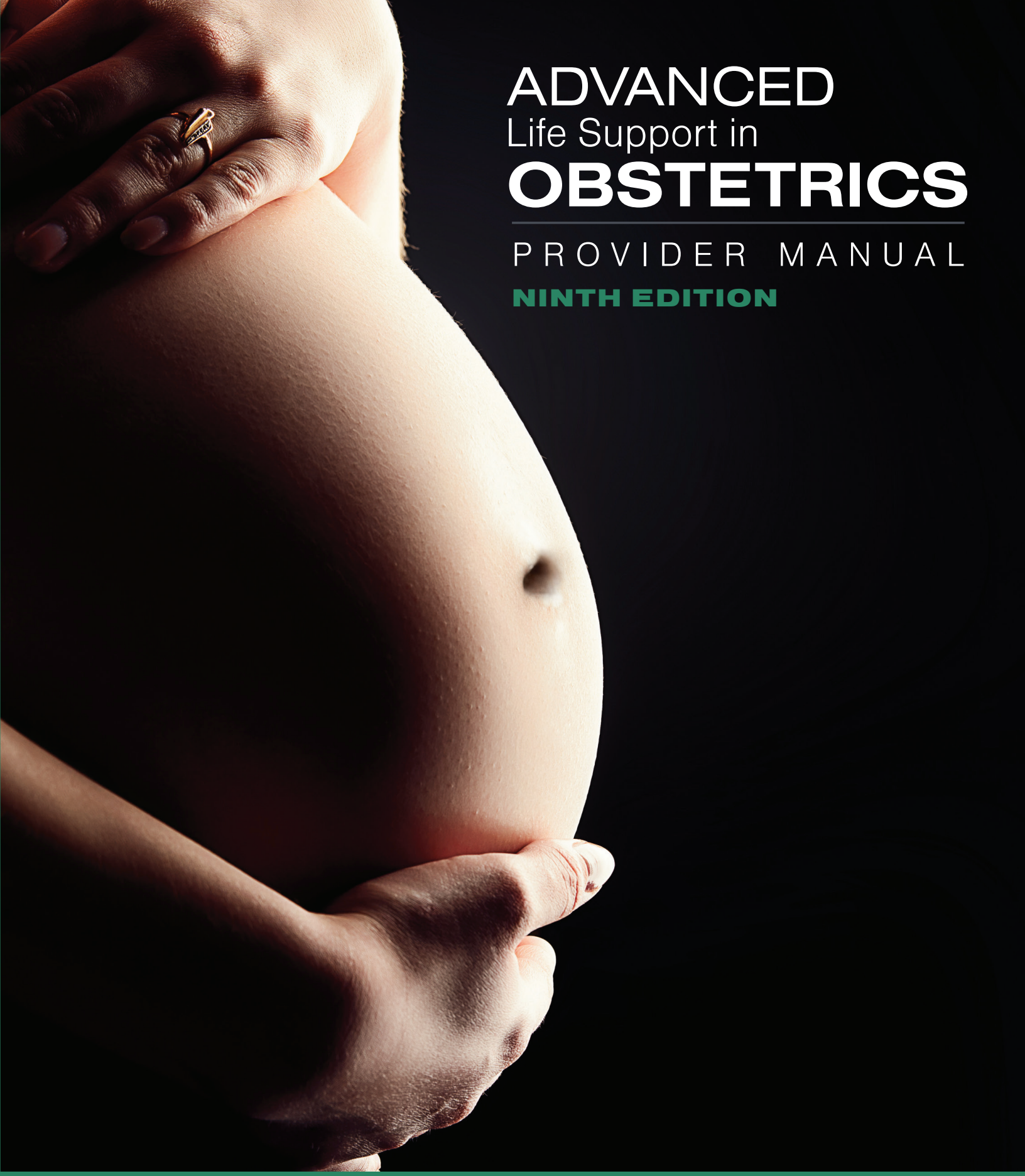


ADVANCED Life Support in OBSTETRICS

PROVIDER MANUAL

NINTH EDITION

ALSO

Editors

Lawrence Leeman, MD, MPH
Medical Editor

Lee Dresang, MD
Jeffrey D. Quinlan, MD, FAAFP
Susanna R. Magee, MD, MPH
Associate Medical Editors

AAFP Staff

Sarah Meyers, MHA
CME Maternity Care Manager, Continuing Professional Development Division

Gaylynn Butts, BSN, RN
ALSO Program Strategist, Editorial Assistant, Continuing Professional Development Division

Carla Cherry
ALSO Program Specialist, Continuing Professional Development Division

Jennifer Head
ALSO Program Specialist, Continuing Professional Development Division

Elaine Kierl Gangel
Managing Editor, Journal Media Division

Brandon Nelson
Associate Editor, Journal Media Division

Stacey Herrmann
Production Design Manager, Journal Media Division

R. Shawn Martin
Executive Vice President and Chief Executive Officer

Clif Knight, MD, FAAFP
Senior Vice President for Education

Medical Illustrations by Lisa Clark

Authors

Janice M. Anderson, MD, FAAFP
Postpartum Hemorrhage

R. Eugene Bailey, MD, FAAFP
Intrapartum Fetal Surveillance

Melissa Beagle, MD
First-Trimester Pregnancy Complications

Rebecca L. Benko, MD, FAAFP
Preterm Labor and Prelabor Rupture of Membranes

Timothy Canavan, MD, MSc
Third- and Fourth-Degree Perineal Lacerations

Brendon Cullinan, MD
Maternal Resuscitation and Trauma

Mark Deutchman, MD, FAAFP
Diagnostic Ultrasound in Labor and Delivery, First-Trimester Pregnancy Complications

Lee Dresang, MD
Safety in Maternity Care, Venous Thromboembolism in Pregnancy

Ann E. Evensen, MD, FAAFP
Postpartum Hemorrhage

Robert W. Gobbo, MD, FAAFP
Shoulder Dystocia

Jessica T. Goldstein, MD, FAAFP
Cardiac Complications of Pregnancy

Gretchen Heinrichs, MD, OB/GYN
Late Pregnancy Bleeding

Kim Hinshaw, MBBS, FRCOG
Shoulder Dystocia

Caroline S. Homer, MScMed, PhD
Birth Crisis

Sarah Jones, MD, FAAFP
Venous Thromboembolism in Pregnancy

Paul Koch, MD
Diagnostic Ultrasound in Labor and Delivery

Lawrence M. Leeman, MD, MPH
Hypertensive Disorders of Pregnancy, Malpresentations, Malpositions, and Multiple Gestation

Paul Lewis, RM
Birth Crisis

Susanna R. Magee, MD, MPH, FAAFP
Assisted Vaginal Delivery

Neil J. Murphy, MD
Maternal Resuscitation and Trauma, Cesarean Delivery

Lauren Plante, MD, MPH
Cardiac Complications of Pregnancy

Stephen Ratcliffe, MD, MSPH
Labor Dystocia

Elizabeth Rochin, PhD, RN
Safety in Maternity Care

Jose A. Rojas-Suarez, MD
Maternal Sepsis

Sara Shields, MD, FAAFP
Labor Dystocia

Niza Suarez Rueda, MD
Maternal Sepsis

Mary Beth Sutter, MD, FAAFP
Assisted Vaginal Delivery

Barbara A. True, RN
Intrapartum Fetal Surveillance

Johanna B. Warren, MD
Shoulder Dystocia

Kerry Watrin, MD
Preterm Labor and Prelabor Rupture of Membranes

Helen Welch, CNM
Birth Crisis

Conflicts of interest: *It is the policy of the AAFP that all individuals in a position to control content disclose any relationships with commercial interests upon nomination/invitation of participation. Disclosure documents are reviewed for potential conflicts of interest and, if identified, conflicts are resolved prior to confirmation of participation. Only those participants who had no conflict of interest or who agreed to an identified resolution process prior to their participation were involved in this CME activity.*

The following individual(s) in a position to control content for this activity have disclosed the following relevant financial relationships:

• Mark Deutchman, MD disclosed a consultant or advisory board relationship with Signostics – Echonous Ultrasound, research/grant support with Signostics – Echonous Ultrasound (diagnostic ultrasound), stock/bond holdings with Signostics – Echonous Ultrasound (diagnostic ultrasound), and he holds a US patent on a medical device (fetal vacuum extractor) with Cooper Surgical.

Related Chapter(s): Diagnostic Ultrasound

All other individuals in a position to control content for this activity have indicated they have no relevant financial relationships to disclose.

Preface

The ALSO Provider Course is an educational program designed to assist health professionals in developing and maintaining the knowledge and skills needed to effectively manage the emergencies which arise in maternity care. The course includes required reading, lectures, and hands-on workstations. Evaluation is by a written exam and skills assessment stations. There are many appropriate ways of managing emergencies. The treatment guidelines presented in ALSO do not necessarily represent the only way to manage problems and emergencies. Instead, these guidelines are presented as reasonable methods of management in obstetrical emergencies. Each maternity care clinician must ultimately exercise his or her own professional judgement in deciding on appropriate action in emergency situations. Completion of the ALSO Provider Course does not imply competency to perform the procedures discussed in the course materials.

Overall Course Objectives

- Discuss methods of managing pregnancy and birth urgencies and emergencies, which standardizes the skills of practicing maternity care providers.
- Demonstrate content and skill acquisition as evidenced by successful completion of course examination, skills workstations, and group testing stations.
- Provide safe team leadership through various emergency obstetric scenarios.
- Demonstrate effective team communication strategies focusing on patient safety.

Copyright

Course Disclaimer

The material presented at this course is being made available by the AAFP for educational purposes only. This material is not intended to represent the only, nor necessarily best, methods or procedures appropriate for the medical situations discussed, but rather is intended to present an approach, view, statement, or opinion of the faculty which may be helpful to others who face similar situations. The AAFP disclaims any and all liability for injury, or other damages, resulting to any individual attending this course and for all claims which may arise out of the use of the techniques demonstrated therein by such individuals, whether these claims shall be asserted by a physician, or any other person. Every effort has been made to ensure the accuracy of the data presented at this course. Physicians may care to check specific details, such as drug doses and contraindications, etc, in standard sources prior to clinical application. The AAFP does not certify competence upon completion of the ALSO Provider Course, nor does it intend this course to serve as a basis for requesting new or expanded privileges.

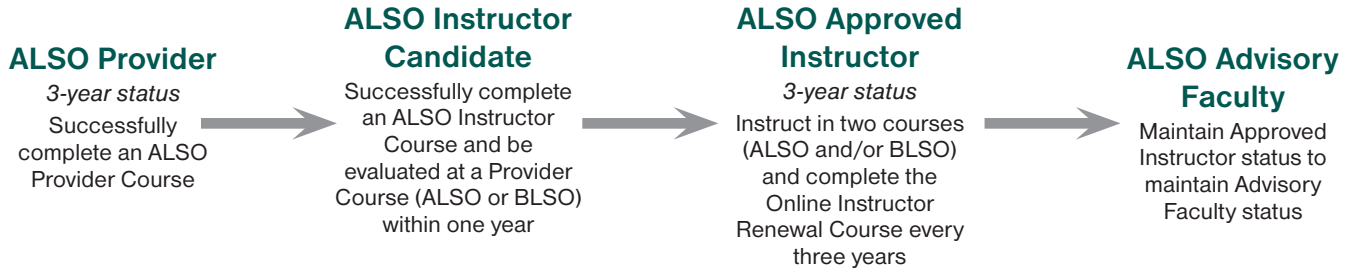
The American Academy of Family Physicians (AAFP) wishes to acknowledge the initial development of the ALSO Program by the University of Wisconsin Department of Family Medicine and the original national ALSO Development Group of family physicians, obstetricians, and nurses, which formed in 1991. The ALSO Program, originally conceptualized by James R. Damos, MD, was developed under the leadership of Dr Damos and John W. Beasley, MD. The AAFP acquired the ALSO Program in 1993.

The curriculum demonstrates the evidence, and quality of that evidence, on which any recommendations of care are based.

The current ALSO Provider Manual continues to be an ongoing process and is reviewed on a 3-year cycle.

11400 Tomahawk Creek Parkway, Leawood, Kansas 66211-2672
1-800-274-2237 • 913-906-6000

Advanced Life Support in Obstetrics (ALSO®)
Status Life Cycle



ALSO Providers and Approved Instructors are responsible for maintaining their own status.

Wallet Cards include status start and expiration dates and can be accessed in AAFP accounts/transcripts.

Please review the ALSO Guidelines document at www.aafp.org/also for further details.

To find out more about the ALSO program and upcoming courses, please visit www.aafp.org/also.

Table of Contents
November 2020

Safety in Maternity Care

Learning Objectives

1. Discuss need for a patient safety focus and team-based approach to maternity care
2. Demonstrate teamwork tools that improve safety, including closed loop communication and application of the steps of evidence-based mnemonics
3. Explain risk management issues in obstetrics and possible solutions (The Five Cs)

Introduction

On January 15, 2009, US Airways flight 1549 lost thrust in both engines. The plane landed on the Hudson River near New York City, and all 155 individuals aboard survived. Teamwork and communication were cited as key factors in the excellent outcome. Preflight training and simulations prepared airline personnel for their roles when the accident occurred. Communication was effective between pilots, crew members, passengers, ground control, and rescuers. Everyone contributed to the successful outcome.

The odds of being killed in an airline crash has decreased to just 1 in 4.7 million flights.[1] Much of this success has been attributed to Crew Resource Management (CRM), which focuses on safety, protocols, excellent communication, checklists, and other tools.[1] A 1979 National Aeronautics and Space Administration workshop introduced CRM to aviation in response to a 1978 crash of a United Airlines DC-8 in Portland, where a pilot was unable to maintain awareness of critical aspects of flying under stressful conditions.[2]

Successful aviation safety strategies, such as CRM, can be applied to medical care. The focus on saving lives and improving outcomes through teamwork, communication, and system improvement is referred to as *patient safety*. The Institute of Medicine defines patient safety as "the prevention of harm to patients."[3]

Advanced Life Support in Obstetrics and Patient Safety

The mission of the Advanced Life Support in Obstetrics (ALSO) program is strongly focused on patient safety. From its inception in 1991, ALSO courses have promoted interdisciplinary teamwork and brought together maternity care providers in the US and over 60 other countries. The courses ideally include participants from different disciplines and settings, which may provide a more realistic approach to team-based training. Attending physicians (family medicine, obstetrics/gynecology, and emergency medicine), midwives, nurses, residents, and students have participated in ALSO courses. Providers from rural and urban, and academic and community programs can learn from each others' experiences and perspectives.

ALSO added a *Safety in Maternity Care* chapter to its curriculum in 2002. The chapter highlights the importance of addressing teamwork and systems issues in the provision of quality medical care. ALSO transitioned to a flipped classroom model in 2017 and eliminated all lectures from the in-person portion of the course, except the *Safety in Maternity Care* lecture. Even before the transition, courses began with the *Safety in Maternity Care* lecture to emphasize the importance of the teamwork, communication, and systems lessons that are taught throughout the course's content.

ALSO promotes safety by teaching a standardized approach to obstetric emergency situations. Standardization is a key patient safety element that can reduce variation in practice and duplication of time and resources, and provide reliability of patient care procedures. Knowledge of the content, practice of manual skills, and use of mnemonics reduce the likelihood of error and the incidence of maternal and fetal morbidity and mortality.

Importance of Safety in Maternity Care

Approximately 303,000 women died from childbirth related causes worldwide in 2015.[4] A United Nations Sustainable Development Goal is to reduce global maternal mortality from 216 per 100,000 live births in 2015 to less than 70 per 100,000 by 2030.[4] Childbirth is the most common reason for hospital admission,

accounting for 11%, and cesarean delivery is the most common operative procedure performed in the United States.[5] With more than 4 million births occurring in the United States annually,[5] this equates to more than 80,000 adverse obstetric events. Public health and hygiene improvements, advancements in technology, development of targeted drugs, increased training of nurses and physicians, and the development of a regionalized approach to perinatal care have combined to reduce the overall risk of death and disability related to childbirth in the past century.

However, as discussed later in this chapter, maternal mortality has increased in the United States, even though it has decreased in most low- and high-resource countries since 1990.[6] An exception is California, where maternal mortality decreased during this period. California's success often is attributed to its patient safety bundles, which are being implemented nationwide with the hope of achieving similar improved outcomes.[6,7]

According to The Joint Commission, between 2004 and 2014, communication was the root cause in 48% of maternal and 70% of perinatal sentinel events.[8] A root cause is the fundamental reason for an adverse event and a point where an intervention may have avoided the adverse outcome. Technology and medical knowledge continue to advance, but women and infants continue to die or experience adverse outcomes. When this occurs, poor communication often is the root cause. If the communication and actions of maternity care providers can be improved, lives can be saved. Communication and teamwork skills are taught at the start of the live portion of the ALSO course and practiced throughout the required workstations, where clinicians address obstetric emergencies as cohesive teams in a simulated in-situ patient care setting.

Even highly trained and dedicated medical professionals make mistakes. Fortunately, most errors do not result in harm, and fatal errors are relatively rare.[9] Nonetheless, an estimated 44,000 to 98,000 Americans die each year of preventable medical errors.[10,11] A 2013 study showed this number to be between 210,000 and 400,000, with nonlethal errors being 10 to 20 times more common.[12] This means that preventable medical errors are the third leading cause of death in the United States, after heart disease and cancer.[13] Seven percent of hospital patients experience a serious medication error; more Americans die each year due to medical errors than of breast cancer, AIDS, or vehicle collisions.[11] The cost associated with medical errors is estimated at $17 billion to $29 billion annually.[11]

"We cannot change the human condition, but we can change the conditions under which humans work."[14]

Assessment of human factors has become a core process in the review of preventable errors. According to The Joint Commission, human failures cause 80% to 90% of errors.[15] The ability to recognize the integral connections that procedures, technology, and humans form within health care is essential in the reduction of preventable errors. A component of this strategy is the use of simulation and team-based training.

Team training has been a requirement of The Joint Commission since the National Safety Patient Goals became effective in 2003; these goals require hospitals to "incorporate(s) methods of team training to foster an interdisciplinary, collaborative approach to the delivery of patient care."[16] In addition, staff must participate in education and training that incorporate team communication, collaboration, and coordination of care.[17]

Although some adverse outcomes cannot be prevented, even with exemplary care provided under the best of circumstances, a significant proportion of these outcomes result from communication and system problems. One study showed that 87% of adverse events and potential adverse events were preventable, and that poor teamwork, protocol violation, and staff unavailability were the most common problems.[9]

"A team of experts does not make an expert team."[18]

Most maternity care units involve so many providers that a patient care team rarely involves the same people. For example, a maternity care unit with 81 obstetricians, 50 registered nurses, 16 anesthesiologists, 12 neonatal nurse practitioners, 14 surgical technologists, and 35 nurse anesthetists could result in 381 million different teams.[19] This high variability in team membership is a key threat to patient safety. Even the most knowledgeable and skilled specialist cannot function to the best of his or her ability without support from a wide array of colleagues. Because working with the exact same team is such a rarity, it is not effective to train a particular team to work well together. Instead, all members of a

health care team should be trained in effective, standardized communication techniques, so that every clinician is prepared to function within each of the many teams with which they will interact.

Evidence for Teamwork Improving Outcomes

A growing body of evidence shows that improving teamwork improves outcomes. The University of Minnesota and the Fairview Health System in Minneapolis have provided an evidence-based framework for the dissemination of in-situ simulations to enhance interdisciplinary communication and teamwork.[19,20] A 2011 study documented a persistent and statistically significant 37% decrease in perinatal morbidity at a hospital with standardized teamwork training and regular in-situ simulations compared with no change at a hospital with standardized teamwork training alone and a control hospital where neither were taught.[21] The Weighted Adverse Outcome Score (WAOS) and maternal severity index improved 50% after the implementation of teamwork training on a maternity care unit at the Harvard-affiliated Beth Israel Deaconess Medical Center.[22]

A randomized controlled trial comparing the American Academy of Pediatrics Neonatal Resuscitation Program course with and without additional teamwork training showed that individuals who underwent standardized teamwork training in conjunction with the course demonstrated improved teamwork behavior at the end of the course.[23]

Standardized team training may not be enough, however. "The best team training in the world will not yield the desired outcomes unless the organization is aligned to support it. The next frontier lies in making effective teamwork, as seen in high-performance teams, an essential element in high-reliability organizations."[24]

Essential Elements of a Strong Maternity Care Team

Childbirth is an intense physical and emotional experience. As such, the maternity care team, with its focus on the pregnant woman, plays a vital role in well-being and outcomes. A woman's family members and support network often have an important and integral role. The health care team includes the birth attendant, nurses, support personnel (eg, nursing assistants), and consultants. The presence of a doula or professional support person and continuous labor support can increase the probability of spontaneous vaginal delivery and reduce the need for drugs and instrument delivery.[25]

Clinician strategies for supporting pregnant women include listening, anticipating potential problems, discussing options, reviewing birth plans, conferring at each decision point, and assessing for entrenched health beliefs, expectations, and concerns. Patient-centered interviewing, caring communication skills, and shared decision-making will promote effective patient-provider communication.[26] Involving women in their own care can improve outcomes, satisfaction, and adherence.[27]

Provider strategies for working with a woman's family and support network include developing relationships with a woman's partner and/or family, encouraging or expecting the woman and her family to be part of the perinatal team, assessing cultural norms and expectations, assessing family dynamics, encouraging attendance at childbirth classes, and acknowledging existing anger or anxiety.

The health care team can improve patient safety and satisfaction through effective communication, a readily available birth attendant, care teams, and consultants who are willing to assist in a timely manner. All team member contributions should be respected and encouraged. Characteristics of effective teams include having shared mental models; having clear roles and responsibilities; having a clear, valued, and shared vision; optimizing resources; giving and receiving assistance; managing and optimizing performance outcomes; having strong team leadership; engaging in a regular discipline of feedback; developing a powerful sense of collective trust and confidence; and creating mechanisms for cooperation and coordination.[28] Impediments to team function include personality conflicts, competitive pressures, fixed beliefs about abilities or roles, biases regarding management, and inadequate resources.

Occasionally, the provider and woman do not agree on the care plan. If this conflict cannot be resolved to both parties' satisfaction, transfer of care may be the preferred option. Documentation is always important, especially in cases of conflict. Providers should document that they explained the implications of the patient's decisions to the patient. In addition, frequent conversations with the care team with the patient present are important to continue development of transparency and clarity regarding the care plan and anticipat-

ing subsequent steps that might be necessary to promote a positive outcome.

When conflict occurs, several strategies can help. First, separate the people from the problem: be *hard* on the problem, *soft* on the people. Focus on what is right for the patient, not who is right; this includes focusing on interests, not positions, and focusing on concerns and desired outcomes. Create options for mutual gain by brainstorming to yield win-win solutions. Insisting on the use of objective criteria provides the basis for further improvement.[29]

At a system level, a rapid response team can be created to quickly assemble people with essential skills to respond to emergencies. An important part of developing an effective response team involves identifying appropriate triggers for activating the team. Early activation can improve outcomes.[30] Protocol should designate the role of different team members. Team function can be optimized through simulations, feedback, and quality review when activation occurs.

Teamwork Tools

Like the medical management and technical skills taught in the ALSO course, teamwork can be taught and learned. Important concepts and tools that can improve teamwork and patient safety include situational awareness, standardized language, closed-loop communication, mutual respect, and a shared mental model. It is important to have a standardized approach to teamwork tools within each hospital or health care organization that is supported by all levels of leadership.

Situational Awareness

In an emergency, it is easy to fixate on one particular task and lose sight of the overall situation. For example, a clinician may fixate on fetal heart rate decelerations and overlook elevated maternal blood pressure levels, headache, and hyperreflexia prior to an eclamptic seizure. Another clinician may focus on stopping preterm contractions, but miss signs and symptoms of an abruption and worsen the condition by administering a tocolytic. A clinician may focus on difficult family dynamics and fail to prepare the team to manage a shoulder dystocia despite a large estimated fetal weight and prolonged second stage of labor.

Team members can help each other remain aware of active issues and potential complications by cross monitoring. Early briefings followed by *huddles* when new issues arise can ensure that all team members have the same understanding of the situation. Situational monitoring is an important patient safety tool that facilitates situational awareness.

The acronym **STEP** (**S**tatus of patient, **T**eam members, **E**nvironment, **P**rogress towards goal) can be used to remember important components of situational monitoring.

Standardized Language

Inadequate communication at shift change can compromise patient safety. For example, failing to mention the presence of meconium at a sign-out that occurs just prior to delivery may result in inadequate newborn resuscitation preparation.

Call-outs. Call-outs are a strategy used to quickly inform all team members simultaneously when new critical events occur, particularly during an emergency when several caregivers are at the bedside. When managing a postpartum hemorrhage, a call-out of high blood pressure can alert the managing provider that methylergonovine is contraindicated. A call-out addressing the insertion of a Foley catheter may alert another team member to halt the process knowing the patient has a latex allergy.

SBAR. An acronym for **S**ituation, **B**ackground, **A**ssessment, and **R**ecommendation, SBAR is a standard communication technique for conveying critical information.[31,32] Use of SBAR in one institution resulted in a 72% to 88% improvement in updating patient medication lists on admission, and a 53% to 89% improvement in having a corrected medication list on discharge.[32] The rate of adverse events decreased from 89.9 per 1,000 patient days to 39.96 per 1,000 patient days.[32] SBAR can be an effective tool for communicating critical patient care information to any new team member who enters a room, a nurse calling out to a secretary to phone someone to come to a room, physician-nurse communication at shift changes, and between different specialty care providers.[33]

Situation – What is going on with the patient?

Background – What is the clinical background or context?

Assessment – What do I think the problem is?

Recommendation – What would I do to correct it?

Miscommunications in the transfer of care from one provider or care team to another can result in

life-threatening errors. Effective patient *handoffs* should include interactive communications, limited interactions, a process for verification, and an opportunity to review relevant historical data.[34]

Handoffs. Handoffs occur not only between providers, but also between levels of care or different hospital units such as labor and delivery and postpartum. One of the significant challenges in many countries is having an organized and respectful process for transferring a patient from her community care provider to prehospital transport, and timely referral and transport to the appropriate level of hospital care.

Closed-Loop Communication

Closed-loop communication means that the individual receiving a message confirms or *repeats back* what they have heard from the individual sending the message, so that he/she can affirm that the message is correct or offer a correction. This is a three-step process that ensures clarity and accountability. Closed-loop communication also allows for a clear, shared mental model of the care plan and the assurance that someone is handling the request.

For example, a physician may request 10 units of oxytocin intramuscularly after delivery of the anterior shoulder. The nurse would repeat back that the physician requested 10 units of oxytocin intramuscularly after delivery of the anterior shoulder as confirmation that the message was understood. The physician then *closes the loop* by confirming that, yes, this is what they requested. Without closed-loop communication, messages may be missed or misinterpreted. In this example, the oxytocin may not have been administered or an incorrect dose may have been administered.

Shared Mental Model

Situational awareness, standardized language, and closed-loop communication can allow a team to have a shared mental model. Without a shared mental model, teamwork and patient safety can be compromised. For example, the HELPER[4] mnemonic for shoulder dystocia taught in the ALSO course can create a shared mental model, where nurses and physicians work together via the McRoberts maneuver, suprapubic pressure, and other interventions to avoid fetal injury or mortality.

HELPER[4] is an acronym for Call for **H**elp, **E**valuate and **E**xplain for **E**pisiotomy, **L**egs—McRoberts Maneuver, Suprapubic **P**ressure, Consider **E**pisiotomy, and **R**^{4}: *R*emoval of the posterior arm, use of the *R*otatory internal maneuvers (Rubin II, Woods screw, and reverse Woods screw), *R*oll the patient (Gaskin maneuver), and *R*epeat all maneuvers. This R^4 portion of the mnemonic allows the clinician to use the maneuver(s) in the order they judge to be most effective and appropriate given the clinical circumstances.

Mutual Respect

The ability to communicate clearly and effectively is an essential element of teamwork. Circumstances may require escalation of care strategies to ensure the best outcomes for the woman, the infant, and the care team. The ability to state a concern, offer a solution, and agree on next steps in the care plan is a critical component of patient safety. Intimidating and disruptive behavior undermine patient safety and should not be tolerated.[35]

The Two-Challenge Rule and CUS Words are two communication strategies designed to give voice to all team members.

Two-Challenge Rule. The Two-Challenge Rule[36] allows a team member to clearly articulate a concern regarding a perceived or real patient safety breach. The first challenge is made in the form of a question. The second challenge is made in the form of a statement and can be offered by the same clinician or by another member of the care team. The second challenge is focused on advocating for the needs of the woman.

For example, a senior resident may be preparing to perform a non-emergent manual extraction of a placenta in a woman without epidural analgesia. An accompanying medical student may say, "I don't think the patient has adequate anesthesia." If the resident proceeds, a second statement from the medical student regarding the need for better pain control should signal the senior resident and care team to suspend the procedure and administer additional anesthesia or explain to the student why additional anesthesia is not indicated or feasible.

CUS Words. CUS Words is a communication strategy, where every individual in a care unit is trained to listen when the specific words are spoken, as follows:

1. "I'm **C**oncerned"
2. "I'm **U**ncomfortable"
3. "This is a **S**afety issue"[28]

This strategy may benefit any clinician who requires additional support when caring for a

patient. For example, if a nurse on a care team says they are concerned about a fetal heart rate, that it makes them uncomfortable, and that it is a safety issue, the team should respond by evaluating whether a change in management is indicated.

Briefings, Huddles, and Debriefings

Briefings. Briefings are held before any patient care episode to allow team members to review risk factors, designate roles, and ensure that everyone has a shared mental model regarding how to proceed. Briefings are a way to plan ahead. Briefing prior to labor admission of a woman with gestational diabetes and an apparently large fetus, for example, can prepare the team for who will perform which task if a shoulder dystocia occurs.

Huddles. Huddles are brief gatherings of care team members to discuss patient status and the management plan when issues arise during patient care. Examples of events that should precipitate a huddle are development of high blood pressure levels, fever, and concerning fetal heart tracings during labor. A huddle may take place in person or via teleconference, if a key team member is not physically present when the huddle is needed. Huddles are a way to solve problems in the moment.

Debriefings. Debriefings allow team members to learn from patient care episodes, regardless of the outcome. Team members can quickly answer the questions:

1. What went well, and why?
2. What could have gone better, and why?
3. What would you do differently next time?

During debriefings, it can be helpful to discuss three levels of emergency care management:

1. Medical management
2. Teamwork
3. System or process/protocol issues

Discussion may naturally drift toward medical management. Team leaders can guide the discussion back to teamwork and system issues. Debriefings can allow the team to perform process improvement.

Debriefing can include root cause analysis after a sentinel event. A 2015 statement by The Joint Commission defines a sentinel event as "a patient safety event (not primarily related to the natural course of the patient's illness or underlying condition) that reaches a patient and results in any of the following: death, permanent harm, severe temporary harm." In obstetrics, severe temporary harm is defined as receiving 4 or more units of blood products (subsequently revised to 4 or more units of red blood cells) and/or admission to an intensive care unit.[37]

Near Misses and Positive Outcomes

Debriefings and root cause analysis are also encouraged for *near misses* and severe maternal mortality that do not constitute a sentinel event. There is a much more frequent opportunity for quality improvement if debriefings and system analysis occurs with near misses and not only when outcomes are poor. The introduction of ALSO training to a Colombian hospital in 2007 led to an increase in the near miss incidence ratio (10.5 in 2005 to 2006, to 11.3 in 2008 to 2009) and severe maternal mortality cases (83 in 2005 to 2006, to 161 in 2008 to 2009). However, the maternal mortality ratio (MMR) decreased (114 deaths per 100,000 live births in 2005 to 2006, to 28 in 2008 to 2009) along with the mortality index (9.8% [9/92] in 2005 to 2006, to 2.4% [4/165] in 2008 to 2009).[38] The increase in near misses could be due to the lives saved of laboring women with obstetric emergencies, who might have otherwise died.

Debriefings can also be useful for reinforcing positive practices after deliveries in which everything went well. Team members can be congratulated for communicating and acting effectively. A new positive practice, such as skin-to-skin time for a woman and infant after delivery, can be noted and replicated on a system-wide level. Debriefings can be part of creating a *culture of safety*. When all team members cannot debrief, available team members can still meet. Absent team members (eg, anesthesiologist, neonatal resuscitation staff) can be debriefed afterward via telephone.

Fatigue

Fatigue can affect patient safety factors including memory, speed, and mood.[39] Fatigue has been cited as a root cause of maternal and neonatal injury.[8] With standardized testing, adults with fewer than 5 hours of sleep per night have difficulty with short-term memory, retention, and concentration.[39] Federal Railroad Administration data indicates that fatigue is causative in approximately 29% of train crashes.[40] Resident work-hour requirements are an attempt to prevent fatigue-related medical errors.

Individuals can ensure they are fit for work by reviewing the I'M SAFE (**I**llness, **M**edication, **S**tress, **A**lcohol and Drugs, **F**atigue, **E**ating and **E**limination) checklist.[28]

Systems and colleagues can monitor to ensure work conditions allow for self-care. Employee assistance programs should be high-quality and accessible. Work-hour limits, such as those introduced for medical residents, may prevent the fatigue often involved with medical errors.[41] Work hours and facilities can facilitate employees eating and eliminating, so they will be performing optimally while working.

Medication Errors

On average, US patients experience one medication error per patient per hospitalization day.[42] Some can result in mortalities. This happened in 2006 when a healthy, 16-year-old woman, who was in active labor, was admitted to a hospital in Madison, Wisconsin.[43] She tested positive for group B streptococcus and requested epidural analgesia. The anesthesiologist placed the epidural infusion bag on the counter and left the room. A nurse entered the room and hung the epidural bag, thinking it contained penicillin. Despite efforts to resuscitate her, the young woman died. Her infant survived after a resuscitative hysterotomy.

Electronic medical records (EMRs) are helpful in reducing errors due to poor legibility and can identify drug allergies and drug interactions. Prescribing errors can be reduced by avoiding nonstandard abbreviations and using the "always lead, never follow" rule of placing a zero before numbers less than one and not placing a zero after a decimal point.[42] EMR alerts can prevent errors such as unrecognized drug interactions. However, too many alerts may lead to desensitization: 49% to 96% of alerts are overridden.[44]

Medication errors are common after transitions in care. These errors can be reduced through systematic, careful medication reconciliation on admission, transfer, and discharge.

Distraction can lead to errors. Areas for dispensing medications can be established as noise-free, distraction-free zones.

As with other aspects of patient safety, communication problems often are at the root of errors. Using closed-loop communication can be lifesaving.

Health Information Technology

Health information technology (IT) can be a valuable patient safety tool beyond its role in the safe prescribing of medications. Examples include: facilitating provider communication, tracking and reporting data, providing point-of-care reading material, promoting adherence to practice guidelines, and increasing patient engagement.[45] Use of EMR problem lists can improve interconception care by alerting primary care providers to conditions such as hypertensive disorders of pregnancy and gestational diabetes, which place a woman at higher lifetime risk of hypertension and diabetes, respectively.

For data to be useful, it must be interpreted and acted on appropriately. Use of health IT has risks, including the possible compromise of patient privacy as well as the use of documentation templates that may introduce and duplicate information that is inaccurate or not reviewed.

Larger databases can produce more powerful research and recommendations. Two organizations promoting safety in maternity care using IT are the California Maternal Quality Care Collaborative (CMQCC) (https://www.cmqcc.org) and the Family Medicine Education Consortium (FMEC) IMPLICIT: Interventions to Minimize Preterm and Low birth weight Infants through Continuous Improvement Techniques network (https://fmec.memberclicks.net/implicit).

System-Level Change Versus Blaming Individuals

Reducing medical errors to improve patient safety is a high priority in the United States and other countries. Traditionally, medical culture expects perfection. The typical tactic to fix errors is to ascribe individual blame.

Although there is a tendency to scapegoat an individual when things go wrong, there usually are numerous factors and system issues that lead to the adverse outcome. Blaming the individual does not address those other factors and allows the error to be perpetuated. For example, firing an employee who makes an error at the end of a double shift does not fix the work-hour structure that will likely result in fatigue and errors occurring again.

Examples of ways to effect change at a system level include using checklists and protocols, which have been documented to improve outcomes through standardization of practice.[46] One health

care system incorporated a mandatory field into its EMR requiring a sponge count after obstetric procedures when a patient was found to have a retained laparotomy sponge 1 week after normal vaginal delivery.[47] Other health care systems are using *vaginal wanding* to avoid retained sponges. This method involves scanning individual sponges, which are equipped with radiofrequency tags, to account for all internal gauze or laparotomy sponges used during a delivery.

In the early days of aviation, plane crashes often were blamed on pilot error without much further analysis. Blaming the faulty, and usually deceased, pilot did not do much to prevent further crashes from happening. The aviation industry made minimal progress in safety and reliability until they developed a broader notion of safety and considered the multiplicity of factors underlying airplane crashes and pilot errors. Aviation safety improved through a "collective sense of urgency for maintaining safety and a mutual understanding that all team members will state their observations, opinions, and recommendations, and actively solicit and consider input from other team members."[48]

Efforts to reduce non-medically indicated, early-term labor inductions and cesarean deliveries are an example of a successful system-level patient safety intervention. Delivery before 39 weeks' gestation is associated with increased respiratory distress syndrome, transient tachypnea of the newborn, ventilator use, pneumonia, respiratory failure, newborn intensive care unit admission, hypoglycemia, 5-minute Apgar score less than 7, and neonatal mortality.[49] A hospital hard-stop where elective deliveries are not allowed by hospital personnel was the most effective approach to reducing non-indicated near-term deliveries.[50]

An important tenet of aviation safety is empowering each member of the flight team to identify and correct potential errors.[51] Teams are trained to speak up if they feel any member is at risk of error. The aviation industry has found that this helps overcome the effects of its traditionally hierarchical organization, which otherwise tends to discourage error reporting by subordinates. The medical profession has a similarly hierarchical organization and must overcome this tendency toward silence. CUS Words and the Two-Challenge Rule are tools for overcoming hierarchy and improving communication.

Community Birthing

One example where system-level interventions are needed to improve patient safety is community birthing, including home and free-standing birth center deliveries. A 2012 Cochrane review showed there is no strong evidence from randomized trials to favor planned hospital birth or planned home birth for low-risk pregnant women; however, the study notes that observational studies increasingly suggest that in countries where home birth is integrated into the health system, home birth for low-risk women results in fewer interventions and complications.[52] Lack of role clarity and poor communication are the biggest predictors of preventable maternal and neonatal outcomes, including death. Seamless coordination of care and interprofessional communication results in better maternal and child outcomes.[53]

Maternal Mortality

As mentioned previously, although maternal mortality has decreased in most low- and high-resource countries since the 1990 United Nations Millennium Development Goals were issued, maternal mortality in the United States has increased.[56] From 1990 to 2015, the world's maternal mortality ratio (MMR) decreased from 385 to 216 per 100,000 live births. In the least developed countries, the ratio decreased from 903 to 436.[54] In contrast, between 2000 and 2014, the US MMR for 48 states (excluding California and Texas) and Washington, DC increased from 18.8 to 23.8.[6]

Reasons for the increase are complex and include many factors, one of which is improvements in reporting strategies. In 2003, a pregnancy question was added to the US standard death certificate. US states gradually adopted the revised certificate and by 2014, 44 states and Washington, DC were using it.[6] This question ascertains whether maternal mortality occurred within 42 days after delivery, which is consistent with the World Health Organization's definition of maternal mortality; many states did not previously report deaths after delivery.[6]

The increase in US MMR is not only due to increased reporting, because some states had increases in MMR during periods when no changes were made to reporting systems. Rural access, poverty, immigration, cesarean delivery, obesity, diabetes, advanced maternal age, substance abuse, cardiac-related conditions, and

racial disparities are other possible causes of the increase in US MMR.[55-57] Defunding of women's health also has been associated with the increase in maternal mortality in certain states, including Indiana, Alabama, Arkansas, Arizona, Florida, Louisiana, Kansas, Missouri, Oklahoma, Texas, and Wisconsin.[58]

Despite the increasing US MMR, California's rate decreased from 21.5 to 15.1 from 2003 to 2014.[6] Some have attributed the improved outcomes in California to systems changes introduced by the CMQCC patient safety bundles.[7] A 2017 study of 99 hospitals (256,541 annual births) showed that the use of a standardized postpartum hemorrhage (PPH) bundle resulted in a 20.8% reduction in severe maternal morbidity compared with 48 comparison hospitals (81,089 annual births) with a 1.2% reduction (P <0.0001).[59] Hospitals with a previous PPH protocol had a higher reduction in severe maternal morbidity (17.5% versus 11.7%).[59]

Patient Safety Bundles

The Council on Patient Safety in Women's Health Care, a joint multidisciplinary collaboration of national health care organizations, has developed patient safety bundles through the Alliance for Innovation on Maternity Health (AIM) (*Table 1*).[60] The safety bundles follow a 4R structure: 1) **R**eadiness, 2) **R**ecognition and prevention, 3) **R**esponse and 4) **R**eporting/systems learning.[60] Each patient bundle contains structured goals to ensure standardized care for each of the bundle elements (pages 13 and 14). The Agency for Healthcare Research and Quality (AHRQ) also has developed a toolkit for improving perinatal safety.[61]

Patient Safety and Malpractice Risk

An additional anticipated benefit of a reduction in adverse obstetric outcomes is a decrease in malpractice loss for physicians and hospitals providing maternity care. Throughout the United States, pregnancy and birth-related malpractice claims are the highest of all malpractice loss expenses; it is not surprising that these losses have caused many hospitals and physicians to discontinue providing maternity care. It is estimated that approximately $80 billion per year is spent on practicing defensive medicine.[62] Preventing patient errors is an important part of a multifaceted approach to resolving what is perceived as a current malpractice crisis.[63]

The cost of malpractice insurance can affect the ability to provide maternity care and the satisfaction of physicians who pay high insurance premiums. Multivariate regression analysis of a survey of obstetricians and gynecologists practicing in Michigan, with 365 respondents, showed paying more than $50,000 per year for liability insurance was associated with lower career satisfaction (odds ratio = 0.35; 95% confidence interval = 0.13 to 0.93) compared with insurance coverage provided by an employer.[64]

Pregnancy is unique from a liability standpoint in several ways: 1) two patients are involved: the woman and her fetus, 2) the woman usually is healthy when she presents for care, and 3) she and her family often have expectations of a perfect infant and birth experience.

An unhappy patient usually is the trigger for a lawsuit.[65] This may reflect the patient's or her

Table 1. AIM-Supported Patient Safety Bundles and Safety Tools

Patient Safety Bundles
Maternal Mental Health: Depression and Anxiety
Maternal Venous Thromboembolism Prevention
Obstetric Care for Women with Opioid Use Disorder
Obstetric Hemorrhage
Postpartum Care Basics for Maternal Safety
From Birth to the Comprehensive Postpartum Visit
From Maternity to Well-Woman Care
Prevention of Retained Vaginal Sponges After Birth
Reduction of Peripartum Racial/Ethnic Disparities
Safe Reduction of Primary Cesarean Birth
Severe Hypertension in Pregnancy
Patient Safety Tools
Maternal Early Warning Criteria
Patient Family and Staff Support After a Severe Maternal Event
Severe Maternal Morbidity Review
Summary After a Severe Maternal Event

Information from Council on Patient Safety in Women's Health Care. 2019. Available at http://safehealthcareforeverywoman.org/.

family's feelings of disappointment at the outcome, the kind or cost of care she received, or the cost of caring for a child with a disability.

Malpractice litigation takes a significant toll on all individuals involved. Lawsuits usually take many years to resolve. There are uncounted costs, including decreases in the number of practicing maternity care providers. Defensive medical practices, time lost in litigation activities, increased wariness toward patients, and emotional turmoil are costly results of litigation. Loss of access to maternity care is also exacerbated, especially in rural areas.[66]

Malpractice litigation is common. Seventy-three percent of respondents to a 2015 survey of fellows of the American College of Obstetricians and Gynecologists indicated that they had been sued with an average of 2.59 claims per obstetrician.[67] Costs of litigation and awards continue to increase in the United States and Canada. The likelihood of a lawsuit appears to be directly related to the number of deliveries a provider performs rather than to quality or specialty. Family physicians are not exempt. It is a myth that poor people sue more frequently.[68]

The most common primary allegations of obstetric claims is a neurologically impaired infant (27.4%), and stillbirth or neonatal death (15%).[67] Among the neurologically impaired infant claims, delivery was by cesarean delivery (55.2%), vaginal delivery (40.5%) and labor after cesarean (LAC) (2.0%).[67] Other factors associated with lawsuits include electronic fetal monitoring (22.1%), shoulder dystocia/brachial plexus injury (14.2%), actions of residents (10.6%), and lack of communication among health care providers (10.5%).[67]

Risk management is a strategy that attempts to prevent or minimize patient injuries, decreases the chance of successful malpractice litigation when an injury does occur, and attempts to reduce the amount of the award in a successful claim. Risk management strategies in hospitals have used early case reporting to attempt to decrease claims. Malpractice claims are not sensitively or specifically identified by these strategies. Newer strategies focus on root cause analysis to prevent future adverse outcomes. The Veterans Health Administration system has used a novel approach of intense case finding, coupled with apology and negotiation, with injured patients.[69] They have successfully decreased overall claims costs while compensating injured patients even before initiation of lawsuits.

Malpractice Insurance Rates

Professional liability insurance companies may offer discounts on medical malpractice premiums to maternity care provider clients who take the ALSO Provider course or other maternity care training designed to reduce liability (eg, fetal monitoring courses). Over the years, some malpractice insurance carriers, such as Northwest Physicians Mutual, required providers to take the ALSO course to qualify for coverage. The Northwest Region of the Doctors Company, which purchased Northwest Physicians Mutual, currently provides a malpractice insurance premium discount to providers who take ALSO courses (D. Zimmer, written communication, August 2018).[70]

The Five Cs of Risk Management

ALSO teaches The Five Cs of risk management: **C**ompassion, **C**ommunication, **C**ompetence, **C**harting,[70] and **C**onfession.

Compassion. Every lawsuit begins with a dissatisfied patient. This dissatisfaction often starts before the event leading to the lawsuit. Patients find it more difficult to sue someone they like and who they think cares about them. Allow the woman to choose from a variety of options of care when possible. Allow her to share her concerns. Open-ended questions that can improve empathy include "Tell me more," "How did you feel?", "Anything else?", and "What concerns do you have?"[71]

Communication. Spending more time with patients may result in fewer lawsuits. Patients do not want to feel rushed. Patients who receive adequate explanations about their conditions and test results are more satisfied. Patients do not want to feel that their maternity care provider ignored their concerns.

The simple act of sitting rather than standing when talking with patients improves patient perception of provider communication skills.[72] When providers sit rather than stand, patients report more time was spent at the bedside, improved satisfaction, and a better understanding of their own health condition.[73]

Communication implies being available to the patient and maternity care team. A woman in labor essentially takes precedence over any other patient.

Informed consent is an important tool to use in helping women and families understand and share some of the uncertainty and risk inherent in pregnancy. Informed consent is inherently imperfect: it "depends on there being a shared understanding of the language used to describe the risks and benefits of the appropriate available options."[74] Closed-loop communication and the combination of verbal description, numerical data, and graphical representation can facilitate collective understanding of risks and benefits.

Strategies for facilitating communication include:[75]

- Speaking slowly and using plain, nonmedical language
- Limiting the amount of information provided and repeating the information
- Using *teach-back* or *show-me* techniques (asking the patient to repeat any instructions given) to confirm that the patient understands what has been explained
- Encouraging patients to ask questions
- Providing written materials to reinforce oral explanations.

Competence. The clinician must know his or her ability in any given situation. Honesty and ensuring that interventions are solidly indicated are key features of competence. The provider must possess skill, training, experience and the ability to provide comfort to provide appropriate care. Consultation or referral should be obtained and appropriately documented when these criteria are not met.

Charting. Many lawsuits are filed against maternity care providers and lost because of inadequate documentation in the medical record.[76] The medical record serves as the principal witness when legal action is filed. A suit usually is litigated years after the suit is initiated, and memories fade. Records should be dated, timed, complete, contemporaneous, accurate, and objective. Recording errors should be addressed, corrected, and explained; they should never be ignored or covered up. Even an uncomplicated vaginal delivery should have a complete and legible record. Dictated reports should be read, corrected, and signed. Avoid inflammatory, incorrect, and vague terms such as fetal *distress* and *asphyxia*.

In one study, with 54% of malpractice suits involving shoulder dystocia, the factor influencing damages was lack of clear documentation of events surrounding the management of the dystocia.[76] Damages were awarded in only 25% of the suits because of deviation from standard of care.[76]

Confession. Discussing mistakes with the patient has been actively discouraged in the past. However, many studies confirm that one of the more common reasons for filing a suit is a suspected cover-up.[77] A survey of patients at an academic internal medicine clinic found that almost all patients wanted their physicians to disclose even minor errors.[78]

These Five Cs of risk management are a reminder of strategies that can decrease malpractice risk. More importantly, they serve as strategies for the maternity care provider to ensure satisfying, safe care for pregnant women and their families.

An American College of Obstetricians and Gynecologists committee opinion makes seven patient safety recommendations: 1) develop a commitment to encourage a culture of patient safety, 2) implement recommended safe medicine practices, 3) reduce the likelihood of surgical errors, 4) improve communication with health care providers, 5) improve communication with patients, 6) establish a partnership with patients to improve safety, and 7) make safety a priority in every aspect of practice.[79]

Simulations

Simulations can take place in a simulation laboratory or in situ in maternity care units. In-situ simulations have the advantage of better replicating patient care challenges and system issues, which may not arise in a simulation laboratory.[19]

Simulations can be used to practice the communication and teamwork concepts taught in this chapter in the context of managing obstetric emergencies. Simulations can be executed with equal effectiveness using patient volunteers, low-fidelity manikins, or high-fidelity manikins. The ALSO course incorporates simulations in the maternal resuscitation workshop and group testing scenarios.

Simulations allow multidisciplinary teams to practice managing obstetric emergencies when patient lives are not at risk. In one study, in-situ simulations involving all staff and providers that were held 2 to 3 times per year at one hospital led to a significant and persistent 37% decrease in perinatal morbidity compared with hospitals with didactic training only or no training.[21] With simulations, teams have a briefing to discuss roles

before managing a labor. The team then manages an emergency. Finally, the team debriefs, focusing on what went well and why, what did not go well and why, and what can be done to make things better in the future. Video recording of the entire simulation can provide a powerful tool for use in debriefing sessions. Providers may see themselves and others quite differently when reviewing videotaped management. In-situ simulations allow latent system errors to be identified and corrected before they become active errors leading to patient harm.

Patient Safety in Low-Resource Settings

In low-resource settings, teamwork and communication can save lives just as in higher-resource settings. System issues have a greater effect where there is a lack of infrastructure including ambulance services, roads, telephones, clinics and hospitals, electronic medical records, and blood products and medication. Delays that lead to maternal morbidity and mortality can be categorized as those in: 1) seeking medical care, 2) getting to a medical facility, and 3) receiving quality care after arriving at a medical facility.[80] In terms of the 4 Rs of patient safety bundles, readiness and early recognition are of particular importance and emphasis. More information on safety in maternity care and other issues in developing countries is available through the Global ALSO Program (available at www.aafp.org/globalalso).

Summary

Women and/or their infants die or experience permanent injury because of preventable errors. Routine use of briefings, huddles, and debriefings can help avoid communication errors, which account for more than 70% of medical errors. Teamwork tools include situational awareness, standardized language, closed-loop communication, and development of shared mental models. Tools such as the Two-Challenge Rule and CUS Words empower all individuals involved in patient care to speak up and influence care when they perceive errors are occurring. ALSO mnemonics help team members approach the situation similarly when emergencies arise. Following The Five Cs can reduce the risk of malpractice litigation through improved patient care. Learners are encouraged and challenged to incorporate team thinking during ALSO training and subsequent practice. ALSO simulations can be implemented to enhance team function for more effective management of obstetric emergencies.

Acknowledgement

This chapter is a modification of the previous version of *Safety in Maternity Care* written by Lee T. Dresang, MD, Stan Davis, MD, Kristi K. Miller, MS, RN, and Lesley Atwood, MD.

Nursing Considerations: Safety in Maternity Care

- Advocate for and listen to the patient, reminding others that she is a part of the team
- Identify strategies you can use for conflict resolution
- Champion efforts at your institution for briefings, huddles, debriefings, team training, "in-situ" drills and ALSO Provider courses
- Be aware of your institution's process for debriefing following near misses, severe maternal/neonatal morbidity, maternal/neonatal mortality (root cause analysis), and risk management resources
- Utilize teamwork tools to improve safety: Two-challenge rule, CUS Words, call-out, SBAR, handoff, closed loop communication, The Five Cs

COUNCIL ON PATIENT SAFETY IN WOMEN'S HEALTH CARE

safe health care for every woman

PATIENT SAFETY BUNDLE

Reduction of Peripartum Racial/Ethnic Disparities

READINESS

Every health system

- Establish systems to accurately document self-identified race, ethnicity, and primary language.
  - Provide system-wide staff education and training on how to ask demographic intake questions.
  - Ensure that patients understand why race, ethnicity, and language data are being collected.
  - Ensure that race, ethnicity, and language data are accessible in the electronic medical record.
  - Evaluate non-English language proficiency (e.g. Spanish proficiency) for providers who communicate with patients in languages other than English.
  - Educate all staff (e.g. inpatient, outpatient, community-based) on interpreter services available within the healthcare system.
- Provide staff-wide education on:
  - Peripartum racial and ethnic disparities and their root causes.
  - Best practices for shared decision making.
- Engage diverse patient, family, and community advocates who can represent important community partnerships on quality and safety leadership teams.

RECOGNITION

Every patient, family, and staff member

- Provide staff-wide education on implicit bias.
- Provide convenient access to health records without delay (paper or electronic), at minimal to no fee to the maternal patient, in a clear and simple format that summarizes information most pertinent to perinatal care and wellness.
- Establish a mechanism for patients, families, and staff to report inequitable care and episodes of miscommunication or disrespect.

October 2016

COUNCIL ON PATIENT SAFETY IN WOMEN'S HEALTH CARE

safe health care for every woman

PATIENT SAFETY BUNDLE

Reduction of Peripartum Racial/Ethnic Disparities

RESPONSE

Every clinical encounter

- Engage in best practices for shared decision making.
- Ensure a timely and tailored response to each report of inequity or disrespect.
- Address reproductive life plan and contraceptive options not only during or immediately after pregnancy, but at regular intervals throughout a woman's reproductive life.
- Establish discharge navigation and coordination systems post childbirth to ensure that women have appropriate follow-up care and understand when it is necessary to return to their health care provider.
  - Provide discharge instructions that include information about what danger or warning signs to look out for, whom to call, and where to go if they have a question or concern.
  - Design discharge materials that meet patients' health literacy, language, and cultural needs.

REPORTING & SYSTEMS LEARNING

Every clinical unit

- Build a culture of equity, including systems for reporting, response, and learning similar to ongoing efforts in safety culture.
- Develop a disparities dashboard that monitors process and outcome metrics stratified by race and ethnicity, with regular dissemination of the stratified performance data to staff and leadership.
- Implement quality improvement projects that target disparities in healthcare access, treatment, and outcomes.
- Consider the role of race, ethnicity, language, poverty, literacy, and other social determinants of health, including racism at the interpersonal and system-level when conducting multidisciplinary reviews of severe maternal morbidity, mortality, and other clinically important metrics.
  - Add as a checkbox on the review sheet: Did race/ethnicity (i.e. implicit bias), language barrier, or specific social determinants of health contribute to the morbidity (yes/no/maybe)? And if so, are there system changes that could be implemented that could alter the outcome?

Standardization of health care processes and reduced variation has been shown to improve outcomes and quality of care. The Council on Patient Safety in Women's Health Care disseminates patient safety bundles to help facilitate the standardization process. This bundle reflects emerging clinical, scientific, and patient safety advances as of the date issued and is subject to change. The information should not be construed as dictating an exclusive course of treatment or procedure to be followed. Although the components of a particular bundle may be adapted to local resources, standardization within an institution is strongly encouraged.

The Council on Patient Safety in Women's Health Care is a broad consortium of organizations across the spectrum of women's health for the promotion of safe health care for every woman.

October 2016

Reprinted from Council on Patient Safety in Women's Health Care. AIM-Supported Patient Safety Bundles. Reduction of Peripartum Racial/Ethnic Disparities. Available at https://safehealthcareforeverywoman.org.

References

1. Ornato JP, Peberdy MA. Applying lessons from commercial aviation safety and operations to resuscitation. *Resuscitation*. 2014;85(2):173-176.
2. Nielsen P, Mann S. Team function in obstetrics to reduce errors and improve outcomes. *Obstet Gynecol Clin North Am*. 2008;35(1):81-95, ix.
3. Institute of Medicine. *Patient Safety: Achieving a New Standard for Care*. Washington DC: The National Academies Press; 2004.
4. Alkema L, Chou D, Hogan D, et al; United Nations Maternal Mortality Estimation Inter-Agency Group collaborators and technical advisory group. Global, regional, and national levels and trends in maternal mortality between 1990 and 2015, with scenario-based projections to 2030: a systematic analysis by the UN Maternal Mortality Estimation Inter-Agency Group. *Lancet*. 2016;387(10017):462-474.
5. Podulka J, Stranges E, Steiner C. Hospitalizations related to childbirth, 2008: statistical brief #110. In: *Healthcare Cost and Utilization Project (HCUP) Statistical Briefs*. Rockville, MD: Agency for Health Care Policy and Research (US); 2006-2011.
6. MacDorman MF, Declercq E, Cabral H, Morton C. Recent increases in the U.S. maternal mortality rate: disentangling trends from measurement issues. *Obstet Gynecol*. 2016;128(3):447-455.
7. California Maternal Quality Care Collaborative. 2018. Available at https://www.cmqcc.org/.
8. The Joint Commission. Sentinel event data: root causes by event type 2004-2014. Available at http://www.jointcommission.org.
9. Forster AJ, Fung I, Caughey S, et al. Adverse events detected by clinical surveillance on an obstetric service. *Obstet Gynecol*. 2006;108(5):1073-1083.
10. Committee on Quality of Health Care in America, Institute of Medicine. Crossing the quality chasm: a new health system for the 21st century. Washington, DC: National Academies Press; 2001.
11. Committee on Quality of Health Care In Medicine; Institute of Medicine. To err is human: building a safer health system. Washington, DC: National Academy Press; 2000.
12. James JT. A new, evidence-based estimate of patient harms associated with hospital care. *J Patient Saf*. 2013; 9(3):122-128.
13. Kochanek KD, Murphy SL, Xu J, Arias E. Deaths: final data for 2017. *Natl Vital Stat Rep*. 2019;68(9):1-76..
14. Reason J. Human error: models and management. *BMJ*. 2000;320(7237):768-770.
15. The Joint Commission. Human factors analysis in patient safety systems. *The Source*. 2015;13(4):1,7-10.
16. The Joint Commission. Improving America's hospitals: The Joint Commission's annual report on quality and safety - 2003. Washington, DC: Joint Commission; 2003.
17. The Joint Commission. Improving America's hospitals: The Joint Commission's annual report on quality and safety - 2008. Washington, DC: Joint Commission; 2008.
18. Burke CS, Salas E, Wilson-Donnelly K, Priest H. How to turn a team of experts into an expert medical team: guidance from the aviation and military communities. *Qual Saf Health Care*. 2004;13(Suppl 1):i96-i104.
19. Miller KK, Riley W, Davis S, Hansen HE. In situ simulation: a method of experiential learning to promote safety and team behavior. *J Perinat Neonatal Nurs*. 2008;22(2): 105-113.
20. Davis S, Riley W, Gurses AP, Miller K, Hansen H. Failure modes and effects analysis based on in situ simulations: a methodology to improve understanding of risks and failures. In: Henriksen K, Battles J, Keyes M, Grady M, eds. *Advances in Patient Safety: New Directions and Alternative Approaches (Vol. 3: Performance and Tools)*. Rockville, MD: Agency for Healthcare Research and Quality (US); 2008.
21. Riley W, Davis S, Miller K, Hansen H, Sainfort F, Sweet R. Didactic and simulation nontechnical skills team training to improve perinatal patient outcomes in a community hospital. *Jt Comm J Qual Patient Saf*. 2011;37(8):357-364.
22. Mann S, Marcus R, Sachs B. Lessons from the cockpit: How team training can reduce errors on L&D. *Contemp Ob Gyn*. 2006;51(1):34-45.
23. Thomas EJ, Taggart B, Crandell S, et al. Teaching teamwork during the Neonatal Resuscitation Program: a randomized trial. *J Perinatol*. 2007;27(7):409-414.
24. Salas E, Gregory ME, King HB. Team training can enhance patient safety—the data, the challenge ahead. *Jt Comm J Qual Patient Saf*. 2011;37(8):339-340.
25. Bohren MA, Hofmeyr GJ, Sakala C, Fukuzawa RK, Cuthbert A. Continuous support for women during childbirth. *Cochrane Database Syst Rev*. 2017;7:CD003766.
26. American College of Obstetricians and Gynecologists. ACOG committee cpinion no. 587: effective patient-physician communication. *Obstet Gynecol*. 2014;123(2 Pt 1):389-393.
27. American College of Obstetricians and Gynecologists Committee on Patient Safety and Quality Improvement. ACOG committee opinion no. 490: partnering with patients to improve safety. *Obstet Gynecol*. 2011;117(5): 1247-1249.
28. Agency for Healthcare Research and Quality. TeamSTEPPS: team strategies and tools to enhance performance and patient safety. 2018. Available at https://www.ahrq.gov/teamstepps/index.html.
29. Fisher R, Ury W, Patton B. *Getting to Yes: Negotiating Agreement Without Giving In*. New York, NY: Penguin Books; 2011.
30. American College of Obstetricians and Gynecologists Committee on Patient Safety and Quality Improvement. Committee opinion no. 590: preparing for clinical emergencies in obstetrics and gynecology. *Obstet Gynecol*. 2014;123(3):722-725.
31. McFerran S, Nunes J, Pucci D, Zuniga A. Perinatal Patient Safety Project: a multicenter approach to improve performance reliability at Kaiser Permanente. *J Perinat Neonatal Nurs*. 2005;19(1):37-45.
32. Haig KM, Sutton S, Whittington J. SBAR: a shared mental model for improving communication between clinicians. *Jt Comm J Qual Patient Saf*. 2006;32(3):167-175.

33. Leonard M, Graham S, Bonacum D. The human factor: the critical importance of effective teamwork and communication in providing safe care. *Qual Saf Health Care*. 2004;13(Suppl 1):i85-i90.
34. American College of Obstetricians and Gynecologists. ACOG committee opinion no. 517: communication strategies for patient handoffs. *Obstet Gynecol*. 2012;119 (2 Pt 1):408-411.
35. Committee on Patient Safety and Quality Improvement. Committee opinion no. 683: behavior that undermines a culture of safety. *Obstet Gynecol*. 2017;129(1):e1-e4.
36. Macready N. Two-challenge rule averts errors, improves safety. *OR Manager*. 1999;15(1):12.
37. American College of Obstetricians and Gynecologists. Severe maternal morbidity: clarification of the new Joint Commission sentinel event policy. 2015. Available at http://www.acog.org/About-ACOG/News-Room/Statements/2015/Severe-Maternal-Morbidity-Clarification-of-the-New-Joint-Commission-Sentinel-Event-Policy.
38. Dresang LT, González MM, Beasley J, et al. The impact of Advanced Life Support in Obstetrics (ALSO) training in low-resource countries. *Int J Gynaecol Obstet*. 2015; 131(2):209-215.
39. Committee on Patient Safety and Quality Improvement. ACOG committee opinion no. 730: fatigue and patient safety. *Obstet Gynecol*. 2018;131(2):e78-e81.
40. US Department of Transportation; Federal Railroad Administration. The Railroad Fatigue Risk Management Program at the Federal Railroad Administration: past, present and future. 2006. Available at https://www.fra.dot.gov/Elib/Document/2944.
41. Spritz N. Oversight of physicians' conduct by state licensing agencies. Lessons from New York's Libby Zion case. *Ann Intern Med*. 1991;115(3):219-222.
42. Committee on Patient Safety and Quality Improvement. Committee opinion no. 531: improving medication safety. *Obstet Gynecol*. 2012;120(2 Pt 1):406-410.
43. Smetzer J, Baker C, Byrne FD, Cohen MR. Shaping systems for better behavioral choices: lessons learned from a fatal medication error. *Jt Comm J Qual Patient Saf*. 2010;36(4):152-163.
44. van der Sijs H, Aarts J, Vulto A, Berg M. Overriding of drug safety alerts in computerized physician order entry. *J Am Med Inform Assoc*. 2006;13(2):138-147.
45. Committee on Patient Safety and Quality Improvement; Committee on Practice Management. Committee opinion no. 621: patient safety and health information technology. *Obstet Gynecol*. 2015;125(1):282-283.
46. ACOG Committee on Patient Safety and Quality Improvement. ACOG committee opinion no. 526: standardization of practice to improve outcomes. *Obstet Gynecol*. 2012;119(5):1081-1082.
47. Agrawal A. Counting matters: lessons from the root cause analysis of a retained surgical item. *Jt Comm J Qual Patient Saf*. 2012;38(12):566-574.
48. Lyndon A. Communication and teamwork in patient care: how much can we learn from aviation? *J Obstet Gynecol Neonatal Nurs*. 2006;35(4):538-546.
49. American College of Obstetricians and Gynecologists. ACOG committee opinion no. 765: avoidance of nonmedically indicated early-term deliveries and associated neonatal morbidities. *Obstet Gynecol*. 2019;133(2): e156-e163.
50. Clark SL, Frye DR, Meyers JA, et al. Reduction in elective delivery at <39 weeks of gestation: comparative effectiveness of 3 approaches to change and the impact on neonatal intensive care admission and stillbirth. *Am J Obstet Gynecol*. 2010;203(5):449.e1-449.e6.
51. Nance J. Keynote speech. Presented at: National Forum on Quality Improvement in Healthcare. 1999; New Orleans, LA.
52. Olsen O, Clausen JA. Planned hospital birth versus planned home birth. *Cochrane Database Syst Rev*. 2012;(9):CD000352.
53. Vedam S, Leeman L, Cheyney M, et al. Transfer from planned home birth to hospital: improving interprofessional collaboration. *J Midwifery Womens Health*. 2014; 59(6):624-634.
54. United Nations Children's Fund (UNICEF). Maternal mortality fell by almost half between 1990 and 2015. 2017. Available at http://data.unicef.org/topic/maternal-health/maternal-mortality/.
55. Moaddab A, Dildy G, Brown H, et al. Health care disparity and pregnancy-related mortality in the United States, 2005-2014. *Obstet Gynecol*. 2018;131(4):707-712.
56. Howell EA, Brown H, Brumley J, et al. Reduction of peripartum racial and ethnic disparities: a conceptual framework and maternal safety consensus bundle. *Obstet Gynecol*. 2018;131(5):770-782.
57. Creanga A, Syverson C, Seed K, Callaghan W. Pregnancy-related mortality in the United States, 2011-2013. *Obstet Gynecol*. 2017;130(2):366-373.
58. Boulware DR. Recent increases in the U.S. maternal mortality rate: disentangling trends from measurement issues. *Obstet Gynecol*. 2017;129(2):385-386.
59. Main EK, Cape V, Abreo A, et al. Reduction of severe maternal morbidity from hemorrhage using a state perinatal quality collaborative. *Am J Obstet Gynecol*. 2017; 216(3):298.e1-298.e11.
60. Council on Patient Safety in Women's Health Care. 2018. Available at http://safehealthcareforeverywoman.org/patient-safety-bundles/.
61. Agency for Healthcare Research and Quality. Toolkit for improving perinatal safety. Available at https://www.ahrq.gov/professionals/quality-patient-safety/hais/tools/perinatal-care/index.html.
62. Pearlman MD. Patient safety in obstetrics and gynecology: an agenda for the future. *Obstet Gynecol*. 2006; 108(5):1266-1271.
63. Weinstein L. A multifacited approach to improve patient safety, prevent medical errors and resolve the professional liability crisis. *Am J Obstet Gynecol*. 2006;194(4): 1160-1165, discussion 1165-1167.
64. Xu X, Siefert KA, Jacobson PD, Lori JR, Ransom SB. The impact of malpractice burden on Michigan obstetrician-gynecologists' career satisfaction. *Womens Health Issues*. 2008;18(4):229-237.

65. Huycke LI, Huycke MM. Characteristics of potential plaintiffs in malpractice litigation. *Ann Intern Med*. 1994; 120(9):792-798.

66. Burns LR, Connolly T, DeGraaff RA. Impact of physicians' perceptions of malpractice and adaptive changes on intention to cease obstetrical practice. *J Rural Health*. 1999;15(2):134-146.

67. Carpentieri A, Lumalcuri J, Shaw J, Joseph G. Overview of the 2015 American Congress of Obstetricians and Gynecologists' Survey on Professional Liability. 2015. Available at https://www.acog.org/-/media/Departments/Professional-Liability/2015PLSurveyNationalSummary11315.pdf.

68. Baldwin LM, Greer T, Wu R, Hart G, Lloyd M, Rosenblatt RA. Differences in the obstetric malpractice claims filed by Medicaid and non-Medicaid patients. *J Am Board Fam Pract*. 1992;5(6):623-627.

69. Kraman SS, Hamm G. Risk management: extreme honesty may be the best policy. *Ann Intern Med*. 1999; 131(12):963-967.

70. Roberts RG. Seven reasons family doctors get sued and how to reduce your risk. *Fam Pract Manag*. 2003; 10(3):29-34.

71. American College of Obstetricians and Gynecologists Committee on Ethics. Committee opinion no. 480: empathy in women's health care. *Obstet Gynecol*. 2011; 117(3):756-761.

72. Merel SE, McKinney CM, Ufkes P, Kwan AC, White AA. Sitting at patients' bedsides may improve patients' perceptions of physician communication skills. *J Hosp Med*. 2016;11(12):865-868.

73. Swayden KJ, Anderson KK, Connelly LM, Moran JS, McMahon JK, Arnold PM. Effect of sitting vs. standing on perception of provider time at bedside: a pilot study. *Patient Educ Couns*. 2012;86(2):166-171.

74. Cabeeza PJ, Ramisetty P, Thompson PJ, Khan KS. Risk communication: illusion or reality? *J Obstet Gynaecol*. 2005;25(7):635-637.

75. American Medical Association. Health literacy and patient safety: help patients understand. Reducing the risk by designing a safer, shame-free health care environment. Available at https://www.pogoe.org/sites/default/files/Health%20Literacy%20-%20Reducing%20the%20Risk%20by%20Designing%20a%20Safe,%20Shame-Free%20Health%20Care%20Environment.pdf.

76. Clark SL, Belfort MA, Dildy GA, Meyers JA. Reducing obstetric litigation through alterations in practice patterns. *Obstet Gynecol*. 2008;112(6):1279-1283.

77. Committee on Patient Safety and Quality Improvement. Committee opinion no. 681: disclosure and discussion of adverse events. *Obstet Gynecol*. 2016;128(6): e257-e261.

78. Witman AB, Park DM, Hardin SB. How do patients want physicians to handle mistakes? A survey of internal medicine patients in an academic setting. *Arch Intern Med*. 1996;156(22):2565-2569.

79. American College of Obstetricians and Gynecologists Committee Committee on Patient Safety and Quality Improvement. ACOG committee opinion no. 447: patient safety in obstetrics and gynecology. *Obstet Gynecol*. 2009;114(6):1424-1427.

80. Thaddeus S, Maine D. Too far to walk: maternal mortality in context. *Soc Sci Med*. 1994;38(8):1091-1110.

Intrapartum Fetal Surveillance

Learning Objectives

1. Explain the concepts of structured intermittent auscultation (SIA) and electronic fetal monitoring (EFM).
2. Apply to continuous electronic fetal monitoring (CEFM) the National Institute of Child Health and Human Development (NICHD) terminology.
3. Develop an overall assessment and management plan for EFM and SIA, using the mnemonic DR C BRAVADO.

Introduction

Monitoring the fetal heart rate (FHR), via auscultation or electronic fetal monitoring (EFM), is essential during the labor and delivery process. EFM remains the mainstay technique,[1] but structured intermittent auscultation (SIA) can be safely applied in low-risk pregnancies.[2,3] Studies have shown that although EFM has resulted in a reduction in neonatal seizures there have been no significant decreases in cerebral palsy, infant mortality, or other standard measures of infant well-being.[4] However, EFM has resulted in increased cesarean deliveries and operative vaginal deliveries.[4]

Despite the introduction of standardized fetal monitoring terminology in 1997 and again in 2008 by the Eunice Kennedy Shriver National Institute of Child Health and Human Development (NICHD), use of EFM is limited by interobserver and intraobserver variability in interpretation.[5-7] Management guidelines and algorithms, based on standardized interpretation of fetal monitor tracings, continue to be developed to assist providers with choosing management strategies applicable to their clinical practice settings.[8-10] Therefore, using a standardized language is essential if these evidence-based algorithms and guidelines are to be used effectively. In an effort to improve consistency, many facilities are now requiring maternity staff (medical care providers and nurses) to complete a course on fetal monitoring and/or obtain national certification in fetal monitoring.[11,12] These courses have significantly improved consistency in documentation and interpretation of fetal monitoring strips.[11]

Fetal Heart Rate Surveillance Techniques

Two techniques are available for assessing FHR: SIA using a handheld Doppler device or fetal stethoscope, and EFM via external electronic fetal monitor using a cardiotransducer, or internal electronic fetal monitor using a fetal spiralelectrode (FSE). EFM via external fetal monitor may also be used intermittently during labor; however, there is no defined protocol and no evidence to support this use. Both techniques have advantages and disadvantages, and providers should be aware of these. The decision to choose SIA or EFM starts with assessing maternal medical and obstetric risk factors, as well as fetal risks for uteroplacental insufficiency. In addition, patient and provider preferences, available resources, and departmental policies should be considered. Optimally, a discussion of FHR monitoring techniques should occur before labor, so options can be explored and patient questions answered. Advantages and disadvantages of EFM and SIA should be reviewed and patient preferences determined.

Indications for EFM include maternal medical complications (eg, type 1 diabetes), maternal obstetric complications (eg, preeclampsia), intrapartum complications, use of uterine stimulants, and known fetal conditions (eg, anomalies, anemia, and intrauterine growth restriction [IUGR]). If a pregnancy is considered low-risk, the American College of Obstetricians and Gynecologists (ACOG), the American College of Nurse-Midwives (ACNM), and the Association of Women's Health, Obstetric and Neonatal Nurses (AWHONN) consider intermittent auscultation to be an acceptable choice.[3,13-15] ACOG recommends that women with high-risk conditions undergo EFM.[14]

Structured Intermittent Auscultation

When selecting SIA, the personnel available in the maternity care unit should be considered. SIA often requires a 1:1 nurse-to-patient ratio and a near-constant

presence of the nursing bedside provider to auscultate at the recommended intervals. However, the benefit of this near-constant presence has been shown to improve outcomes and increase patient satisfaction.[16] Patients often have greater freedom of movement with SIA. This ability to ambulate and make frequent position changes assists with enhancing labor progress. One systematic review of 5,218 women showed that women who were mobile or upright during labor reduced the first stage by approximately 1 hour and 20 minutes.[17] In addition, they also had a decreased incidence of cesarean delivery, reduced use of epidural analgesia, and had newborns with fewer neonatal intensive care unit (NICU) admissions.[17] Lastly, nurse training, comfort levels, and skill with SIA should be assessed. Training can be offered to familiarize health care providers with SIA.[13] To encourage SIA in low-risk patients who want this option, facilities should consider developing protocols and training staff in this technique. Advantages and disadvantages of SIA are outlined in *Table 1*.

Table 1. Summary of Advantages and Disadvantages of Systematic Intermittent Auscultation

FHR: Handheld doppler or Fetoscope	Uterine Activity: Palpation
Advantages	**Advantages**
Non-invasive	Noninvasive
Can be used anytime FHR is audible	Detects relative frequency, duration and intensity of contractions
Detects baseline and rhythm	Detects relative uterine resting tone
Detects increases and decreases from FHR baseline	Increased bedside attendance of provider
Allows freedom of movement	No cost
Less costly equipment than CEFM	Allows freedom of movement
Increased bedside attendance by provider	
Evidence indicates outcomes are comparable with those of CEFM	
Lower incidence of cesarean delivery when used	
Disadvantages	**Disadvantages**
Does not produce a tracing	Does not detect accurate intensity of uterine activity (contractions and resting tone)
May miss some cardiac events as not continuous	Maternal obesity may limit accuracy of assessment
Requires skill and training	Subjective between different providers
For low-risk patients	
Ability to hear fetal heart sounds may be limited in obese patients, active patients (maternal and fetal), increased amniotic fluid, and with uterine contractions	
Requires 1:1 nurse-to-patient ratio	

FHR = fetal heart rate; CEFM = continuous electronic fetal monitoring.

To determine baseline FHR using SIA, the FHR should be auscultated in between contractions when the fetus is not moving.[3,13] It is important that providers simultaneously assess the maternal pulse to ensure that the FHR is being auscultated rather than the maternal heart rate (MHR). Count for 15 to 60 seconds at the recommended intervals.

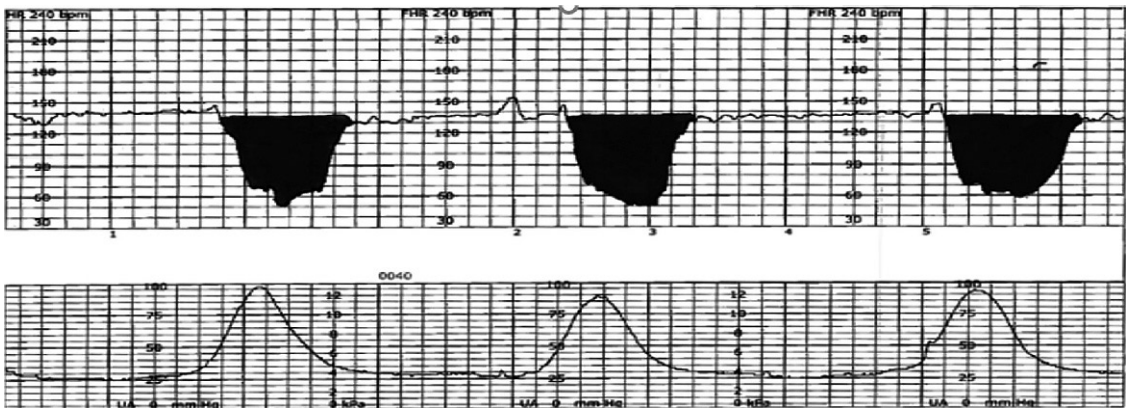
Evidence for the best timing to evaluate FHR changes from the baseline (accelerations or decelerations) is limited. One of the goals of listening through and shortly after a contraction is to determine decelerations associated with uterine activity; however, counting through a contraction may be unachievable and technically challenging (*Figure 1*). One suggested technique for ascertaining these decelerations is to listen during the second half of the contraction and count in multiples of 5- to 15-second increments. This method appears to be the most accurate to assess rate, rhythm, accelerations, and decelerations associated with contractions.[3] However, it must be remembered that SIA cannot determine baseline variability or the type of deceleration pattern. Despite this, researchers comparing SIA with EFM found them to be equivalent in terms of neonatal outcomes.[18,19] Therefore, with low-risk women, based on existing evidence, SIA's limitation of not being able to determine variability or the type of decelerations does not appear to be of clinical significance.[3]

To date, no studies have been conducted to assess the optimal frequency for SIA in the absence of risk factors. Professional organizations differ slightly in their recommendations for SIA frequency (*Table 2*).

General guidelines for performing SIA have been set forth by AWHONN and ACNM (*Table 3*).

Normal findings for SIA include a normal FHR baseline between 110 to 160 beats per minute (BPM), regular rhythm, presence of FHR increases from baseline, and the absence of FHR decreases from baseline.[3,13] These findings indicate a fetus that is well oxygenated and nonacidotic, and can be monitored in a routine manner.[3,13] SIA findings are FHR baselines greater than or equal to 160 BPM or less than or equal to 110 BPM, irregular rhythms, and the presence of gradual or abrupt decreases from the baseline. That may be indicative of a fetus who is not

Figure 1. Calculating Deceleration Area



Reprinted from Cahill AG, Tuuli MG, Stout MJ, et al. A prospective cohort study of fetal heart rate monitoring: deceleration area is predictive of fetal acidemia. Am J Obstet Gynecol*. 2018;218:523.e1-12.*

Table 2. Professional Organization Recommendations for Structured Intermittent Auscultation in Low-Risk Women

	Latent Phase (<4 cm)	Active First Stage	Active Second Stage
ACOG		Every 30 min	Every 15 min
ACOG/AAP Guidelines for Perinatal Care		Every 30 min	Every 15 min
ACNM		Every 15 to 30 min	Every 5 min
AWHONN	At least hourly	Every 15 to 30 min	Every 5 to 15 min
RCOG		Every 15 min	Every 5 min
SOGC	At time of assessment and approximately every hour thereafter	Every 15 to 30 min	Every 5 min

AAP = American Academy of Pediatrics; ACNM = American College of Nurse Midwives; ACOG = American College of Obstetricians and Gynecologists; AWHONN = Association of Women's Health, Obstetric and Neonatal Nurses; RCOG = Royal College of Obstetricians and Gynaecologists; SOGC = Society of Obstetricians and Gynaecologists of Canada.

Information from Lyndon A, Ali LU, eds. Association of Women's Health, Obstetric and Neonatal Nursing. Fetal Heart Monitoring Principles and Practices. 5th ed. Washington DC: Kendall Hunt Professional; 2015. AWHONN Position Statement. Fetal Heart Monitoring. JOGNN. 2015; 44(5): 683-686; American College of Obstetricians and Gynecologists. ACOG Practice Bulletin no. 106: intrapartum fetal heart rate monitoring: nomenclature, interpretation, and general management principles. Obstet Gynecol. 2009;114(1):192-202; Kilpatrick SJ, Papile LA, eds. Guidelines for Perinatal Care. 8th ed. Washington DC: American College of Obstetricians and Gynecologists; 2017; American College of Nurse-Midwives. Intermittent Auscultation for Intrapartum Fetal Heart Rate Surveillance (replaces ACNM Clinical Bulletin #60, Sept/Oct 2015). J Midwifery Womens Health. 2015;60(5):626-632; Royal College of Obstetricians and Gynaecologists. The Use of Electronic Fetal Monitoring. The use and interpretation of cardiotocography in intrapartum fetal surveillance. Evidence-based Clinical Guideline Number 8. London: RCOG Press; 2001; Liston R, Sawchuck D, Young D; Society of Obstetrics and Gynaecologists of Canada; British Columbia Perinatal Health Program. Fetal health surveillance: antepartum and intrapartum consensus guideline. J Obstet Gynaecol Can. 2007;29(9)(Suppl 4):S3-S56. Erratum in J Obstet Gynaecol Can. 2007;29.

optimally oxygenated.[3,13] EFM should be used to verify or clarify these patterns and guide possible intervention strategies.[3] Should the FHR pattern not resolve or become recurrent, EFM should be considered.[3] Strategies for successful implementation of SIA are shown in *Table 4*.

Electronic Fetal Monitoring

Electronic fetal monitoring can be external or internal. External EFM consists of a Doppler ultrasound to capture the FHR and a tocotransducer (or tocodynamometer) to detect uterine activity. The FHR Doppler uses a Doppler signal that is designed to detect wave forms (ie, motion). The Doppler produces sound waves that are transmitted toward a structure (ie, fetal heart). When that structure moves (ie, heart motion) the soundwaves bounce back and return to the Doppler at a different frequency than when they were sent. This phenomenon is called the *Doppler shift*. This shift is then amplified to create a wave form, which is further processed by the monitor's computer. A recording of the FHR is generated from this signal processing.

It is important to remember that external FHR monitoring is not a direct electrocardiogram (ECG) reading of the FHR. As such, a common error when using the external Doppler is to inadvertently detect and record the MHR rather than the FHR. This occurs when the Doppler is placed over a maternal blood vessel. In this case, fetal bradycardia may be erroneously assumed when, in fact, it is the MHR recording. On the other hand, the MHR might be doubled giving the mistaken appearance of fetal tachycardia. In yet another instance, if the woman is tachycardic, thereby having a heart rate within the normal range for a fetus (ie, 110 to 160 BPM), maternal tachycardia can be recorded on the monitor strip. This can lead providers to assume that the FHR is being recorded when, in fact, it is the MHR.

To avoid these errors, external monitors should be frequently adjusted to obtain an adequate tracing and signal quality. In addition, they may be difficult to use in patients who are obese, ambulating, active in bed, or in the second stage of labor. In cases where it is suspected that MHR and not FHR is being recorded, maternal pulse oximetry may be used to differentiate between the two.

The tocotransducer, or *toco,* on the external fetal monitor detects uterine activity. The toco can detect the frequency and duration of the contractions but cannot assess their actual strength. When using the toco, providers must palpate the abdomen to determine contraction strength. Contractions are generally described as mild (+1), moderate (+2), or strong (+3) depending on the firmness of the abdominal wall during the contraction. A common gauge many providers use is mild being comparable to compressing the tip of the nose, moderate being comparable to compressing the chin, and strong being comparable to compressing the forehead.[13]

Table 3. Guidelines for Auscultation

Procedure for Auscultation

1. Palpate the abdomen to determine the position of the fetus (Leopold maneuvers)
2. Place the Doppler over the area of maximum intensity of fetal heart tones, generally over the fetal back
3. Differentiate MHR from FHR by simultaneously palpating maternal radial artery or assessing the MHR electronically
4. Palpate for uterine contraction during period of FHR auscultation to determine relationship of contraction to FHR
5. Count baseline FHR between contractions and when the fetus is not moving. Count FHR for 15 to 60 seconds, per facility protocol, to determine the baseline rate
6. Count FHR after uterine contraction using multiple consecutive 5- to 15-second intervals for 30 to 60 seconds to determine differences between baseline FHR and fetal response to contractions (this may be subject to hospital protocols)

FHR = fetal heart rate; MHR = maternal heart rate.

Information from Lyndon A, Ali LU, eds. Association of Women's Health Obstetric and Neonatal Nursing. Fetal Heart Monitoring Principles and Practices. 5th ed. Washington DC: Kendall Hunt Professional; 2015; American College of Nurse-Midwives. Intermittent Auscultation for Intrapartum Fetal Heart Rate Surveillance (replaces ACNM Clinical Bulletin #11, March 2010). J Midwifery Womens Health. 2015;60(5):626-632.

Table 4. Strategies for Successful Implementation of Structured Intermittent Auscultation

1. The presence of nurses and providers experienced in auscultation, palpation of contractions, and auditory recognition of FHR changes are necessary
2. Institutional policy should be developed to promote SIA and address the technique and frequency of assessment
3. Clinical interventions should follow when concerning findings are present
4. Nurse-to-patient ratio is 1:1
5. User-friendly documentation tools for recording SIA findings
6. Ready availability of auscultation devices
7. Culture embracing normalcy of childbirth and minimization of unnecessary interventions

FHR = fetal heart rate; SIA = structured intermittent auscultation.

Information from Intermittent Auscultation for Intrapartum Fetal Heart Rate Surveillance: American College of Nurse-Midwives. J Midwifery Womens Health. *2015;60(5):626-632; Lyndon A, Ali LU, eds. Association of Women's Health, Obstetric, and Neonatal Nursing.* Fetal Heart Monitoring Principles and Practices. *5th ed. Washington DC: Kendall Hunt Professional; 2015; Committee on Obstetric Practice. Committee Opinion No. 687: Approaches to Limit Intervention During Labor and Birth.* Obstet Gynecol. *2017;129(2):e20-e28.*

Internal EFM consists of a FSE to assess FHR and an intrauterine pressure catheter (IUPC) to record contractions. Rupture of membranes and cervical dilatation are required for placement of these devices. Morbidity, although uncommon, includes maternal and fetal soft tissue injuries and intrauterine infections. Relative contraindications include active maternal genital herpes, HIV infection, hepatitis C virus infection, and any presentation in which placement would not be possible (eg, placenta previa).[13] In addition, FSEs should not be placed in the setting of undiagnosed vaginal bleeding until placenta previa is ruled out by ultrasound. The electrode may be placed on the buttocks in breech presentations, however, care must be taken to avoid placement on the fetal genitalia. The FSE captures the FHR using an ECG signal. In cases of fetal death, the FSE may detect the maternal ECG, amplify it, and record it on the tracing. This may give the false impression that the FHR is being recorded rather than the MHR.[13]

The internal uterine monitor consists of an IUPC. The IUPC allows for the accurate calibration of uterine activity measured in mm Hg. Montevideo units (MVUs), which give an accurate assessment of the work of labor, can then be calculated. MVUs are calculated using the sum of the peak of contractions in a 10-minute window, minus the resting tone after each contraction. MVUs cannot be calculated during the second stage of labor. Many electronic systems will now automatically calculate MVUs, making this an easy measure to assess. Adequate uterine activity is typically MVUs of 180 to 240 mm Hg, with 91% of women with MVUs of 200 to 224 mm Hg successfully delivering vaginally.[20]

Although there are no absolute indications for use of internal fetal and/or internal uterine monitoring devices, providers may use internal methods when external methods are inadequate or to gain additional information about fetal status or uterine activity. For instance, an IUPC may be indicated when an external toco fails to sufficiently detect uterine activity and an objective means to measure uterine activity is needed. An IUPC is indicated in the instance of needing to provide an amnioinfusion. A decision to use an FSE may occur if the external monitor is not adequately detecting the FHR leaving large gaps in the tracing. *Table 5* summarizes the advantages and disadvantages of internal and external EFM methods.

Electronic fetal monitoring at admission. In the United States, women in labor in a hospital maternity care unit will frequently have an EFM placed for approximately 20 minutes. The method of subsequent fetal monitoring is often determined based on the interpretation of this initial 20-minute tracing as well as other factors including risk factors, institutional policies, and patient and provider preferences. In some cases, EFM will last longer if the tracing is concerning. A 2017 systematic review of more than 13,000 women showed that compared with SIA, an EFM tracing in triage for low-risk women showed no benefit and actually increased the risk of cesarean delivery in these women by approximately 20%.[19] In another systematic review of three randomized controlled trials (RCT) including 11,259 women and 11 observational studies including 5,831 women, no significant difference was found in outcomes comparing women who underwent EFM on admission with those who did not.[21]

Electronic fetal monitoring outcomes. The only clinically significant benefit shown with routine EFM is the reduction of neonatal seizures in the immediate newborn period. This appears to be a short-term benefit, because these infants did not have permanent sequelae at the end of 1 year.[4] In the largest RCT of infants with early seizures, there was no significant difference in the rates of perinatal death or cerebral palsy when compared with those who received SIA versus EFM.[16,18] A follow-up at age 4 years showed no significant difference between those with cerebral palsy and those without, leading researchers to conclude that EFM offers little, if any, benefit for reduction in neurologic injury. [16,18,22]

An unrealistic expectation persists that certain FHR tracings will predict neurologic injury.[14,23] The incidence of cerebral palsy has been stable since the introduction of EFM. Cerebral palsy is attributed to events that occur before labor in approximately 70% of cases.[14,22] Only 4% of cases caused by hypoxic ischemic encephalopathy can be directly linked to intrapartum events.[14,23] In newborns with an estimated fetal weight greater than or equal to 5.5 lb (2,500 g), it has been estimated that the positive predictive value of an abnormal FHR tracing for cerebral palsy is 0.14%,[14] and the false-positive rate of EFM for cerebral palsy is greater than 99%.[14] A systematic review comparing SIA to EFM in more than 3,000 women and newborns showed no clear differences in neonatal outcomes

or long-term outcomes for the infant, but showed an increase in cesarean delivery rates in the EFM group.[2] Despite this lack of scientific support, EFM remains in use almost universally in the hospital setting. Continuous monitoring in particular may contribute to increased rates of maternal morbidity due to unnecessary medical interventions, such as cesarean delivery or assisted vaginal delivery, without improving fetal outcomes.[14,25]

Interpreting Electronic Fetal Monitoring Fetal Heart Rate Tracings

Electronic fetal monitoring has been under scrutiny because of the lack of consistent

Table 5. Summary of Advantages/Disadvantages of Internal Versus External Electronic Fetal Monitoring

External EFM FHR: Doppler	Internal EFM FHR: FSE
Advantages	**Advantages**
Noninvasive	Minimal artifact with more continuous tracing
Accurate assessment of FHR variability	Less adjustment of monitor
Can be used antepartum as well as intrapartum	
No contraindication for use except patient refusal	
Disadvantages	**Disadvantages**
Requires frequent readjustment to maintain adequate tracing	Invasive: Requires rupture of membranes and cervical dilatation
Increased artifact	Requires skill in placement
Difficult to use in obese patients	Increases risk of maternal/fetal infection
Maternal and fetal movement affect ability to maintain tracing	Increases risk of fetal soft tissue injury
May be difficult to obtain tracing in second stage or after active pushing begins	May pick up MHR in presence of a dead fetus and record on tracing
May pick up MHR and record on tracing	Limits ambulation
May record MHR in presence of a dead fetus	May be contraindicated in herpes simplex virus, HIV, hepatitis, group B streptococcus, known fetal blood dyscrasias
May half and double rates, especially in presence of fetal tachycardia or bradycardia	May be contraindicated in vaginal bleeding
Cannot "count" above 240 BPM and will generally halve these rates or not record	
Fetal arrhythmias difficult to trace	
Uterine Activity: Tocodynamometer ("Toco")	**Uterine Activity: IUPC**
Advantages	**Advantages**
Noninvasive, does not require rupture of membranes	Accurate assessment of uterine activity and calculation of Montevideo units
Can assess frequency and duration of uterine contractions	Provides access for amnioinfusion
Easy application	
Disadvantages	**Disadvantages**
Cannot assess intensity or resting tone	Invasive
Poor placement could result in inaccurate tracing	Requires skill in placement
	Same contraindications as FSE
	Maternal position and changes in maternal position affect readings
	IUPC may become obstructed with meconium, vernix, or blood and not give reliable reading
	IUPC may become lodged against uterine wall or fetal part and not give reliable reading
	Increased risk of uterine perforation

BPM = beats per minute; EFM = electronic fetal monitoring; FHR = fetal heart rate; FSE = fetal spiral electrode; IUPC = intrauterine pressure catheter; MHR = maternal heart rate.

interpretation of FHR tracings.[5,6,9,25] In 1997, NICHD issued guidelines to "develop standardized and unambiguous definitions for FHR tracings."[6] The goal of this standardization was to set forth research guidelines to enable future studies to determine the predictive value of EFM, which could lead to an evidence-based approach for management of EFM tracings.[6] In 2008, NICHD updated their definitions, interpretation, and research guidelines.[25] ACOG incorporated these guidelines into their 2009 Practice Bulletin *Intrapartum Fetal Heart Rate Monitoring: Nomenclature, Interpretation, and General Management Principles*.[14] AWHONN also incorporated these guidelines into their training materials and practice guidelines.[13,26] In 2010, ACOG released a second Practice Bulletin on management of intrapartum FHR tracings based on the three-tier category system set forth by the NICHD in 2008. This bulletin also included guidelines for management of uterine tachysystole.[7] ACOG describes FHR tracings as visual patterns that should be adaptable to computerized interpretation, and notes that definitions and categorizations should be applied to intrapartum tracings but also can be used in the antepartum period.[14,25]

When performing EFM, it is recommended that the monitor strips be reviewed frequently by a nurse or physician trained to do so and assessments documented periodically. In uncomplicated patients, fetal monitor strips should be reviewed every 30 minutes during the first stage of labor, and every 15 minutes during the second stage. If the pregnancy is complicated (eg, IUGR, preeclampsia), the monitor strip should be reviewed more frequently: a suggested interval is every 15 minutes during the first stage of labor and every 5 minutes during the second stage.[14]

DR C BRAVADO Mnemonic

The mnemonic DR C BRAVADO (**D**efine **R**isk, **C**ontractions, **B**aseline **RA**te, **V**ariability, **A**ccelerations, and **D**ecelerations, interpretation [**O**verall assessment]) was developed specifically for the ALSO program to denote a systematic approach to interpreting EFM FHR tracings. This mnemonic does not compete with or replace the NICHD guidelines defined later, but reinforces their use. When using this mnemonic, the FHR tracing and uterine contractions should be of adequate quality for visual interpretation (*Table 6).*

Table 6. DR C BRAVADO for Electronic Fetal Monitoring Tracings

DR	Define risk (low or high)
C	Contractions (frequency, duration, intensity, resting tone)
BRA	Baseline rate (normal 110-160 bpm, bradycardia, tachycardia)
V	Variability (absent, minimal, moderate, marked)
A	Accelerations
D	Decelerations (early, variable, late, prolonged)
O	Overall assessment (normal, indeterminate, abnormal)

BPM = beats per minute.

Table 7. DR C BRADO for Structured Intermittent Auscultation

DR	Define risk (low or high)
C	Contractions (frequency, duration, intensity, resting tone)
BR	Baseline rate (normal 110-160 bpm, bradycardia, tachycardia)
A	Accelerations (increases from baseline)
D	Decelerations (decreases from baseline)
O	Overall assessment (normal, indeterminate)

BPM = beats per minute.

When using SIA, the mnemonic is DR C BRADO (**D**efine **R**isk, **C**ontractions, **B**aseline **R**ate, **A**ccelerations, **D**ecelerations). In addition, there must be an adequate quality of FHR sounds.[13] Providers are reminded that definitions for decelerations (late, early, and variable decelerations) and variability apply only to EFM tracings and therefore should not be used with SIA.[13,25] However, baseline changes can be accurately determined using SIA (*Table 7*).[3,13]

Define Risk

Before any FHR tracing can be interpreted, the patient's history and the clinical context of the situation need to be evaluated to determine risk.[25] Many FHR characteristics are dependent on gestational age and the woman's physiologic status.

Consideration must be given to drugs, prior antepartum testing, maternal status, and fetal medical conditions (eg, anomalies, growth restriction, arrhythmias).[25] Risk primarily refers to the risk of having or developing uteroplacental insufficiency or the risk of a sudden event such as placental abruption or cord prolapse. Low risk generally includes women with clear amniotic fluid, no unusual intrapartum bleeding, normal prenatal course, normal prenatal testing, nonconcerning initial admission FHR tracing, no conditions with increased risk of developing fetal acidemia during labor, no maternal condition that may affect fetal well-being, and no requirement for oxytocin.[16] The fetus's ability to respond to hypoxic events, based on these risks, must be considered in the overall management of fetal monitoring (SIA or EFM). For example, a tracing with late decelerations might be managed differently in a woman with preeclampsia and long labor compared with that of a woman with normal pregnancy and a normal previous tracing who just received epidural analgesia and has hypotension.

Contractions

Uterine monitoring can be performed internally using an IUPC, externally using a toco, or by palpation to determine the duration and frequency of contractions. Strength of a contraction cannot be determined with the external toco and requires placement of an IUPC or palpation of the abdominal wall. Uterine contraction frequency can be quantified as the number present over a 10-minute period averaged over 30 minutes. Routine use of an IUPC is discouraged due to lack of clinical benefit and increased risk of fever.[27,28]

Uterine activity is classified as normal (five or fewer contractions in a 10-minute period averaged over 30 minutes) or tachysystole (more than five contractions in a 10-minute period averaged over 30 minutes).[14,25] Tachysystole should be qualified as to the presence or absence of decelerations. Tachysystole applies to spontaneous and stimulated labor, but management may differ if the uterine activity is induced rather than spontaneous.[14] *Hyperstimulation* and *hypercontractility* are poorly defined, and therefore use of these terms should be discontinued.[7,14]

Baseline Rate

The baseline heart rate is calculated by averaging the rate rounded to 5-BPM intervals over a 10-minute segment for EFM. Segments that should be excluded are those that have marked variability (more than 25 BPM), are greater than or equal to 25 BPM above or below the baseline, or contain accelerations or decelerations. There must be at least a 2-minute identifiable segment within any 10-minute period. This 2-minute segment does not need to be contiguous.[25] The normal range is 110 to 160 BPM.[13,14,25] When performing SIA, the average baseline rate should be determined between contractions. Changes in baseline are common in term labor and predict morbidity poorly.[29] Causes of a change in baseline rate may include change in fetal status, chorioamnionitis, drugs, maternal fever, position, and prematurity.[7,13,30]

Bradycardia

Bradycardia is defined as a baseline heart rate less than 110 BPM for greater than or equal to 10 minutes.[25] Bradycardia of 80 to 110 BPM with moderate variability may represent normal physiologic variation with increased vagal tone, and is not typically associated with hypoxemia.[13,30] Rates less than 70 BPM may be seen in fetuses with congenital heart disease or myocardial conduction defects.[30] Maternal causes of fetal bradycardia include supine positioning, hypotension, hypoglycemia, tachysystole, or hypothermia. Fetal causes include prolonged cord occlusion, prolapsed cord, rapid descent, or fetal decompensation.[7,13,30]

Tachycardia

Tachycardia is defined as a baseline heart rate greater than 160 BPM for greater than or equal to 10 minutes.[13,14,25] Fetal movement and maternal anxiety, fever, dehydration, ketosis, and beta-adrenergic agonist use can cause fetal tachycardia unassociated with hypoxia.[7,13] Fetal immaturity, thyrotoxicosis, and anemia also can cause fetal tachycardia.[7,13] Fetal tachycardia can also be caused by illicit drug use[7,13] and, in some instances, maternal drug screening is indicated. Persistent tachycardia greater than 180 BPM, especially if maternal fever is present, suggests chorioamnionitis.[31] An FHR baseline greater than 200 BPM is frequently due to fetal arrhythmia or other congenital anomaly.[7] "In isolation, fetal tachycardia is poorly predictive for fetal hypoxemia or acidemia unless accompanied by minimal or absent FHR variability or recurrent decelerations or both."[7] If tachycardia persists in the

preterm fetus, close surveillance and assessment for other causes is warranted.[30]

Variability

The NICHD guidelines state that FHR variability should no longer be described as short-term (beat-to-beat) or long-term, nor should the terms *good*, *increased*, *decreased*, or *average* be used. Definitions to characterize variability are specifically classified as absent (undetectable), minimal (undetectable to 5 BPM or less), moderate (6 to 25 BPM), or marked (greater than 25 BPM) (*Table 8*).[25]

The FHR normally exhibits fluctuations in baseline heart rate activity that is irregular in amplitude and frequency. The presence of variability represents an intact nervous pathway through the cerebral cortex, the midbrain, the vagus nerve, and the normal cardiac conduction system. When the fetus is well oxygenated, the central nervous system responds with moderate variability.[13,21,25] However, minimal or absent variability alone is not necessarily predictive of hypoxemia or metabolic acidemia.[25] The degree of FHR variability is affected by the fetal state and by multiple causes other than uteroplacental insufficiency or acidosis. Fetal sleep cycles, narcotics, steroids, and other drugs (eg, analgesics, anesthetics, barbiturates, tranquilizers, atropine, magnesium sulfate) may decrease FHR variability.[7,32,33] Cardiac conduction defects and congenital central nervous system anomalies can cause a decrease in variability that is not hypoxic in origin.[25,30] In addition, decreased variability can occur with no known cause.

Table 8. Definitions of Fetal Heart Rate Variability

Variability	Amplitude Range
Absent	Undetectable
Minimal	Detectable to ≥5 BPM
Moderate	6 to 25 BPM
Marked	>25 BPM

BPM = beats per minute.

Information from Macones GA, Hankins GD, Spong CY, Hauth J, Moore T. The 2008 National Institute of Child Health and Human Development workshop report on electronic fetal monitoring: update on definitions, interpretation, and research guidelines. Obstet Gynecol. *2008;112(3):661-666.*

However, variability remains the most important predictor of fetal oxygenation and is therefore a vital element of EFM assessment.[25]

The finding most strongly associated with fetal acidemia is absent baseline variability accompanied by recurrent late decelerations, recurrent variable decelerations, or bradycardia.[25] This is an abnormal tracing and requires prompt intervention.[7,25] The significance of marked variability (greater than 25 BPM) is unclear but is likely indicative of a fetus in hypoxic stress that is still able to maintain central oxygenation. The presence of minimal, absent, or marked variability should be further evaluated within the context in which each occurs.

Accelerations

Accelerations are visually apparent, abrupt increases in FHR above the most recent baseline with an onset to peak of less than 30 seconds. The peak of the acceleration is 15 BPM or greater (10 BPM or greater if less than 32 weeks' gestation) and lasts for 15 seconds or more (10 seconds or more if less than 32 weeks' gestation).[14,25] The return to baseline is within 2 minutes. If the acceleration lasts 2 minutes or more but less than 10 minutes, it is defined as a prolonged acceleration. The absence of accelerations does not necessarily indicate fetal acidemia, but may warrant the need for further evaluation.[25] When used in antenatal testing, a contraction stress test or biophysical profile may be used to clarify fetal status in the presence of a nonreactive nonstress test (ie, less than two FHR accelerations in 20 minutes that last 15 BPM for 15 seconds or 10 BPM for 10 seconds).

The presence of accelerations, whether spontaneous or stimulated, is strongly predictive of normal acid-base status at the time of observation.[14,25] When accelerations are seen in association with variable decelerations, they generally indicate partial cord compression and sometimes are referred to as *shoulders*. This occurs when the vein is compressed in the umbilical cord but the thicker-walled arteries remain patent. These accelerations are part of the variable deceleration (ie, physiologic response to cord compression) and therefore should not be used to assess fetal well-being or fetal acid-base status.

Although the presence of accelerations assures the provider of normal acid-base status, the disappearance or absence of FHR accelerations does not necessarily indicate a hypoxic or acidemic fetus.[25]

Drugs and sleep cycles may cause accelerations to disappear.[7,14] Therefore, if accelerations disappear, providers should look for other indicators of compromise (eg, decelerations increasing in depth, duration, or frequency, decreased baseline variability, baseline tachycardia or bradycardia). In addition, providers can attempt to elicit accelerations using scalp or vibroacoustic stimulation. Of note, FHR accelerations should not be elicited (ie, use of scalp stimulation) during FHR decelerations or bradycardia.[13] Such actions only delay initiation of appropriate intrauterine resuscitation measures and can potentially further compromise fetal status.

Decelerations

Decelerations are defined in terms of the rate of onset (abrupt versus gradual) and the relationship to uterine contractions. If decelerations occur with 50% or more of contractions during 20 minutes, they are considered to be recurrent decelerations. If they occur with less than 50% of contractions within 20 minutes, they are called intermittent decelerations.[13,14,25] Use of the word *repetitive*, although used in the past, should be avoided because it implies that decelerations are occurring with every contraction rather than with the majority (50% or greater) of contractions. Decelerations are classified as early, variable, late, or prolonged.[13,14,25]

Early Decelerations

Early decelerations are visually apparent, gradual decreases in FHR (30 seconds or greater from onset of deceleration to nadir) with the nadir of the deceleration occurring at the peak of the contraction.[13,14,25] They are almost always benign if no other abnormalities of the FHR tracing are noted. They occur due to head compression and represent transient local changes in blood flow as a result of stimulus of the vagal nerve centers.

Variable Decelerations

Variable decelerations are visually apparent, abrupt decreases in FHR below the baseline with an onset to nadir of 30 seconds or less.[13,14,25] The decrease in FHR is 15 BPM or greater from the baseline with duration of 15 seconds or more but less than 2 minutes. Variable decelerations may or may not be associated with contractions,[13,14,25] and are most commonly the result of cord compression. They occur due to a sudden rise in peripheral resistance (fetal blood pressure), increased parasympathetic outflow, and slowing of the fetal pacemaker (ie, FHR). Although most variable decelerations occur due to a simple reflex response to increased blood pressure, they can result from decreased arterial oxygen concentration secondary to uteroplacental insufficiency in some instances. Assessing the presence of moderate baseline variability and/or accelerations in the baseline will help the provider differentiate between variable decelerations resulting from simple cord compression and those associated with uteroplacental insufficiency.

Variable decelerations may be accompanied by other characteristics, the clinical significance of which requires further investigation. Some examples include a slow return of the FHR after the end of the contraction, biphasic decelerations, tachycardia after variable deceleration(s), accelerations preceding and/or following a deceleration (sometimes called shoulders or *overshoots*), and fluctuations in the FHR in the nadir of the deceleration.[25]

Variable decelerations are the most common FHR deceleration that occur during labor and are generally associated with normal perinatal outcomes.[7] Assessment of the tracing should include changes in the overall baseline rate and variability, and the recurrence, depth, and duration of the deceleration. The presence of moderate variability or accelerations suggests the absence of acidemia.[7] Management generally involves relieving cord compression by repositioning the woman or amnioinfusion if variables are recurrent. Administering oxygen and reducing or discontinuing uterine stimulants may also be helpful interventions for suspected uteroplacental insufficiency. Although it is a common practice to administer oxygen, there is insufficient evidence to support its use and the current National Institute for Health and Care Excellence (NICE) guideline does not recommend oxygen supplementation.[34]

Late Decelerations

Late decelerations are visually apparent, gradual decreases in FHR (30 seconds or greater from onset to nadir) with the nadir of the deceleration falling after the peak of the contraction. The onset, nadir, and recovery of the deceleration generally occur after the beginning, peak, and ending of the contraction, respectively.[25] Late decelerations are associated with uteroplacental insufficiency and fetal hypoxemia. They can lead to acidemia

and myocardial depression if not corrected. When combined with absent or minimal variability or other FHR abnormalities, there is an increased likelihood of significant fetal compromise; immediate evaluation and intervention are indicated. Subtle, shallow late decelerations are clinically significant but easily missed. They can be detected by holding a straight edge along the baseline.

Prolonged Decelerations

Prolonged decelerations are visually apparent decreases in FHR baseline of 15 BPM or more, lasting 2 minutes or more, but less than 10 minutes.[13,14,25]

A sudden deterioration in the FHR tracing may be seen after vaginal examination, FSE placement, amniotomy, uterine tachysystole secondary to administration of oxytocin or a cervical ripening drug, maternal hypotension (ie, secondary to regional anesthesia), maternal seizures, or fetal movement producing transient cord compression. If the fetus was not previously compromised, recovery will typically occur with discontinuation of the inciting event or drug, position change, and, if indicated, increased intravenous (IV) fluids, maternal oxygen supplementation, or a combination of these measures. When accompanied by changes in variability or baseline, prolonged decelerations are more likely to be associated with fetal acid-base abnormalities. Factors known to cause these changes should be identified and corrected.

Sinusoidal Fetal Heart Rate Tracings

A sinusoidal FHR tracing is a specific pattern that is not consistent with any of the previously discussed definitions. It is generally thought to be due to severe fetal anemia.[30,31,35] It is described as a visually apparent, regular smooth sine wavelike undulating tracing seen within the FHR baseline. Providers are cautioned not to confuse this tracing with FHR variability. The FHR tracing undulates slowly and regularly, generally with a cycle frequency of 3 to 5 cycles (waves)/minute. The tracing must continue for at least 20 minutes,[13,14,25] but providers may need to intervene sooner in emergencies (eg, bleeding from a ruptured vasa previa). A sinusoidal tracing is considered a Category III pattern and therefore highly predictive of acidemia at the time of observation.[13,14,25]

Of note, the term *pseudosinusoidal* is not recognized by the NICHD. In the past, this term was used to denote a tracing that frequently occurred following narcotic administration. In addition, it was sometimes observed during ultrasound and associated with rhythmic fetal movements (eg, rapid breathing, sucking movements of the mouth, hiccoughing, thumb sucking).[35] *Table 9* summarizes the NICHD definitions.

Overall Assessment

Having assessed the contraction pattern and the FHR tracing and defined the risk, an overall assessment of the situation and management plan should be made. The terms *fetal distress* and *birth asphyxia* are inappropriate and have no place in the assessment. In the past, terms describing the FHR tracing were *reassuring* and *nonreassuring*, but since the release of the 2008 NICHD workshop report, the assessment of fetal status has been organized into a three-tier system: Category I, II, or III.[25] During labor, the FHR is dynamic and frequently transitions from one category to another depending on the clinical situation and management.[25] Treatment of the woman should be based on the clinical context and the FHR tracing category, and should include a plan for further fetal surveillance if labor is allowed to continue.

Category I tracings are considered normal and can be monitored routinely. Category II tracings are indeterminate, meaning they are not predictive of abnormal fetal pH status. These tracings require prompt evaluation and implementation of interventions to address the tracing. Category III tracings are abnormal and predictive of abnormal acid-base status at the time of observation.[25] Prompt evaluation, intervention, and consideration of immediate delivery is required if the pattern does not resolve or improve in a timely fashion.

NICHD Fetal Heart Rate Classification System[25]

Category I Fetal Heart Rate Tracings

Category I tracings are normal tracings that are strongly predictive of normal fetal acid-base status at the time of observation and must include all of the following:

- Baseline rate of 110 to 160 BPM
- Moderate baseline variability
- Absence of late or variable decelerations
- Absence or presence of early decelerations
- Absence or presence of accelerations (spontaneous or elicited)

Table 9. NICHD Descriptive Terms for Fetal Heart Rate Characteristics

Term	Definition
Baseline Rate	Approximate mean FHR rounded to increments of 5 BPM during a 10-minute window, excluding accelerations and decelerations and periods of marked FHR variability (>25 BPM). There must be at least 2 minutes of identifiable baseline segment (not necessarily contiguous) in any 10-minute window, or the baseline for that period is indeterminate. In such cases, it may be necessary to refer to the previous 10-minute window for determination of the baseline
Bradycardia	Baseline rate <110 BPM for 10 minutes or longer
Tachycardia	Baseline rate >160 BPM for 10 minutes or longer
Baseline Variability	Determined in a 10-minute window, excluding accelerations and decelerations. Baseline FHR variability is defined as fluctuations in the baseline FHR that are irregular in amplitude and frequency. The fluctuations are visually quantified as the amplitude of the peak-to-trough in BPM
Absent Variability	Amplitude range undetectable
Minimal Variability	Amplitude range visually detectable but ≤5 BPM
Moderate Variability	Amplitude range 6 to 25 BPM
Marked variability	Amplitude range >25 BPM
Acceleration	Visually apparent abrupt increase in FHR. An abrupt increase is defined as an increase from the onset of acceleration to the peak in <30 seconds. To be called an acceleration, the peak must be ≥15 BPM, and the acceleration must last ≥15 seconds from the onset to return. Before 32 weeks' gestation, accelerations are defined as having a peak ≥10 BPM and duration of ≥10 seconds. An acceleration lasting ≥10 minutes is defined as a baseline change
Prolonged Acceleration	A prolonged acceleration is ≥2 minutes but <10 minutes in duration
Early Deceleration	Visually apparent, usually symmetrical, gradual decrease and return of the FHR associated with a uterine contraction. A gradual FHR decrease is defined as one from the onset to the FHR nadir ≥30 seconds. The decrease in FHR is calculated from the onset to the nadir of the deceleration. The nadir of the deceleration occurs at the same time as the peak of the contraction. In most cases the onset, nadir, and recovery of the deceleration is coincident with the beginning, peak, and ending of the contraction, respectively
Late Deceleration	Visually apparent, usually symmetrical, gradual decrease and return of the FHR associated with a uterine contraction. A gradual FHR decrease is defined as one from the onset to the FHR nadir ≥30 seconds. The decrease in FHR is calculated from the onset to the nadir of the deceleration. The nadir of the deceleration is delayed in timing, with the nadir of the decelerations occurring after the peak of the contraction. In most cases the onset, nadir, and recovery of the decelerations occur after the beginning, peak, and ending of the contraction, respectively
Variable Deceleration	Visually apparent abrupt decrease in the FHR. An abrupt decrease in the FHR is defined as from the onset of the deceleration to the beginning of the FHR nadir <30 seconds. The decrease in FHR is calculated from the onset to the nadir of the deceleration. The decrease in the FHR is ≥15 BPM, lasting ≥15 seconds and <2 minutes in duration. When variable decelerations are associated with uterine contractions, onset, depth, and duration commonly vary with successive uterine contractions. Variable decelerations can occur in the absence of contractions
Prolonged Deceleration	Visually apparent decrease in FHR from the baseline that is ≥15 BPM, lasting ≥2 minutes, but <10 minutes. A deceleration that lasts ≥10 minutes is a baseline change
Recurrent	Decelerations are defined as recurrent if they occur with ≥50% of uterine contractions in any 20-minute window
Intermittent	Decelerations occurring with <50% of uterine contractions in any 20-minute segment
Sinusoidal	Visually apparent, smooth, sine wavelike undulating pattern in FHR baseline with a frequency of 3-5 cycles/minute that persists for ≥20 minutes
Normal Uterine Activity	Five or fewer contractions in a 10-minute window, averaged over 30 minutes
Tachysystole	More than five contractions in a 10-minute window, averaged over 30 minutes

BPM = beats per minute; FHR = fetal heart rate.

Information from Macones GA, Hankins GD, Spong CY, Hauth J, Moore T. The 2008 National Institute of Child Health and Human Development workshop report on electronic fetal monitoring: update on definitions, interpretation, and research guidelines. Obstet Gynecol. *2008;112(3):661-666.*

Category II Fetal Heart Rate Tracings

Category II tracings are indeterminate tracings that are not predictive of fetal acid base status and cannot be classified as Category I or Category III. The presence of moderate variability and/or accelerations during a Category II tracing is highly predictive of normal fetal acid base status at the time of observation.[7,14,25] However, Category II tracings require prompt evaluation and implementation of interventions to address the tracing. Category II tracings are common and occur in 80% or more of fetuses during labor.[10,14,25] Some examples of Category II tracings include:

- Tachycardia
- Bradycardia not accompanied by absent variability
- Baseline with minimal or marked variability
- Baseline with absent variability not accompanied by recurrent decelerations
- Recurrent variable decelerations with minimal to moderate variability
- Recurrent late decelerations with moderate variability
- Variable decelerations with slow return, overshoot, or shoulders
- Prolonged decelerations lasting more than 2 minutes but less than 10 minutes
- No acceleration(s) after fetal stimulation

Category III Fetal Heart Rate Tracings

Category III tracings are predictive of abnormal fetal acid-base status at the time they are observed.[25] These require prompt evaluation and expedient interventions to address the pattern. Interventions may include, as appropriate, administration of oxygen, administration of an IV fluid bolus, repositioning of, discontinuation of uterine stimulants, and treatment of maternal hypotension. If these fail to correct the tracing, immediate delivery should be considered. Historically, a 30-minute *decision-to-incision* window for emergency cesarean delivery was thought to be needed to avoid neonatal neurologic injury. However, scientific evidence to support this time frame is lacking.[7,15,23,25] Therefore, the decision-to-incision interval should be the one that best integrates maternal and fetal risks and benefits.[15]

Category III tracings include:

- Sinusoidal pattern

OR

- Absent FHR variability with any of the following:
  - Recurrent late decelerations
  - Recurrent variable decelerations
  - Bradycardia

Table 10 summarizes the NICHD FHR classifications.

Table 10. NICHD Three-Tier Fetal Heart Rate Categories

Category I

Category I FHR tracings include the following:

- Baseline rate: 110-160 BPM
- Baseline FHR variability: moderate
- Late or variable decelerations: absent
- Early decelerations: present or absent
- Accelerations: present or absent

Category II

Category II FHR tracings include all FHR tracings not categorized as Category I or Category III. Category II tracings may represent an appreciable fraction of those encountered in clinical care. Examples of Category II FHR tracings include any of the following:

- Baseline rate
  - Bradycardia not accompanied by absent baseline variability
  - Tachycardia
- Baseline FHR variability
  - Minimal baseline variability
  - Absent baseline variability not accompanied by recurrent decelerations
  - Marked baseline variability
- Accelerations
  - Absence of induced accelerations after fetal stimulation
- Periodic or episodic decelerations
  - Recurrent variable decelerations accompanied by minimal or moderate baseline variability
  - Prolonged deceleration >2 minutes but <10 minutes
  - Recurrent late decelerations with moderate baseline variability
  - Variable decelerations with other characteristics, such as slow return to baseline, 'overshoots,' or 'shoulders'

Category III

Category III FHR tracings include either:

Absent baseline FHR variability and any of the following:

- Recurrent late decelerations
- Recurrent variable decelerations
- Bradycardia
- Sinusoidal pattern

BPM = beats per minute; FHR = fetal heart rate; NICHD = Eunice Kennedy Shriver National Institute of Child Health and Human Development.

Information from Macones GA, Hankins GD, Spong CY, et al. The 2008 National Institute of Child Health and Human Development workshop report on electronic fetal monitoring: update on definitions, interpretation, and research guidelines. Obstet Gynecol. *2008;112(3):661-666.*

When using EFM during labor, providers and nurses should review tracings regularly.[7,10,13] This periodic review includes maintaining an adequate quality tracing, identifying and responding to FHR abnormalities, and appropriately communicating findings. Some institutions now use tools for risk management and patient safety that enhance communication. An example is the SBAR (**S**ituation-**B**ackground-**A**ssessment-**R**ecommendation) technique, which provides clinicians with a communication template.[36] Checklists have been widely used in nonobstetric areas and have recently been used in the labor and delivery area.[37-39] Use of checklists improves communication, standardizes care, and reduces potential errors. Use of a vaginal delivery safety checklist has been shown to improve clinician communication in delivery room settings.[39] Whatever techniques are used, standardized communication surrounding fetal monitoring is imperative to reduce errors and improve patient safety.[11,12]

Many institutions now use computerized charting, flow sheets, clinical pathways, or fetal tracing archival processes to facilitate documentation. Any written information on the printed tracing (ie, emergent interventions/events during labor) should coincide with these automated processes to avoid confusion and minimize litigation risk.[13] Documentation of the tracing may occur at different intervals than the actual assessment and may be in the form of summary notes.[7,13,26]

Documentation of the FHR tracing and categorization during labor should include:

1. FHR data (ie, baseline rate, variability, periodic changes, categorization)
2. Uterine activity characteristics obtained by palpation or pressure transducer (ie, frequency, duration, intensity, resting tone)
3. Specific actions taken when changes occur in FHR or uterine activity
4. Other maternal observations and assessments
5. Maternal and fetal responses to interventions
6. Subsequent return to normal findings
7. Pertinent communication with other care providers

Categorization of Structured Intermittent Auscultation Findings

The Association of Women's Health, Obstetric and Neonatal Nurses and the ACNM have proposed a categorization for interpretation of SIA (*Table 11*).[3,13] There is not a Category III for SIA because this category requires the ability to assess variability, which is not always possible when using SIA.[3,13] In addition, the terms for deceleration tracings (ie, late, variable, early) are reserved for EFM and should not be used with SIA.

Table 11. Categories for Systematic Intermittent Auscultation

Category I - Normal: Includes ALL of the following:

Normal FHR baseline of 110-160 BPM

Regular rhythm

Presence of FHR increases or accelerations from the baseline

Absence of FHR decreases or decelerations from the baseline

Category II - Indeterminate: May include any of the following:

Irregular rhythm

Presence of FHR decreases or decelerations from the baseline

Tachycardia (baseline FHR >160 BPM >10 min)

Bradycardia (baseline FHR <110 BPM >10 min)

BPM = beats per minute; FHR = fetal heart rate.

Information from Lyndon A, Ali LU, eds. Association of Women's Health Obstetric and Neonatal Nursing. Fetal Heart Monitoring Principles and Practices. 5th ed. Washington DC: Kendall Hunt Professional; 2015; American College of Nurse-Midwives. Intermittent Auscultation for Intrapartum Fetal Heart Rate Surveillance (replaces ACNM Clinical Bulletin #11, March 2010). J Midwifery Womens Health. 2015;60(5):626-632.

Categorization and Management of Electronic Fetal Monitoring Tracings

Category I Electronic Fetal Monitoring Tracings

Category I EFM tracings are considered normal and are not associated with fetal acidemia.[7,14,25] Recommendations are to continue the current monitoring (whether SIA or EFM), periodically evaluate the tracing and clinical status along with any underlying risk factors, and change the management strategy if the tracing changes to a Category II or III.

Category III Electronic Fetal Monitoring Tracings

Category III EFM tracings are considered abnormal and predictive of abnormal fetal acid-base

status at the time of observation.[7,14,25] Category III tracings require prompt evaluation and treatment. Recommendations are to correct fetal acidemia to reduce outcomes of neonatal encephalopathy, cerebral palsy, and neonatal acidosis. Preparation for delivery, development of a time frame for delivery, and performance of intrauterine resuscitative measures are essential. If tracings do not improve with appropriate corrective maneuvers, prompt delivery of the fetus is indicated.[7,14,25]

Considerations in preparing for an operative delivery in the presence of a Category III tracing should be made judiciously and expeditiously. The standard rule of 30 minutes from decision-to-incision, although used frequently, has not been shown to reduce adverse neonatal outcomes.[15,25,40] In addition, immediate delivery of a fetus with an unknown duration of a Category III tracing may not improve outcomes if the fetus has already experienced hypoxic injury.[7]

Category II Electronic Fetal Monitoring Tracings

Category II tracings include all tracings that are not classified as Category I or III; these occur at some time in over 80% of labors.[10,41] Because these tracings may represent fetal compromise, recommendations are to evaluate the tracing, perform appropriate corrective measures when indicated, and then re-evaluate. Because Category II tracings represent a wide variety of concerns, the presence of accelerations (spontaneous or induced) or moderate variability is useful in assessing the presence of normal fetal acid-base status. If neither of these characteristics is present after appropriate intrauterine resuscitative measures, or if the tracing worsens or progresses to Category III, consideration should be given to delivery of the fetus. If after appropriate intervention(s), the tracing reverts to Category I, then previous monitoring may be resumed.

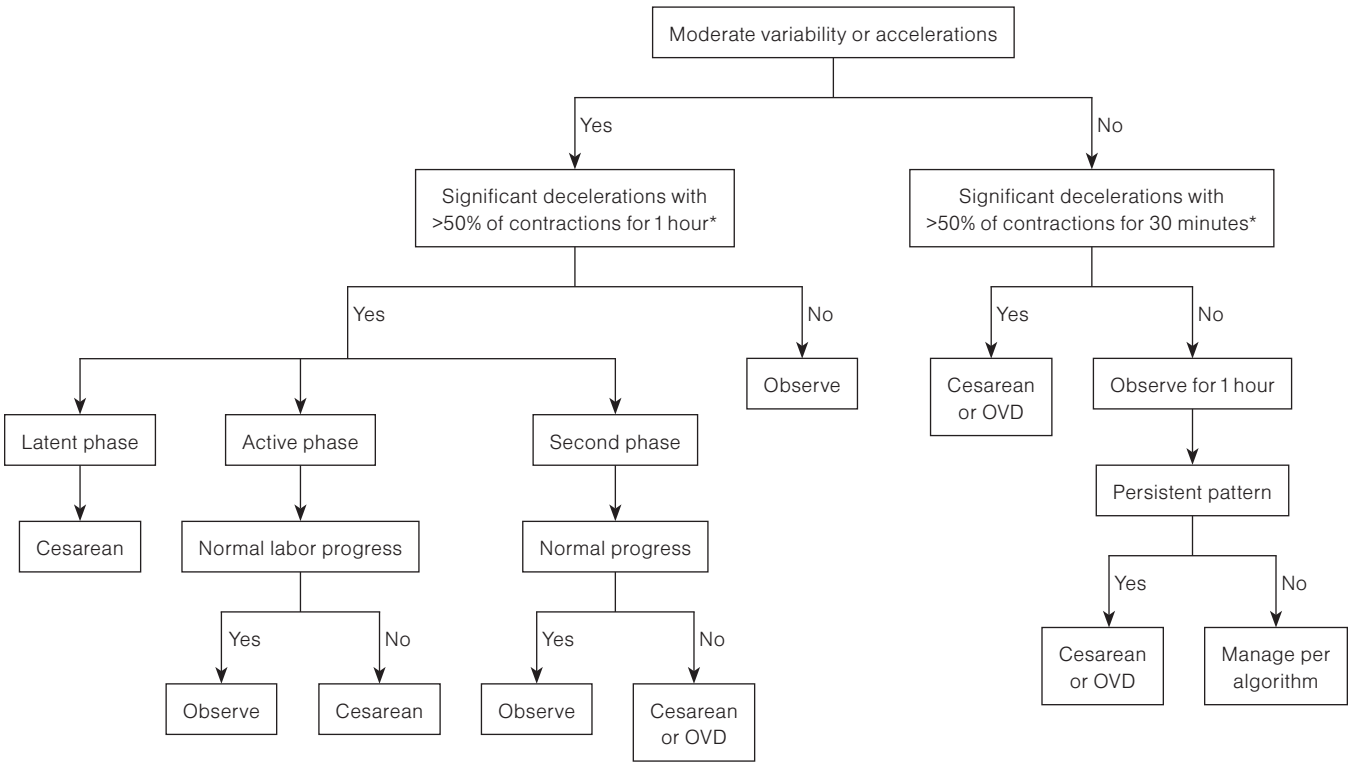
Category II tracings are encountered in over 80% of labors and their management can be challenging without defined management protocols.[10] Category II tracings can range from almost normal to tracings in which acidemia is present or developing rapidly. Without rapid intervention, these tracings can rapidly evolve into Category III tracings. Clinical considerations when managing Category II tracings must include gestational age, fetal growth status (ie, IUGR), maternal medical and obstetric conditions, co-morbidities, labor progress, and available resources and personnel to respond.

Algorithms for Category II Management

Due to the diversity of tracings comprised in Category II, management of this category has been largely based on consensus and individual provider opinion/experience rather than a firm scientific foundation. To assist providers in implementing the most beneficial and appropriate interventions, a number of management algorithms have been proposed.[9,10,32,42-45] All algorithms are based on specific standardized terminology providers need to be aware of when using them. Use of algorithms has been shown to reduce interobserver variability with the potential to identify tracings associated with fetal acidemia earlier.[46] One proposed algorithm for managing Category II tracings (*Figure 2* and *Table 12*)[10] follows the definitions set forth by the NICHD, is supported by a growing body of evidence[10,47] and medical societies,[7,13] and is becoming increasingly used in the United States. The goal of this algorithm is to present a standardized approach to managing Category II tracings that encourage vaginal delivery in fetuses whose FHR tracings show minimal risk of progression to clinically significant acidemia. Given the wide variety of FHR tracings in Category II, this algorithm is not intended to represent a sole management strategy for all Category II tracings, but to serve as a template for management consideration. Use of this algorithm requires application of specific definitions for significant decelerations and takes into account labor phase and labor progress (*Table 8*). This algorithm is only applicable to Category II tracings and is not to be used in extremely premature fetuses.

When a Category II tracing is identified, intrauterine resuscitation measures are performed and the algorithm is delayed for 30 minutes to allow these interventions to improve the tracing. After 30 minutes, if these measures do not alleviate the Category II tracing, the algorithm is started. The algorithm is initiated by an assessment of moderate variability or FHR accelerations because this rules out clinically significant acidemia. From there, the assessment includes the presence or absence of significant decelerations, the stage of labor, and whether labor is progressing normally. If delivery is indicated by the algorithm, ideally,

Figure 2. Algorithm for Management of Category II Fetal Heart Rate Tracings



**That have not resolved with appropriate conservative corrective measures, which may include supplemental oxygen, maternal position changes, intravenous fluid administration, correction of hypotension, reduction or discontinuation of uterine stimulation, administration of uterine relaxant, amnioinfusion, and/or changes in second stage breathing and pushing techniques.*

OVD = operative vaginal delivery.

Reprinted from Clark SL, Nageotte MP, Garite TJ, et al. Intrapartum management of category II fetal heart rate tracings: towards standardization of care. Am J Obstet Gynecol*. 2013;209(2):89-97.*

it would be initiated within 30 minutes of the decision. The algorithm can be discontinued at any time the provider thinks more rapid intervention is indicated.

Five-Tier Classification

In addition to the previously discussed approach for managing Category II tracings, a five-tier classification scheme has also been proposed (*Table 13*).[32,42] Similar to the algorithm in *Table 12,* the purpose of this classification system is to classify FHR monitor tracings according to risk of fetal acidemia, determine the risk of evolution to a more serious tracing, and construct a standard process for FHR tracing management with the ultimate aim of minimizing newborn acidemia without excessive obstetric intervention. A grid categorizes all possible heart rate patterns based on baseline rate (ie, normal, tachycardia, bradycardia), type of decelerations (ie, early, late, variable, prolonged), and quantity of variability (ie, undetectable, minimal, moderate, marked). All definitions use the NICHD nomenclature.

In the five-tier approach, each FHR tracing has been color-coded to represent no threat of acidemia (green, no intervention required) to severe threat for acidemia (red, emergent delivery recommended) (*Table 14*). Three intermediate categories - blue, yellow, orange - are NICHD Category II tracings and represent increasing concern and response for evolving acidemia (*Table 10*).[32,42]

A 2012 study compared the NICHD three-tier system to the proposed five-tier system.[32] For tracings categorized by orange or red, there was 79% sensitivity and 100% specificity for a pH level less than 7 with no false positives. Using the five-tier system, 79% of fetal acidemia was correctly identified in the orange and red tracings, compared with

only 12% in the NICHD Category III. All tracings with pH levels greater than 7 were correctly categorized as blue, green, or yellow. The five-tier system also better identified tracings that resulted in lower Apgar scores, reduced admission to the NICU, and reduced need for oxygen supplementation.[32] However, another study reports no difference between very normal or very abnormal tracings using this system. This study concluded that it is yet to be determined whether one system is superior in predicting fetal acidemia.[45] The complexity of this five-tier system has made practical application difficult; however, mobile applications are available to help with simplifying application of this algorithm in the clinical setting.

Intrauterine Resuscitative Measures

Regardless of the classification scheme used, applicable intrauterine resuscitative measures should be undertaken for any concerning FHR tracing (*Table 15*).[7,48] The goals of these corrective measures should be aimed at the underlying suspected cause of the abnormality in the FHR tracing. If the FHR tracing is a Category III and does not resolve quickly, plans for an expedient delivery should be made.[7,14,25] Category II tracings need to be assessed in light of the entire clinical picture. Some Category II tracings may require emergent delivery if there is no benefit from interventions.

Possible interventions include:

- Change in maternal position to lateral positioning (left or right) or on hands and knees
- Administer maternal oxygen
- Administer IV fluid bolus
- Reduce uterine contraction frequency
- Discontinue oxytocin or cervical ripening drugs
- Administer tocolytic drugs
- Initiate amnioinfusion with recurrent variable decelerations
- If prolapsed umbilical cord is noted, continuous elevation of the presenting fetal part until operative delivery occurs
- Modify pushing efforts in the second stage of labor
- Assess maternal vital signs to detect hypotension
- Perform vaginal examination to assess progress of labor and rule out prolapsed umbilical cord
- If recent epidural analgesia administration and hypotension with new late decelerations are present, prepare for possible administration of ephedrine

Good evidence exists for the use of lateral positioning for intrauterine resuscitation.[49] Lateral positioning reduces compression of the inferior vena cava and aorta. Repositioning the woman may also reduce compression of the umbilical cord. Several studies indicate that lateral positioning on either side is superior when compared with supine positioning.[48]

Table 12. Management of Category II Fetal Heart Rate Tracings: Clarifications for Use of Algorithm

1. Variability refers to predominant baseline FHR pattern (marked, moderate, minimal, absent) during a 30-minute evaluation period, as defined by NICHD.
2. Marked variability is considered same as moderate variability for purposes of this algorithm.
3. Significant decelerations are defined as any of the following:
   - Variable decelerations lasting longer than 60 seconds and reaching a nadir more than 60 bpm below baseline.
   - Variable decelerations lasting longer than 60 seconds and reaching a nadir less than 60 bpm regardless of the baseline.
   - Any late decelerations of any depth.
   - Any prolonged deceleration, as defined by the NICHD. Due to the broad heterogeneity inherent in this definition, identification of a prolonged deceleration should prompt discontinuation of the algorithm until the deceleration is resolved.
4. Application of algorithm may be initially delayed for up to 30 minutes while attempts are made to alleviate category II pattern with conservative therapeutic interventions (eg, correction of hypotension, position change, amnioinfusion, tocolysis, reduction or discontinuation of oxytocin).
5. Once a category II FHR pattern is identified, FHR is evaluated, and algorithm is applied every 30 minutes.
6. Any significant change in FHR parameters should result in reapplication of algorithm.
7. For category II FHR patterns in which algorithm suggests delivery is indicated, such delivery should ideally be initiated within 30 minutes of decision for cesarean.
8. If at any time tracing reverts to category I status, or deteriorates for even a short time to category III status, the algorithm no longer applies. However, algorithm should be reinstituted if category I pattern again reverts to category II.
9. In fetus with extreme prematurity, neither significance of certain FHR patterns of concern in more mature fetus (eg, minimal variability) or ability of such fetuses to tolerate intrapartum events leading to certain types of category II patterns are well defined. This algorithm is not intended as guide to management of fetus with extreme prematurity.
10. Algorithm may be overridden at any time if, after evaluation of patient, physician believes it is in the best interest of the fetus to intervene sooner.

FHR = fetal heart rate; NICHD = Eunice Kennedy Shriver National Institute of Child Health and Human Development.

Reprinted from Clark SL, Nageotte MP, Garite TJ, et al. Intrapartum management of category II fetal heart rate tracings: towards standardization of care. Am J Obstet Gynecol. *2013;209(2):89-97.*

Table 13. Proposed Five-Tier Fetal Heart Rate Classification System

Category	Risk of Acidemia	Risk of Evolution	Action
Green Mostly Category I	None	Very low	None
Blue Category II	No central fetal academia	Low	Conservative techniques, prepare for delivery
Yellow Category II	No central fetal acidemia, but FHR suggestive of intermittent reductions in oxygen which may result in fetal oxygen debt	Moderate	Conservative techniques and increased surveillance
Orange Category II	Borderline/acceptably low; fetus at potential risk of decompensation	High	Conservative techniques, prepare for urgent delivery
Red Category III	Unacceptably high; evidence of actual or impending damaging fetal asphyxia	Already present	Delivery

FHR = fetal heart rate.

Information from Parer JT, Ikeda T. A framework for standardized management of intrapartum fetal heart rate patterns. Am J Obstet Gynecol. *2007;197(1):26.e1-26.e6.*

Administration of maternal oxygen with the goal of improving fetal oxygenation remains a common intervention, yet study results regarding its benefit for the fetus remain controversial and may even suggest a possible detrimental effect.[48-54] A recent Cochrane review found that there was not enough evidence to support or refute the use of oxygen for intrauterine resuscitation.[55] Notably, NICE, endorsed by the Royal College of Obstetricians and Gynecologists (RCOG), states that oxygen should not be administered for intrauterine resuscitation because it may harm the fetus.[33] Due to controversy in the literature, it is reasonable to administer oxygen after other appropriate intrauterine resuscitation techniques have failed and to discontinue its use after the desired fetal response has been achieved.[13,51]

Administration of an IV fluid bolus is another common technique thought to increase intravascular volume, uteroplacental perfusion, and thus, fetal oxygenation.[41,48,49] One study showed that fetal oxygenation saturation improved with a 500 mL bolus of lactated Ringer solution, and a 1,000 mL bolus produced the greatest increase. The positive effect of the bolus continued for 15 minutes after administration.[51]

Modification of pushing efforts can have a significant effect on the FHR tracing. Coached sustained valsalva pushing (ie, closed glottis, "hold your breath and count to 10" three times with each contraction) can have a deleterious effect on maternal and fetal oxygenation,[48,56,57] with a resulting increase in number and severity of FHR decelerations.[58,59] A preventive measure would be to avoid pushing until the woman feels the urge to push (ie, *laboring down*), thereby minimizing the length of the active pushing phase and fetal exposure to the hypoxic stress of pushing. Modification of pushing efforts also includes temporarily discontinuing pushing to allow the fetus to recover, or to push with every second or third contraction.[13,56,60,61] A recent systematic review of 884 women with epidural analgesia evaluated delayed versus immediate pushing. In the delayed pushing group, active pushing was decreased by 19 minutes and there were no differences in perineal lacerations or episiotomies and an increased number of spontaneous vaginal births.[62] Of note, delayed pushing was associated with an increased incidence of lower umbilical cord pH levels; however, there was no difference in 5-minute Apgar scores less than 7 or NICU admissions.[62]

Amnioinfusion

A recent systematic review showed inconclusive evidence for use of amnioinfusion to reduce recurrent variable decelerations during labor.[49] In some reviews, amnioinfusion was associated with a reduction in cesarean delivery rates, FHR

decelerations, 5-minute Apgar scores less than 7, postpartum endometritis, and the incidence of neonatal and maternal hospital stays greater than 3 days.[63] In addition, mean cord umbilical pH was higher. However, no improvement in long-term neonatal outcomes was detected.

Although typically considered safe, amnioinfusion carries a few precautions and potential complications. Amnioinfusion is indicated only for recurrent variable decelerations and is not indicated for late decelerations, fetal bradycardia, thick meconium, or oligohydramnios with a normal heart rate tracing.[7,63,64]

Amnioinfusion should also not be attempted when cesarean delivery is indicated, such as in transverse lie or placenta previa. It should never be undertaken when doing so would result in a delay of an indicated definitive treatment. With breech presentation, multiple gestations, or when placental abruption is suspected, caution should be taken in performing amnioinfusion. Complications include umbilical cord prolapse, rupture of a previous

Table 14. Risk Categories for Fetal Acidemia Related to Variability, Baseline Rate, and Presence of Recurrent Decelerations

Variable	No	Early	Mild VD	Moderate VD	Severe VD	Mild LD	Moderate LD	Severe LD	Mild PD	Moderate PD	Severe PD
Moderate (normal) variability											
Tachycardia	B	B	B	Y	O	Y	Y	O	Y	Y	Y
Normal	G	G	G	B	Y	B	Y	Y	Y	Y	O
Mild bradycardia	Y	Y	Y	Y	O	Y	Y	O	Y	Y	O
Moderate bradycardia	Y	Y			O		O	O			O
Severe bradycardia	O	O			O			O			O
Minimal variability											
Tachycardia	B	Y	Y	O	O	O	O	R	O	O	O
Normal	B	B	Y	O	O	O	O	R	O	O	R
Mild bradycardia	O	O	R	R	R	R	R	R	R	R	R
Moderate bradycardia	O	O			R		R	R			R
Severe bradycardia	R	R			R			R			R
Absent variability											
Tachycardia	R	R	R	R	R	R	R	R	R	R	R
Normal	O	R	R	R	R	R	R	R	R	R	R
Mild bradycardia	R	R	R	R	R	R	R	R	R	R	R
Moderate bradycardia	R	R			R		R	R			R
Severe bradycardia	R	R			R			R			R
Sinusoidal R											
Marked variability Y											

B = blue; G = green; LD = late decelerations; O = orange; PD = prolonged decelerations; R = red; VD = variable decelerations; Y = yellow.

Reprinted from Parer JT, Ikeda T. A framework for standardized management of intrapartum fetal heart rate patterns. Am J Obstet Gynecol. *2007;197(1):26.e1-26.e6.*

cesarean scar, amniotic fluid embolism, acute uterine hypertonus with a Category II or Category III FHR tracing, and acute polyhydramnios.

In the past, amnioinfusion was used to dilute thick meconium as a prophylactic measure to prevent newborn meconium aspiration. RCTs did not confirm the efficacy of this approach and ACOG no longer considers this an indication for amnioinfusion.[64] Although amnioinfusion is no longer indicated for meconium in high-resource settings, where EFM is available, it does seem to be indicated in low-resource settings, where monitoring capabilities are limited.[65]

Guidelines for Performing Amnioinfusion

Amnioinfusion can be performed by continuous or intermittent techniques. A RCT trial showed there was no difference between the two techniques for resolving variable decelerations.

For continuous infusion:

1. Perform a vaginal examination to determine presentation and dilation, and to rule out cord prolapse
2. Obtain informed consent
3. Place the patient in the left-lateral position. Place an IUPC and consider placing an FSE. If available, use a double lumen catheter for saline infusion
4. If a double lumen catheter is not available, attach an 18-gauge needle to IV tubing connected to normal saline or lactated Ringer's solution. Attach extension tubing filled with distilled water between the IUPC and the transducer. Insert the 18-gauge needle into the side port of the extension tubing. Alternatively, insert a second single lumen catheter (one for IUPC and one for amnioinfusion)
5. Infuse fluid, administering 250 to 500 mL initially, followed by 50 to 60 mL/hour maintenance infusion until FHR abnormalities resolve.

Because resting tone will be increased while the infusion is occurring, elevated baseline tone before the infusion is a contraindication to its use.

Management of Tachysystole

Good evidence exists for the use of tocolytic drugs for intrauterine resuscitation in cases of

Table 15. Potential Intrauterine Resuscitative Measures for Category II or Category III Tracings

Goal	Associated FHR Changes	Potential Interventions
Improve uteroplacental blood flow	Recurrent late decelerations Prolonged decelerations Minimal or absent FHR variability	Lateral positioning Administer maternal oxygen[a] Administer intravenous fluid bolus Discontinue or reduce uterine stimulants Administer tocolytic drugs Correct maternal hypotension Modify maternal expulsive (pushing) efforts
Reduce uterine activity	Tachysystole with Category II or Category III tracing	Lateral positioning Administer intravenous fluid bolus Discontinue or reduce uterine stimulants Administer tocolytic drugs
Alleviate/reduce umbilical cord compression	Recurrent variable decelerations Prolonged decelerations Bradycardia	Reposition to where FHR is most improved Discontinue uterine stimulants Initiate amnioinfusion if variable decelerations recurrent Modify maternal expulsive (pushing) efforts Check for prolapsed cord. If identified, continuously elevate presenting part until operative delivery occurs.

FHR = fetal heart rate.

[a]refer to page 17 regarding lack of evidence.

Note: always check cervix, maternal vital signs.

Information from American College of Obstetricians and Gynecologists. Practice bulletin no. 116: Management of intrapartum fetal heart rate tracings. Obstet Gynecol. 2010;116(5):1232-1240.

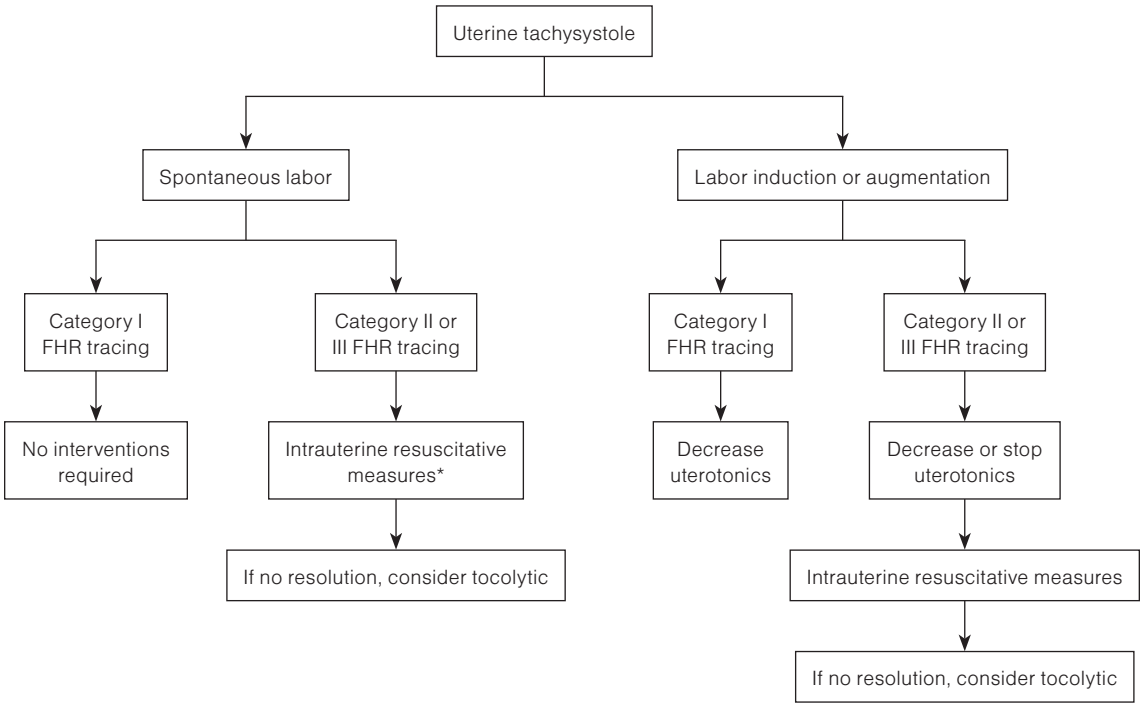
tachysystole.[49,66,67] Uterine contractions cause a cessation of uterine blood flow and thus oxygen delivery to the fetus. In most healthy fetuses, this temporary decline is well tolerated. However, uterine tachysystole has been shown to cause a progressive decline in fetal oxygenation and arterial blood gases at birth,[68-72] increased risk of operative delivery and lower Apgar scores, as well as an increase in NICU admissions.[71] Tocolysis should be considered, especially in women with tachysystole-associated Category II or Category III tracings that do not respond to standard intrauterine resuscitative measures.[7,72] A recent Cochrane review showed that betamimetic therapy reduced the number of FHR abnormalities.[66] If tachysystole is induced and a Category II or III tracing is present, oxytocin should be decreased or discontinued.[7] Other interventions for tachysystole may include maternal repositioning and IV fluid bolus.[7,13,72] Evaluation for placental abruption should occur. Tocolysis is contraindicated when abruption is suspected because it may worsen the abruption.[51] Use of tocolytic drugs is not without risk. Although they may prompt uterine relaxation, improving placental blood flow and thereby fetal oxygenation, they may also have an adverse effect on maternal cardiovascular status and place the woman at increased risk of postpartum hemorrhage.

An algorithm for the management of uterine tachysystole is shown in *Figure 3.*

Ancillary Testing for Category II and III Fetal Heart Rate Tracings

When a concerning FHR pattern develops, fetal scalp pH testing is performed to assess fetal acid-base status. It is rarely performed in the United States because it has been replaced with a simple assessment of moderate FHR variability or the presence of FHR accelerations. If accelerations are not present, fetal stimulation may be used to elicit accelerations. Current evidence indicates the presence of moderate variability or accelerations reliably predicts the absence of fetal metabolic

Figure 3. Management Algorithm for Uterine Tachysystole



FHR = fetal heart rate.

Reprinted from American College of Obstetricians and Gynecologists. Practice bulletin no. 116: Management of intrapartum fetal heart rate tracings. Obstet Gynecol. *2010;116(5):1232-1240.*

acidemia at the time these characteristics are observed.[25] A meta-analysis showed that if there is absent or minimal variability without spontaneous accelerations, the presence of an acceleration after scalp stimulation or fetal acoustic stimulation indicates that the fetal pH level is greater than 7.[7,73] Providers are cautioned not to attempt to elicit accelerations during decelerations as this may further compromise the fetus and potentially lead to a delay in definitive management.[13]

Global Perspective

Although this chapter has focused on NICHD guidelines, which have had significant effects on EFM classification and interpretation in the United States, providers, especially those who may practice in international settings, should be aware of the other nomenclatures and classification systems (*Table 16* and *Figure 4*). The NICE guidelines (endorsed by RCOG) were published in 2001 and updated in 2017.[34] The International

Table 16. Comparison of Guideline Definitions of Fetal Heart Rate

Definition	National Institute of Child Health and Human Development (NICHD) (Endorsed by ACOG, AAFP, SMFM, AWHONN, and ACNM)	International Federation of Gynecology and Obstetrics (FIGO)	National Institute for Health and Care Excellence (NICE) (Endorsed by RCOG)
FHR Baseline	Mean FHR rounded to increments of 5 BPM during a 10-minute window, excluding period or episodic changes, periods of marked FHR variability, or segments of baseline that differ >25 BPM. There must be at least 2 minutes of identifiable baseline segments (not necessarily contiguous) in any 10-minute window or the baseline for that period is indeterminate. In which case, refer to the prior 10-minute window Normal: 110-160 BPM Tachycardia: >160 BPM that persists for at least 10 minutes Bradycardia: <110 BPM that persists for at least 10 minutes	Mean level of the most horizontal and less oscillatory FHR segments. Estimate in periods of 10 minutes expressed as BPM. Baseline may vary between subsequent 10-minute sections Normal: 110-160 BPM Tachycardia: >160 BPM lasting more than 10 minutes Bradycardia <110 BPM lasting more than 10 minutes	Mean level of the FHR when this stable, excluding accelerations and decelerations. It is determined over a period of 5-10 minutes and expressed in BPM Normal: 110-160 BPM Tachycardia: >160 BPM 161-180 BPM moderate tachycardia Bradycardia: <100 BPM 100-109 BPM is moderate bradycardia
Variability	Fluctuations in the baseline FHR, in a 10-minute window excluding accelerations and decelerations, that are irregular in amplitude and frequency. The fluctuations are visually quantified as the amplitude of the peak-to-trough in BPM Absent: Amplitude range undetectable Minimal: >undetectable - ≤5 BPM Moderate: 6-25 BPM Marked: >25 BPM	Oscillations in the FHR signal, evaluated as the average bandwidth amplitude of the signal in 1-minute segments. Normal: a bandwidth of 5-25 BPM Reduced: <5 BPM for more than 50 minutes in baseline segments or for >3 minutes during decelerations Increased (saltatory pattern): a bandwidth value >25 BPM lasting more than 30 minutes	Minor fluctuations in baseline FHR occurring at 3-5 cycles/minute. It is measured by estimating the difference in BPM between the highest peak and the lowest trough of fluctuation in a 1-minute segment Normal: ≥5 BPM between contractions Nonreassuring: <5 BPM for 30-50 minutes >25 BPM for 15-25 minutes Abnormal: <5 BPM for >50 minutes >25 BPM >25 minutes Sinusoidal

continues

AAFP = American Academy of Family Physicians; ACNM = American College of Nurse-Midwives; ACOG = The American College of Obstetricians and Gynecologists; AWHONN = Association of Women's Health, Obstetric and Neonatal Nurses; BPM = beats per minute; FHR = fetal heart rate; RCOG Royal College of Obstetricians and Gynaecologists; SMFM = Society for Maternal-Fetal Medicine.;

Table 16. Comparison of Guideline Definitions of Fetal Heart Rate *(continued)*

Definition	National Institute of Child Health and Human Development (NICHD) (Endorsed by ACOG, AAFP, SMFM, AWHONN, and ACNM)	International Federation of Gynecology and Obstetrics (FIGO)	National Institute for Health and Care Excellence (NICE) (Endorsed by RCOG)
Accelerations	Visually apparent abrupt increase in FHR defines as increase from the onset of the acceleration to the peak in <30 seconds. Duration of acceleration must be <2 minutes >32 weeks increase in FHR must be ≥15 BPM for ≥15 seconds <32 weeks increase in FHR must be ≥10 BPM for ≥10 seconds Prolonged acceleration: ≥2 minutes but <10 minutes. Acceleration ≥10 minutes is a baseline change	Transient increase in FHR of ≥15 BPM last ≥15 seconds but <10 minutes <32 weeks accelerations in FHR may only be 10 BPM for 10 seconds	Transient increase in FHR of ≥15 BPM last ≥15 seconds
Decelerations	Decrease in FHR from onset to nadir of deceleration described as abrupt or gradual Late deceleration: Symmetrical gradual decrease of FHR. Gradual decrease defined as ≥30 seconds from onset to nadir of deceleration. Deceleration is delayed in timing with nadir occurring after the peak of the contraction with a duration ≤2 minutes Early deceleration: Symmetrical gradual decrease of FHR. Gradual decrease defined as ≥30 seconds from onset to nadir of deceleration. Nadir occurs at the peak of the contraction with a duration ≤2 minutes Variable deceleration: Abrupt decrease in the FHR of <30 seconds. Decrease must be at least 15 BPM lasting 15 seconds with a duration <2 minutes Prolonged deceleration: >2 minutes but ≤10 minutes at least 15 BPM. Decelerations >10 minutes are a baseline change	Decreases in the FHR below baseline of >15 BPM lasting >15 seconds Late deceleration: U-shaped and/or with reduced variability with gradual onset and/or gradual return to baseline and/or reduced variability within the decelerations. Gradual onset and return occurs when more than 30 seconds elapses between the beginning/end of a deceleration and its nadir. When contractions adequately monitored, late decelerations start >20 second after the onset, have a nadir after the acme, and a return after the end of the contraction Early deceleration: Decelerations that are shallow, short-lasting with normal variability within the deceleration and are coincident with contractions Variable decelerations: V-shaped decelerations that exhibit a rapid drop (onset to nadir <30 seconds), good variability within the deceleration, rapid recovery to the baseline, varying in size, shape, and relationship to uterine contractions Prolonged deceleration: >3 minutes	Transient episodes of slowing of the FHR below the baseline level of ≥15 BPM lasting ≥15 seconds Late deceleration: Uniform repetitive periodic slowing of the FHR with onset mid to end of contraction and nadir >20 seconds after the peak of the contraction and ending after the contraction. Includes decelerations <15 BPM if nonaccelerative trace with baseline variability <5 BPM Early deceleration: Uniform repetitive periodic slowing of the FHR with onset early in the contraction and return to baseline at the end of the contraction Variable deceleration: Variable, intermittent periodic slowing of the FHR with rapid onset and recovery. Time relationships with contraction cycle are variable and may occur in isolation. Sometimes they resemble other types of deceleration patterns in timing and shape Prolonged deceleration: An abrupt decrease in FHR to levels below the baseline that last at least 60-90 seconds. These decelerations become pathological if they cross 2 contractions (ie, >3 minutes)

continues

AAFP = American Academy of Family Physicians; ACNM = American College of Nurse-Midwives; ACOG = The American College of Obstetricians and Gynecologists; AWHONN = Association of Women's Health, Obstetric and Neonatal Nurses; BPM = beats per minute; FHR = fetal heart rate; RCOG Royal College of Obstetricians and Gynaecologists; SMFM = Society for Maternal-Fetal Medicine.

Table 16. Comparison of Guideline Definitions of Fetal Heart Rate *(continued)*

Definition	National Institute of Child Health and Human Development (NICHD) (Endorsed by ACOG, AAFP, SMFM, AWHONN, and ACNM)	International Federation of Gynecology and Obstetrics (FIGO)	National Institute for Health and Care Excellence (NICE) (Endorsed by RCOG)
Sinusoidal	Visually apparent smooth sine wavelike undulating pattern in FHR baseline with a frequency of 3-5 cycles/minute that persists for ≥20 minutes	A regular smooth undulating signal, resembling a sine wave, with an amplitude of 5-15 BPM and a frequency of 3-5 cycles/minute. This pattern lasts more than 30 minutes and coincides with absent accelerations	A regular oscillation of the baseline long-term variability resembling a sine wave. This smooth, undulating pattern, lasting at least 10 minutes that has a relative fixed period of 3-5 cycles/minute and amplitude of 5-15 BPM above and below the baseline. Baseline variability is absent
Pseudosinusoidal	Not defined	A pattern resembling the sinusoidal pattern but with more jagged *saw-tooth* appearance, rather than the smooth sine waveform Seldom exceeds 30 minutes and is characterized by normal patterns before and after	Not defined
Uterine Activity	Normal uterine activity: ≤5 contractions in 10 minutes averaged over a 30-minute window Tachysystole: >5 contractions in 10 minutes averaged over a 30-minute window. Term applies to spontaneous and stimulated labor *Hyperstimulation* and *hypercontractility* are not defined and should be abandoned	Normal uterine activity: not defined Tachysystole: >5 contractions in 10 minutes in 2 successive 10-minute periods or averaged over a 30-minute period	Not defined, although does use the word uterine hyperstimulation

AAFP = American Academy of Family Physicians; ACNM = American College of Nurse-Midwives; ACOG = The American College of Obstetricians and Gynecologists; AWHONN = Association of Women's Health, Obstetric and Neonatal Nurses; BPM = beats per minute; FHR = fetal heart rate; RCOG Royal College of Obstetricians and Gynaecologists; SMFM = Society for Maternal-Fetal Medicine.

Information from National Collaborating Centre for Women's and Children's Health commissioned by the National Institute for Health and Care Excellence (NICE). Intrapartum care for healthy women and babies. 2017 Retrieved from https://www.nice.org.uk/guidance/cg190/chapter/Recommendations; Ayres-de-Campos D, Spong CY, Chandraharan E. FIGO consensus guidelines on intrapartum fetal monitoring: Cardiotocography. Int J of Gyn Obst*. 2015;131:13-24; Santo S, Ayres-de-Campos A, Costa-Santos C, et al. Agreement and accuracy using the FIGO, ACOG, and NICE cardiotocography interpretation guidelines.* Acta Obst Gyn Scand*. 2017. 96:166-175; Macones GA, Hankins GD, Spong CY, et al. The 2008 National Institute of Child Health and Human Development workshop report on electronic fetal monitoring: update on definitions, interpretation, and research guidelines.* Obstet Gynecol*. 2008;112(3):661-666.*

Federation of Gynecology and Obstetrics (FIGO) guidelines were introduced in 1987 and last updated in 2015, and are the only international consensus classification.[74]

No general recommendation has been made that any one nomenclature or classification system is preferable to another. One comparative study evaluated the accuracy and agreement of the FIGO, NICE, and NICHD classifications. Overall, the NICHD guidelines were less reliable and sensitive, but had higher specificity for the prediction of fetal acidemia and showed the best agreement among Category II classifications. With the FIGO and NICE guidelines, there is higher reliability, a trend toward higher sensitivity, and lower specificity in the prediction of fetal acidemia. Reliability of any of the guidelines was only slight to fair.[75]

Another study compared the five-tier and FIGO classification systems. The FIGO system showed a greater sensitivity and lower specificity to detect neonatal acidemia and severe metabolic

acidemia compared with the five-tier system. Interobserver agreement was moderate for both systems, but the five-tier system performed better in each specific category.[32,75]

In addition, use of the five-tier system has been shown to result in improved team communication compared with use of the NICHD system alone. Specifically, one study showed that it was a more effective tool for communicating nonreassuring fetal status and increased interobserver agreement on tracing interpretation (*Table 18*).[76]

Although there are some overall similarities among these EFM nomenclatures and classification systems, important differences exist. Providers should familiarize themselves with these differences in order to communicate effectively with other team members and provide appropriate care in international settings. *Table 16* and *Figure 5* summarize the differences between the NICHD, NICE, FIGO, and five-tier systems. Providers who plan to practice in international settings are encouraged to familiarize themselves with the terminology and classification systems used in their particular venue.

Notably, in some international settings, SIA may be the preferred method of monitoring the fetus during labor. In many low-resource settings, it may be the only option available for fetal assessment. In addition, SIA may be performed with a fetoscope rather than a handheld Doppler. Therefore, skills in SIA should be obtained before accepting an international assignment. More information on intrapartum fetal surveillance and other topics relevant to low-resource settings are available at www.aafp.org/globalalso.

Areas of Current Research

A prospective trial of 8,580 births conducted between 2010 and 2015 showed that women whose newborns had acidemia were more likely to be nulliparous and older, have higher body mass indexes and/or pregestational diabetes, and were more likely to have labor induced with prostaglandin or a Foley catheter bulb. The total deceleration area (area of FHR tracing monitored during decelerations [*Figure 2*]) for the 120 minutes preceding delivery was the most predictive of fetal acidemia. The Youden maximal cut point for this study was 42,152, with a sensitivity of 63.4% and specificity of 67.2%, resulting in a number needed to treat of five cesarean deliveries to prevent one case of acidemia. In addition, acceleration was an independent factor for normal pH levels with a sensitivity of 63.4% and specificity of 67.2%.[77]

Research is being conducted to enhance the efficacy of EFM by using computerized interpretation and to develop newer methodologies to monitor fetal well-being during labor.

Fetal hypoxemia results in biphasic changes in the ST segment of the fetal electrocardiogram (FECG) waveform and an increase in the T/QRS ratio. ST segment automated analysis software can record the frequency of ST events and, when combined with changes in EFM, has the potential to determine whether intervention during the labor process is warranted. The need for membrane rupture and internal fetal scalp monitoring is one drawback to this technique. Several studies have evaluated the effect of FECG analysis on reducing operative vaginal deliveries, fetal scalp sampling, neonatal encephalopathy, perinatal or neonatal death, seizures, 5-minute Apgar scores less than 7, neonatal intubation, NICU admission, and fetal acidosis (pH levels less than 7.05).[78-81] To date, RCTs and meta-analyses of over 26,500 women using FECG waveform analysis have failed to show an improvement in neonatal outcomes or operative delivery rates. There appears to be a modest reduction in metabolic acidosis; however, this decrease may be primarily due to the differences in the methodology and quality of these studies. The clinical significance of this observed modest reduction in metabolic acidosis is controversial, and further research is needed to confirm this finding.[82-85]

Another area of research is the use of computer analysis of key components of the fetal tracing,[86-88] or decision analysis, for the interpretation of the EFM tracing.[88] These studies have not been shown to improve clinical outcomes using computerized analysis.[86,87,89]

Fetal pulse oximetry was developed using an internal monitoring device, which requires the rupture of membranes, to continuously monitor fetal oxygenation saturation during labor. Trials have not shown significant differences in cesarean delivery rates or neonatal outcomes (Apgar scores, cord pH levels less than 7.0, seizures, intubation in the delivery room, stillbirth, death, NICU admission), and a recent Cochrane review noted that current data provided little support for use of fetal pulse oximetry.[83,90-92]

Figure 4. Comparison of Fetal Heart Rate Classification Systems

NICHD (Endorsed by ACOG, AAFP AWHONN, CNM, SMFM)

Category I (FHR tracings including all the following)
- Baseline rate: 110-160 BPM
- Baseline variability: 6-25 BPM
- LD and VD absent
- ED present or absent
- Accelerations present or absent

Category II (includes all FHR tracings not categorized as Category I or III. They include any of the following)
- Baseline
  - Baseline bradycardia not accompanied by absent variability
  - Baseline tachycardia
- Variability
  - Minimal variability
  - Absent variability with no recurrent decelerations
  - Marked variability
- Accelerations
  - Absence of induced accelerations after fetal stimulation
- Periodic or sporadic decelerations
  - Recurrent VD with minimal or moderate variability
  - PD (2-10 minutes)
  - Recurrent LD with moderate variability
  - VD with other characteristics such as slow return to baseline, overshoots, or shoulders

FIGO

Normal pattern
- Baseline heart rate between 110-150 BPM
- Amplitude of variability 5-25 BPM

Suspicious pattern
- Baseline rate 150-170 BPM OR 100-110 BPM
- Amplitude of variability 5-10 BPM for more than 40 minutes
- Increased variability above 25 BPM
- VD

NICE (Endorsed by RCOG)

Normal (CTG including all 4 features)
- Baseline rate 110-160 BPM
- Variability ≥5 BPM
- No decelerations
- Accelerations present

Suspicious (CTG where one of the following features is present and all others fall into the reassuring category)
- Baseline rate
  - 100-109 BPM
  - 161-180 BPM
- Baseline variability
  - <5 BPM for 40-90 minutes
- Decelerations
  - Typical VD with >50% of contractions occurring for >90 minutes
  - Single PD for up to 3 minutes
- Accelerations
  - The absence of accelerations with an otherwise normal trace is of uncertain significance

Five-Tier

Green
- Baseline normal
- Variability moderate
- No deceleration, ED or mild VD

Blue (low risk of acidemia)
- Moderate variability
  - Baseline tachycardia and no early or mild VD
  - Baseline normal and moderate VD or mild LD
- Minimal variability
  - Baseline tachycardia and no decelerations
- Normal baseline and no or ED

Yellow (No central fetal acidemia, but FHR suggestive of intermittent reductions in oxygen which may result in fetal oxygen debt)
- Moderate variability
  - Baseline tachycardia and moderate VD or mild/moderate LD or mild/moderate/severe PD
  - Normal baseline and severe VD or moderate/severe LD or mild/moderate PD
  - Mild bradycardia and no ED or mild/moderate VD or mild/moderate LD or mild/moderate VD
- Minimal variability
  - Baseline tachycardia and ED or mild VD
  - Baseline normal and mild VD
- Marked variability

Orange (borderline/acceptably low fetal acidemia and fetus potentially on the verge of decompensation)
- Moderate variability
  - Baseline tachycardia and severe VD or severe LD
  - Normal baseline and severe PD
  - Mild bradycardia and severe VD or severe LD or severe PD
  - Moderate bradycardia and severe VD or moderate/severe LD or severe PD
  - Severe bradycardia and no or ED or severe VD/LD/PD
- Minimal variability
  - Baseline tachycardia and moderate/severe VD or mild/moderate LD or mild/moderate PD
  - Normal baseline and moderate/severe VD or mild/moderate LD or mild/moderate PD
  - Mild/moderate bradycardia and no or ED
  - Absent variability
- Normal baseline and no decelerations

CTG = Cardiotocography; FHR = fetal heart rate; BPM = beats per minute; AAFP = American Academy of Family Physicians; ACNM = American College of Nurse-Midwives; ACOG = American College of Obstetricians and Gynecologists; AWHONN = Association of Women's Health, Neonatal and Obstetric Nurses; RCOG = Royal College of Obstetricians and Gynaecologists; SMFM = Society for Maternal Fetal Medicine; NICHD = Eunice Kennedy Shriver National Institute of Child Health and Human Development; FIGO = International Federation of Gynecology and Obstetrics; NICE = National Institute for Health and Care Excellence; VD = variable deceleration; ED = early deceleration; LD = late deceleration; PD = prolonged deceleration.

Information from National Collaborating Centre for Women's and Children's Health commissioned by the National Institute for Health and Care Excellence (NICE). Intrapartum care for healthy women and babies. 2017. Available at https://www.nice.org.uk/guidance/cg190/chapter/Recommendations; Ayres-de-Campos D, Spong CY, Chandraharan E. FIGO consensus guidelines on intrapartum fetal monitoring: Cardiotocography. Int J of Gyn Obst. *2015;131:13-24; Santo S. Ayres-de-Campos A, Costa-Santos C, et al. Agreement and accuracy using the FIGO, ACOG, and NICE cardiotocography interpretation guidelines.* Acta Obst Gyn Scand. *2017. 96:166-175; Parer JT, Ikeda T. A framework for standardized management of intrapartum fetal heart rate patterns.* Am J Obstet Gynecol. *2007;197(1):26.e1-26.e6; Macones GA, Hankins GD, Spong CY, et al. The 2008 National*

Category III (FHR tracings include either)
- Absent baseline FHR variability and any of the following:
  - Recurrent LD
  - Recurrent VD
  - Bradycardia
  - Sinusoidal pattern

Pathological pattern
- Baseline rate <100 BPM or >170 BPM
- Persistence of variability <5 BPM for >40 minutes
- Severe VD or severe repetitive ED

PD
- LD: the most ominous trace is a steady baseline without baseline variability and with small decelerations after each contraction
- A sinusoidal pattern

Pathologic (CTG with one or more of the following features or two or more features in the previous category)
- Baseline rate
  - <100 BPM
  - >180 BPM
- Sinusoidal pattern
  - ≥10 minutes
  - Baseline variability <5 BPM for ≥90 minutes

Decelerations
- Atypical VD with >50% contractions for >30 minutes
- LD for >30 minutes
- PD >3 minutes

Red (Unacceptably high acidemia, evidence of actual or impending damaging fetal asphyxia)

Minimal variability
- Baseline tachycardia and severe LD
- Baseline normal and severe LD/PD
- Mild bradycardia and mild/moderate/severe VD or mild/moderate/severe LD or mild/moderate/severe PD
- Moderate bradycardia and severe VD or moderate/severe LD or severe PD
- Severe bradycardia and no or ED or severe VD/LD/PD

Absent variability
- Baseline tachycardia regardless of no decelerations or presence of any decelerations
- Baseline normal and the presence of any deceleration
- Mild bradycardia regardless of no or presence of any deceleration
- Moderate bradycardia and no decelerations or ED or severe VD/LD/PD or moderate LD
- Severe bradycardia and no decelerations or ED or severe VD/LD/PD

Sinusoidal

Institute of Child Health and Human Development workshop report on electronic fetal monitoring: update on definitions, interpretation, and research guidelines. Obstet Gynecol. *2008;112(3):661-666.*

Summary

Initiation of fetal monitoring starts with assessment of maternal and fetal risk. Because EFM has a low positive predictive value and can result in increased rates of cesarean delivery, intermittent auscultation is recommended for low-risk pregnancies. However, hospital policy as well as staff availability and experience must be considered before using this technique. Clinicians should be ready to switch to EFM if a high-risk situation develops or if the provider is unable to adequately assess fetal status using SIA.

If EFM is selected, interpretation needs to be conducted in conjunction with fetal scalp or acoustic stimulation and with consideration of the clinical background, the overall tracing, and stage of labor. Outcomes are likely still unaffected using this technique, even in high-risk pregnancies. Efforts have recently been undertaken to standardize the definitions, interpretation, and general management of FHR tracings. DR C BRAVADO is a helpful mnemonic for defining risk and EFM interpretation. It is critical that institutions and hospitals ensure that all labor and delivery personnel are trained in FHR surveillance and interpretation and management of findings. Communication among team members is critical, and tools or strategies to maximize accuracy and completeness of transfer of information should be used (ie, SBAR or checklists) to minimize medical errors and maximize patient safety. Use of a vaginal delivery safety checklist has been proposed to enhance communication and teamwork by ensuring all delivery room personnel have a shared mental model regarding the patient's status and plan of care.[39]

Table 18. Comparing NICHD and ABC System (derived from Five-Tier System)

Measure	NICHD N=69	ABC system N=83	*P* Value
Effective tool to communicate nonreassuring fetal status	43%	80%	<0.01
Agree ≥75% on tracing interpretation with other providers	64%	79%	0.046

Reprinted from Triebwasser JE, Colvin R, Macones GA, Cahill AG. Nonreassuring Fetal Status in the Second Stage of Labor: Fetal Monitoring Features and Association with Neonatal Outcomes. Am J Perinatol. *2016;33(7):665-670.*

Regardless of the technology used, the patient-provider relationship is paramount during the labor process. Clinicians should not allow any monitoring approach to substitute for personal attention to the woman and fetus throughout labor.

Regular monitoring and compliance with all aspects of fetal surveillance should be undertaken. Multidisciplinary quality assurance committees should be comprised of physicians, nurses, administrators, and other pertinent staff for successful implementation. In addition, encouraging all staff involved with fetal monitoring to obtain certification or receive the same fetal monitoring training will facilitate standardized language and documentation surrounding fetal monitoring with the potential to improve patient outcomes.[11,12]

Nursing Considerations: Intrapartum Fetal Surveillance

- Use a standardized approach for communicating with team members about CEFM
- Understand indications for CEFM and support a policy for use of SIA in low-risk pregnancies in your institution
- Advocate for standard EFM training for all maternity providers in your institution to improve consistency in interdisciplinary documentation and interpretation of EFM tracings

CEFM = continuous electronic fetal monitoring; EFM = electronic fetal monitoring.

References

1. Graham EM, Petersen SM, Christo DK, Fox HE. Intrapartum electronic fetal heart rate monitoring and the prevention of perinatal brain injury. *Obstet Gynecol*. 2006; 108(3 Pt 1):656-666.
2. Martis R, Emilia O, Nurdiati DS, Brown J. Intermittent auscultation (IA) of fetal heart rate in labour for fetal well-being. *Cochrane Database Syst Rev*. 2017;2(2): CD008680.
3. Intermittent Auscultation for Intrapartum Fetal Heart Rate Surveillance: American College of Nurse-Midwives. *J Midwifery Womens Health*. 2015;60(5):626-632.
4. Alfirevic Z, Devane D, Gyte GML, Cuthbert A. Continuous cardiotocography (CTG) as a form of electronic fetal monitoring (EFM) for fetal assessment during labour. *Cochrane Database Syst Rev*. 2017;2(2):CD006066.
5. Chauhan SP, Klauser CK, Woodring TC, et al. Intrapartum nonreassuring fetal heart rate tracing and prediction of adverse outcomes: interobserver variability. *Am J Obstet Gynecol*. 2008;199(6):623.e1-623.e5.
6. Electronic fetal heart rate monitoring: research guidelines for interpretation. National Institute of Child Health and Human Development Research Planning Workshop. *Am J Obstet Gynecol*. 1997;177(6):1385-1390.
7. American College of Obstetricians and Gynecologists. Practice Bulletin no. 116: management of intrapartum fetal heart rate tracings. *Obstet Gynecol*. 2010;116(5):1232-1240.
8. Martí Gamboa S, Giménez OR, Mancho JP, et al. Diagnostic accuracy of the FIGO and the 5-tier fetal heart rate classification systems in the detection of neonatal acidemia. Am J Perinatol. 2017;34(5):508-514.
9. Parer JT, Hamilton EF. Comparison of 5 experts and computer analysis in rule-based fetal heart rate interpretation. *Am J Obstet Gynecol*. 2010;203(5):451.e1-451.e7.
10. Clark SL, Nageotte MP, Garite TJ, et al. Intrapartum management of category II fetal heart rate tracings: towards standardization of care. *Am J Obstet Gynecol*. 2013;209(2):89-97.
11. Govindappagari S, Zaghi S, Zannat F, et al. Improving interprofessional consistency in electronic fetal heart rate interpretation. *Am J Perinatol*. 2016;33(8):808-813.
12. Nageotte MP, Tomlinson MW, Okeefe M. A credentialing test for EFM. *Contemp Ob Gyn*. 2016;61(5):19-24.
13. Lyndon A, Ali LU, eds. Association of Women's Health, Obstetric, and Neonatal Nursing. *Fetal Heart Monitoring Principles and Practices*. 5th ed. Washington, DC: Kendall Hunt Professional; 2015.
14. American College of Obstetricians and Gynecologists. ACOG Practice Bulletin no. 106: intrapartum fetal heart rate monitoring: nomenclature, interpretation, and general management principles. *Obstet Gynecol*. 2009; 114(1):192-202.
15. Kilpatrick SJ, Papile LA, eds. *Guidelines for Perinatal Care*. 8th ed. Washington DC: American College of Obstetricians and Gynecologists and American Academy of Pediatrics; 2017.
16. Committee on Obstetric Practice. Committee Opinion no. 687: approaches to limit intervention during labor and birth. *Obstet Gynecol*. 2017;129(2):e20-e28.
17. Lawrence A, Lewis L, Hofmeyr G, Styles C. Maternal positions and mobility during first stage labour. *Cochrane Database of Syst Revs*. 2013;(10):CD003934.
18. Grant A, O'Brien N, Joy MT, et al. Cerebral palsy among children born during the Dublin randomised trial of intrapartum monitoring. *Lancet*. 1989;2(8674):1233-1236.
19. Devane D, Lalor JG, Daly S, et al. Cardiotocography versus intermittent auscultation of fetal heart on admission to labour ward for assessment of fetal wellbeing. *Cochrane Database Syst Rev*. 2017;1(1):CD005122.
20. Hauth JC, Hankins GD, Gilstrap LC III, et al. Uterine contraction pressures with oxytocin induction/augmentation. *Obstet Gynecol*. 1986;68(3):305-309.
21. Bix E, Reiner LM, Klovning A, Oian P. Prognostic value of the labour admission test and its effectiveness compared with auscultation only: a systematic review. *BJOG*. 2005;112(12):1595-1604.
22. American College of Obstetricians and Gynecologists. Neonatal encephalopathy and cerebral palsy: executive summary. *Obstet Gynecol*. 2004;103(4):780-781.
23. The American College of Obstetricians and Gynecologists and American Academy of Pediatrics. *Neonatal Encephalopathy and Neurologic Outcome*. 2nd ed. Washington DC: American College of Obstetricians and Gynecologists; 2014.
24. Alfirevic Z, Devane D, Gyte GM. Continuous cardiotocography (CTG) as a form of electronic fetal monitoring (EFM) for fetal assessment during labour. *Cochrane Database Syst Rev*. 2013;(5):CD006066.
25. Macones GA, Hankins GD, Spong CY, et al. The 2008 National Institute of Child Health and Human Development workshop report on electronic fetal monitoring: update on definitions, interpretation, and research guidelines. *Obstet Gynecol*. 2008;112(3):661-666.
26. Association of Women's Health, Obstetric, and Neonatal Nurses. AWHONN position statement: fetal heart monitoring. *J Obstet Gynecol Neonatal Nurs*. 2015; 44(5):683-686.
27. Bakker JJ, Janssen PF, van Halem K, et al. Internal versus external tocodynamometry during induced or augmented labour. *Cochrane Database Syst Rev*. 2013; 8(8):CD006947.
28. Harper LM, Shanks AL, Tuuli MG, et al. The risks and benefits of internal monitors in laboring patients. *Am J Obstet Gynecol*. 2013;209(1):38.e1-38.e6.
29. Yang M, Stout MJ. López JD, et al. Association of Fetal Heart Rate Baseline Change and Neonatal Outcomes. *Am J Perinatol*. 2017;34(9):879-886.
30. Freeman RK, Garite TJ, Nageote MP, Mille LA. *Fetal Heart Rate Monitoring*. 4th ed. New York, NY: Wolters Kluwer, Lippincott Williams & Wilkins; 2012.
31. Gabbe SG, Niebyl JR, Galan HL, et al. Intrapartum Fetal Evaluation. In: Gabbe SG, Niebyl JR, Galan HL, et al. *Obstetrics: Normal and Problem Pregnancies*. 6th ed. Philadelphia, PA: Saunders; 2012.
32. Coletta J, Murphy E, Rubeo Z, Gyamfi-Bannerman C. The 5-tier system of assessing fetal heart rate tracings is superior to the 3-tier system in identifying fetal acidemia. *Am J Obstet Gynecol*. 2012;206(3):226.e1-226.e5.

33. Perinatology.com. Intrapartum Fetal Heart Rate Monitoring. Available at http://perinatology.com/Fetal%20Monitoring/Intrapartum%20Monitoring.htm.
34. National Institute for Health and Care Excellence (NICE). Intrapartum care for healthy women and babies. 2017. Available at https://www.nice.org.uk/guidance/cg190/chapter/Recommendations.
35. Modanlou HD, Murata Y. Sinusoidal heart rate pattern: reappraisal of its definition and clinical significance. *J Obstet Gynaecol Res*. 2004;30(3):169-180.
36. Institute for Healthcare Improvement. SBAR Tool: Situation-Background-Assessment-Recommendation. 2018. Available at http://www.ihi.org/resources/Pages/Tools/SBARTechniqueforCommunicationASituationalBriefingModel.aspx.
37. Clark SL, Meyers JA, Frye DK, et al. Recognition and response to electronic fetal heart rate patterns: impact on newborn outcomes and primary cesarean delivery rate in women undergoing induction of labor. *Am J Obstet Gynecol*. 2015;212(4):494.e1-494.e6.
38. Clark S, Belfort M, Saade G, et al. Implementation of a conservative checklist-based protocol for oxytocin administration: maternal and newborn outcomes. *Am J Obstet Gynecol*. 2007;197(5):480.e1-480.e5.
39. True BA, Cochrane CC, Sleutel MR, et al. Developing and testing a vaginal delivery safety checklist. *J Obstet Gynecol Neonatal Nurs*. 2016;45(2):239-248.
40. Bloom SL, Leveno KJ, Spong CY, et al; National Institute of Child Health and Human Development Maternal-Fetal Medicine Units Network. Decision-to-incision times and maternal and infant outcomes. *Obstet Gynecol*. 2006;108(1):6-11.
41. Jackson M, Holmgren CM, Esplin MS, et al. Frequency of fetal heart rate categories and short-term neonatal outcome. *Obstet Gynecol*. 2011;118(4):803-808.
42. Parer JT, Ikeda T. A framework for standardized management of intrapartum fetal heart rate patterns. *Am J Obstet Gynecol*. 2007;197(1):26.e1-26.e6.
43. Sadaka A, Furuhashi M, Minami H, et al. Observation on validity of the five-tier system for fetal heart rate pattern interpretation proposed by Japan Society of Obstetricians and Gynecologists. *J Matern Fetal Neonatal Med*. 2011;24(12):1465-1469.
44. Okai T, Ikeda T, Kawarabayashi T, et al; Perinatology Committee of the Japan Society of Obstetrics and Gynecology. Intrapartum management guidelines based on fetal heart rate pattern classification. *J Obstet Gynaecol Res*. 2010;36(5):925-928.
45. Gyamfi Bannerman C, Grobman WA, Antoniewicz L, et al. Assessment of the concordance among 2-tier, 3-tier, and 5-tier fetal heart rate classification systems. *Am J Obstet Gynecol*. 2011;205(3):288.e1-288.e4.
46. Uccella S, Cromi A, Colombo GF, et al. Interobserver reliability to interpret intrapartum electronic fetal heart rate monitoring: Does a standardized algorithm improve agreement among clinicians? *J Obstet Gynaecol*. 2015;35(3):241-245.
47. Clark SL, Hamilton EF, Garite TJ, et al. The limits of electronic fetal heart rate monitoring in the prevention of neonatal metabolic acidemia. *Am J Obstet Gynecol*. 2017;216(2):163e.1-163.e6.
48. Garite TJ, Simpson KR. Intrauterine resuscitation during labor. *Clin Obstet Gynecol*. 2011;54(1):28-39.
49. Bullens LM, van Runnard Heimel PJ, van der Hout-van der Jagt MB, Oei SG. Interventions for intrauterine resuscitation in suspected fetal distress during term labor: a systematic review. *Obstet Gynecol Surv*. 2015;70(8):524-539.
50. Haydon ML, Gorenberg DM, Nageotte MP, et al. The effect of maternal oxygen administration on fetal pulse oximetry during labor in fetuses with nonreassuring fetal heart rate patterns. *Am J Obstet Gynecol*. 2006;195(3):735-738.
51. Garabedian C, Butruille L, Drumez E, et al. Interobserver reliability of 4 fetal heart rate classifications. *J Gynecol Obstet Hum Reprod*. 2017;46(2):131-135.
52. Hamel MS, Anderson BL, Rouse DJ. Oxygen for intrauterine resuscitation: of unproved benefit and potentially harmful. *Am J Obstet Gynecol*. 2014;211(2):124-127.
53. Garite TJ, Nageotte MP, Parer JT. Should we really avoid giving oxygen to mothers with concerning fetal heart rate patterns? *Am J Obstet Gynecol*. 2015;212(4):459-460, 459.e1.
54. Hamel MS, Hughes BL, Rouse DJ. Whither oxygen for intrauterine resuscitation? *Am J Obstet Gynecol*. 2015;212(4):461-462, 461.e1.
55. Fawole B, Hofmeyr GJ. Maternal oxygen administration for fetal distress. *Cochrane Database Syst Rev*. 2012;12(12):CD000136.
56. Association of Women's Health, Obstetric, and Neonatal Nursing. *Nursing Care and Management of the Second Stage of Labor: Evidence-Based Clinical Practice Guideline*. 2nd ed. Washington DC; 2008.
57. Yildirim G, Beji NK. Effects of pushing techniques in birth on mother and fetus: a randomized study. *Birth*. 2008;35(1):25-30.
58. Simpson KR, James DC. Effects of immediate versus delayed pushing during second-stage labor on fetal well-being: a randomized clinical trial. *Nurs Res*. 2005;54(3):149-157.
59. Hansen SL, Clark SL, Foster JC. Active pushing versus passive fetal descent in the second stage of labor: a randomized controlled trial. *Obstet Gynecol*. 2002;99(1):29-34.
60. Brancato RM, Church S, Stone PW. A meta-analysis of passive descent versus immediate pushing in nulliparous women with epidural analgesia in the second stage of labor. *J Obstet Gynecol Neonatal Nurs*. 2008;37(1):4-12.
61. Hanson L. Second-stage labor care: challenges in spontaneous bearing down. *J Perinat Neonatal Nurs*. 2009;23(1):31-39, quiz 40-41.
62. Lemos A, Amorim MMR, Dornelas de Andrade A, et al. Pushing/bearing down methods for the second stage of labour. *Cochrane Database Syst Rev*. 2017;3(3):CD009124.

63. Hofmeyr GJ, Lawrie TA. Amnioinfusion for potential or suspected umbilical cord compression in labour. *Cochrane Database Syst Rev*. 2012;1(1):CD000013.
64. Rinehart BK, Terrone DA, Barrow JH, et al; ACOG Committee Obstetric Practice. Randomized trial of intermittent or continuous amnioinfusion for variable decelerations. *Obstet Gynecol*. 2000;96(4):571-574.
65. Hofmeyr GJ, Xu H, Eke AC. Amnioinfusion for meconium-stained liquor in labour. *Cochrane Database Syst Rev*. 2014;1(1):CD000014.
66. Kulier R, Hofmeyr GJ. Tocolytics for suspected intrapartum fetal distress. *Cochrane Database Syst Rev*. 2000; (2):CD000035.
67. Leathersich SJ, Vogel JP, Tran TS, Hofmeyr GJ. Acute tocolysis for uterine tachysystole or suspected fetal distress. *Cochrane Database Syst Rev*. 2018;7:CD009770.
68. Ross MG. Labor and fetal heart rate decelerations: relation to fetal metabolic acidosis. *Clin Obstet Gynecol*. 2011;54(1):74-82.
69. Bakker PCAM, Kurver PHJ, Kuik DJ, Van Geijn HP. Elevated uterine activity increases the risk of fetal acidosis at birth. *Am J Obstet Gynecol*. 2007;196(4):313.e1-313.e6.
70. Simpson KR, James DC. Effects of oxytocin-induced uterine hyperstimulation during labor on fetal oxygen status and fetal heart rate patterns. *Am J Obstet Gynecol*. 2008;199(1):34.e1-34.e5.
71. Heuser CC, Knight S, Esplin MS, et al. Tachysystole in term labor: incidence, risk factors, outcomes, and effect on fetal heart tracings. *Am J Obstet Gynecol*. 2013; 209(1):32.e1-32.e6. Erratum in *Am J Obstet Gynecol*. 2014;210(2):162.
72. Simpson KR, Miller L. Assessment and optimization of uterine activity during labor. *Clin Obstet Gynecol*. 2011; 54(1):40-49.
73. Skupski DW, Rosenberg CR, Eglinton GS. Intrapartum fetal stimulation tests: a meta-analysis. *Obstet Gynecol*. 2002;99(1):129-134.
74. Ayres-de-Campos D, Spong CY, Chandraharan E; FIGO Intrapartum Fetal Monitoring Expert Consensus Panel. FIGO consensus guidelines on intrapartum fetal monitoring: Cardiotocography. *Int J Gynaecol Obstet*. 2015; 131(1):13-24.
75. Santo S, Ayres-de-Campos D, Costa-Santos C, et al. Da Graça LM; FM-Compare Collaboration. Agreement and accuracy using the FIGO, ACOG and NICE cardiotocography interpretation guidelines. *Acta Obstet Gynecol Scand*. 2017;96(2):166-175.
76. Triebwasser JE, Colvin R, Macones GA, Cahill AG. Nonreassuring Fetal Status in the Second Stage of Labor: Fetal Monitoring Features and Association with Neonatal Outcomes. *Am J Perinatol*. 2016;33(7):665-670.
77. Cahill AG, Tuuli MG, Stout MJ. et al. A prospective cohort study of fetal heart rate monitoring: deceleration area is predictive of fetal acidemia. *Am J Obstet Gynecol*. 2018;218(5):523.e1-523.e12.
78. Neilson JP. Fetal electrocardiogram (ECG) for fetal monitoring during labour. *Cochrane Database Syst Rev*. 2013;5(5):CD000116.
79. Devoe LD, Ross M, Wilde C, et al. United States multicenter clinical usage study of the STAN 21 electronic fetal monitoring system. *Am J Obstet Gynecol*. 2006; 195(3):729-734.
80. Vayssiere C, Haberstich R, Sebahoun V, et al. Fetal electrocardiogram ST-segment analysis and prediction of neonatal acidosis. *Int J Gynaecol Obstet*. 2007;97(2): 110-114.
81. Ojala K. Vääräsmäki M, Mäkikallio K, et al. A comparison of intrapartum automated fetal electrocardiography and conventional cardiotocography—a randomised controlled study. *BJOG*. 2006;113(4):419-423.
82. Belfort MA, Saade GR, Thom E, et al; Eunice Kennedy Shriver National Institute of Child Health and Human Development Maternal–Fetal Medicine Units Network. A randomized trial of intrapartum fetal ECG ST-Segment analysis. *N Engl J Med*. 2015;373(7):632-641.
83. Amer-Wahlin I, Kwee A. Combined cardiotocographic and ST event analysis: A review. *Best Pract Res Clin Obstet Gynaecol*. 2016;30:48-61.
84. Bloom SL, Belfort M, Saade G; Eunice Kennedy Shriver National Institute of Child Health and Human Development Maternal-Fetal Medicine Units Network. What we have learned about intrapartum fetal monitoring trials in the MFMU Network. *Semin Perinatol*. 2016;40(5):307-317.
85. Blix E, Brurberg KG, Reierth E, et al. ST waveform analysis versus cardiotocography alone for intrapartum fetal monitoring: a systematic review and meta-analysis of randomized trials. *Acta Obstet Gynecol Scand*. 2016; 95(1):16-27.
86. Salamalekis E, Hintipas E, Salloum I, et al. Computerized analysis of fetal heart rate variability using the matching pursuit technique as an indicator of fetal hypoxia during labor. *J Matern Fetal Neonatal Med*. 2006;19(3):165-169.
87. Giannubilo SR, Buscicchio G, Gentilucci L, et al. Deceleration area of fetal heart rate trace and fetal acidemia at delivery: a case-control study. *J Matern Fetal Neonatal Med*. 2007;20(2):141-144.
88. Westgate JA, Wibbens B, Bennet L, et al. The intrapartum deceleration in center stage: a physiologic approach to the interpretation of fetal heart rate changes in labor. *Am J Obstet Gynecol*. 2007;197(3): 236.e1-236.e11.
89. Greene MF. Obstetricians still await a deus ex machina. *N Engl J Med*. 2006;355(21):2247-2248.
90. East CE, Brennecke SP, King JF, et al; FOREMOST Study Group. The effect of intrapartum fetal pulse oximetry, in the presence of a nonreassuring fetal heart rate pattern, on operative delivery rates: a multicenter, randomized, controlled trial (the FOREMOST trial). *Am J Obstet Gynecol*. 2006;194(3):606.e1-606.e16.
91. Bloom SL, Spong CY, Thom E, et al; National Institute of Child Health and Human Development Maternal-Fetal Medicine Units Network. Fetal pulse oximetry and cesarean delivery. *N Engl J Med*. 2006;355(21): 2195-2202.
92. East CE, Begg L, Colditz PB, Lau R. Fetal pulse oximetry for fetal assessment in labour. *Cochrane Database Syst Rev*. 2014;10(10):CD004075.

Preterm Labor and Prelabor Rupture of Membranes

Learning Objectives

1. Discuss risk factors associated with preterm labor (PTL) and preterm prelabor rupture of membranes (PPROM).
2. Identify patients who may benefit from antenatal progesterone.
3. Outline evaluation and management of PTL and PPROM.
4. Discuss neonatal group B streptococcal prevention strategies.

Introduction

Preterm birth (PTB) occurs in approximately 10% of pregnancies in the United States.[1] The causes are multifactorial and complex. Identifying effective preventive interventions for those at risk can reduce PTBs. Previous PTB is the most important historical risk factor for subsequent PTB.[2] Progesterone may be used to decrease the likelihood of PTB in patients with a previous PTB or short cervical length (CL).[3] When a patient presents with preterm contractions, assessing CL on transvaginal ultrasound (TVU) and fetal fibronectin (fFN) levels may help determine the risk of PTB.[4] Antenatal corticosteroids (ACSs) are the most important intervention to improve neonatal outcomes in women with preterm labor (PTL).[5] Tocolytic drugs may delay PTB, which allows time to administer steroids and transfer the patient to a facility with a neonatal intensive care unit (NICU).[6] Management of preterm prelabor rupture of membranes (PPROM) may include administering antibiotics or ACSs, or, depending on gestational age at presentation, inducing labor.[7]

Epidemiology and Pathophysiology

Worldwide, PTB rates vary from 5% in Northern Europe to 18% in sub-Saharan Africa.[1,8] The incidence of PTB in the United States, defined as occurring before 37 weeks' gestation, was 9.85% in 2016.[9]

Approximately 45% of PTBs occur with spontaneous PTL with intact membranes. Twenty five percent of PTBs are associated with PPROM and the remaining 30% are due to delivery for medical indications.[10,11] PTB has complex multifactorial causes. Etiologies of PTL include mechanical (overdistension, cervical incompetence), infection-inflammatory, immunological, vascular-placental, and hormonal (especially progesterone deficiency).[12,13]

In 2015, the Eunice Kennedy Shriver National Institute of Child Health and Human Development (NICHD) and the Genomic and Proteomic Network for Preterm Birth Research developed a classification system with active phenotypes showing prevalent causes of PTB including maternal stress (43%), PPROM (29%), familial factors (12%), maternal comorbidities (6%), and infection, hemorrhage, and placental dysfunction (10%).[14] Research is identifying maternal genetic and epigenetic propensities, proteomic expression, vaginal microbiome, and environmental factors leading to increased risk of PTB. One example is the TNF-2 allele of the tumor necrosis factor gene, which, when present, yields a 1.6 odds ratio (OR) of PTB in whites and a 2.5 OR in blacks. This risk is higher if the environmental cofactor of symptomatic bacterial vaginosis (BV) is present.[1] Social determinants of health also affect PTB rates, and include income, unemployment, malnutrition, stress, and lack of social support. In the United States, significant disparities exist in the rates between racial ethnic groups. In 2016, the PTB rate for non-Hispanic white infants was 9% compared with 14% for non-Hispanic black infants.[15]

Risk Factors for Preterm Birth

Up to 50% of PTBs occur in pregnancies with no known risk factors, and more than 50% of women with identifiable risk factors for PTB will ultimately deliver at term.[16,17] A 2015 Cochrane review of risk scoring systems failed to identify an eligible study and noted the role of risk scoring systems is unknown.[18] However, recognizing and responding to risks can lower PTB when there are evidence-based effective interventions and allow for transfer to a higher level of care. Risk factors and interventions for PTB are listed in *Tables 1* and *2*.

Risks with low prevalence but high rates of PTB include multiple gestation, unicornate uterus, and history of incompetent cervix.

Previous PTB is the most important identifiable risk factor for recurrent PTB. Women with a singleton

Table 1. Risk Factors for Preterm Labor

Maternal Characteristics	Pregnancy Characteristics
Non-Hispanic black race	Multiple gestation
BMI <19 kg/m^2	Polyhydramnios or oligohydramnios
BMI ≥30 kg/m^2	Uterine anomalies
Low economic status	Previous cesarean delivery
Stressful life events	Shortened cervix
Fatigue-inducing work	Use of assisted reproductive technology
Age <20 years or >35 years	Vaginal bleeding from placental abruption or placenta previa
Maternal Medical History	Threatened abortion current pregnancy
Previous PTB	Maternal abdominal surgery during pregnancy
Previous surgical abortions (>1 versus none)	Interpregnancy interval <6 months
Infection	Cocaine or heroin use
Bacterial vaginosis	Alcohol use (>10 drinks/week)
Asymptomatic bacteriuria	Tobacco use
UTI/pyelonephritis	Maternal depression during pregnancy
Intrauterine infection	Presence of thyroid autoantibodies
Periodontal infection	Low vitamin D level

BMI = body mass index; PTB = preterm birth; UTI = urinary tract infection.

Information from Purisch SE, Gyamfi-Bannerman C. Epidemiology of preterm birth. Semin Perinatol. *2017;41(7):387-391; Martin JA, Hamilton BE, Osterman MJK. Births in the United States, 2016.* NCHS Data Brief. *2017;(287):1-8.*

pregnancy after a previous singleton PTB have a 20% risk of recurrence (95% confidence interval [CI] = 19.9-20.6).[2] In contrast, after a term delivery, women have a 2.7% risk of PTB in the subsequent pregnancy. More specifically, after delivery between 32 to 36 weeks' gestation, a subsequent pregnancy has a 14.7% risk of PTB (95% CI = 5.84-6.42), and after delivery before 28 weeks' gestation, a subsequent pregnancy has a 26% risk of PTB (95% CI = 10.8-15.9).[19] Multiple previous PTBs further increase risk.

Multiple gestation is a strong predictor for PTB with the majority of twin pregnancies delivering at less than 37 weeks' gestation because of spontaneous PTL or medical indications for delivery.[20] Higher order multiples further increase the risk of PTB. In 2011, 11% of twin, 36% of triplet, and more than 67% of quadruplet pregnancies delivered before 32 weeks' gestation.[21]

Risk factors for PTB are listed in *Table 1*.

Laboratory identified exposures increasing the OR of PTB include a maternal serum alpha fetoprotein greater than the 90th percentile (OR = 8.3), severe anemia (OR = 2.2), urinary tract infection (UTI) (OR = 2.0), and BV (OR = 2.0),[22] and the presence of antithyroid antibodies (OR = 2.07).[23]

Laboratory and clinical values can be combined to assess PTB risk. Two risk factors for PTB are shortened CL and the presence of vaginal fFN. They have most often been assessed in high-risk screening of symptomatic women with threatened PTL. Alternatively, they have been assessed as a screening tool of low-risk asymptomatic nulliparous women. Between 16 and 22 weeks' gestation, the average CL for normal healthy pregnant women is between 3.7 and 4 cm, whereas short CL of 2 cm or less is found in approximately 1% of women and 2.5 cm or less in approximately 2%.[24] Cervical shortening in the second trimester is associated with increased risk of PTB.[25] When measuring CL, TVU should be used because transabdominal ultrasound may miss up to 57% of cervices shorter than 2.5 cm.[26] The shorter the cervix and the earlier in gestation the shortening is detected, the higher the risk of PTB.[27] In women without vaginal bleeding, the rate of change in cervical shortening, when measured by sequential cervical ultrasound, is associated with increased risk of PTB (OR = 1.2; 95% CI = 1.1-1.4).[28]

Common pregnancy medical conditions ending in PTB are multiple pregnancy, hypertensive disorders of pregnancy with severe features, antenatal hemorrhage due to placenta previa or abruption, intrauterine growth rate, cervical incompetence, and infection. Women with a previous medically indicated PTB are at increased risk of a subsequent medically indicated PTB and are also at risk of a subsequent spontaneous PTB.[29]

Effective Evidence-Based Interventions

Prevention can be categorized in four main interventions: immunizations, chemoprophylaxis-procedures, screening tests, and counseling. Most of the effective interventions are focused on women at high risk of PTB.

Immunizations

In a meta-analysis of large vaccine trials, influenza vaccine has been shown to decrease PTB by 13%.[30]

Chemoprophylaxis Procedures

Antenatal progesterone. Antenatal progesterone has been shown to lower PTB rates in women

with a history of PTB and a cervix length of 2.5 cm or less on TVU. After 17-hydroxyprogesterone (17OHP) is started, it should not be stopped because discontinuation increases the risk of recurrent PTB.[31] Progesterone is not effective in women with multiple pregnancy.[3] Progesterone dose and administration are presented in *Table 3*.[32-34]

For women with a previous PTB, a 2013 Cochrane review (updated 2015) noted a 36% decrease in PTB at less than 34 weeks' gestation and 45% at less than 37 weeks' gestation. Treated pregnancies led to 60% less need for assisted ventilation and incurred 70% less necrotizing enterocolitis and 55% less neonatal death. The

Table 2. Effective Interventions to Prevent Preterm Birth

Intervention Category	PTB High-Risk Subgroup	Intervention	RR Effect Size
Immunization	All pregnant in flu season	Influenza vaccine	RR = 0.87 (95% CI = 0.77-0.98)
Chemoprophylaxis and preventive procedures	Prior PTB	Progesterone	See *Table 3*
	Short cervix ≤2.5 cm on transvaginal US		
	Prior PTB and short cervix History of incompetent cervix	Cerclage	RR = 0.77 (95% CI = 0.66-0.89)
	High risk of preeclampsia	Low-dose ASA	RR = 0.86 (95% CI = 0.76-0.98)
	Undernourished	Zinc	RR = 0.86 (95% CI = 0.76-0.97)
	Hypothyroidism	Euthyroid replacement	RR = 0.28 (95% CI = 0.20-0.80)
	Assisted reproduction	Techniques to avoid multiple gestation	
Screening	Pregnant women with asymptomatic bacteriuria	Antibiotics	RR = 0.27 (95% CI = 0.11-0.62)
Counseling	Smoking	Smoking cessation	
	Illicit drug use	Drug treatment and abstinence	
	All postpartum women to prevent short interpregnancy interval	Family planning and reliable contraception	

ASA = acetylsalicylic acid; CI = confidence interval; PTB = preterm birth; RR = relative risk; US = ultrasound.

(Note: Keeping track of the many content recommendations in prenatal care can be difficult. One method is to apply the 4-box categorization of preventive interventions to be addressed at each visit, and recognize whether an intervention applies to all pregnant women or a specific high-risk group)

Information from various sources.

Table 3. Progesterone Formulation and Dosage for the Prevention of Preterm Birth

Indication	Progestogen	Dosage
Prior PTB	17-alpha hydroxyprogesterone caproate	250 mg IM weekly from 16 to 36 weeks' gestation
CL <2 cm at <24 weeks' gestation without prior PTB	Vaginal progesterone gel, 90 mg Vaginal progesterone capsule, 200 mg	Daily from diagnosis of short CL until 36 weeks' gestation

CL = cervical length; IM = intramuscular; PTB = preterm birth.

Information from Society for Maternal-Fetal Medicine Publications Committee, with assistance of Vincenzo Berghella. Progesterone and preterm birth prevention: translating clinical trials data into clinical practice. Am J Obstet Gynecol. 2012;206(5):376-386; Iams JD. Clinical practice. Prevention of preterm parturition. N Engl J Med. 2014;370(3):254-261; Committee on Practice Bulletins—Obstetrics, The American College of Obstetricians and Gynecologists. Practice Bulletin no. 130: prediction and prevention of preterm birth. Obstet Gynecol. 2012;120(4):964-973.

review noted no difference in the route and timing of the progesterone but called for further trials to determine the optimal timing, dose, and route.[23] The 2017 Society for Maternal-Fetal Medicine (SMFM) recommendation is to use intramuscular (IM) 17OHP for women with a history of PTB starting at 16 to 20 weeks' gestation and continuing until 36 weeks' gestation. IM 17OHP should be used rather than vaginal progesterone because studies including the 2016 OPPTIMUM study (n = 1,228), have found negative effects of vaginal progesterone in asymptomatic women with a previous PTB.[35]

Women with no history of PTB but with a shortened cervix (2 cm or less on ultrasound) should receive vaginal progesterone from the time of diagnosis until 36 weeks' gestation.[32-34] Two large trials[36,37] and three meta-analyses have shown decreased incidence of PTB and neonatal morbidity and mortality with this intervention (*Table 4*).[3,38,39] A 2016 systematic review and meta-analysis of individual patient data (n = 974) showed a 38% decrease in PTB at less than 33 weeks' gestation with vaginal progesterone compared with placebo when treating a short cervix of 2.5 cm or less.[40,41]

Low-dose aspirin. Aspirin administered to women at high risk of preeclampsia had an absolute risk reduction of 2% to 4% of PTB (relative risk [RR] 0.86; 95% CI = 0.76-0.98).[42]

Cerclage. A 2014 American College of Obstetricians and Gynecologists (ACOG) Practice Bulletin noted cerclage placements are indicated for a history of cervical incompetence, which includes one or more second trimester pregnancy losses with painless dilation not due to abruption or labor. A second indication is a physical examination showing painless dilation in the second trimester. A third indication is a singleton pregnancy with a history of PTB and a CL of 2.5 cm or less is an indication for cerclage.[43] Women with a history of PTB, who are also being treated with 17OHP, should have CL evaluated by TVU every 2 weeks from 16 to 23 6/7 weeks' gestation. If CL is less than 2.5 cm, cervical cerclage can be offered.[32-34] Cerclage is not effective in women with singleton or multiple gestations with a short cervix but without a prior spontaneous PTB. A 2017 Cochrane review found cerclage for all indications reduced PTB in patients at less than 37 weeks' gestation and less than 34 weeks' gestation by 23% (average RR = 0.77; 95% CI = 0.66-0.89). There were too few trials of the subgroups to further define indications.[44]

Cervical pessary. The larger trials of cervical pessary have not shown a decrease in PTB.[45]

Screening Tests

Universal transvaginal cervical length screening. Universal screening by TVU to measure CL of women with a singleton gestation and no prior PTB is a subject of debate. The 2016 SMFM recommendations caution against generalization from high- to low-risk groups and emphasizes potential harms of screening, recommending "practitioners who decide to implement universal cervical length screening follow strict guidelines."[46]

Table 4. Progesterone and Cervical Cerclage for the Prevention of Preterm Birth

Indication	PTB RR (CI)	Neonatal Morbidity and Mortality
Vaginal progesterone for short cervix	Delivery <33 weeks: 0.62 (0.47-0.81)	Newborn RDS: 0.47 (0.27-0.81)
17OHP for previous PTB	Delivery <34 weeks: 0.31 (0.14-0.69)	Mortality: 0.50 (0.33-0.75)
Cerclage for short cervix and previous PTB	Delivery <32 weeks: 0.63 (0.45-0.88)	Mortality: 0.58 (0.35-0.98)

17OHP = 17 alpha-hydroxyprogesterone caproate; CI = confidence interval; PTB = preterm birth; RDS = respiratory distress syndrome; RR = relative risk.

Information from Dodd JM, Jones L, Flenady V, et al. Prenatal administration of progesterone for preventing preterm birth in women considered to be at risk of preterm birth. Cochrane Database Syst Rev. 2013;7(7): CD004947; Conde-Agudelo A, Romero R, Nicolaides K, et al. Vaginal progesterone vs. cervical cerclage for the prevention of preterm birth in women with a sonographic short cervix, previous preterm birth, and singleton gestation: a systematic review and indirect comparison metaanalysis. Am J Obstet Gynecol. 2013;208(1):42.e1-42.e18; Romero R, Nicolaides K, Conde-Agudelo A, et al. Vaginal progesterone in women with an asymptomatic sonographic short cervix in the midtrimester decreases preterm delivery and neonatal morbidity: a systematic review and metaanalysis of individual patient data. Am J Obstet Gynecol. 2012;206(2):124.e1-124.e19; Romero R, Conde-Agudelo A, Da Fonseca E, et al. Vaginal progesterone for preventing preterm birth and adverse perinatal outcomes in singleton gestations with a short cervix: a meta-analysis of individual patient data. Am J Obstet Gynecol. 2018;218(2):161-180.

Fetal fibronectin. Fetal fibronectin is an extracellular matrix glycoprotein found at the maternal-fetal interface, and in normal pregnancies it is almost undetectable in vaginal secretions. With disruption of this interface, which occurs before the onset of labor, fFN is released into vaginal secretions. fFN has a low positive predictive value (13% to 30%) for delivery within 7 to 10 days in symptomatic patients and a high negative predictive value (99%) for delivery within 14 days of detection.[47,48] fFN is primarily used after 20 weeks' gestation for threatened PTL. It can be tested as qualitative positive/negative or quantitative as less than 10 ng/mL, less than 50 ng/mL, and greater than 200 ng/mL, with higher amounts corresponding to higher risk of PTB. In a study of 9,410 asymptomatic nulliparous women, fFN was present in 7.3% of women at 16 to 22 weeks' gestation, and 8% of women at 22 to 30 weeks' gestation who eventually progressed to PTB, showing a poor positive predictive value.[24] A 2019 Cochrane review showed that management based on knowledge of fFN status did not produce a decrease in PTB prior to 34 weeks' gestation.[49]

Screening for vaginal infections. The U.S. Preventive Services Task Force recommends against screening for BV in women with a low risk of infection and concludes that evidence is insufficient to recommend for or against screening for BV in high-risk patients.[50] A 2013 Cochrane review based on 21 trials with 7,847 women found no effect on PTB with the treatment of BV.[51] A 2015 Cochrane review of one trial of 4,155 Austrian women screened for vaginal infections before 20 weeks' gestation (ie, BV, trichomonas, candida) found a 2% difference in PTB (RR = 0.55; 95% CI = 0.41-0.75).[52]

If screening for BV is incorporated into clinical practice, consideration should be given to screening before 22 weeks' gestation. If results are positive for BV, treatment with 2% clindamycin vaginal cream nightly for 6 days is consistent with the treatment regimen in the Austrian trial. In patients with recurrence, administering clindamycin 300 mg orally twice a day for 7 days should be considered.[52] Treatment with metronidazole is not recommended. Other infections such as trichomonas, chlamydia, and/or gonorrhea are also associated with increased risk of PTB, although evidence is inconclusive about whether treatment for these infections reduces the risk of PTB.[53]

Screening for asymptomatic bacteriuria. Pyelonephritis, symptomatic lower UTI, and asymptomatic bacteriuria have all been associated with increased risk of PTB.[53] Treatment of asymptomatic bacteriuria may reduce the risk of PTB (RR = 0.27; 95% CI = 0.11-0.62).[54]

Counseling

Motivational interviewing and shared decision making help patients choose healthy life habits and avoid those that promote PTB.

Tobacco smoking cessation. Discontinuing tobacco smoking has been shown to lower PTB.[55] Maternal cigarette smoking has a dose dependent association with PTB; smoking 10 to 20 cigarettes/day has a RR of 1.2 to 1.5, and smoking more than 20 cigarettes/day has a RR of 1.5 to 2.[56] A prospective study of 2,504 nulliparous women found the risk of PTB and small-for-gestational-age infants was similar for women who discontinued smoking before 15 weeks' gestation and those who did not smoke, emphasizing the importance of counseling and intervention for this modifiable risk factor.[57] Programs using rewards plus social support were found to be most effective.[58]

Lifestyle modifications. Avoidance of recreational drugs and illicit substances is advised.

Group prenatal visits have been promoted by the Centers for Disease Control and Prevention (CDC) to increase social support and decrease stress. A 2015 Cochrane review of four studies and 2,350 women receiving group versus conventional care noted no statistical significance in lowering PTB (RR = 0.75; 95% CI = 0.57-1).[59]

A short interpregnancy interval is associated with increased risk of PTB, and family planning counseling and long-acting reliable contraception can prolong this interval.[60,61]

Additional Considerations

Nongenitourinary infection, such as pneumonia and appendicitis, have also been associated with PTB. Periodontal infection doubles the risk of PTB; however, antenatal treatment does not appear to alter outcomes.[62] Whether cervical excisional procedures, such as cone biopsy or loop electrosurgical excision, increase the risk of PTB remains a subject of debate. A recent meta-analysis found that women with a prior loop electrosurgical excision have a similar risk of PTB as women with cervical dysplasia without an excisional

procedure, suggesting a shared underlying risk factor.[46,63] System-based interventions include using assisted reproduction techniques, which lower the risk of multiple gestations and elimination of elective deliveries before 39 weeks' gestation.

Assessment of Preterm Labor in the Symptomatic Patient

The diagnosis of PTL is based on the clinical criteria of regular uterine contractions with associated cervical change in dilation and/or effacement between 20 and 36 6/7 weeks' gestation.[6] Early diagnosis of PTL allows the ability to transfer a laboring woman to a facility with a NICU, administer glucocorticoids, consider magnesium sulfate (MgSO4) for neuroprotection, and initiate prophylactic treatment for GBS. However, diagnosing PTL remains challenging. In one observational study of women hospitalized for threatened PTL, 38% went on to deliver during their first admission, but more than half of those who were discharged continued their pregnancy until term.[64]

The initial assessment of women presenting with signs or symptoms of threatened PTL includes obtaining an accurate history, physical examination, and assessment of fetal and maternal well-being. The initial assessment should include determining whether membranes are ruptured, if infection is present and stratifying risk of PTB (*Table 5*).[3] History should include confirming accurate dating of gestational age, history of PTB, PTB risk factors, and any evidence of pertinent coexisting conditions such as abruption or preeclampsia. The patient should be assessed for frequency of contractions, pelvic pressure, back pain, leakage of fluid, vaginal bleeding, and symptoms of infection. Physical examination should include a sterile speculum examination to assess for rupture of membranes (ROM) and obtaining vaginal swab samples to test for infection and a GBS swab sample, if not previously completed, as well as a fFN swab sample if desired. A digital sterile vaginal examination should be avoided until ROM and placenta previa have been ruled out. Fetal well-being should be evaluated with external fetal monitoring. Ultrasound can be used for assessment of fetal size, presentation, amniotic fluid, placental location, and CL if the operator is technically experienced.[3]

Table 5. Initial Assessment of Women With Preterm Labor

Questions	Assessment
Is the gestational age less than 37 weeks?	Verify dates using clinical history and ultrasonography
Is the patient in labor?	Observe for regular contractions accompanied by progressive dilation and cervical effacement
Are the membranes ruptured?	History of leaking fluid: observed leakage or pooling of fluid from cervical os on sterile speculum examination
	Positive nitrazine test result
	Arborization or ferning of fluid on microscopy
	Positive amniotic protein test result (eg, placental alpha microglobulin-1
	Ultrasound assessment shows low amniotic fluid
	Ultrasound-guided transabdominal instillation of indigo carmine dye into the amniotic sac, if available, shows dye outside of the amniotic sac
Is there an infection?	Evaluate for group B streptococcus carrier status, urinary tract infection, bacterial vaginosis, and sexually transmitted infections (trichomoniasis, gonorrhea, or chlamydia); treat as appropriate
What is the likelihood that the patient will deliver prematurely?	Negative fetal fibronectin test results and cervical length of at least 3 cm on ultrasonography have a low likelihood of delivery within 14 days

Adapted with permission from Sayres WG Jr. Preterm labor. Am Fam Physician. *2010;81(4):480, with additional information from Hassan SS, Romero R, Vidyadhari D, et al. PREGNANT Trial. Vaginal progesterone reduces the rate of preterm birth in women with a sonographic short cervix: a multicenter, randomized, double-blind, placebo-controlled trial.* Ultrasound Obstet Gynecol. *2011;38(1):18-31.*

Are the Membranes Ruptured?

A sterile speculum examination assists in evaluation of membrane integrity and facilitates collection of fluid for a fFN test. Direct observation of amniotic fluid leaking from the cervical os and pooling in the vaginal vault is diagnostic of ROM. Gentle fundal pressure or having the patient cough during the examination may facilitate leakage.

Amniotic fluid, when allowed to air dry on a slide, will demonstrate *fern*ing or arborization on microscopic evaluation. A false-positive test result for fern ing is possible if cervical mucus or water-based lubricant is inadvertently tested. The normal vaginal environment has a pH of 4.5 to 6.0, whereas the pH of amniotic fluid is 7.1 to 7.3. Amniotic fluid will therefore change the color of nitrazine paper from yellow to blue.[65] A false-positive nitrazine test is possible if blood, semen, or BV is present.[65]

In equivocal cases, ROM may be diagnosed with a test for placental alpha microglobulin-1 protein in cervicovaginal fluid. In a large meta-analysis, the test for placental alpha macroglobulin-1 was 96% sensitive and 98.9% specific for ROM.[66] In some cases of prolonged ROM, minimal fluid may be present for analysis making diagnosis difficult. Oligohydramnios on ultrasound supports the diagnosis, but is not diagnostic, nor does a normal amniotic fluid level rule out ROM. If the diagnosis remains uncertain, ultrasound-guided transabdominal instillation of indigo carmine into the amniotic fluid may be used. The passage of blue-dyed fluid into the vagina, staining a Papanicolaou test or tampon, is diagnostic of ROM.[65] It should be noted that this technique is rarely used and is a risk for ROM if membranes are still intact.

Is Infection Present?

A high index of suspicion for infection should be maintained when evaluating a woman with preterm contractions. UTI testing should be obtained with an initial urinalysis and subsequent urine culture as indicated. Vaginal swab samples for yeast, BV, trichomonas, gonorrhea, and chlamydia should be considered. Subclinical chorioamnionitis, infection without the classic findings of fever, uterine tenderness, foul-smelling discharge, and maternal tachycardia, may be present.[67] A vaginal-rectal swab sample should be obtained to test for GBS. If delivery appears likely and GBS culture is unavailable or positive, GBS prophylaxis should be administered.[68]

What is the Likelihood of Preterm Birth?

Correctly identifying women with symptoms of PTL who are at high risk of PTB allows for appropriate interventions and treatment. It also allows women who are at low risk of PTB to avoid overtreatment. Unfortunately, accurately predicting PTB remains challenging despite the availability of various management algorithms and guidelines.

Preterm birth is more likely with six or more contractions/hour, cervical dilation of 3 cm or greater, cervical effacement of 80% or more, vaginal bleeding, or ROM.[69] Although frequency of uterine contractions is significantly related to risk of PTB, contraction frequency alone is not sensitive and has a low positive predictive value for PTB.[70] Assessment of the cervix via digital examination is subjective and also has a low predictive value for PTB.[71] Women with an initial equivocal examination should undergo further assessment, which can include TVU of CL, repeat fFN test, or a combination of these. The evaluation obtained for PTL is dependent on clinical resources available and local practice patterns.[72] It should be noted that the positive predictive value of a positive fFN test result or a short cervix alone is poor and should not be used exclusively to direct management in the setting of acute symptoms.[6]

Fetal fibronectin testing may be obtained between 24 and 34 weeks' gestation and is most useful to identify women who are at low risk of delivery in the following 10 to 14 days. The test should not be performed when there is active vaginal bleeding or when intercourse, digital vaginal examination, or endovaginal ultrasound has occurred in the preceding 24 hours because these can yield a false positive test result.[73] More recently, quantitative fFN has been studied and may prove to be more useful in predicting PTB in symptomatic patients, including its use in combination with CL.[74]

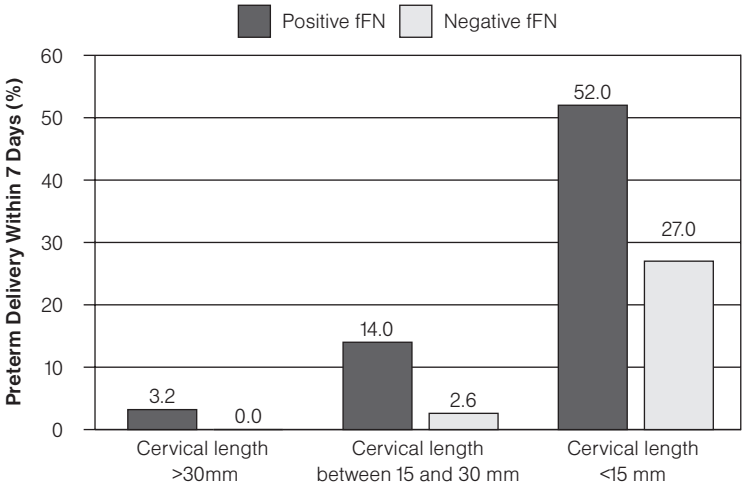
The presence of a shortened CL has been associated with PTB, although the utility of CL measurement by ultrasound in assessing threatened PTL is not as clear. A retrospective study of 1,077 women presenting with preterm contractions found that although cervical shortening was associated with increased risk of delivery in the following 7 to 14 days, the overall accuracy of CL alone to predict PTB was poor.[75] Another prospective study of 665 women combined CL

and fFN testing to risk stratify women presenting for PTL. This study found that women with CL of at least 3 cm had a low risk of delivery within 7 days (0.7%), regardless of the fFN result. Women with a CL less than 1.5 cm had a high risk of delivery within 7 days (27% if fFN was negative, and 52% if fFN was positive). For women with an intermediate CL between 1.5 cm and 3 cm, a negative fFN result correctly identified women at low risk (less than 5%) of delivery within 7 days (*Figure 1*).[76] These results are consistent with results from a similar study conducted in 2005.[77] By using tests such as fFN and CL measurements, which have high negative predictive values, it may be possible to identify women at low risk of PTB. However, there is no test for early PTL with a high positive predictive value, and making an accurate diagnosis of PTL remains a challenge.

Management of Preterm Labor

After PTL has been diagnosed, several interventions should be undertaken to improve neonatal outcomes. These interventions include maternal transfer to a facility with a higher-level nursery if indicated, administration of corticosteroids, antibiotic prophylaxis of neonatal GBS infection, consideration of MgSO4 for neuroprotection, and preparation for PTB.

Figure 1. Risk of Preterm Delivery Within 7 Days Using Fetal Fibronectin and Cervical Length for Risk Stratification



Information from van Baaren GJ, Vis JY, Wilms FF, et al. Predictive Value of cervical Length Measurement and Fibronectin Testing in Threatened Preterm Labor. Obstet Gynecol. *2014;123(6):1185-1192.*

Table 6. Antenatal Corticosteroid Effects on Fetal Outcomes in Preterm Labor at 24 to 33 6/7 Weeks' Gestation

Outcome	RR (95% CI)
Neonatal mortality	0.69 (0.59-0.81)
Need for mechanical ventilation	0.68 (0.56-0.84)
Respiratory distress syndrome	0.66 (0.56-0.77)
Systemic infections in the first 48 hours of life	0.60 (0.41-0.88)
Intraventricular hemorrhage	0.55 (0.40-0.76)
Necrotizing enterocolitis	0.50 (0.32-0.78)

ACS = antenatal corticosteroids; CI = confidence interval; PTL = preterm labor; RR = relative risk.

Information from Roberts D, Dalziel S. Antenatal corticosteroids for accelerating fetal lung maturation for women at risk of preterm birth. Cochrane Database Syst Rev. *2017;(3):CD004454.*

Antenatal Corticosteroid Treatment

Treatment with ACSs decreases neonatal mortality and morbidity as described in *Table 6*. A single course of an ACS is recommended for women between 24 and 33 6/7 weeks' gestation who are at risk of PTB within 7 days.[78] ACSs may be considered for women between 23 and 24 weeks' gestation based on the family's decision regarding resuscitation (see the section on infants at the threshold of viability).[6,78] The optimal benefits of ACSs appear greatest at 2 to 7 days after the initial dose and therefore should not be administered unless there is substantial clinical concern for imminent PTB.[79] A single repeat course of ACSs administered to women who are less than 34 weeks' gestation improves respiratory distress syndrome by 17% (number needed to treat [NNT] = 17) and poor infant outcomes by 16% (NNT = 30) without affecting long-term outcomes when administered 7 or more days after the first ACS course.[79] ACOG recommends a single repeat dose of ACSs be considered in women who are less than 34 weeks' gestation who are at risk of delivery within 7 days and who have received the first ACS course at least 14 days prior. A rescue course could be considered as early as 7 days after the first dose, if indicated by the clinical scenario.[6,78] Treatment with ACSs does not increase the risk of maternal mortality, chorioamnionitis, or puerperal sepsis.[5,6]

Table 7. Antenatal Corticosteroids for Fetal Maturation

Corticosteroid	Dosage
Betamethasone	Two doses of 12 mg IM administered 24 hours apart
Dexamethasone	Four doses of 6 mg IM administered every 6 hours

IM = intramuscularly.

Information from Clinical Pharmacology. Available at www.clinicalpharmacology.com.

Treatment with ACSs has not shown benefit for chronic lung disease, mean birthweight, death in childhood, neurodevelopmental delay in childhood, or death in adulthood.[5]

A single course of betamethasone can be considered in women between 34 to 36 6/7 weeks' gestation who are at risk of PTB within 7 days and have not received a previous course of ACSs.[78] A 2016 multicenter randomized controlled trial (RCT) noted that betamethasone reduced neonatal respiratory complications (RR = 0.80; 95% CI = 0.66-0.97; *P* = 0.03) when administered to women with threatened PTL (at least 3 cm dilated or 75% effaced), ROM, or indicated planned late PTB between 34 and 36 6/7 weeks' gestation.[80] Neonatal hypoglycemia was noted to be more common in the betamethasone group than placebo group (24% versus 15%; RR = 1.60; 95% CI = 1.37-1.87; *P* <0.001).[80]

Corticosteroid dosages are listed in *Table 7*. In general, dexamethasone and betamethasone show similar results in respiratory distress and perinatal mortality, but one meta-analysis showed a decrease in intraventricular hemorrhage and length of NICU stay with use of dexamethasone compared with betamethasone.[81] There is evidence that even a single dose of either drug is beneficial, so it is recommended that a first dose be administered even if it is unlikely that the patient will receive subsequent doses. However, there is no evidence of improved outcomes with accelerated dosing (ie, shortening the dosage interval to complete the course before an imminent delivery).[6,81]

Magnesium Sulfate for Neuroprotection

Magnesium sulfate administered immediately before and at the time of delivery of a preterm infant decreases the rate of cerebral palsy (RR = 0.68; 95% CI = 0.54-0.87; NNT = 63).[82] Various trials have shown this clear benefit but have varied regarding the regimen used in MgSO4 administration. A 2012 Cochrane review did not show superiority of any regimen over another and called for further studies to define the optimal dosing.[83,84] Two commonly used regimens are listed in *Table 8* and are applicable to women between 24 and 32 weeks' gestation at high risk of delivery within 24 hours.[83,85]

Tocolysis

Tocolytic drugs are used for short-term pregnancy prolongation (up to 48 hours), with the goal of allowing time for administration of ACS, MgSO4 for neuroprotection, antibiotics for GBS prophylaxis, and maternal transfer if necessary. They may be useful in pregnancies between viability and 34 weeks estimated gestational age with established PTL, and in the absence of evidence of

Table 8. Magnesium Sulfate for Fetal Neuroprotection

Magnesium Sulfate Loading Dose	Maintenance Dose	Repeat Treatment
4 g over 20 minutes	1 g/hour continued until birth or for 24 hours	No immediate repeat doses
6 g over 20 minutes	2 g/hour continued until birth or for 12 hours	If less than 6 hours have elapsed since discontinuation of infusion, restart maintenance dose. If more than 6 hours have elapsed, rebolus and start maintenance dose

Information from Reeves SA, Gibbs RS, Clark SL. Magnesium for fetal neuroprotection. Am J Obstet Gynecol. *2011;204(3):202.e1-202.e4; Magee L, Sawchuck D, Synnes A, von Dadelszen P. SOGC Clinical Practice Guideline. Magnesium sulphate for fetal neuroprotection.* J Obstet Gynaecol Can. *2011; 33(5):516-529.*

maternal or fetal compromise (ie, chorioamnionitis, severe preeclampsia, maternal instability, fetal demise, lethal anomaly, worrisome fetal status). After cessation of labor, there is no evidence of benefit of long-term tocolysis for further prolongation of pregnancy.[6] First-line tocolytic drugs include calcium channel blockers (eg, nifedipine), beta-adrenergic receptor agonists (eg, terbutaline), and prostaglandin inhibitors/nonsteroidal anti-inflammatory drugs (NSAIDs) (eg, indomethacin).[6] Pharmaceutical treatment options are listed in *Table 9*.

Using a single tocolytic drug rather than a combination of two drugs has traditionally been recommended to decrease the risk of adverse effects; however, a 2019 RCT comparing com-

Table 9. Pharmaceutical Agents for Tocolysis

Drug (Class)	Dosage	Comments	Contraindications and Adverse Effects
Nifedipine (calcium channel blocker)	30 mg oral loading dose, then 10 to 20 mg every 4 to 6 hours	Nifedipine decreases the incidence of delivery before 48 hours. Neonatal mortality not affected. Decreased incidence of neonatal respiratory distress syndrome, necrotizing enterocolitis, intraventricular hemorrhage, and jaundice	Contraindication: maternal hypotension Maternal adverse effects: flushing, headache, dizziness, nausea, transient hypotension No fetal adverse effects noted
Terbutaline (betamimetic)	0.25 mg subcutaneously every 20 minutes for up to three doses	Betamimetic drugs may delay delivery for 48 hours, but neonatal outcomes are variable and maternal adverse effects are common Injectable terbutaline may have a narrow role in treatment of uterine tachysystole or in emergent situations FDA warns against long-term or oral use because of maternal adverse events and lack of efficacy	Maternal contraindications: heart disease, poorly controlled diabetes, thyrotoxicosis Maternal adverse effects: Cardiac arrhythmias, pulmonary edema, myocardial ischemia, hypotension, tachycardia Hyperglycemia, hyperinsulinemia, hypokalemia, antidiuresis, altered thyroid function Physiologic tremor, palpitations, nervousness, nausea/vomiting, fever, hallucinations Fetal and neonatal adverse effects: tachycardia, hypoglycemia, hypocalcemia, hyperbilirubinemia, hypotension, intraventricular hemorrhage
Indomethacin (NSAID)	Loading dose: 50 mg rectally or 50 to 100 mg orally Maintenance dose: 25 to 50 mg orally every 4 hours for 48 hours	NSAIDs theoretically intervene more proximally in the labor *cascade* than the other agents. Efficacy appears similar to other agents Maternal adverse-effect profile is favorable Other NSAIDs (sulindac, ketorolac) may be used	Contraindications: maternal renal or hepatic impairment, active peptic ulcer disease, oligohydramnios Maternal adverse effects: nausea, heartburn Fetal adverse effects: constriction of the ductus arteriosus (not recommended after 32 weeks' gestation), pulmonary hypertension, reversible decrease in renal function with oligohydramnios, intraventricular hemorrhage, hyperbilirubinemia, necrotizing enterocolitis
Magnesium Sulfate	4 to 6 g bolus over 20 minutes, then 1 to 2 g/hour (3 g/hour maximum)	In widespread use in the United States, meta-analysis fails to show improvement in outcomes. Comparison studies show similar effectiveness to other agents in delay of delivery May be used for fetal neuroprotection at less than 32 weeks' gestation. Dosing regimens vary; use hospital protocols	Contraindication: myasthenia gravis Maternal adverse effects: flushing, lethargy, headache, muscle weakness, diplopia, dry mouth, pulmonary edema, cardiac arrest. Toxicity rare with serum level <10 mg/dL. Respiratory depression and subsequent arrest can occur at levels >10 to 12 mg/dL Newborn adverse effects: lethargy, hypotonia, respiratory depression, demineralization with prolonged use

FDA = Food and Drug Administration; NSAID = nonsteroidal anti-inflammatory drug.

Information from Clinical Pharmacology. Available at www.clinicalpharmacology.com.

bination therapy and treatment with individual drugs in women with PTL showed administration of nifedipine and indomethacin in combination was more effective in discontinuing contractions within 2 hours and resulted in increased gestational age at delivery and likelihood of attaining 37 weeks estimated gestational age.[86] Additional studies are needed before routine use of multiple tocolytics can be recommended.

Calcium channel blockers. Nifedipine decreases the likelihood of delivering within 48 hours (RR = 0.30; 95% CI = 0.21-0.43). Nifedipine may have advantages over other tocolytics. Compared with betamimetics, nifedipine increased time before delivery, decreased maternal adverse events, and improved neonatal outcomes (very PTB, respiratory distress syndrome, necrotizing enterocolitis, intraventricular hemorrhage, neonatal jaundice, and NICU admissions). Compared with MgSO4, nifedipine showed decreased maternal adverse events and decreased NICU admissions.[87]

Beta-adrenergic receptor agonists/Betamimetics. Betamimetics (eg, terbutaline, ritodrine) are effective in delaying delivery for 48 hours.[88] Studies have not shown an improvement in measures of fetal outcomes, but maternal adverse effects and adverse events are significant.[88] Terbutaline is the most commonly used betamimetic for tocolysis. In 2011, the Food and Drug Administration issued a warning against the use of terbutaline for tocolysis longer than 48 to 72 hours because of the lack of demonstrated efficacy and the potential for serious maternal cardiac complications and mortality.[89] Injectable terbutaline may have a narrow role for tocolysis in emergent settings or with uterine tachysystole.[6]

Prostaglandin inhibitors/Nonsteroidal anti-inflammatory drugs. Indomethacin increases the likelihood of delivery at greater than 37 weeks' gestation and average gestational age at delivery (weighted mean difference of 3.53 weeks) with low maternal adverse effects.[90] Because NSAIDs can interfere with fetal prostaglandin synthesis, concerns have been raised regarding fetal safety. Meta-analyses of observational studies have shown conflicting results regarding fetal safety (mostly no effects), but have raised concerns about severe intraventricular hemorrhage, necrotizing enterocolitis, and periventricular leukomalacia.[91-93] More research and RCTs are needed in this area. According to one decision analysis, prostaglandin inhibitors may be the optimal first-line treatment for PTL before 32 weeks' gestation.[94] Because of the risk of premature closure of the ductus arteriosus, NSAIDs should not be used for more than 48 hours or beyond 32 weeks' gestation.

Magnesium sulfate use for tocolysis has not been shown to prolong pregnancy or improve neonatal outcomes compared with placebo or other tocolytics.[95] In addition, a Cochrane meta-analysis showed an increased risk of fetal and neonatal mortality of borderline significance (RR = 4.56; 95% CI = 1-20.86) and an increase in length of NICU admission compared with calcium channel blockers (but not in comparison to NSAIDs).[95,96]

In women receiving MgSO4 for neuroprotection who continue to labor after administration, consideration may be given to adding another tocolytic. However, a Cochrane meta-analysis was unable to reach any conclusions regarding combinations of tocolytics regarding safety and efficacy, citing a lack of large well-designed trials.[97] Caution should be used if administering calcium channel blockers and MgSO4 because of theoretical maternal cardiac complications.[6]

Neonatal Group B Streptococcus Prophylaxis

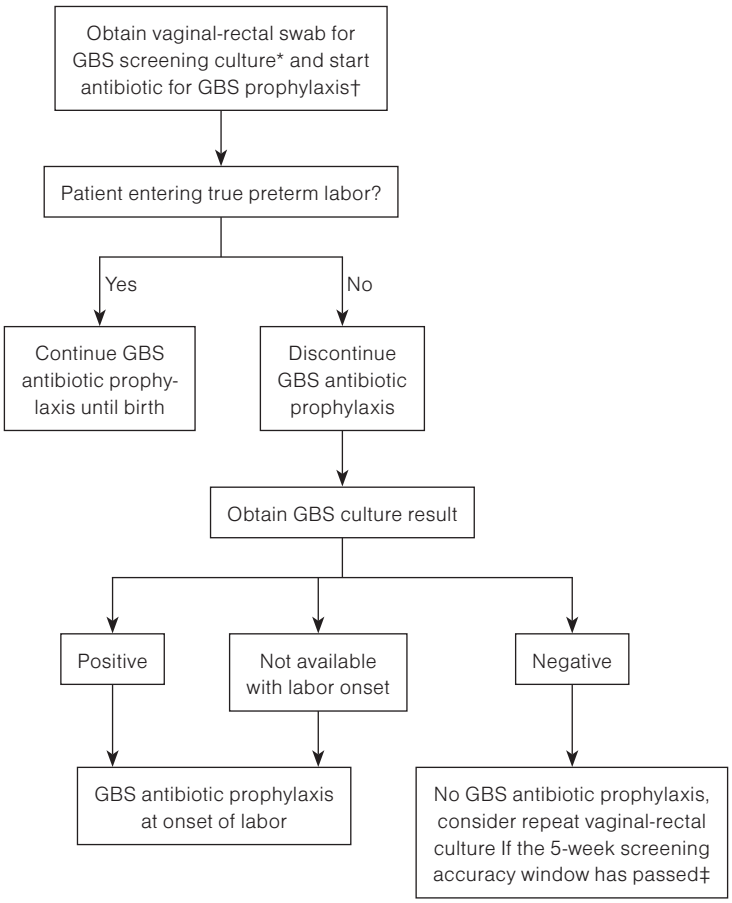
The incidence of neonatal GBS infection and mortality has dropped significantly since the wide adoption of the CDC guidelines for the prevention of neonatal GBS disease. However, GBS remains the leading cause of mortality due to infection among neonates.[68] Several large retrospective record reviews show gaps in adherence to current recommendations, especially in PTL, PPROM, and the use of clindamycin as opposed to cefazolin in patients who are allergic to penicillin.[98,99]

A vaginal-rectal swab for GBS culture should be obtained when women present with PTL or PPROM if results from prior testing obtained within the last 5 weeks are not available. Intrapartum antibiotics (penicillin or ampicillin) should be started on admission and continued until birth, or until it is determined that the woman is not in true PTL or a negative GBS culture result becomes available. In cases of PPROM when antibiotics are used for prolonging latency, antibiotic coverage should include coverage for GBS.

Women with a penicillin allergy should receive cefazolin unless the allergic response was due to

anaphylaxis, angioedema, respiratory distress, or urticaria.[68] Clindamycin and vancomycin are the antibiotics of last resort for women with life-threatening penicillin allergies. GBS isolates show increasing resistance to clindamycin, and clindamycin has poor penetration into amniotic fluid.[98] It should be used only when the GBS isolate can be shown to be sensitive to clindamycin and erythromycin. Vancomycin with dosing based on maternal weight should be used in women with the serious penicillin allergies described and unknown GBS status, or GBS strains resistant to clindamycin. CDC algorithms for screening and GBS prophylaxis treatment of women with threatened PTB are presented in *Figures 2* and *3.*

Figure 2. Management of Women With Preterm Labor <37 0/7 Weeks of Gestation



Abbreviation: GBS, group B streptococcus.

**If a patient has undergone vaginal-rectal GBS screening culture within the preceding 5 weeks, the results of that culture should guide management. Women colonized with GBS should receive Intrapartum antibiotic prophylaxis. Although a negative GBS culture is considered valid for 5 weeks, the number of weeks Is based on early-term screening and data In preterm gestations is lacking.*

†See Figure 3 for recommended antibiotic regimens.

‡A negative GBS culture Is considered valid for 5 weeks. However, the number of weeks is based on early-term screening and data In preterm gestations Is lacking. If a patient with preterm labor is entering true labor and had a negative GBS culture more than 5 weeks previously, she should be rescreened and treated according to this algorithm at that time.

Reprinted from American College of Obstetricians and Gynecologists. ACOG Committee Opinion No. 782. Prevention of group B streptococcal early-onset disease in newborns. Obstet Gynecol. *2019;134:e19–40.*

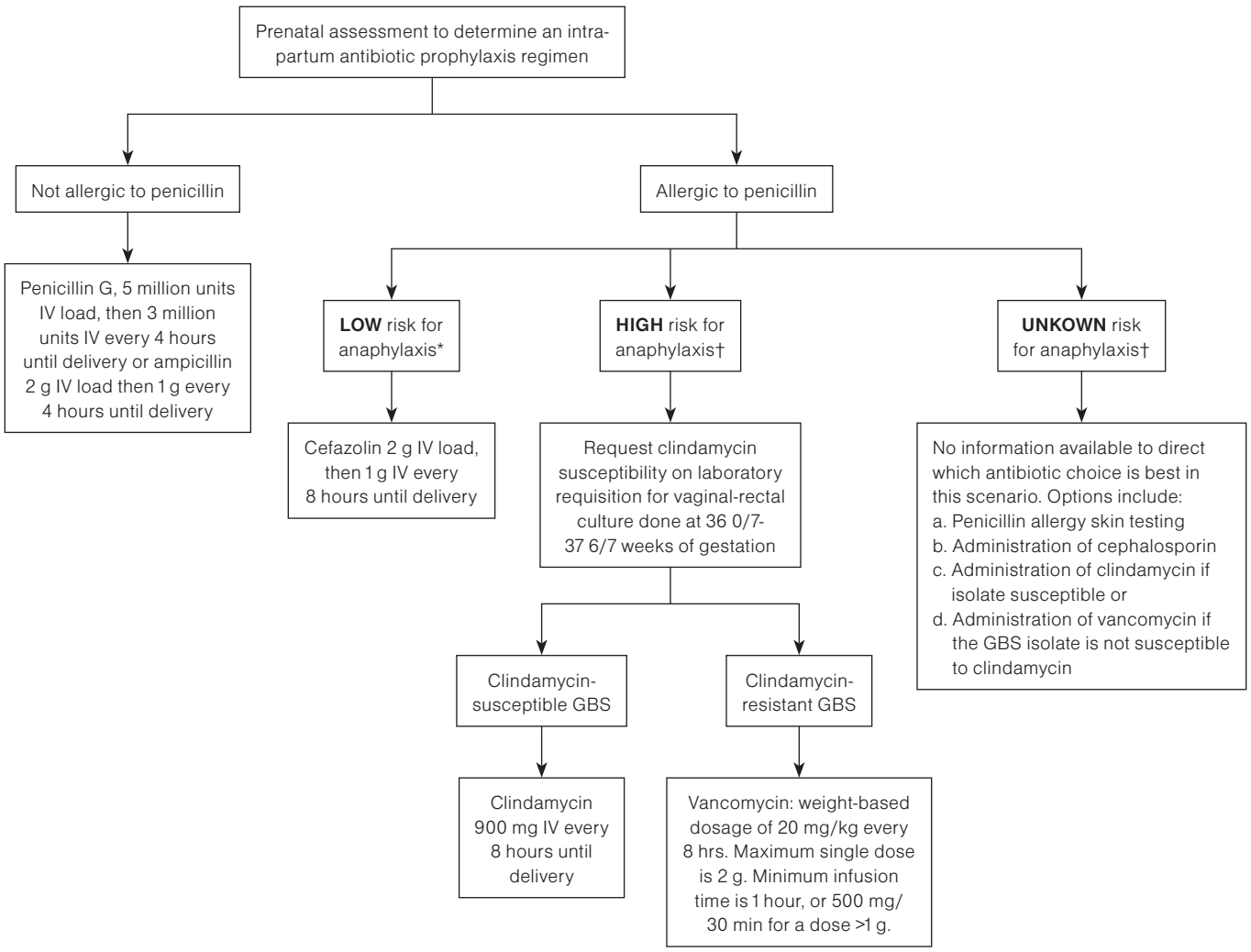
In 2019, ACOG released updated clinical recommendations.[100] These include obtaining routine vaginal-rectal swab samples at 36 to 36 6/7 weeks' gestation rather than starting at 35 weeks' gestation as previously recommended. Maternal vancomycin dosing should now be weight-based using 20 mg/kg intravenously (IV) every 8 hours with a maximum of 2 g for each dose. Women presenting in labor with unknown GBS status should be offered the option of starting GBS prophylaxis if they had a positive maternal GBS culture result, in a previous pregnancy. A shared decision making process is appropriate in this scenario.

Preterm Prelabor Rupture of Membranes

Preterm prelabor rupture of membranes, previously referred to as preterm premature rupture of membranes, precedes 25% to 30% of PTBs.[11] The pathophysiology leading to PPROM is multifactorial and appears to be different from those leading to PTL. Risk factors for PROM are similar to those for PTL with intact membranes.[7] The earlier in pregnancy PPROM occurs, the more likely its etiology is associated with infection.[65] Delivery is likely within a week; however, the earlier in pregnancy the rupture occurs, the greater the potential latency period.[101] Clinically evident intra-amniotic infection will develop in 13% to 60% of cases, the likelihood of which is increased by digital vaginal examination.[65] The most significant risks to the fetus after PPROM are complications of prematurity. Intrauterine complications include umbilical cord compression, abruption of the placenta, infection, and pulmonary developmental abnormalities.[102] Infection may lead to maternal morbidity and likely has a role in initiation of labor.

The initial clinical examination and evaluation of a patient with suspected PPROM is similar to that of PTL and includes a sterile speculum examination with confirmation of ROM, screening for infection, and ongoing assessment of maternal

Figure 3. Determination of Antibiotic Regimen for Group B Streptococcus Prophylaxis in Labor



Abbreviations: GBS, group B streptococcus; IV, intravenous.

**Individuals with a history of any of the following: a non-urticarial maculopapular (morbilliform) rash without systemic symptoms; family history of penicillin allergy but no personal history; nonspecific symptoms such as nausea, diarrhea, yeast vaginitis; patient reports history but has no recollection of symptoms or treatment.*

†Individuals with a history of any of the following after administration of a penicillin: urticarial rash (hives), intense pruritis, anaphylaxis, angioedema, laryngeal edema, respiratory distress, hypotension, immediate flushing or rare delayed reactions, such as eosinophilia and systemic symptoms/drug-induced hypersensitivity syndrome, Stevens-Johnson syndrome, or toxic epidermal necrolysis. Individuals with recurrent reactions, reactions to multiple beta-lactam antibiotics, or those with positive skin testing also are considered high risk.

Reprinted from American College of Obstetricians and Gynecologists. ACOG Committee Opinion No. 782. Prevention of group B streptococcal early-onset disease in newborns. Obstet Gynecol. *2019;134:e19–40.*

and fetal well-being. fFN and CL, however, are typically not assessed.

Management of Preterm Prelabor Rupture of Membranes

As in the case of PTL with intact membranes, the management of PPROM necessitates a balance between the advantages of delaying delivery and the risks of potential complications associated with continuation of pregnancy. Initial management includes transfer to a higher-level NICU as deemed appropriate, ACSs for fetal lung maturity, MgSO4 for neuroprotection, GBS prophylaxis as indicated, and antibiotics to prolong latency (time from ROM to delivery). Specific recommendations based on gestational age are discussed in more detail

below. Patients should receive ongoing monitoring for signs of infection and complications including abruption, cord prolapse, and malpresentation.

In general, tocolysis is not recommended in PPROM as it has not been associated with maternal or neonatal benefits.[103] Antepartum fetal surveillance recommendations are based primarily on expert opinion and local practice. The nonstress test and biophysical profile have been used to assess for fetal compromise.[7,104]

Antenatal Corticosteroid Treatment

Antenatal corticosteroid administration in the setting of PPROM reduces the risk of neonatal respiratory distress syndrome, intraventricular hemorrhage, and necrotizing enterocolitis without an increase in the incidence of maternal and neonatal infection.[5,105] In women with PPROM between 24 and 33 6/7 weeks' gestation, a single course of ACSs is recommended.[78] It may also be considered for pregnant women starting at 23 weeks' gestation (see additional discussion in the section on infants at the threshold of viability). There is insufficient evidence to recommend for or against administering a repeat or rescue course of ACS with PPROM.[78] As previously discussed, a single course of betamethasone can be considered for women between 34 to 36 6/7 weeks' gestation at risk of PTB within 7 days who have not received a previous course of ACSs.[78]

Magnesium Sulfate for Neuroprotection

Women with PPROM before 32 weeks' gestation who are at risk of imminent delivery should be considered candidates for MgSO4 for fetal neuroprotection.[65] Additional details regarding MgSO4 for neuroprotection were discussed previously in this chapter.

Antibiotic Therapy

Administration of antibiotics in women with PPROM is associated with prolongation of pregnancy, decreased fetal and maternal infections, and decreased fetal morbidity.[106] The optimal antibiotic regimen for PPROM is unknown. In women with PPROM, who are less than 34 weeks' gestation, a 7-day course of antibiotics, including IV ampicillin and erythromycin for 48 hours followed by a 5-day course of oral amoxicillin and erythromycin, is typically recommended.[65,107] A 1-g oral dose of azithromycin has been substituted in some protocols for erythromycin with no apparent change in outcomes.[108,109] The antibiotics and dosages used in a large NICHD trial are listed in *Table 10*.[107] The use of amoxicillin-clavulanic acid has been associated with increased rates of necrotizing enterocolitis and should not be used.[65,106]

Gestational Age and Management of Preterm Prelabor Rupture of Membranes

34 to 36 6/7 weeks' gestation. If GBS status is unknown, start antibiotics pending results. Consider a single course of ACSs with betamethasone as previously discussed. A 2016 RCT showed that expectant management of PPROM between 34 and 36 6/7 weeks' gestation resulted in significantly lower rates of respiratory distress, mechanical ventilation, days spent in NICU, and cesarean delivery but significantly higher rates of antepartum or intrapartum hemorrhage, intrapartum fever, use of postpartum antibiotics, and longer hospital stay compared with immediate delivery.[110] The authors concluded that offering expectant management for women with late PPROM is reasonable.[110] A 2017 Cochrane review showed improved maternal and infant outcomes in expectant management in pregnancies greater than 34 weeks' gestation, specifically relating to respiratory

Table 10. Antibiotic Therapy in Preterm Prelabor Rupture of Membranes

Antibiotic	Dosage
Initial Therapy	
Ampicillin	2 g IV every 6 hours for 48 hours
Erythromycin	250 mg IV every 6 hours for 48 hours
Follow-Up Therapy	
Amoxicillin	250 mg orally every 8 hours for 5 days
Erythromycin	333 mg orally every 8 hours for 5 days

IV = intravenously.

Information from Mercer BM, Miodovnik M, Thurnau GR, et al; National Institute of Child Health and Human Development Maternal-Fetal Medicine Units Network. Antibiotic therapy for reduction of infant morbidity after preterm premature rupture of the membranes. A randomized controlled trial. JAMA. 1997;278(12):989-995.

distress syndrome and maternal infections.[111] A 2018 ACOG Practice Bulletin notes the optimal gestational age for delivery after PROM remains controversial and currently recommends delivery rather than expectant management for all women with ROM at 34 weeks' gestation or greater.[65] If expectant management is continued beyond 34 weeks' gestation in women with PROM, the balance between risks and benefits should be carefully considered and discussed with the patient.[65]

24 to 33 6/7 weeks' gestation. Administer antibiotics and corticosteroids. Add MgSO4 for neuroprotection when in labor if less than 32 weeks gestational age. Monitor for infection and other intrauterine fetal complications. If there is no evidence of fetal compromise and labor does not begin spontaneously, these pregnancies are managed expectantly until they reach 34 weeks' gestation.

23 to 23 6/7 weeks' gestation. Consider administering antibiotics and corticosteroids based on parental choice after counseling with neonatology and maternal-fetal medicine clinicians regarding outcomes of interventions, including cesarean delivery and resuscitation, at periviable gestational age. If resuscitation is planned, add MgSO4 for neuroprotection when in labor.[65]

Delivery of the Preterm Infant

Location of Delivery

Premature delivery at a facility with an appropriate-level NICU results in better neonatal outcomes. If delivery is not imminent and appropriate NICU services are unavailable, then maternal transfer is indicated.[112,113] A discussion with the receiving hospital should include recommendations for drugs to be administered prior to transport including ACSs, antibiotics, tocolysis, and MgSO4.

Timing of Delivery

Timing of delivery is based on considerations as previously discussed for PTL and PPROM. Ideally appropriate interventions and maternal transport to an appropriate level of care can be achieved before delivery. However, if delivery is imminent or urgently indicated, plans should be in place for neonatal resuscitation and transport to the appropriate level of care.

Route of Delivery

The choice of vaginal or cesarean delivery should be made based on standard obstetric indications. The rate of cesarean delivery is higher for preterm than for term deliveries, because the indications for surgery are more common in prematurity. Preterm fetuses are more likely to have a malpresentation and are less able to handle the potential stresses of labor. Continuous fetal monitoring is important to detect signs of fetal intolerance of labor. Neither prophylactic episiotomy nor forceps delivery has been shown to benefit the preterm fetus. Vacuum-assisted delivery should not be performed at less than 34 weeks' gestation, because of the risk of intracranial hemorrhage.[114,115]

Delayed Umbilical Cord Clamping

For the preterm infant, delayed clamping of the umbilical cord has been shown to decrease intraventricular hemorrhage, necrotizing enterocolitis and the need for neonatal transfusion.[116,117] Delayed cord clamping does not increase the risk of postpartum hemorrhage.[117] Given the benefits to most newborns, umbilical cord clamping should be delayed in vigorous term and preterm infants for at least 30 to 60 seconds after birth.[117] Required neonatal resuscitation efforts should not be delayed to allow for delayed cord clamping.

Additional Considerations

Infants at the threshold of viability. Periviable birth has been defined as infants born at 20 to 25 6/7 weeks' gestation.[118] These infants are at high risk of neonatal mortality and severe long-term disability. However, the difference in maturity of even a few days can significantly affect these risks. Parents of these infants should receive careful counseling and accurate information to assist them in making decisions regarding tocolysis, labor management, resuscitation, and NICU interventions.[112] The NICHD Neonatal Research Network has developed a web-based tool to estimate outcomes among live-born infants at 22 to 25 weeks' gestation that is available at https://neonatal.rti.org.[119] This tool was developed from outcomes information prospectively collected from 1997 to 2003 based on gestational age, weight, sex, and exposure to ACSs. Additional outcomes data has subsequently been reported in a prospective registry of infants at 22 to 28 weeks' gestation between 1993 and 2012. This registry noted a modest reduction in several morbidities, although bronchopulmonary dysplasia increased.[120] Survival increased most markedly for infants born between

23 to 24 weeks' gestation, and survival without major morbidity increased for infants between 25 to 28 weeks' gestation.[120]

Best practice recommendations and general guidance for obstetric interventions for threatened and imminent periviable birth are also available in a joint Obstetric Care Consensus statement on periviable birth from ACOG and SMFM.[119] Recommendations include antepartum transport to an appropriate level of care, family counseling provided by a multidisciplinary team (eg, obstetrics and maternal-fetal medicine providers, neonatologist) and establishing a predelivery plan with the patient and her family. Providers and patients should understand that the response of an individual neonate to resuscitation can never be known with certainty.[119] Ultimately, the decisions regarding interventions during the periviable period are complex and involve ongoing shared decision making with the patient and her family.

Term prelabor rupture of membranes. ROM before the onset of labor occurs in 8% of pregnancies beyond 37 weeks' gestation. In most women, PROM at term is generally followed by the prompt onset of spontaneous labor and delivery.[65,121] The initial evaluation of a patient suspected of term PROM is the same as PPROM and should include an assessment of fetal well-being, confirmation of fetal position, and evaluation for any signs or symptoms of infection. Digital cervical examination is typically avoided as it has been associated with an increased risk of infection.

Maternal and neonatal infection are the primary concerns when term PROM is not followed quickly by spontaneous labor. In general, labor induction is recommended when term PROM occurs and labor does not follow.[65,122] However, the differences in maternal and neonatal outcomes are minimal for induction versus expectant management. If the woman and fetus are well, expectant management of term PROM may be acceptable with appropriate counseling regarding potential risk.[122] In a 2017 systematic review, planned induction, compared with expectant management, showed a reduction in chorioamnionitis and endometritis without an increase in the risk of operative delivery.[122] Planned induction also appeared to decrease NICU admissions but had no significant effect on the number of neonatal infections.[122] The authors judged the quality of the trials and evidence to be low. Women with GBS colonization should receive antibiotic prophylaxis and be encouraged to proceed with induction to reduce the risk of neonatal GBS infection.[65,123] Routine administration of antibiotics in the absence of GBS infection does not appear to reduce neonatal sepsis, maternal infections, stillbirth, or neonatal mortality.[124] In one trial, women were noted to prefer induction to expectant management.[121]

Global Perspective

Low-income countries often do not have the resources needed for the prevention and management of PTL. Infants born at 32 weeks' gestation, who may have survived in other parts of the world, may die of respiratory failure because ventilatory support and surfactant are unavailable, and infrastructure for rapid transport to a tertiary care facility is lacking. Complex social and medical infrastructure necessary for technology-based care and follow-up of preterm neonates may not exist in these settings. In 2012, a coalition of experts released *Born Too Soon: The Global Action Report on Preterm Birth*. The report lists the first country-level estimates of PTB and revealed that there are approximately 15 million PTBs/year[125] with over 85% occurring in Asia and Africa.[126] In 2014, the stakeholders who created that report also released *Every Newborn: An Action Plan to Avoid Preventable Deaths*.[127] The components of the action plan for PTB included worldwide access to newborn resuscitation, low-cost corticosteroids,[128] and *kangaroo care* in which maternal skin-to-skin care is used in place of an incubator. Others have urged caution with introducing corticosteroids until there is evidence supporting benefit in low-resource settings.[129] An RCT on ACS treatment was conducted in six countries: Argentina, Guatemala, India, Kenya, Pakistan, and Zambia.[130] The study showed no benefit from ACS treatment and increased rates of maternal infection. Additional research is needed to clarify the role of ACS treatment in low-resource settings.

Conclusion

It is possible to identify some patients at high risk of PTB, and a subset of these may benefit from preventive interventions such as antenatal progesterone. In the triage of patients presenting with preterm contractions, it is possible to stratify their risk of subsequent PTB. The major role of tocoly-

sis is to delay delivery during the 48 hours necessary for full therapeutic effect of ACSs, MgSO4 for neuroprotection, GBS prophylaxis as indicated, and to allow maternal transport if needed. Management of PPROM presents its own set of challenges and is primarily based on gestational age. ACSs and antibiotics are useful in some cases. PTL remains an area of intense research activity and therapeutic evolution.

Nursing Considerations: Preterm Labor and Prelabor Rupture of Membranes

- Advocate for antenatal corticosteroids, magnesium sulfate for neuroprotection, group B streptococcus prophylaxis, tocolysis, and maternal transfer as appropriate
- For women with prelabor rupture of membranes, develop guidelines for frequent assessment of maternal vital signs, including temperature, and to limit cervical examinations
- If delivery is imminent, facilitate a neonatal intensive care unit consult prior to delivery for parents to ask questions and develop realistic expectations

References

1. Purisch SE, Gyamfi-Bannerman C. Epidemiology of preterm birth. *Semin Perinatol*. 2017;41(7):387-391.
2. Kazemier BM, Buijs PE, Mignini L, et al. EBM CONNECT. Impact of obstetric history on the risk of spontaneous preterm birth in singleton and multiple pregnancies: a systematic review. *BJOG*. 2014;121(10): 1197-1208, discussion 1209.
3. Dodd JM, Jones L, Flenady V, et al. Prenatal administration of progesterone for preventing preterm birth in women considered to be at risk of preterm birth. *Cochrane Database Syst Rev*. 2013;7(7):CD004947.
4. Sayres WG Jr. Preterm labor. *Am Fam Physician.* 2010;84(4):480.
5. Roberts D, Brown J, Medley N, Dalziel SR. Antenatal corticosteroids for accelerating fetal lung maturation for women at risk of preterm birth. *Cochrane Database Syst Rev*. 2017;3(3):CD004454.
6. American College of Obstetricians and Gynecologists' Committee on Practice Bulletins—Obstetrics. Practice Bulletin no. 171: management of preterm labor. *Obstet Gynecol*. 2016;128(4):e155-e164.
7. Simhan HN, Canavan TP. Preterm premature rupture of membranes: diagnosis, evaluation and management strategies. *BJOG*. 2005;112(Suppl 1):32-37.
8. Harrison MS, Goldenberg RL. Global burden of prematurity. *Semin Fetal Neonatal Med*. 2016;21(2):74-79.
9. Martin JA, Hamilton BE, Osterman MJK. Births in the United States, 2016. *NCHS Data Brief*. 2017;(287):1-8.
10. Ananth CV, Joseph KS, Oyelese Y, et al. Trends in preterm birth and perinatal mortality among singletons: United States, 1989 through 2000. *Obstet Gynecol.* 2005;105(5 Pt 1):1084-91.
11. Goldenberg RL, Culhane JF, Iams JD, Romero R. Epidemiology and causes of preterm birth. *Lancet*. 2008; 371(9606):75-84.
12. Romero R, Espinoza J, Kusanovic JP, et al. The preterm parturition syndrome. *BJOG.* 2006;113 Suppl 3:17-42.
13. Gotsch F, Gotsch F, Romero R, et al. The preterm parturition syndrome and its implications for understanding the biology, risk assessment, diagnosis, treatment and prevention of preterm birth. *J Matern Fetal Neonatal Med.* 2009;22 Suppl 2:5-23.
14. Frey HA, Klebanoff MA. The epidemiology, etiology, and costs of preterm birth. *Semin Fetal Neonatal Med*. 2016; 21(2):68-73.
15. Centers for Disease Control and Prevention. Preterm Birth Review. 2018. Available at https:cdc.gov/features/prematurebirth/index.html.
16. Muglia LJ, Katz M. The enigma of spontaneous preterm birth. *N Engl J Med*. 2010;362(6):529-535.
17. Liong S, Di Quinzio MK, Fleming G, et al. Prediction of spontaneous preterm labour in at-risk pregnant women. *Reproduction*. 2013;146(4):335-345.
18. Davey MA, Watson L, Rayner JA, Rowlands S. Risk-scoring systems for predicting preterm birth with the aim of reducing associated adverse outcomes. *Cochrane Database Syst Rev*. 2015;(10):CD004902.
19. Lykke JA, Paidas MJ, Langhoff-Roos J. Recurring complications in second pregnancy. *Obstet Gynecol*. 2009; 113(6):1217-1224.
20. Stock S, Norman J. Preterm and term labour in multiple pregnancies. *Semin Fetal Neonatal Med*. 2010;15(6): 336-341.
21. Martin JA, Hamilton BE, Ventura SJ, et al. Births: final data for 2011. *Natl Vital Stat Rep*. 2013;62(1):1-69, 72.
22. Goldenberg RL, Iams JD, Mercer BM, et al. National Institute of Child Health and Human Development Maternal-Fetal Medicine Units Network. What we have learned about the predictors of preterm birth. *Semin Perinatol*. 2003;27(3):185-193.
23. Thangaratinam S, Tan A, Knox E, et al. Association between thyroid autoantibodies and miscarriage and preterm birth: meta-analysis of evidence. *BMJ*. 2011;342: d2616.
24. Esplin MS, Elovitz MA, Iams JD, et al. nuMoM2b Network. Predictive accuracy of serial transvaginal cervical lengths and quantitative vaginal fetal fibronectin levels for spontaneous preterm birth among nulliparous women. *JAMA*. 2017;317(10):1047-1056.
25. Iams JD, Goldenberg RL, Meis PJ, et al. National Institute of Child Health and Human Development Maternal Fetal Medicine Unit Network. The length of the cervix and the risk of spontaneous premature delivery. *N Engl J Med*. 1996;334(9):567-572.
26. Hernandez-Andrade E, Romero R, Ahn H, et al. Transabdominal evaluation of uterine cervical length during pregnancy fails to identify a substantial number of women with a short cervix. *J Matern Fetal Neonatal Med*. 2012;25(9):1682-1689.
27. Berghella V, Roman A, Daskalakis C, et al. Gestational age at cervical length measurement and incidence of preterm birth. *Obstet Gynecol*. 2007;110(2 Pt 1):311-317.
28. Behrendt N, Gibbs RS, Lynch A, et al. Rate of change in cervical length in women with vaginal bleeding during pregnancy. *Obstet Gynecol*. 2013;121(2 Pt 1):260-264.
29. Ananth CV, Vintzileos AM. Medically indicated preterm birth: recognizing the importance of the problem. *Clin Perinatol*. 2008;35(1):53-67, viii.
30. Nunes MC, Aqil AR, Omer SB, Madhi SA. The effects of influenza vaccination during pregnancy on birth outcomes: a systematic review and meta-analysis. *Am J Perinatol*. 2016;33(11):1104-1114.
31. Rebarber A, Ferrara LA, Hanley ML, et al. Increased recurrence of preterm delivery with early cessation of 17-alpha-hydroxyprogesterone caproate. *Am J Obstet Gynecol*. 2007;196(3):224.e1-224.e4.
32. Society for Maternal-Fetal Medicine Publications Committee with assistance of Vincenzo Berghella. Progesterone and preterm birth prevention: translating clinical trials data into clinical practice. *Am J Obstet Gynecol*. 2012;206(5):376-386.
33. Iams JD. Clinical practice. Prevention of preterm parturition. *N Engl J Med*. 2014;370(3):254-261.
34. Committee on Practice Bulletins—Obstetrics, The American College of Obstetricians and Gynecologists. Practice Bulletin no. 130: prediction and prevention of preterm birth. *Obstet Gynecol*. 2012;120(4):964-973.

35. Norman JE, Marlow N, Messow CM, et al; OPPTIMUM study group. Vaginal progesterone prophylaxis for preterm birth (the OPPTIMUM study): a multicentre, randomised, double-blind trial. *Lancet*. 2016;387(10033): 2106-2116.
36. Fonseca EB, Celik E, Parra M, et al; Fetal Medicine Foundation Second Trimester Screening Group. Progesterone and the risk of preterm birth among women with a short cervix. *N Engl J Med*. 2007;357(5):462-469.
37. Hassan SS, Romero R, Vidyadhari D, et al. PREGNANT Trial. Vaginal progesterone reduces the rate of preterm birth in women with a sonographic short cervix: a multicenter, randomized, double-blind, placebo-controlled trial. *Ultrasound Obstet Gynecol*. 2011;38(1):18-31.
38. Conde-Agudelo A, Romero R, Nicolaides K, et al. Vaginal progesterone vs. cervical cerclage for the prevention of preterm birth in women with a sonographic short cervix, previous preterm birth, and singleton gestation: a systematic review and indirect comparison metaanalysis. *Am J Obstet Gynecol*. 2013;208(1):42.e1-42.e18.
39. Romero R, Nicolaides K, Conde-Agudelo A, et al. Vaginal progesterone in women with an asymptomatic sonographic short cervix in the midtrimester decreases preterm delivery and neonatal morbidity: a systematic review and metaanalysis of individual patient data. *Am J Obstet Gynecol*. 2012;206(2):124.e1-124.e19.
40. Romero R, Conde-Agudelo A, Da Fonseca E, et al. Vaginal progesterone for preventing preterm birth and adverse perinatal outcomes in singleton gestations with a short cervix: a meta-analysis of individual patient data. *Am J Obstet Gynecol*. 2018;218(2):161-180.
41. Crowther CA, Ashwood P, McPhee AJ, et al; PROGRESS Study Group. Vaginal progesterone pessaries for pregnant women with a previous preterm birth to prevent neonatal respiratory distress syndrome (the PROGRESS Study): a multicentre, randomised, placebo-controlled trial. *PLoS Med*. 2017;14(9):e1002390.
42. Henderson JT, Whitlock EP, O'Connor E, et al. Low-dose aspirin for prevention of morbidity and mortality from preeclampsia: a systematic evidence review for the U.S. Preventive Services Task Force. *Ann Intern Med*. 2014;160(10):695-703.
43. American College of Obstetricians and Gynecologists. ACOG Practice Bulletin no. 142: cerclage for the management of cervical insufficiency. *Obstet Gynecol*. 2014; 123(2 Pt 1):372-379.
44. Alfirevic Z, Stampalija T, Medley N. Cervical stitch (cerclage) for preventing preterm birth in singleton pregnancy. *Cochrane Database Syst Rev*. 2017;6(6): CD008991.
45. Society for Maternal-Fetal Medicine (SMFM) Publications Committee. The role of cervical pessary placement to prevent preterm birth in clinical practice. *Am J Obstet Gynecol*. 2017;216(3):B8-B10.
46. McIntosh J, Feltovich H, Berghella V, Manuck T; Society for Maternal-Fetal Medicine (SMFM). Electronic address: pubs@smfm.org. The role of routine cervical length screening in selected high- and low-risk women for preterm birth prevention. *Am J Obstet Gynecol*. 2016;215(3):B2-B7.
47. Anwar A, Lindow SW, Greaves L, et al. The use of fetal fibronectin in suspected pre-term labour. *J Obstet Gynaecol*. 2014;34(1):45-47.
48. Honest H, Bachmann LM, Gupta JK, et al. Accuracy of cervicovaginal fetal fibronectin test in predicting risk of spontaneous preterm birth: systematic review. *BMJ*. 2002;325(7359):301.
49. Berghella V, Saccone G. Fetal fibronectin testing for reducing the risk of preterm birth. *Cochrane Database Syst Rev*. 2019;7:CD006843.
50. U.S. Preventive Services Task Force. Screening for bacterial vaginosis in pregnancy to prevent preterm delivery: U.S. Preventive Services Task Force recommendation statement. *Ann Intern Med*. 2008;148(3): 214-219.
51. Brocklehurst P, Gordon A, Heatley E, Milan SJ. Antibiotics for treating bacterial vaginosis in pregnancy. *Cochrane Database Syst Rev*. 2013;1(1):CD000262.
52. Sangkomkamhang US, Lumbiganon P, Prasertcharoensuk W, Laopaiboon M. Antenatal lower genital tract infection screening and treatment programs for preventing preterm delivery. *Cochrane Database Syst Rev*. 2015;(2):CD006178.
53. Cunnington M, Kortsalioudaki C, Heath P. Genitourinary pathogens and preterm birth. *Curr Opin Infect Dis*. 2013; 26(3):219-230.
54. Smaill FM, Vazquez JC. Antibiotics for asymptomatic bacteriuria in pregnancy. *Cochrane Database Syst Rev*. 2015;(8):CD000490.
55. Cnattingius S, Granath F, Petersson G, Harlow BL. The influence of gestational age and smoking habits on the risk of subsequent preterm deliveries. *N Engl J Med*. 1999;341(13):943-948.
56. Institute of Medicine (US) Committee on Understanding Premature Birth and Assuring Healthy Outcomes. Behrman RE, Butler AS, eds. *Preterm Birth: Causes, Consequences, and Prevention*. Washington, DC: National Academies Press; 2007.
57. McCowan LM, Dekker GA, Chane E, et al; SCOPE consortium. Spontaneous preterm birth and small for gestational age infants in women who stop smoking early in pregnancy: prospective cohort study. *BMJ*. 2009;338: b1081. Erratum in *BMJ*. 2009;338:b1558.
58. Coleman T, Chamberlain C, Davey MA, et al. Pharmacological interventions for promoting smoking cessation during pregnancy. *Cochrane Database Syst Rev*. 2012; 9(9):CD010078.
59. Catling CJ, Medley N, Foureur M, et al. Group versus conventional antenatal care for women. *Cochrane Database Syst Rev*. 2015;(2):CD007622.
60. Lonhart JA, Mayo JA, Padula AM, et al. Short interpregnancy interval as a risk factor for preterm birth in non-Hispanic black and white women in California. *J Perinatol*. 2019;39(9):1175-1181.
61. White K, Teal SB, Potter JE. Contraception after delivery and short interpregnancy intervals among women in the United States. *Obstet Gynecol*. 2015;125(6):1471-1477.
62. Offenbacher S, Boggess KA, Murtha AP, et al. Progressive periodontal disease and risk of very preterm delivery. *Obstet Gynecol*. 2006;107(1):29-36.

63. Conner SN, Frey HA, Cahill AG, Macones GA, Colditz GA, Tuuli MG. Loop electrosurgical excision procedure and risk of preterm birth: a systematic review and meta-analysis. *Obstet Gynecol*. 2014;123(4):752-761.
64. McPheeters ML, Miller WC, Hartmann KE, et al. The epidemiology of threatened preterm labor: a prospective cohort study. *Am J Obstet Gynecol*. 2005;192(4):1325-1329, discussion 1329-1330.
65. Committee on Practice Bulletins-Obstetrics. ACOG Practice Bulletin no. 188: prelabor rupture of membranes. *Obstet Gynecol*. 2018;131(1):e1-e14.
66. Ramsauer B, Vidaeff AC, Hösli I, et al. The diagnosis of rupture of fetal membranes (ROM): a meta-analysis. *J Perinat Med*. 2013;41(3):233-240.
67. Ratcliffe SD, Baxley EG, Cline MK, Sakornbut EL, eds. *Family Medicine Obstetrics*. 3rd ed. Philadelphia, PA: Elsevier, 2008. 448-457.
68. Verani JR, McGee L, Schrag SJ; Division of Bacterial Diseases, National Center for Immunization and Respiratory Diseases, Centers for Disease Control and Prevention (CDC). Prevention of perinatal group B streptococcal disease—revised guidelines from CDC, 2010. *MMWR Recomm Rep*. 2010;59(RR-10):1-36.
69. Iams JD. Prediction and early detection of preterm labor. *Obstet Gynecol*. 2003;101(2):402-412.
70. Berghella V, Iams JD, Newman RB, et al; National Institute of Child Health and Human Development Network of Maternal-Fetal Medicine Units. Frequency of uterine contractions in asymptomatic pregnant women with or without a short cervix on transvaginal ultrasound scan. *Am J Obstet Gynecol*. 2004;191(4):1253-1256.
71. Gomez R, Galasso M, Romero R, et al. Ultrasonographic examination of the uterine cervix is better than cervical digital examination as a predictor of the likelihood of premature delivery in patients with preterm labor and intact membranes. *Am J Obstet Gynecol*. 1994;171(4):956-964.
72. Society for Maternal Fetal Medicine. Preterm Birth Toolkit. Available at https://www.smfm.org/publications/231-smfm-preterm-birth-toolkit.
73. Ramsey PS, Andrews WW. Biochemical predictors of preterm labor: fetal fibronectin and salivary estriol. *Clin Perinatol*. 2003;30(4):701-733.
74. Bruijn MMC, Kamphuis EI, Hoesli IM, et al. The predictive value of quantitative fibronectin testing in combination with cervical length measurement in symptomatic women. *Am J Obstet Gynecol*. 2016;215(6):793.e1-793.e8.
75. Melamed N, Hiersch L, Domniz N, et al. Predictive value of cervical length in women with threatened preterm labor. *Obstet Gynecol*. 2013;122(6):1279-1287.
76. van Baaren GJ, Vis JY, Wilms FF, et al. Predictive value of cervical length measurement and fibronectin testing in threatened preterm labor. *Obstet Gynecol*. 2014;123(6):1185-1192.
77. Gomez R, Romero R, Medina L, et al. Cervicovaginal fibronectin improves the prediction of preterm delivery based on sonographic cervical length in patients with preterm uterine contractions and intact membranes. *Am J Obstet Gynecol*. 2005;192(2):350-359.
78. Committee on Obstetric Practice. Committee Opinion no. 713: antenatal corticosteroid therapy for fetal maturation. *Obstet Gynecol*. 2017;130(2):e102-e109.
79. Crowther CA, McKinlay CJD, Middleton P, Harding JE. Repeat doses of prenatal corticosteroids for women at risk of preterm birth for improving neonatal health outcomes. *Cochrane Database Syst Rev*. 2015;(7):CD003935.
80. Gyamfi-Bannerman C, Thom EA, Blackwell SC, et al; NICHD Maternal–Fetal Medicine Units Network. Antenatal betamethasone for women at risk for late preterm delivery. *N Engl J Med*. 2016;374(14):1311-1320.
81. Brownfoot FC, Gagliardi DI, Bain E, et al. Different corticosteroids and regimens for accelerating fetal lung maturation for women at risk of preterm birth. *Cochrane Database Syst Rev*. 2013;8(8):CD006764.
82. Doyle LW, Crowther CA, Middleton P, et al. Magnesium sulphate for women at risk of preterm birth for neuroprotection of the fetus. *Cochrane Database Syst Rev*. 2009;(1):CD004661.
83. Reeves SA, Gibbs RS, Clark SL. Magnesium for fetal neuroprotection. *Am J Obstet Gynecol*. 2011;204(3):202.e1-202.e4.
84. Bain E, Middleton P, Crowther CA. Different magnesium sulphate regimens for neuroprotection of the fetus for women at risk of preterm birth. *Cochrane Database Syst Rev*. 2012;2(2):CD009302.
85. Magee L, Sawchuck D, Synnes A, von Dadelszen P; MAGNESIUM SULPHATE FOR FETAL NEUROPROTECTION CONSENSUS COMMITTEE. MATERNAL FETAL MEDICINE COMMITTEE. SOGC Clinical Practice Guideline. Magnesium sulphate for fetal neuroprotection. *J Obstet Gynaecol Can*. 2011;33(5):516-529.
86. Kashanian M, Shirvani S, Sheikhansari N, Javanmanesh F. A comparative study on the efficacy of nifedipine and indomethacin for prevention of preterm birth as monotherapy and combination therapy: a randomized clinical trial. *J Matern Fetal Neonatal Med*. 2019:1-6.
87. Flenady V, Wojcieszek AM, Papatsonis DNM, et al. Calcium channel blockers for inhibiting preterm labour and birth. *Cochrane Database Syst Rev*. 2014;6(6):CD002255.
88. Neilson JP, West HM, Dowswell T. Betamimetics for inhibiting preterm labour. *Cochrane Database Syst Rev*. 2014;2(2):CD004352.
89. US Food and Drug Administration. FDA Drug Safety Communication: New warnings against use of terbutaline to treat preterm labor. Available at http://www.fda.gov/Drugs/DrugSafety/ucm243539.htm.
90. Khanprakob T, Laopaiboon M, Lumbiganon P, Sangkomkamhang US. Cyclo-oxygenase (COX) inhibitors for preventing preterm labour. *Cochrane Database Syst Rev*. 2012;10(10):CD007748.
91. Loe SM, Sanchez-Ramos L, Kaunitz AM. Assessing the neonatal safety of indomethacin tocolysis: a systematic review with meta-analysis. *Obstet Gynecol*. 2005;106(1):173-179.

92. Hammers AL, Sanchez-Ramos L, Kaunitz AM. Antenatal exposure to indomethacin increases the risk of severe intraventricular hemorrhage, necrotizing enterocolitis, and periventricular leukomalacia: a systematic review with metaanalysis. *Am J Obstet Gynecol*. 2015;212(4): 505.e1-505.e13.

93. Amin SB, Sinkin RA, Glantz JC. Metaanalysis of the effect of antenatal indomethacin on neonatal outcomes. *Am J Obstet Gynecol*. 2007;197(5):486.e1-486.e10.

94. Haas DM, Imperiale TF, Kirkpatrick PR, et al. Tocolytic therapy: a meta-analysis and decision analysis. *Obstet Gynecol*. 2009;113(3):585-594.

95. Crowther CA, Brown J, McKinlay CJD, Middleton P. Magnesium sulphate for preventing preterm birth in threatened preterm labour. *Cochrane Database Syst Rev*. 2014;8(8):CD001060.

96. Mercer BM, Merlino AA; Society for Maternal-Fetal Medicine. Magnesium sulfate for preterm labor and preterm birth. *Obstet Gynecol*. 2009;114(3):650-668.

97. Vogel JP, Nardin JM, Dowswell T, et al. Combination of tocolytic agents for inhibiting preterm labour. *Cochrane Database Syst Rev*. 2014;7(7):CD006169.

98. Fairlie T, Zell ER, Schrag S. Effectiveness of intrapartum antibiotic prophylaxis for prevention of early-onset group B streptococcal disease. *Obstet Gynecol*. 2013; 121(3):570-577.

99. American College of Obstetricians and Gynecologists Committee on Obstetric Practice. ACOG Committee Opinion no. 485: Prevention of early-onset group B streptococcal disease in newborns. *Obstet Gynecol*. 2011;117(4):1019-1027.

100. Prevention of group B streptococcal early-onset disease in newborns: ACOG Committee Opinion, number 782. *Obstet Gynecol*. 2019;134(1):e19-e40.

101. Melamed N, Hadar E, Ben-Haroush A, et al. Factors affecting the duration of the latency period in preterm premature rupture of membranes. *J Matern Fetal Neonatal Med*. 2009;22(11):1051-1056.

102. Mercer BM, Crocker LG, Boe NM, Sibai BM. Induction versus expectant management in premature rupture of the membranes with mature amniotic fluid at 32 to 36 weeks: a randomized trial. *Am J Obstet Gynecol*. 1993; 169(4):775-782.

103. Mackeen AD, Seibel-Seamon J, Muhammad J, et al. Tocolytics for preterm premature rupture of membranes. *Cochrane Database Syst Rev*. 2014;2(2):CD007062.

104. Sharp GC, Stock SJ, Norman JE. Fetal assessment methods for improving neonatal and maternal outcomes in preterm prelabour rupture of membranes. *Cochrane Database Syst Rev*. 2014;10(10):CD010209.

105. Harding JE, Pang J, Knight DB, Liggins GC. Do antenatal corticosteroids help in the setting of preterm rupture of membranes? *Am J Obstet Gynecol*. 2001;184(2): 131-139.

106. Kenyon S, Boulvain M, Neilson JP. Antibiotics for preterm rupture of membranes. *Cochrane Database Syst Rev*. 2013;12(12):CD001058.

107. Mercer BM, Miodovnik M, Thurnau GR, et al; National Institute of Child Health and Human Development Maternal-Fetal Medicine Units Network. Antibiotic therapy for reduction of infant morbidity after preterm premature rupture of the membranes. A randomized controlled trial. *JAMA*. 1997;278(12):989-995.

108. Pierson RC, Gordon SS, Haas DM. A retrospective comparison of antibiotic regimens for preterm premature rupture of membranes. *Obstet Gynecol*. 2014;124(3): 515-519.

109. Haas D. Preterm Premature Rupture of Membranes: Erythromycin Versus Azithromycin a Randomized Trial Comparing Their Efficacy to Prolong Latency (PEACE Trial). Available at http://clinicaltrials.gov/show/NCT01556334.

110. Morris JM, Roberts CL, Bowen JR, et al. PPROMT Collaboration. Immediate delivery compared with expectant management after preterm pre-labour rupture of the membranes close to term (PPROMT trial): a randomised controlled trial. *Lancet*. 2016;387(10017):444-452.

111. Bond DM, Middleton P, Levett KM, et al. Planned early birth versus expectant management for women with preterm prelabour rupture of membranes prior to 37 weeks' gestation for improving pregnancy outcome. *Cochrane Database Syst Rev*. 2017;3(3):CD004735.

112. American College of Obstetricians and Gynecologists. ACOG Practice Bulletin: clinical management guidelines for obstetrcian-gynecologists: number 38, September 2002. Perinatal care at the threshold of viability. *Obstet Gynecol*. 2002;100(3):617-624.

113. Warner B, Musial MJ, Chenier T, Donovan E. The effect of birth hospital type on the outcome of very low birth weight infants. *Pediatrics*. 2004;113(1 Pt 1):35-41.

114. American College of Obstetrics and Gynecology. Operative vaginal delivery. Clinical management guidelines for obstetrician-gynecologists. *Int J Gynaecol Obstet*. 2001;74(1):69-76.

115. Cargill YM, MacKinnon CJ, Arsenault MY, et al; Clinical Practice Obstetrics Committee. Guidelines for operative vaginal birth. *J Obstet Gynaecol Can*. 2004;26(8): 747-761.

116. Rabe H, Diaz-Rossello JL, Duley L, Dowswell T. Effect of timing of umbilical cord clamping and other strategies to influence placental transfusion at preterm birth on maternal and infant outcomes. *Cochrane Database Syst Rev*. 2012;8(8):CD003248.

117. Committee on Obstetric Practice. Committee Opinion no. 684: delayed umbilical cord clamping after birth. *Obstet Gynecol*. 2017;129(1):e5-e10.

118. Raju TN, Mercer BM, Burchfield DJ, Joseph GF Jr. Periviable birth: executive summary of a joint workshop by the Eunice Kennedy Shriver National Institute of Child Health and Human Development, Society for Maternal-Fetal Medicine, American Academy of Pediatrics, and American College of Obstetricians and Gynecologists. *Obstet Gynecol*. 2014;123(5):1083-1096.

119. American College of Obstetricians and Gynecologists; Society for Maternal-Fetal Medicine. Obstetric Care Consensus no. 6: periviable birth. *Obstet Gynecol*. 2017; 130(4):e187-e199.

120. Stoll BJ, Hansen NI, Bell EF, et al; Eunice Kennedy Shriver National Institute of Child Health and Human Development Neonatal Research Network. Trends in care practices, morbidity, and mortality of extremely preterm neonates, 1993-2012. *JAMA*. 2015;314(10): 1039-1051.

121. Hannah ME, Ohlsson A, Farine D, et al; TERMPROM Study Group. Induction of labor compared with expectant management for prelabor rupture of the membranes at term. *N Engl J Med*. 1996;334(16):1005-1010.

122. Middleton P, Shepherd E, Flenady V, et al. Planned early birth versus expectant management (waiting) for prelabour rupture of membranes at term (37 weeks or more). *Cochrane Database Syst Rev*. 2017;1(1):CD005302.

123. Hannah ME, Ohlsson A, Wang EE, et al. Maternal colonization with group B streptococcus and prelabor rupture of membranes at term: the role of induction of labor. TermPROM Study Group. *Am J Obstet Gynecol*. 1997;177(4):780-785.

124. Wojcieszek AM, Stock OM, Flenady V. Antibiotics for prelabour rupture of membranes at or near term. *Cochrane Database Syst Rev*. 2014;10(10):CD001807.

125. Lawn JE, Kinney MV, Belizan JM, et al; Born Too Soon Preterm Birth Action Group. Born too soon: accelerating actions for prevention and care of 15 million newborns born too soon. *Reprod Health*. 2013;10(Suppl 1):S6.

126. March of Dimes. White Paper on Preterm Birth: The Global and Regional Toll. 2009. Available at http://www.marchofdimes.org/materials/white-paper-on-preterm-birth.pdf.

127. World Health Organization. Every Newborn: an action plan to end preventable deaths. 2014. Available at https://www.who.int/maternal_child_adolescent/documents/every-newborn-action-plan/en/.

128. Lawn JE, Blencowe H, Oza S, et al; Lancet Every Newborn Study Group. Every Newborn: progress, priorities, and potential beyond survival. *Lancet*. 2014;384(9938): 189-205.

129. Azad K, Costello A. Extreme caution is needed before scale-up of antenatal corticosteroids to reduce preterm deaths in low-income settings. *Lancet Glob Health*. 2014;2(4):e191-e192.

130. Althabe F, Belizán JM, McClure EM, et al. A population-based, multifaceted strategy to implement antenatal corticosteroid treatment versus standard care for the reduction of neonatal mortality due to preterm birth in low-income and middle-income countries: the ACT cluster-randomised trial. *Lancet*. 2015;385(9968):629-639.

Hypertensive Disorders of Pregnancy

Learning Objectives

1. Compare and contrast the hypertensive disorders of pregnancy: chronic hypertension, gestational hypertensions, preeclampsia, or preeclampsia on chronic hypertension.
2. Formulate a plan for diagnosis and treatment.

Introduction

Pregnancy complicates many medical conditions because of the complex interactions between the woman, the condition, and the treatment. Understanding these interactions is crucial in optimizing outcomes for the woman and infant. The woman is the priority in any medical emergency, because the fetus is dependent on her for physiologic support. For example, in managing an eclamptic seizure, every effort is directed toward supporting maternal vital functions and using necessary critical care interventions.[1,2] Concern for the fetus is shown by choosing expectant management for preeclampsia without severe features when the fetal gestational age is less than 37 weeks' gestation,[3-6] administering antenatal corticosteroids when delivery is indicated prior to 37 weeks' gestation,[7,8] and careful blood pressure (BP) management to avoid iatrogenic uteroplacental insufficiency due to hypotension.[9] This chapter focuses on four potentially life-threatening medical complications: preeclampsia with severe features, eclampsia, HELLP syndrome, and acute fatty liver of pregnancy (AFLP). The hypertensive disorders are the most common medical complications of pregnancy, whereas AFLP is an uncommon disorder unique to pregnancy that causes significant morbidity and mortality.[10,11]

Classification of Hypertensive Disorders of Pregnancy

Worldwide, hypertensive disorders represent the most common medical complication of pregnancy, affecting up to 10% of pregnancies.[10] From 2011 to 2015, hypertensive disorders accounted for 7.1% of maternal deaths in the United States.[12] The goal of classifying hypertensive disorders during pregnancy is to differentiate diseases preceding conception as opposed to those specific to pregnancy. The 2013 report by the American College of Obstetrics and Gynecologists (ACOG) Task Force on Hypertension in Pregnancy modified some components of prior hypertensive disorders in pregnancy classifications. However, it maintained a precise and practical classification system that considers hypertension during pregnancy in only four categories: chronic hypertension (of any cause), chronic hypertension with superimposed preeclampsia, preeclampsia-eclampsia, and gestational hypertension.[10]

Chronic Hypertension

During pregnancy, chronic hypertension is defined as high BP known to predate conception or detected prior to 20 weeks' gestation.[10,13] Criteria includes an elevated BP greater than or equal to 140/90 mm Hg on two occasions at least 4 hours apart prior to pregnancy or at less than 20 weeks' gestation or which persists beyond 12 weeks' postpartum. Chronic hypertension is associated with adverse perinatal outcomes, including preeclampsia, intrauterine growth restriction (IUGR), and placental abruption. The severity of maternal BP at 20 weeks' gestation is associated with worse outcomes.[14]

Pharmacotherapy for mild to moderate chronic hypertension in pregnancy has no proven fetal benefit and has not been shown to prevent preeclampsia.[9,15] Excessively lowering the BP may theoretically result in decreased placental perfusion and adverse perinatal outcomes. However, the Control of Hypertension in Pregnancy Study, which randomized women to tight control (diastolic BP [DBP] goal of 85 mm Hg) or less-tight control (DBP goal of 100 mm Hg), did not show adverse outcomes with tight control.[15] When BP is greater than 160/105 mm Hg, per ACOG,[10] pharmacologic treatment is indicated to prevent maternal end-organ damage.[10,16] A lower BP threshold is appropriate for treating women who already manifest target organ damage, such as renal insufficiency and left ventricular hypertrophy.[10]

Methyldopa, labetalol, and nifedipine are the oral drugs most commonly used for severe, chronic hypertension in pregnancy as reported in the 2013 report

by the ACOG Task Force on Hypertension in Pregnancy.[10] An open label randomized controlled trial (RCT) comparing these three agents showed nifedipine to be most effective, with nifedipine and labetalol more effective than methyldopa; however, all three remain acceptable options.[17] Angiotensin-converting enzyme (ACE) inhibitors and angiotensin II receptor antagonists should not be used because of the association with IUGR, neonatal renal failure, oligohydramnios, effects of oligohydramnios (eg, limb abnormalities, cranial ossification defects, pulmonary hyperplasia), and neonatal death.[10] The beta blocker atenolol has also been associated with IUGR.[10] Thiazide diuretics may be continued if used before pregnancy, but they must be discontinued if they exacerbate the intravascular fluid depletion of preeclampsia or if chronic hypertension becomes complicated by superimposed preeclampsia.[18,19] Therefore, thiazide diuretics are not first-line antihypertensive drugs for chronic hypertension in pregnancy.

Chronic hypertension during pregnancy is most commonly referred to as mild (BP greater than 140/90 mm Hg) or severe (BP 160/110 mm Hg or greater); however, the 2019 ACOG Pratice Bulletin acknowledges the new American College of Cardiology/American Heart Association definition of mild hypertension as systolic BP (SBP) of 130 to 139 mm Hg or diastolic BP of 80 to 89 mm Hg.[13,20] Women in the new mild hypertension category are recommended to have closer observation but not to initiate aspirin for preeclampsia prevention unless there are other risk factors.[13] Women in active labor with uncontrolled severe chronic hypertension require treatment with intravenous (IV) labetalol or hydralazine in doses similar to those used for preeclampsia with severe features as discussed later.[18] Although IV drugs have traditionally been recommended over oral drugs, a small 2013 RCT showed a more rapid lowering of BP with oral nifedipine than with IV labetalol.[21] Oral nifedipine is considered to be a therapeutic option for acute-onset, severe hypertension during pregnancy or postpartum in the 2019 ACOG Committee Opinion.[18]

Chronic Hypertension With Superimposed Preeclampsia

Women with chronic hypertension should be monitored carefully for the development of super-imposed preeclampsia and IUGR.[10] The development of proteinuria or a sudden sustained increase in proteinuria, a sudden increase in BP in a woman whose hypertension has previously been well controlled, or development of the severe features of preeclampsia —right upper quadrant pain, headache, vision changes, pulmonary edema, rise in creatinine or transaminase level, thrombocytopenia (platelet count less than 100,000/mL)—are diagnostic for superimposed preeclampsia.[10] If the only manifestations are elevated BP to levels less than 160/110 mm Hg along with proteinuria, the subcategory classification is considered to be superimposed preeclampsia *without* severe features. With the additional presence of any organ dysfunction as evidenced by the severe features mentioned previously, the subcategory classification is considered to be superimposed preeclampsia *with* severe features. Both variants are classified as chronic hypertension with superimposed preeclampsia, but management is guided by the subcategory. Fetal growth should be assessed by serial ultrasonography starting after 24 weeks' gestation to screen for developing IUGR.[10]

Although evidence is lacking for an optimal interval for fetal growth ultrasonography assessments, every 4 weeks is a reasonable option if there is no evidence of IUGR or superimposed preeclampsia. Antenatal surveillance (eg, modified biophysical [nonstress test with amniotic fluid index] or biophysical profile) is recommended for women with chronic hypertension who require antihypertensive drugs or who have IUGR or superimposed preeclampsia.[13]

Gestational Hypertension

The National High Blood Pressure Education Program Working Group on High Blood Pressure in Pregnancy has recommended that *gestational hypertension* replace the term *pregnancy-induced hypertension*.[10] Women who develop hypertension after 20 weeks' gestation and do not have significant proteinuria or other criteria for preeclampsia should be diagnosed with gestational hypertension. Gestational hypertension is a provisional diagnosis used for a heterogeneous group of women including: those who will eventually develop proteinuria or other preeclampsia criteria and be diagnosed with preeclampsia during the pregnancy or postpartum, those who will have persistent hypertension after 12 weeks and be diagnosed with chronic hypertension, and those who do not develop preeclampsia and

whose BP normalizes in the postpartum period. Women in the last group are ultimately diagnosed with *transient hypertension of pregnancy*.[10]

Gestational hypertension is not a benign category. Approximately 50% of women diagnosed with gestational hypertension between 24 and 35 weeks' gestation ultimately develop preeclampsia.[22] Expectant management of gestational hypertension can reduce the increased cesarean delivery rate that occurs with inductions.[23] If BP levels progress to the severe range (SBP greater than 160 mm Hg or DBP greater than 110 mm Hg), then management similar to preeclampsia with severe features is required even if the patient does not have proteinuria. This is because women with severe gestational hypertension have worse perinatal outcomes than women with preeclampsia without severe features.[24] ACOG recommends induction at 37 weeks' gestation if delivery has not already occurred.[19] A retrospective analysis comparing women with gestational hypertension, preeclampsia without severe features, and mild chronic hypertension showed higher rates of maternal admission to an intensive care unit, postpartum hemorrhage, and blood transfusion in the gestational hypertension group.[25]

Preeclampsia Without Severe Features

Preeclampsia is a multi-organ disease process characterized by new-onset hypertension and proteinuria or severe features of preeclampsia in the second half of pregnancy in a woman with previously normal BP. To meet diagnostic criteria for preeclampsia, SBP must be 140 mm Hg or greater or DBP 90 mm Hg or greater on at least two occasions no less than 4 hours apart.[10] BP should be measured at each prenatal visit using appropriate techniques. This includes using an appropriately sized cuff, the patient being seated in an upright position with her legs uncrossed, and instructing the patient to relax and not talk during the measurement. The patient's arm and back should be supported so that the middle of the BP cuff is at the level of the right atrium. If the initial BP level is elevated, then a repeat measurement should be obtained after at least 5 minutes. Although previously considered diagnostic of preeclampsia, an increase in SBP of 30 mm Hg or DBP of 15 mm Hg is no longer included in the diagnostic criteria of preeclampsia because similar increases are common in uncomplicated pregnancies.[10] The U.S. Preventive Services Task Force (USPSTF) 2017 recommendations support measuring BP throughout pregnancy (B Recommendation) but do not support routine urine *dipstick* testing for proteinuria.[26]

The diagnostic threshold for proteinuria is 300 mg in a 24-hour specimen or 0.3 with a urine protein/creatinine ratio.[10] A dipstick reading lacks sensitivity or specificity for proteinuria, but if a 24-hour analysis or urine protein/creatinine ratio is not available, then random urine dipstick measurements greater than or equal to 1+ (30 mg/dL) are consistent with the presence of proteinuria, and measurements of 2+ have greater specificity.[13,27] A quantitative determination is the gold standard, however, because urine dipsticks can be affected by dehydration and bacteriuria. A catheterized urinalysis may avoid protein due to contamination, though a traumatic catheterization can introduce protein from blood. In selected clinical circumstances, a shorter period of timed urine collection (eg, 6 or 12 hours) to quantitate protein is another alternative.[28] A random urine protein/creatinine ratio that is less than 0.21 had an 83% negative predictive value in one study of 138 women with proteinuria.[29] There is not a universally accepted negative cutoff, but most studies have used ratios of 0.15 to 0.5.[30] Proteinuria is not useful for screening because it occurs late in preeclampsia after kidney and liver damage has already occurred, and the amount of protein in the urine is not predictive of how the disease will progress.

Edema supports the diagnosis of preeclampsia when it is pronounced and generalized (affecting the face or hands) but is no longer a diagnostic criterion. Rapid weight gain may be a sign of pronounced edema and should alert the provider to at least consider the diagnosis. Many women with preeclampsia never have edema, whereas nondependent edema occurs in 10% to 15% of women who remain normotensive during pregnancy.[10]

The diagnosis of preeclampsia can be made without proteinuria if any of the following severe features of pregnancy are present: platelet levels less than 100,000/mL, serum creatinine levels greater than 1.1 mg/dL or a doubling of serum creatinine levels from baseline (if known) without another etiology, pulmonary edema, or elevation of transaminase levels to twice the normal level. The presence of new-onset cerebral or vision symptoms or severe persistent right upper quadrant or epigastric pain that is unresponsive

to drugs and not accounted for by an alternative diagnosis also is sufficient to diagnose preeclampsia in the setting of elevated BP.[10]

The etiology of preeclampsia remains unknown and no single causal factor links all theories (*Table 1*). Growing evidence suggests the disease is a multi-organ disease and not just high BP and proteinuria. It is evident that the placenta has a central role in preeclampsia.[10] Despite the identification of many biomarkers and clinical risk factors (*Table 2*), a study of nulliparous women showed the predictive benefit of these factors to be modest and none are routinely indicated.[31-33] A ratio of soluble fms-like tyrosine kinase-1 to placental growth factor of 38 has a negative predictive value of 99.3% (95% CI = 97.9-99.9).[32] Its use to rule out preeclampsia is recommended by the National Institute for Health and Care Excellence (NICE) in the United Kingdom, but only for the negative predictive value.[33] The test is currently available in many countries, but has not been approved for use in the United States.

Randomized controlled trials have not found a role for routine prenatal supplementation with calcium, omega-3 fatty acids, antioxidant vitamins E and C, or vitamin D to prevent preeclampsia.[34] Calcium supplementation may decrease the incidence of hypertension, preeclampsia, and maternal death among women at high risk of developing those conditions and women with low calcium intake.[35] The World Health Organization (WHO) recommends routine supplementation with 1.5 to 2.0 g/day of elemental calcium for women with low calcium intake.[36] Women in the United States or other high-resource countries are unlikely to have low calcium intake because of the widespread supplementation in food. The ACOG Task Force on Hypertension in Pregnancy recommends against supplementation except in populations at risk of low calcium intake.[10,35]

Antiplatelet agents (eg, low-dose aspirin) do have a role in preeclampsia prevention for high-risk women. A Cochrane review of low-dose aspirin use in women at increased risk of preeclampsia showed a 17% reduction in the risk of developing preeclampsia (number needed to treat [NNT] = 72). In the subgroup of women at highest risk because of a history of preeclampsia with severe features, diabetes, chronic hypertension, or renal or autoimmune disease, only 19 women needed to be treated with low-dose aspirin to prevent one case of preeclampsia.[37]

In 2013, the ACOG Task Force on Hypertension in Pregnancy recommended initiation of 60 to 80 mg of aspirin in the late first trimester for women with histories of preeclampsia, prior pregnancies with delivery before 34 weeks' gestation, or who had preeclampsia in more than one previous pregnancy.[10] In 2014, the USPSTF released a systematic review and a recommendation for the

Table 1. Theories Associated With Preeclampsia Pathophysiology

Genetic predisposition (maternal, paternal, thrombophilia)
Immunologic phenomena
Abnormal placental implantation (defects in trophoblasts and spiral arterioles)
Vascular endothelial damage and oxidative stress
Angiogenic factors (low level of placental growth factor)

Information from various sources.

Table 2. Preeclampsia Risk Factors and Approximate Increased Risk

Family history of preeclampsia (first-generation relative) = 3×
Nulliparity = 3×
Maternal age greater than 40 years = 1.6×
Multiple gestation = 3×
Preeclampsia in a prior pregnancy (particularly if severe or before 32 weeks' gestation) = 7×
Elevated body mass index = 2×
Diabetes (preexisting) = 3×
Chronic hypertension and/or renal disease
Systemic lupus erythematosus/antiphospholipid syndrome

Note: Previously, young maternal age was previously considered a risk factor, but this was not supported by a systematic review.

Information from Duckitt K, Harrington D. Risk factors for pre-eclampsia at antenatal booking: systematic review of controlled studies. BMJ. *2005;330(7491):565; Milne F, Redman C, Walker J, et al. The pre-eclampsia community guideline (PRECOG): how to screen for and detect onset of pre-eclampsia in the community.* BMJ. *2005;330(7491):576-580;*

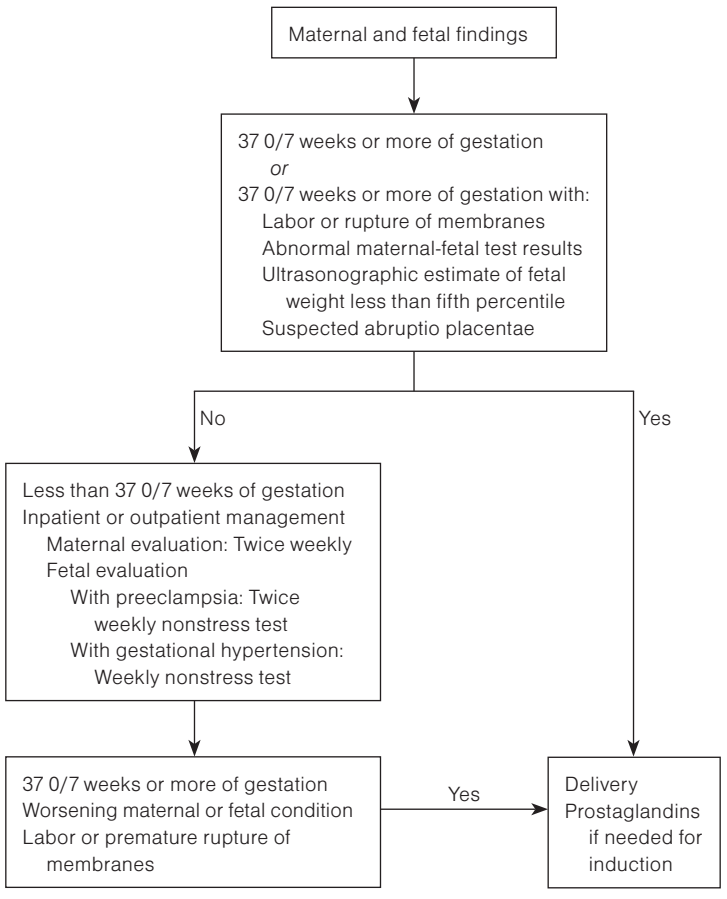
use of aspirin for preeclampsia prevention that included a wider range of indications including history of preeclampsia, multifetal gestation, chronic hypertension, diabetes (type 1 or type 2), renal disease, or autoimmune disease (eg, systematic lupus erythematosus, antiphospholipid syndrome).[38] A decision analysis showed that the USPSTF recommendation would treat 23.5% of pregnant women[39] and reduce the incidence of preeclampsia from 4.18% to 3.83%, whereas the ACOG recommendation would treat 0.35% of pregnant women and only decrease the incidence of preeclampsia from 4.18% to 4.17%.[39] The USPSTF recommendations were more cost effective as well, and ACOG released a practice advisory in 2016 supporting the USPSTF recommendations.[40]

The optimal dose of aspirin and timing for initiation of treatment remain controversial. The Combined Multimarker Screening and Randomized Patient Treatment with Aspirin for Evidence-Based Preeclampsia Prevention (ASPRE) trial used a complex screening algorithm including uterine artery Doppler measurements, plasma protein A, and growth factor measurements as well as the medical and obstetric risk factors used in earlier studies.[41] ASPRE used a higher dose of aspirin (150 mg) and initiation of treatment at 11 to 14 weeks' gestation. The study's treatment versus placebo arm showed a lower rate of preterm preeclampsia of 1.6% versus 4.3% (odds ratio 0.38; 95% CI = 0.20-0.74). A secondary analysis of the ASPRE trial in 2017 showed that the effectiveness of low-dose aspirin to prevent preterm preeclampsia was dependent on a high level of compliance as 0.9% of women with greater than 90% compliance developed preterm preecclampsia compared with 3.3% in the group with less than 90% compliance consistent with a dose-response effect. A meta-analysis of 45 RCTs showed a dose-response relationship based on aspirin dose (100 mg versus 60 mg) and time of initiation (before or after 16 weeks' gestation).[42,43] Based on these studies, the use of 100 to 150 mg may be preferable to the 81 mg recommended by USPSTF in 2014.

Expectant management of women with preeclampsia without severe features may include twice weekly BP testing, weekly laboratory tests (complete blood count [CBC], alanine aminotransferase [ALT], aspartate transaminase [AST], and creatinine), twice weekly nonstress tests (NST), weekly amniotic fluid assessment or weekly biophysical profiles, and ultrasonography for fetal growth monitoring every 3 to 4 weeks.[10] Although uric acid and lactate dehydrogenase (LDH) are commonly assessed in women with possible preeclampsia these tests are not part of the criteria for diagnosis of preeclampsia with severe features. Fetal umbilical artery Doppler studies are recommended as part of antenatal surveillance for women with preeclampsia when IUGR has been detected.[10]

The decision to induce labor or perform cesarean delivery involves balancing prematurity-related risks with the risk of worsening preeclampsia. Delivery is typically indicated for women with preeclampsia or gestational hypertension at 37 weeks' gestation (*Figure 1*). This recommendation is based

Figure 1. Management of Gestational Hypertension or Preeclampsia Without Severe Features



Reprinted from American College of Obstetricians and Gynecologists; Task Force on Hypertension in Pregnancy. Hypertension in pregnancy. Report of the American College of Obstetricians and Gynecologists' Task Force on Hypertension in Pregnancy. Obstet Gynecol. *2013;122(5):1122-1131.*

on the Hypertension and Pre-eclampsia Intervention Trial At Term (HYPITAT) RCT of induction versus expectant management and the recommendations of a 2011 workshop sponsored by the Society for Maternal-Fetal Medicine (SMFM) and the National Institute of Child Health and Human Development.[44,45] A secondary analysis of HYPITAT showed greater benefit of labor induction on preventing high-risk maternal situations and reducing the cesarean delivery rate in women with an unfavorable cervical examination, presumably because these women were more remote from spontaneous labor.[46] An economic analysis of HYPITAT showed cost savings from labor induction compared with expectant monitoring.[47] A 2017 Cochrane review of delivery versus expectant management from 34 weeks' gestation to term for women with preeclampsia or gestational hypertension without severe features showed maternal benefit from earlier delivery. However, planned early delivery was associated with the adverse neonatal outcomes of respiratory distress syndrome (relative risk [RR] 2.2; 95% CI = 1.2-4.2 [three studies]) and neonatal intensive care unit (NICU) admission (RR 1.7; 95% CI = 1.1-2.4 [four studies]).[48]

Preeclampsia With Severe Features

The distinction between preeclampsia and preeclampsia with severe features is based on the degree of BP elevation, presence of specific abnormal laboratory findings, or the presence of clinical symptoms resulting from involvement of the kidneys, brain, liver, lungs, and cardiovascular system. Proteinuria is no longer a criterion for preeclampsia with severe features, because higher levels of protein are not an indicator of disease severity.[10]

Diagnostic criteria for preeclampsia with severe features are listed in *Table 3*.[27] Preeclampsia with severe features may result in multi-system deterioration that can be gradual or sudden. Severe headache, vision disturbances, and progressive hyperreflexia may signal impending generalized seizures (eclampsia). Increasing peripheral vascular resistance stresses the cardiovascular system, and pulmonary edema may result. A decreased glomerular filtration rate may progress to oliguria and acute renal failure. Hemodilution and increased creatinine clearance typically lowers pregnancy creatinine levels; levels above 0.9 mg/dL in pregnancy are abnormal.[49] Liver manifestations include elevated transaminase levels, subcapsular hemorrhage with right upper quadrant pain, and capsular rupture with life-threatening intraabdominal bleeding. Preeclampsia-related coagulopathies include HELLP syndrome and disseminated intravascular coagulation (DIC). Obstetric complications include IUGR, abruption, and fetal or maternal death.[6]

Table 3. Diagnostic Criteria for Preeclampsia With Severe Features

SBP ≥140 mm Hg or DBP ≥90 mm Hg on at least two occasions taken 4 hours apart

Any of the following signs and symptoms:

- Progressive renal insufficiency (serum creatinine >1.1 mg/dL or double the baseline)
- Cerebral or visual disturbances
- Pulmonary edema
- Impaired liver function (transaminases 2× normal), right upper quadrant pain or epigastric pain
- Thrombocytopenia (<100,000/mL)

DBP = diastolic blood pressure; SBP = systolic blood pressure.

Information from American College of Obstetricians and Gynecologists. ACOG Practice Bulletin no. 202: gestational hypertension and preeclampsia. Obstet Gynecol. *2019;133(1):e1-e25.*

The progression of preeclampsia is only reversed by delivery. Patients with preeclampsia with severe features should be admitted to the hospital, placed on bed rest, and carefully monitored.[10] The overall treatment goals are to prevent seizures, lower BP to prevent maternal cerebral hemorrhage and myocardial infarction, and expedite delivery based on a decision that considers disease severity and fetal maturity.

Sample admitting orders for preeclampsia with severe features are outlined in *Table 4.* Fluid management requires special care. Excessive fluid administration can result in pulmonary edema, ascites, and cardiopulmonary overload, whereas insufficient fluid can exacerbate an already constricted intravascular volume and lead to further end-organ ischemia. If urine output falls below 25 to 30 mL/hour, lactated Ringer solution or normal saline should be administered.[50] IV fluid should be administered at a dosage of 100 to 125 mL/hour,[50,51] and total oral and IV fluid intake should not exceed 150 mL/hour.[51] A Foley catheter allows accurate monitoring of urine output. A Swan-

Ganz catheter may optimize fluid management if pulmonary edema and renal failure are present but should not be routinely used. Maternal-fetal medicine consultation is recommended if a Swan-Ganz catheter is being considered.[50]

Plasma volume is reduced among women with preeclampsia, which suggests that increasing plasma volume with colloid solution might improve uteroplacental circulation and perinatal outcomes. However, risk-benefit data regarding this practice are lacking.[52]

In addition to the basic laboratory investigation for preeclampsia, women with signs and symptoms of severe disease may be evaluated with LDH testing, peripheral blood smear, and laboratory tests for evidence of hemolysis and DIC, depending on the clinical scenario.

Delivery Decisions in Women With Preeclampsia With Severe Features

Delivery is the only known cure for preeclampsia. Decisions regarding the timing and mode of delivery are based on a combination of maternal and fetal factors. Fetal factors include gestational age, evidence of lung maturity, and signs of fetal compromise on antenatal assessment. Maternal factors include the degree to which the hypertension is controllable and any clinical or laboratory signs of impending decompensation. For patients with resistant severe hypertension, eclampsia, pulmonary edema, placental abruption, or other signs of maternal or fetal deterioration, delivery is indicated after maternal stabilization without waiting the full 48 hours for antenatal corticosteroids, regardless of gestational age.[27] Women at less than 34 weeks' gestation should be delivered after receiving 48 hours of antenatal corticosteroids and if they have platelet counts less than 100,000/mL, transaminase levels twice the normal value, IUGR, umbilical artery reversed end-diastolic flow, or new or worsening renal dysfunction.[10,27] If maternal and fetal conditions allow, attempting to delay labor and administer corticosteroids is recommended for preeclampsia in the setting of preterm prelabor rupture of membranes or preterm labor at less than 34 weeks' gestation.[10]

The Maternal-Fetal Medicine Units Network Antenatal Late Preterm Steroids trial showed strong neonatal benefits when antenatal corticosteroids were administered in women who were between 34 and 36 6/7 weeks' gestation, at high risk of a late preterm delivery, and had not received a prior course of antenatal corticosteroids. The incidence of severe respiratory complications decreased from 12.1% in the placebo group to 8.1% in the betamethasone group (RR 0.67;

Table 4. Admitting Orders for Severe Preeclampsia With Severe Features

Bed rest with seizure precautions
Vital signs (BP, pulse, respirations), every 15 minutes until stable, then hourly or per hospital protocol
Neurological assessments (headache, deep tendon reflexes, visual changes, clonus) every 15 minutes until stable, then hourly or per hospital protocol
Gastroenterological assessments (RUQ/epigastric pain, nausea, vomiting) every 15 minutes until stable, then hourly or per hospital protocol
Respiratory assessments (lung sounds, productive cough, dyspnea) every 15 minutes until stable, then hourly or per hospital protocol
Accurate intake and output; Foley catheter and IV access if needed. Ensure minimum urine output of 30 mL/hour. Total fluid intake (IV and oral) should not exceed 125 mL/hour or 3,000 mL/day
Continuous EFM for contractions and FHR assessment
Laboratory tests:
Dipstick urine for protein on admission and urine protein/creatinine ratio (not needed with definitive finding of severe features)
Begin 24-hour urine collection for total urine protein and creatinine (not needed with definitive finding of severe features)
CBC
Serum creatinine
Serum AST or ALT
Uric acid
Serum LDH
(See *Table 5* for information on magnesium sulfate)
Hydralazine 5 or 10 mg IV over 2 minutes. If BP remains elevated after 20 minutes, then administer an additional 10 mg IV. If BP remains elevated after 20 minutes, then change to IV labetalol
or
Labetalol 20 mg IV over 2 minutes. If BP remains elevated after 10 minutes, double the dose to 40 mg. If BP remains elevated after 10 more minutes, then double the dose to 80 mg. If BP remains elevated, then change to IV hydralazine. The maximum dose of IV labetalol is 300 mg in a 24-hour period
or
Nifedipine 10 mg orally. If BP remains elevated after 20 minutes, administer an additional 20 mg dose orally. May repeat 20 mg dose orally in 20 minutes for a total of 50 mg
If BP remains elevated after all of the above medications are administered consider critical care, MFM, or anesthesia consultation

ALT = alanine aminotransferase; AST = aspartate transaminase; BP = blood pressure; CBC = complete blood cell count; EFM = external fetal monitoring; FHR = fetal heart rate; IV = intravenous; LDH = lactate dehydrogenase; MFM = maternal-fetal medicine; RUQ = right upper quadrant.

Information from various sources.

95% CI = 0.53-0.84; *P*<0.001).[53] ACOG and SMFM do not recommend deferring delivery to complete a steroid course before initiating delivery in women with preeclampsia with severe features in the late preterm period.[7,8] When the decision is made to proceed with delivery or a patient presents in labor with preeclampsia with severe features, magnesium sulfate (MgSO4), if not already being administered, should be initiated for seizure prophylaxis with a bolus and ongoing infusion as described in *Table 5*.

There is limited data regarding the optimal treatment of women with preeclampsia with severe features between 24 and 34 weeks' gestation. The 2013 Cochrane review is based on only four RCTs with a total of 425 women.[3] The use of expectant management with close maternal and fetal surveillance in a hospital with perinatal and neonatology services appears to have decreased neonatal morbidity and length of stay in the NICU. However, many women are not candidates for expectant management or may need urgent delivery because of complications including eclampsia, HELLP syndrome, pulmonary edema, renal insufficiency, abnormal fetal surveillance, or placental abruption.[3,4,6,27] In one study, bed rest and close monitoring of women between 28 to 32 weeks' gestation with preeclampsia prolonged pregnancy by an average of 15 days, which resulted in fewer days in the NICU and fewer cases of neonatal respiratory distress syndrome and necrotizing enterocolitis without increasing maternal morbidity.[6] The largest RCT is the MEXPRE Latin study, which was a multisite study of eight centers in Latin America. Despite a delay in delivery of 10.3 versus 2.2 days, the study did not show neonatal benefit of expectant management.[54] Of note, the varied criteria for intervention led to many more deliveries for uncontrolled BP than in a smaller US study.[6,54] A commentary accompanying the MEXPRE study recommended that pregnant women with preeclampsia with severe features should be delivered after administration of corticosteroids in countries with limited resources, rather than attempting continued expectant management.[54,55]

Attempted vaginal delivery is recommended for women who have preeclampsia with severe features if there is no evidence of maternal or fetal compromise or other obstetric contraindications.[10] Potential indications for cesarean delivery may include status epilepticus, severe BP ranges resistant to drug treatment, or other situations indicating worsening maternal condition remote from delivery (eg, pulmonary edema, severe thrombocytopenia). Some experts recommend cesarean delivery in women with fetuses younger than 30 weeks' gestation when the cervix is not ripe, but a trial of induction may be considered.[10,27]

Fetal Surveillance

Assessment for uteroplacental insufficiency may be achieved using NSTs, amniotic fluid measurements, and biophysical profiles. Umbilical artery Doppler systolic-to-diastolic ratios may detect early uteroplacental insufficiency, and this examination is indicated for the fetus with IUGR. The presence of reversed end diastolic umbilical artery flow is an indication for delivery after corticosteroids are administered, if less than 34 weeks' gestation.[10] Fetal monitoring frequency varies depending on the clinical context. A common strategy for preeclampsia without severe features at less than 37 weeks' gestation includes perform-

Table 5. Magnesium Sulfate for Hypertensive Disorders of Pregnancy

MgSO4 loading dose: 4 to 6 g mixed in 100 mL, administered IV over 15 to 20 minutes, followed by a continuous infusion of 2 g/hour

Monitor:
- Vital signs
- Deep tendon reflexes
- Mental status
- Respiratory status
- Total fluid intake
- Total urine output
- FHR status

Magnesium levels (therapeutic range = 4.8-9.6 mg/dL) should be checked every 8 hours or as needed if renal dysfunction is present (elevated creatinine >0.9 mg/dL, or decreased urine output <30 mL/hr), loss of reflexes, or other symptoms of magnesium toxicity

FHR = fetal heart rate; IV = intravenous; MgSO4 = magnesium sulfate.

Information from American College of Obstetricians and Gynecologists. ACOG Practice Bulletin no. 202: gestational hypertension and preeclampsia. Obstet Gynecol. *2019;133(1):e1-e25; Euser AG, Cipolla MJ. Magnesium sulfate for the treatment of eclampsia: a brief review.* Stroke. *2009;40(4):1169-1175.*

ing twice weekly NSTs and a weekly amniotic fluid index (AFI) measurement with a biophysical profile for follow-up of nonreactive NSTs.[10] Those diagnosed with preeclampsia *with* severe features should be admitted to a hospital for close observation and undergo daily fetal monitoring. Women with gestational hypertension at less than 37 weeks' gestation may receive a weekly NST and AFI.[10] Ultrasonography for assessment of fetal growth should be repeated every 3 to 4 weeks.[10]

Corticosteroids are administered to accelerate lung maturity for fetuses between 24 and 34 weeks' gestation, either betamethasone (two doses of 12 mg administered intramuscularly 24 hours apart) or dexamethasone (four doses of 6 mg administered intramuscularly 12 hours apart).[56]

Eclampsia

The generalized seizures of eclampsia represent a life-threatening emergency that requires immediate attention while honoring the concept of *primum non nocere* or *first do no harm*.

Pathophysiology

Eclampsia is defined as the onset of seizures in pregnant women with hypertension. The precise mechanism leading to seizures is unknown, but it may include cerebral edema, transient vasoconstriction, ischemia, or microinfarcts.[1]

Clinical Course

Eclampsia may be preceded by worsening of the signs and symptoms of preeclampsia with severe features, or may appear unexpectedly in a patient with preeclampsia that lacked severe features and with minimally elevated or normal BP. In one large series, 15% of the women had DBP less than 90 mm Hg.[57] It is rare for eclampsia to occur before 20 weeks' gestation in the absence of gestational trophoblastic disease. Neurological symptoms often precede eclamptic seizures as shown by a study of 46 women with eclampsia at a Tanzanian hospital; 80% of these women had a preceding headache and 45% had visual changes.[58]

Eclamptic seizures typically last from 60 to 90 seconds, and the patient is without respiratory effort during this time. A postictal phase may follow with confusion, agitation, and combativeness. The timing of an eclamptic seizure can be antepartum (38% to 53%), intrapartum (18% to 36%), or postpartum (11% to 44%).[1]

Management

An eclamptic seizure can be dramatic and disturbing. The attending clinician is challenged to remain calm and avoid unnecessary interventions that can result in iatrogenic complications.[1,2]

1. Do not attempt to shorten or abolish the initial seizure by using drugs such as diazepam or phenytoin. These drugs can lead to respiratory depression, aspiration, or frank respiratory arrest, particularly when they are administered repetitively or used in combination with MgSO4. Further, phenytoin is less effective than MgSO4 in preventing recurrent eclamptic seizures.[59]

2. Protect the airway and minimize the risk of aspiration by placing the woman on her left side and suctioning her mouth. Summon a clinician skilled in intubation to be immediately available.[2] The adult cardiopulmonary resuscitation recovery position involves the patient being positioned as laterally as possible. Allow for observation of breathing and avoid any pressure on the chest.[60] This position helps a semiconscious or unconscious individual breathe and permits fluids to drain from the nose and throat to avoid aspiration; in addition, it maximizes venous return. Administer supplemental oxygen at 10 L via nonrebreather facemask during the seizure.

3. Prevent maternal injury. Falls from the bed can result in contusions or fractures, and head injury may result from violent seizure activity. Close observation, soft padding, and use of hospital bed rails may help prevent these complications.

4. Administer MgSO4 to control seizures. If the patient with preeclampsia has already received a prophylactic loading dose of MgSO4 and is receiving a continuous maintenance infusion when the seizure occurs, an additional 2 g IV should be infused over 15 to 20 minutes. Otherwise, a 6-g IV loading dose of MgSO4 should be administered over 15 to 20 minutes, followed by a maintenance dose of 2 g/hour. No more than 8 g should be infused over a 1-hour period.[1,2] A serum magnesium level may be obtained 4 to 6 hours after the loading dose and the maintenance infusion should be adjusted accordingly to obtain a therapeutic range of 4.8 to 9.6 mg/dL, although there is limited evidence regarding the optimal therapeutic range.[27] Examine the patient hourly for presence of deep tendon reflexes (DTRs) and adequate urine output.

After the seizure has ended, continue supplemental oxygen until the patient is fully responsive.

When the patient has stabilized, plan for prompt delivery. Avoid performing an immediate cesarean delivery for a self-limited seizure episode. Anticipate that the fetal heart rate will demonstrate bradycardia or decelerations during the immediate postseizure period (eg, 10 minutes with a gradual return of moderate variability).

Maternal and Fetal Outcomes in Eclampsia

The perinatal mortality rate from eclamptic seizures in high-resource areas is less than 1%; however, it is higher in low-resource settings. In a 2008 Moroccan study, a rate of 6.7% was shown,[61] and a 7.5% rate was shown in a 2011 study in Nigeria.

From 2006 to 2010, 9.4% of US pregnancy-related deaths were due to hypertensive disorders of pregnancy with a decrease to 7.4% between 2011 and 2013.[62,63] In an early US study, approximately 50% of preeclampsia/eclampsia-related deaths were caused by abruption, DIC, aspiration pneumonia, and cardiopulmonary arrest, all of which are serious causes of morbidity and mortality in women with eclampsia.[61,64]

Most fetal eclampsia-related morbidity and mortality result from prematurity, growth restriction, and placental abruption. During an eclamptic seizure, the fetus will frequently manifest hypoxia-related bradycardia. The fetus typically recovers after the seizure ends.

In rural or remote areas, maternity care clinicians need to balance the risk of transferring the unstable patient with preeclampsia/eclampsia and the benefit of higher level of care offered at tertiary maternal and neonatal care facilities. When the woman has been adequately treated with MgSO4 and the woman and fetus are stabilized, a successful transfer can be made. Close coordination of care with consultants at the receiving institution is mandatory.

Postpartum Management of Preeclampsia

Most patients with preeclampsia benefit promptly from delivery with decreased BP, diuresis, and general clinical improvement. Eclampsia may occur postpartum, with the greatest risk of postpartum eclampsia occurring in the first 48 hours.[1] MgSO4 administration should continue for 24 hours after delivery, or occasionally longer if the clinical situation warrants.[1,27,65] There is limited evidence supporting the 24-hour postpartum MgSO4 recommendation, with a 2018 RCT failing to show benefit; however, the study had limited authority as postpartum eclampsia is an infrequent occurrence.[66] Patients receiving MgSO4 require ongoing monitoring of BP and urine output, because they are at risk of pulmonary edema due to IV fluid overload, mobilization of *third space* fluids, and decreased renal function.

Hypertension may worsen in the days after delivery as fluid in the third space returns to the vasculature. For this reason, ACOG recommends observation in the hospital for 72 hours after delivery with gestational hypertension and preeclampsia or the equivalent monitoring at home.[10] Because there are no longer concerns for fetal well-being regarding BP lowering, antihypertensive treatment is recommended for women whose postpartum SBP is 150 mm Hg or greater or whose DBP is 100 mm Hg or greater on at least two occasions at least 4 hours apart. If SBP is 160 mm Hg or greater or DBP is 110 mm Hg or greater, antihypertensive treatment should be initiated within 60 minutes of diagnosis.[18] Studies have not found a clinically relevant effect of nonsteroidal anti-inflammatory drugs on postpartum BP and these agents remain first-line pain management medications,[67,68] particularly given the desire to minimize the need for postpartum opioids in the setting of the current epidemic of opioid use disorder.[69]

Despite a lack of high-quality studies on postpartum hypertensive management,[70] oral nifedipine or labetalol are commonly used and safe for breastfeeding. If needed, IV labetalol or hydralazine may be used as described for intrapartum management.[18] Patients should be evaluated in the office 7 to 10 days after hospital discharge or sooner if they are symptomatic.[10]

HELLP Syndrome

The acronym HELLP describes a variant of severe preeclampsia with severe features characterized by **H**emolysis, **E**levated **L**iver enzymes, and **L**ow **P**latelets.[71] HELLP syndrome poses significant challenges to maternity care clinicians. First, they must maintain a high index of suspicion for the diagnosis, particularly in pregnant patients who are remote from term and may not be hypertensive; and second, they must manage the life-threatening, multi-organ system complications. Research has yet to explain why a small subset of women

with preeclampsia with severe features develop HELLP syndrome.

Risk Factors and Clinical Presentation of HELLP Syndrome

HELLP syndrome occurs in less than 1% of pregnancies, but approximately 16% of pregnancies are complicated by preeclampsia with severe features.[72] The clinical presentation of HELLP syndrome is quite variable.

In one study, 70% of the women were pregnant and 30% were postpartum at the onset of HELLP syndrome. Of the antenatal patients 18% were term, 71% preterm (27 to 36 weeks' gestation), and 11% extremely preterm (less than 27 weeks' gestation).[72] The most common presenting reports are right upper quadrant or epigastric pain, nausea, and vomiting. Many patients will have a history of malaise or nonspecific symptoms suggesting an acute viral syndrome.[73] A subset of patients present with headache and vision disturbances consistent with preeclampsia with severe features. Advanced coagulopathy may cause hematuria or gastrointestinal bleeding. Physical findings include right upper quadrant and epigastric tenderness. Because 12% to 18% of women with HELLP syndrome are normotensive and 13% do not have proteinuria,[73] clinicians must consider HELLP syndrome in patients who lack these classic findings of preeclampsia.

Differential Diagnosis of HELLP Syndrome

One of the most difficult challenges posed by HELLP syndrome is its extensive differential diagnosis. The differential diagnosis of right upper quadrant pain includes cholecystitis, hepatitis, AFLP, gastroesophageal reflux, gastroenteritis, and pancreatitis. Urinalysis or kidney function abnormalities may suggest pyelonephritis, hemolytic uremic syndrome, or ureteral calculi. Other causes of thrombocytopenia in pregnancy include gestational thrombocytopenia, pseudothrombocytopenia, HIV, immune thrombocytopenic purpura, systemic lupus erythematosus, antiphospholipid syndrome, hypersplenism, DIC, thrombotic thrombocytopenic purpura, hemolytic uremic syndrome, congenital thrombocytopenias, and alcohol and other drug use.[74] A high index of suspicion is the key to diagnosing HELLP syndrome. Any patient with reports of right upper quadrant or epigastric pain, nausea, vomiting, or any signs of preeclampsia should be evaluated with a CBC, platelet count, and liver enzyme levels.[75]

Laboratory Diagnosis and Classification of HELLP Syndrome

Laboratory tests are used for diagnosis and as an indicator of severity in HELLP syndrome. A decreasing platelet count and increasing serum LDH level (indicative of hemolysis and liver dysfunction) reflect the severity of the disease. Thrombocytopenia also forms the basis of a commonly used classification system.[51] To diagnose HELLP syndrome, thrombocytopenia, elevated liver function tests, and hemolysis must all be present. If there is isolated thrombocytopenia or only elevated liver enzyme levels, then the diagnosis is preeclampsia with severe features. *Table 6* lists some commonly used laboratory criteria for the diagnosis of HELLP syndrome.[73]

In addition, when the platelet count is less than 50,000/mL or concerns develop regarding active bleeding due to coagulopathy,[9] then fibrinogen, fibrin degradation products or D-dimer, prothrombin, and partial thromboplastin times should be assessed to rule out superimposed DIC.

Table 6. Criteria for Laboratory Diagnosis of HELLP Syndrome

Hemolysis
Abnormal peripheral blood smear (evidence of damaged erythrocytes: schistocytes, burr cells, helmet cells)
Serum bilirubin ≥1.2 mg/dL, LDH ≥600 IU/L
Elevated Liver Enzymes
Transaminases (AST and/or ALT) > twice the upper limit of normal
Low Platelet Count
<100,000/mcL

ALT = alanine aminotransferase; AST = aspartate aminotransferase; LDH = lactate dehydrogenase.

Information from American College of Obstetricians and Gynecologists; Task Force on Hypertension in Pregnancy. Hypertension in pregnancy. Report of the American College of Obstetricians and Gynecologists' Task Force on Hypertension in Pregnancy. Obstet Gynecol. *2013;122(5):1122-1131; Barton JR, Sibai BM. Diagnosis and management of hemolysis, elevated liver enzymes, and low platelets syndrome.* Clin Perinatol. *2004;31(4):807-833, vii; Magann EF, Martin JN Jr. Twelve steps to optimal management of HELLP syndrome.* Clin Obstet Gynecol. *1999;42(3):532-550.*

Management of HELLP Syndrome

Management of HELLP syndrome follows the general guidelines for preeclampsia with severe features. All women with HELLP syndrome should receive MgSO4 from the time of hospital admission until at least 24 hours postpartum.[73]

Management issues specific to HELLP syndrome include the following:

1. Corticosteroids. Although a few small RCTs have shown improvement in laboratory measurements (particularly platelet counts) with the use of high-dose steroids,[73] a Cochrane review did not show improved maternal or fetal outcomes beyond the known benefits of corticosteroids in fetuses at less than 34 weeks' gestation.[76] The only randomized, double-blind, placebo-controlled clinical trial failed to show any improved maternal outcomes with antepartum or postpartum use of dexamethasone, except for a reduced time to platelet count recovery in women with platelet counts below 50,000/mcL.[77] Increased platelet counts may allow for the use of regional anesthesia.[78] High-dose corticosteroids are not recommended for routine use in women beyond 34 weeks' gestation or during the postpartum period.

2. Blood products. Fresh frozen plasma, platelets, and packed red blood cells may be needed to correct coagulation defects or acute hemorrhage. Women with platelet counts greater than 50,000/mcL are unlikely to experience excessive bleeding.[74] Intrapartum platelet transfusions are indicated in the presence of significant bleeding (eg, ecchymosis, bleeding from puncture sites, bleeding gums) or before a cesarean delivery if the platelet count is less than 50,000/mcL.[74] Physicians may consider platelet transfusion before an anticipated vaginal delivery if the platelet count is less than 10,000 to 20,000/mcL. However, evidence-based guidelines are lacking, and the unpredictable timing of birth may prevent this in practice. Regional anesthesia is generally considered safe in women with platelet counts above 70,000/mcL and may be reasonable at lower thresholds.[74]

3. Spontaneous rupture of a subcapsular liver hematoma. This is a life-threatening complication that must be suspected in any patient with HELLP syndrome who develops shock and massive ascites. Emergent laparotomy may be life-saving. A subcapsular hematoma may be suggested by right upper quadrant, epigastric pain, or shoulder pain. The diagnosis is confirmed by computed tomography (CT) or ultrasonography. If unruptured, the hematoma may be monitored with serial ultrasonography or CT scans in a facility with a readily available vascular or general surgeon and a blood bank prepared for massive transfusions.[75]

Delivery and Postpartum Management of HELLP Syndrome

The decision regarding timing of delivery is weighted toward earlier delivery for women with HELLP syndrome than for women with preeclampsia with severe features without HELLP syndrome. Specifically, infants greater than 28 weeks' gestation are routinely delivered 24 to 48 hours after the first maternal dose of dexamethasone or betamethasone is administered.[75] The frequency of repeating blood tests will vary based on severity and rate of progression of the disease, but every 6 to 8 hours is typical during pregnancy and every 12 hours postpartum until evidence of resolution.[27] Conservative management of HELLP syndrome remains experimental, and the clinical course is too rapid to wait for the complete steroid course before initiating delivery for most women.[10,73]

The choice between vaginal and cesarean delivery should be based on obstetric factors (eg, parity and cervical ripeness), fetal maturity, and the severity of medical complications.[73,75] Cesarean delivery carries special risks, such as bleeding due to thrombocytopenia and difficulty controlling BP because of depleted intravascular volume. The surgeon may elect to place a subfascial drain or perform secondary skin closure because of expected continued oozing. After delivery, some women with HELLP syndrome experience a period of clinical and laboratory deterioration before recovery. MgSO4 infusion is continued for at least 24 hours. The platelet count typically reaches its nadir and the LDH level peaks 24 to 48 hours after delivery.[79] Unfortunately, postpartum deterioration sometimes progresses to include hepatic rupture, renal failure, pulmonary edema, ascites, pleural effusion, postpartum hemorrhage, acute respiratory distress syndrome, DIC, or death. These patients may require prolonged intensive care with continuous cardiac monitoring, central lines, respirator support, dialysis, and other major interventions. Clinical signs of recovery include decreasing BP levels, mobilization of fluid from peripheral edema, ascites, or pleural effusions, and subsequent diuresis.

Pharmacologic Treatment for Hypertensive Disorders of Pregnancy

Magnesium Sulfate

Magnesium sulfate helps prevent seizures in women with preeclampsia,[80-82] and is more effective in preventing recurrent seizures in eclamptic patients than phenytoin, diazepam, or a lytic cocktail (chlorpromazine, promethazine, and meperidine).[59,81,83-85] The Magpie trial showed that 63 women with severe preeclampsia need to receive MgSO4 prophylaxis to prevent one eclamptic seizure[82] and a Cochrane review showed a NNT of 100 for prevention of preeclampsia when used in all women with preeclampsia.[86]

Women with preeclampsia without severe features should be monitored closely and MgSO4 administered only if they develop severe features.[10] Assuming that 50% of seizures would be prevented with the use of MgSO4, as was assumed in the Magpie trial,[82] 400 women with mild preeclampsia would need to be treated to prevent one eclamptic seizure.[87] ACOG recommends that women with preeclampsia who are not symptomatic and have BP levels less than 160/110 mm Hg should not universally receive MgSO4 for seizure prophylaxis; however, this recommendation is based on low-quality evidence and some physicians and hospitals may choose to use MgSO4 as seizure prophylaxis in women with preeclampsia without severe features.[27] When MgSO4 is not used, it is important to remain vigilant, because BP levels are only mildly elevated in 30% to 60% of women who develop eclampsia.[1] Postpartum initiation of MgSO4 may be required in women who did not require intrapartum MgSO4 and in those with new-onset hypertension with cerebral symptoms (eg, headache, blurred vision), new-onset preeclampsia with severely elevated BP levels (greater than 160/110 mm Hg), or eclampsia.

Magnesium sulfate works by slowing neuromuscular conduction and depressing central nervous system irritability. It does not significantly affect BP levels. A quarter of women have adverse effects, most commonly flushing.[86] *Table 5* presents a standard dosing regimen.

Magnesium sulfate is excreted by the kidneys. Women with normal renal function do not require routine monitoring of serum magnesium levels. However, women with absent reflexes, elevated serum creatinine levels, or decreased urine output (less than 30 mL/hour) should have magnesium levels tested every 6 hours after the loading dose has been administered to determine if adjustments in the maintenance infusion rate are necessary.[1,88] A therapeutic magnesium level is 4.8 to 9.6 mg/dL. Loss of patellar reflexes occurs when the level reaches 8 to 10 mg/dL, somnolence occurs at 10 to 12 mg/dL, and respiratory depression at 12 to 17 mg/dL.[89]

Magnesium toxicity can lead to respiratory paralysis, central nervous system depression, and cardiac arrest. With magnesium overdose, vital functions are lost in a predictable sequence. If DTRs are present, magnesium concentrations are rarely toxic.[88] The MgSO4 infusion should be discontinued and magnesium levels tested immediately if DTRs are absent, the respiratory rate is less than 12 breaths/minute, or urine output is less than 30 mL/hour.[1,88] Maternal deaths have resulted from overdose due to administration of improperly prepared solutions.[90] The antidote for MgSO4 overdose is 1 g of calcium gluconate (10 mL of a 10% solution) infused IV over 2 minutes.[50] Avoid rapid IV administration or extravasation. Use calcium gluconate with caution in women with renal failure, severe hypophosphatemia, or acidosis.

Antihypertensive Drugs

The optimal level of BP control in pregnancies complicated by hypertension is unknown.[9,91] Less tight control may decrease the risk of infants being small for gestational age, but may potentially increase the risk of respiratory distress syndrome, severe hypertension, antenatal hospitalization, and proteinuria at delivery.[9,16] In a retrospective review of 28 women with preeclampsia with severe features who experienced cerebrovascular accidents, more than 95% had SBP over 160 mm Hg, but only 12.5% had DBP over 110 mm Hg.[92]

There are several antihypertensive drugs to choose from, depending on whether the goal is acute or chronic control. For acute management, IV labetalol and hydralazine are commonly used.[10,93] Doses for IV labetalol, hydralazine, and oral nifedipine are listed in *Table 4*. A Cochrane review of drugs for treating severe hypertension in pregnancy showed no evidence that one drug had superior effectiveness.[93] The role of hydralazine as a first-line treatment has been questioned by a meta-analysis showing increased maternal hypo-

tension, tachycardia, and headaches compared with other antihypertensive drugs.[91] The need for IV antihypertensive drugs, in repeated doses or by continuous infusion, indicates a patient who is unstable and likely to need continuous monitoring and careful management.

Oral nifedipine or labetalol are alternatives to IV drugs when severely elevated BP levels require treatment. Traditionally, IV drugs have been preferred to rapidly lower BP with careful titration to avoid maternal and fetal effects of an excessive decrease in BP. In two studies, oral nifedipine has been shown to control BP more rapidly than IV labetelol,[21,94] and a third trial showed equivalent time to adequate BP control.[95] Nifedipine has been shown to cause a greater increase in cardiac index and urinary output than labetalol,[93,94] as well as decreased systemic vascular resistance. The use of these three antihypertensive drugs is supported by the ACOG 2019 Committee Opinion,[18] NICE guidelines,[16] and a Cochrane review.[92] Oral labetalol at a dose of 200 mg is considered an alternative by ACOG for lowering severely elevated BP levels when IV drugs are not an option, and is recommended in the NICE guidelines.[16,18] If BP levels remain greater than 160/110 mm Hg and IV drugs are still not an option, then 200 mg oral labetalol can be repeated.[18] It is recommended that each maternity care unit choose a single first-line drug and have alternatives available for women with elevated BP levels that are refractory to the selected drug. IV labetalol, IV hydralazine, or immediate-release oral nifedipine use in women who are pregnant or postpartum does not require cardiac monitoring.

For preeclampsia with severe features in women undergoing expectant management before 34 weeks' gestation, oral labetalol and nifedipine are acceptable options.[10] Delivery is recommended for women with preeclampsia with severe features at 34 weeks' gestation or greater.[10]

Standardized Management of Severe Hypertensive Disorders in Pregnancy

In 2017, the National Partnership for Maternal Safety published the Severe Hypertension in Pregnancy patient safety bundle. The bundle has four action domains for standardizing management: readiness, recognition and prevention, response, and reporting and systems learning (pages 16 and 17). A study of 23 California hospitals showed the benefit of standardizing management of severe hypertension. Using the California Maternal Quality Care Collaborative Preeclampsia Toolkit, the hospitals achieved a 42.6% decrease in incidence of eclampsia and a 16.7% decrease in severe maternal morbidity.[96]

Acute Fatty Liver of Pregnancy

Acute fatty liver of pregnancy is a rare condition that occurs in the third trimester and may be initially diagnosed as HELLP syndrome because of similarities in clinical and laboratory findings. The incidence of AFLP is approximately 1 in 7,000 to 15,000 pregnancies. In the 1980s, maternal mortality was as high as 85%, but earlier recognition and prompt delivery lowered the mortality rate to the current level of 10% to 15% by the 2000s.[11]

The pathophysiology of AFLP involves abnormal hepatic mitochondrial function that leads to accumulation of fat droplets in hepatocytes and culminates in sudden hepatic failure if left untreated. The etiology is unknown. Pregnant women who have fetuses with long-chain L-3-hydroxyacyl-CoA dehydrogenase (LCHAD) deficiency, a mutation affecting fatty acid oxidation, have an increased incidence of AFLP. Newborns of women with AFLP should be tested for LCHAD deficiency because affected newborns have a 75% to 90% mortality rate, which can be decreased significantly through dietary treatment.[97]

AFLP presents in the third trimester with symptoms that include nausea and vomiting (71% to 75%), upper abdominal pain (43% to 50%), malaise (31%), and jaundice (29% to 37%).[98,99] Physical examination findings are nonspecific, and the liver size is normal or small. With disease progression, liver failure develops with signs of coagulopathy, asterixis, encephalopathy, and coma. Ascites (due to portal hypertension), pancreatitis, and gastrointestinal bleeding secondary to severe vomiting, esophagitis, and associated coagulation disorders may be present.

Differential Diagnosis

Most women with AFLP are misdiagnosed on initial hospital admission. Preeclampsia and hepatitis are the most common initial diagnoses.[98] Many clinical features of AFLP overlap with those of preeclampsia and HELLP syndrome, and patients may have both conditions. Approximately half of patients with AFLP will have hypertension,

proteinuria, or edema. Acute hepatitis and liver damage secondary to drugs or toxins should also be considered in the differential diagnosis.

The diagnosis of AFLP is heavily dependent on laboratory findings. Early in the disease course, bilirubin levels are elevated (usually less than 5 mg/dL) and may be detected in the urine. The international normalized ratio and activated partial thromboplastin time are prolonged, whereas the platelet count is only mildly decreased (100,000 to 150,000/mcL). This contrasts with HELLP syndrome, where significant thrombocytopenia is an early finding and bilirubin is typically normal.[51,100] In AFLP, the AST and ALT are typically elevated, but not to the extent that would be expected with acute infectious hepatitis. Appropriate serologic tests for acute infectious hepatitis can further clarify the diagnosis. In one case series, all women with AFLP had laboratory evidence of DIC, including markedly decreased antithrombin III levels.[98] Hypoglycemia is common in AFLP and can help distinguish HELLP syndrome from AFLP, but its absence does not exclude AFLP. A case series of 51 women showed kidney injury in almost all cases, with 76% having creatinine levels of 1.5 mg/dL or higher.[101] Radiologic tests are of limited usefulness in diagnosing AFLP because ultrasonography, CT scans, and magnetic resonance imaging of the liver all have high false negative rates.[98] Liver biopsy can confirm the diagnosis of AFLP but is invasive and not typically necessary to proceed with treatment.[98]

Treatment

The most important treatment for AFLP is delivery, because the disease does not resolve while the patient is still pregnant and severe complications can develop if delivery is delayed. As is the case with preeclampsia and HELLP syndrome, the choice between vaginal and cesarean delivery should be based on obstetric factors, fetal maturity, and the severity of medical complications.[98] Hepatotoxic general anesthetics should be avoided. Coagulopathy should be corrected, but infusion of antithrombin has not been shown to improve clinical outcomes.[98] Hypoglycemia may be corrected with infusions of 10% dextrose supplemented by boluses of 50% dextrose.[98] If diagnosis and delivery are accomplished early, postpartum improvement is typically rapid. The Parkland Hospital study showed resolution of ongoing hepatic necrosis occurring within a few days of delivery and clinical improvement to be common by 3 to 4 days postpartum. However, laboratory evidence of AFLP can persist for 7 to 10 days or more.[101] Rarely, liver transplantation has been required for multisystem failure that does not improve with delivery.[102] If AFLP continues to worsen after delivery, plasmapheresis may be used, and it showed promising results in a Chinese case series of 39 women.[103] The rarity of AFLP and usual postpartum clinical improvement make clinical trials unlikely.

Summary

Multiple medical complications can evolve during pregnancy. This chapter aims to better clinician understanding of the risk factors, diagnosis, and management of hypertensive disorders of pregnancy, eclampsia, HELLP syndrome, and AFLP. The key to diagnosing these conditions is clinical vigilance coupled with appropriate laboratory or imaging studies. A common clinical challenge is balancing maternal and fetal well-being in diagnostic and treatment decisions.

Nursing Considerations: Hypertensive Disorders of Pregnancy

- Educate women about signs and symptoms to report
- Think safety: appropriate blood pressure cuff size, seizure precautions, monitor urine output, medication adverse effects, and antidote for magnesium sulfate toxicity
- Advocate for unit huddles for high-risk patients and post-event debriefs
- Facilitate team efforts for optimizing patient safety, including in-situ drills
- Champion the Maternal Safety Bundle: Severe Hypertension in Pregnancy in your institution

COUNCIL ON PATIENT SAFETY
IN WOMEN'S HEALTH CARE
safe health care for every woman

PATIENT SAFETY BUNDLE

Hypertension

READINESS

Every Unit

- Standards for early warning signs, diagnostic criteria, monitoring and treatment of severe preeclampsia/eclampsia (include order sets and algorithms)
- Unit education on protocols, unit-based drills (with post-drill debriefs)
- Process for timely triage and evaluation of pregnant and postpartum women with hypertension including ED and outpatient areas
- Rapid access to medications used for severe hypertension/eclampsia: Medications should be stocked and immediately available on L&D and in other areas where patients may be treated. Include brief guide for administration and dosage.
- System plan for escalation, obtaining appropriate consultation, and maternal transport, as needed

RECOGNITION & PREVENTION

Every Patient

- Standard protocol for measurement and assessment of BP and urine protein for all pregnant and postpartum women
- Standard response to maternal early warning signs including listening to and investigating patient symptoms and assessment of labs (e.g. CBC with platelets, AST and ALT)
- Facility-wide standards for educating prenatal and postpartum women on signs and symptoms of hypertension and preeclampsia

May 2015

COUNCIL ON PATIENT SAFETY
IN WOMEN'S HEALTH CARE
safe health care for every woman

PATIENT SAFETY BUNDLE

Hypertension

RESPONSE

Every case of severe hypertension/preeclampsia

- Facility-wide standard protocols with checklists and escalation policies for management and treatment of:
  - Severe hypertension
  - Eclampsia, seizure prophylaxis, and magnesium over-dosage
  - Postpartum presentation of severe hypertension/preeclampsia
- Minimum requirements for protocol:
  - Notification of physician or primary care provider if systolic BP =/> 160 or diastolic BP =/> 110 for two measurements within 15 minutes
  - After the second elevated reading, treatment should be initiated ASAP (preferably within 60 minutes of verification)
  - Includes onset and duration of magnesium sulfate therapy
  - Includes escalation measures for those unresponsive to standard treatment
  - Describes manner and verification of follow-up within 7 to 14 days postpartum
  - Describe postpartum patient education for women with preeclampsia
- Support plan for patients, families, and staff for ICU admissions and serious complications of severe hypertension

REPORTING/SYSTEMS LEARNING

Every unit

- Establish a culture of huddles for high risk patients and post-event debriefs to identify successes and opportunities
- Multidisciplinary review of all severe hypertension/eclampsia cases admitted to ICU for systems issues
- Monitor outcomes and process metrics

Note: "Facility-wide" indicates all areas where pregnant or postpartum women receive care. (E.g. L&D, postpartum critical care, emergency department, and others depending on the facility).

Standardization of health care processes and reduced variation has been shown to improve outcomes and quality of care. The Council on Patient Safety in Women's Health Care disseminates patient safety bundles to help facilitate the standardization process. This bundle reflects emerging clinical, scientific, and patient safety advances as of the date issued and is subject to change. The information should not be construed as dictating an exclusive course of treatment or procedure to be followed. Although the components of a particular bundle may be adapted to local resources, standardization within an institution is strongly encouraged.

The Council on Patient Safety in Women's Health Care is a broad consortium of organizations across the spectrum of women's health for the promotion of safe health care for every woman.

May 2015

Reprinted from Council on Patient Safety in Women's Health Care. AIM-Supported Patient Safety Bundles. Severe Hypertension in Pregnancy. Available at https://safehealthcareforeverywoman.org/.

References

1. Sibai BM. Diagnosis, prevention, and management of eclampsia. *Obstet Gynecol*. 2005;105(2):402-410.
2. Aagaard-Tillery KM, Belfort MA. Eclampsia: morbidity, mortality, and management. *Clin Obstet Gynecol*. 2005; 48(1):12-23.
3. Churchill D, Duley L, Thornton JG, et al. Interventionist versus expectant care for severe pre-eclampsia between 24 and 34 weeks' gestation. *Cochrane Database Syst Rev*. 2018;10:CD003106.
4. Odendaal HJ, Pattinson RC, Bam R, et al. Aggressive or expectant management for patients with severe preeclampsia between 28-34 weeks' gestation: a randomized controlled trial. *Obstet Gynecol*. 1990;76(6): 1070-1075.
5. Publications Committee, Society for maternal-Fetal Medicine; Sibai BM. Evaluation and management of severe preeclampsia before 34 weeks' gestation. *Am J Obstet Gynecol*. 2011;205(3):191-198.
6. Sibai BM, Mercer BM, Schiff E, Friedman SA. Aggressive versus expectant management of severe preeclampsia at 28 to 32 weeks' gestation: a randomized controlled trial. *Am J Obstet Gynecol*. 1994;171(3):818-822.
7. Society for Maternal-Fetal Medicine (SMFM) Publications Committee. Implementation of the use of antenatal corticosteroids in the late preterm birth period in women at risk for preterm delivery. *Am J Obstet Gynecol*. 2016; 215(2):B13-B15. Erratum in *Am J Obstet Gynecol*. 2016; 215(2):B14.
8. Committee on Obstetric Practice. Committee Opinion no. 713. Antenatal corticosteroid therapy for fetal maturation. *Obstet Gynecol*. 2017;130(2):e102-e109.
9. Abalos E, Duley L, Steyn DW, Gialdini C. Antihypertensive drug therapy for mild to moderate hypertension during pregnancy. *Cochrane Database Syst Rev*. 2018; 10:CD002252.
10. American College of Obstetricians and Gynecologists; Task Force on Hypertension in Pregnancy. Hypertension in pregnancy. Report of the American College of Obstetricians and Gynecologists' Task Force on Hypertension in Pregnancy. *Obstet Gynecol*. 2013;122(5): 1122-1131.
11. Liu J, Ghaziani TT, Wolf JL. Acute fatty liver disease of pregnancy: updates in pathogenesis, diagnosis, and management. *Am J Gastroenterol*. 2017;112(6):838-846.
12. Petersen EE, Davis NL, Goodman D, et al. Vital signs: pregnancy-related deaths, United States, 2011-2015, and strategies for prevention, 13 States, 2013-2017. *MMWR Morb Mortal Wkly Rep*. 2019;68(18):423-429.
13. American College of Obstetricians and Gynecologists. ACOG Practice Bulletin no. 203: chronic hypertension in pregnancy. *Obstet Gynecol*. 2019;133(1):e26-e50.
14. Ankumah N-A, Cantu J, Jauk V, et al. Risk of adverse pregnancy outcomes in women with mild chronic hypertension before 20 weeks of gestation. *Obstet Gynecol*. 2014;123(5):966-972.
15. Magee LA, von Dadelszen P, Rey E, et al. Less-tight versus tight control of hypertension in pregnancy. *N Engl J Med*. 2015;372(5):407-417.
16. National Collaborating Centre for Women's and Children's Health (UK). Hypertension in Pregnancy: *The Management of Hypertensive Disorders During Pregnancy.* London: RCOG Press; 2010.
17. Easterling T, Mundle S, Bracken H, et al. Oral antihypertensive regimens (nifedipine retard, labetalol, and methyldopa) for management of severe hypertension in pregnancy: an open-label, randomised controlled trial. *Lancet*. 2019;394(10203):1011-1021.
18. American College of Obstetricians and Gynecologists. ACOG Committee Opinion no. 767: Emergent therapy for acute-onset, severe hypertension during pregnancy and the postpartum period. *Obstet Gynecol*. 2019; 133(2):e174-e180.
19. Churchill D, Beevers GD, Meher S, Rhodes C. Diuretics for preventing pre-eclampsia. *Cochrane Database Syst Rev*. 2007;(1):CD004451.
20. Whelton PK, Carey RM, Aronow WS, et al. 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. *Hypertension*. 2018;71(6):e13-e115. Erratum in *Hypertension*. 2018;71(6):e140-e144.
21. Shekhar S, Sharma C, Thakur S, Verma S. Oral nifedipine or intravenous labetalol for hypertensive emergency in pregnancy: a randomized controlled trial. *Obstet Gynecol*. 2013;122(5):1057-1063.
22. Barton JR, O'brien JM, Bergauer NK, et al. Mild gestational hypertension remote from term: progression and outcome. *Am J Obstet Gynecol*. 2001;184(5):979-983.
23. Gofton EN, Capewell V, Natale R, Gratton RJ. Obstetrical intervention rates and maternal and neonatal outcomes of women with gestational hypertension. *Am J Obstet Gynecol*. 2001;185(4):798-803.
24. Buchbinder A, Sibai BM, Caritis S, et al; National Institute of Child Health and Human Development Network of Maternal-Fetal Medicine Units. Adverse perinatal outcomes are significantly higher in severe gestational hypertension than in mild preeclampsia. *Am J Obstet Gynecol*. 2002;186(1):66-71.
25. Cruz MO, Gao W, Hibbard JU. Obstetrical and perinatal outcomes among women with gestational hypertension, mild preeclampsia, and mild chronic hypertension. *Am J Obstet Gynecol*. 2011;205(3):260.e1-260.e9.
26. Bibbins-Domingo K, Grossman DC, Curry SJ, et al; US Preventive Services Task Force. Screening for preeclampsia: US Preventive Services Task Force Recommendation Statement. *JAMA*. 2017;317(16):1661-1667.
27. American College of Obstetricians and Gynecologists. ACOG Practice Bulletin no. 202: gestational hypertension and preeclampsia. *Obstet Gynecol*. 2019;133(1): e1-e25.
28. Davison JM, Homuth V, Jeyabalan A, et al. New aspects in the pathophysiology of preeclampsia. *J Am Soc Nephrol*. 2004;15(9):2440-2448.
29. Rodriguez-Thompson D, Lieberman ES. Use of a random urinary protein-to-creatinine ratio for the diagnosis of significant proteinuria during pregnancy. *Am J Obstet Gynecol*. 2001;185(4):808-811.

30. Côté AM, Brown MA, Lam E, et al. Diagnostic accuracy of urinary spot protein:creatinine ratio for proteinuria in hypertensive pregnant women: systematic review. *BMJ*. 2008;336(7651):1003-1006.
31. Kenny LC, Black MA, Poston L, et al. Early pregnancy prediction of preeclampsia in nulliparous women, combining clinical risk and biomarkers: the Screening for Pregnancy Endpoints (SCOPE) international cohort study. *Hypertension*. 2014;64(3):644-652.
32. Zeisler H, Llurba E, Chantraine F, et al. Predictive value of the sFlt-1:PlGF ratio in women with suspected pre-eclampsia. *N Engl J Med*. 2016;374(1):13-22.
33. National Institute for Health and Care Excellence. PlGF-based testing to help diagnose suspected pre-eclampsia (Triage PlGF test, Elecsys immunoassay sFlt-1/PlGF ratio, DELFIA Xpress PlGF 1-2-3 test, and BRAHMS sFlt-1 Kryptor/BRAHMS PlGF plus Kryptor PE ratio). 2016. Available at https://www.nice.org.uk/guidance/dg23/resources/plgfbased-testing-to-help-diagnose-suspected-preeclampsia-triage-plgf-test-elecsys-immunoassay-sflt1plgf-ratio-delfia-xpress-plgf-123-test-and-brahms-sflt1-kryptorbrahms-plgf-plus-kryptor-pe-ratio-pdf-1053688576453.
34. Villar J, Purwar M, Merialdi M, et al; WHO Vitamin C and Vitamin E trial group. World Health Organisation multicentre randomised trial of supplementation with vitamins C and E among pregnant women at high risk for pre-eclampsia in populations of low nutritional status from developing countries. *BJOG*. 2009;116(6):780-788.
35. Hofmeyr GJ, Lawrie TA, Atallah AN, Torloni MR. Calcium supplementation during pregnancy for preventing hypertensive disorders and related problems. *Cochrane Database Syst Rev*. 2018;10:CD001059.
36. World Health Organization. Guideline: *Calcium supplementation in pregnant women*. Geneva: World Health Organization; 2013.
37. Duley L, Henderson-Smart DJ, Meher S, King JF. Antiplatelet agents for preventing pre-eclampsia and its complications. *Cochrane Database Syst Rev*. 2007;(2):CD004659.
38. Henderson JT, Whitlock EP, O' Conner E, et al. Low-Dose Aspirin for Prevention of Morbidity and Mortality from Preeclampsia: A Systematic Evidence Review for the U.S. Preventive Services Task Force. Rockville, MD: Agency for Healthcare Research and Quality; 2014.
39. Werner EF, Hauspurg AK, Rouse DJ. A cost-benefit analysis of low-dose aspirin prophylaxis for the prevention of preeclampsia in the United States. *Obstet Gynecol*. 2015;126(6):1242-1250.
40. The American College of Obstetricians and Gynecologists. Practice advisory on low-dose aspirin and prevention of preeclampsia: updated recommendations. 2016. Available at http://www.losolivos-obgyn.com/info/md/acog/Low-dose%20aspirin,%20ACOG%20Practice%20Advisory%202016.pdf.
41. Rolnik DL, Wright D, Poon LC, et al. Aspirin versus placebo in pregnancies at high risk for preterm preeclampsia. *N Engl J Med*. 2017;377(7):613-622.
42. Wright D, Poon LC, Rolnik DL, et al. Aspirin for Evidence-Based Preeclampsia Prevention trial: influence of compliance on beneficial effect of aspirin in prevention of preterm preeclampsia. *Am J Obstet Gynecol*. 2017;217(6):685.e1-685.e5.
43. Roberge S, Nicolaides K, Demers S, et al. The role of aspirin dose on the prevention of preeclampsia and fetal growth restriction: systematic review and meta-analysis. *Am J Obstet Gynecol*. 2017;216(2):110-120.e.
44. Koopmans CM, Bijlenga D, Groen H, et al; HYPITAT study group. Induction of labour versus expectant monitoring for gestational hypertension or mild pre-eclampsia after 36 weeks' gestation (HYPITAT): a multicentre, open-label randomised controlled trial. *Lancet*. 2009;374(9694):979-988.
45. Spong CY, Mercer BM, D'alton M, et al. Timing of indicated late-preterm and early-term birth. *Obstet Gynecol*. 2011;118(2 Pt 1):323-333.
46. Tajik P, van der Tuuk K, Koopmans CM, et al. Should cervical favourability play a role in the decision for labour induction in gestational hypertension or mild pre-eclampsia at term? An exploratory analysis of the HYPITAT trial. *BJOG*. 2012;119(9):1123-1130.
47. Vijgen SM, Koopmans CM, Opmeer BC, et al; HYPITAT study group. An economic analysis of induction of labour and expectant monitoring in women with gestational hypertension or pre-eclampsia at term (HYPITAT trial). *BJOG*. 2010;117(13):1577-1585.
48. Cluver C, Novikova N, Koopmans CM, West HM. Planned early delivery versus expectant management for hypertensive disorders from 34 weeks gestation to term. *Cochrane Database Syst Rev*. 2017;1:CD009273.
49. Ramin SM, Vidaeff AC, Yeomans ER, Gilstrap LC. Chronic renal disease in pregnancy. *Obstet Gynecol*. 2006;108(6):1531-1539.
50. Dildy GA. Complications of preeclampsia. In: Dildy GA, Belfort MA, Saade GR, Phelan JP, Hankins GDV, Clark SL, eds. *Critical Care Obstetrics*. 4th ed. Malden, Massachusetts: Blackwell Science; 2004.
51. Magann EF, Martin JN Jr. Twelve steps to optimal management of HELLP syndrome. *Clin Obstet Gynecol*. 1999;42(3):532-550.
52. Duley L, Williams J, Henderson-Smart DJ. Plasma volume expansion for treatment of women with pre-eclampsia. *Cochrane Database Syst Rev*. 2000;(2):CD001805.
53. Gyamfi-Bannerman C, Thom EA, Blackwell SC, et al; NICHD Maternal–Fetal Medicine Units Network. Antenatal betamethasone for women at risk for late preterm delivery. *N Engl J Med*. 2016;374(14):1311-1320.
54. Vigil-De Gracia P, Reyes Tejada O, Calle Miñaca A, et al. Expectant management of severe preeclampsia remote from term: the MEXPRE Latin Study, a randomized, multicenter clinical trial. *Am J Obstet Gynecol*. 2013;209(5):425.e1-425.e8.
55. Sibai BM. What to expect from expectant management in severe preeclampsia at <34 weeks gestation: pregnancy outcomes in developed vs developing countries. *Am J Obstet Gynecol*. 2013;209(5):400-401.

56. American College of Obstetricians and Gynecologists. Practice Bulletin no. 159: Management of preterm labor. *Obstet Gynecol*. 2016;127(1):e29-e38.
57. Mattar F, Sibai BM. Eclampsia. VIII. Risk factors for maternal morbidity. *Am J Obstet Gynecol*. 2000;182(2): 307-312.
58. Cooray SD, Edmonds SM, Tong S, et al. Characterization of symptoms immediately preceding eclampsia. *Obstet Gynecol*. 2011;118(5):995-999.
59. Eclampsia trial collaborative group. Which anticonvulsant for women with eclampsia? Evidence from the Collaborative Eclampsia Trial. *Lancet*. 1995;345(8963): 1455-1463.
60. American Heart Association. Guidelines 2000 for cardiopulmonary resuscitation and emergency cardiovascular care. Part 3: adult basic life support. The American Heart Association in collaboration with the International Liaison Committee on Resuscitation. *Circulation*. 2000;102(8)(Suppl):I22-I59.
61. Miguil M, Chekairi A. Eclampsia, study of 342 cases. *Hypertens Pregnancy*. 2008;27(2):103-111.
62. Centers for Disease Control and Prevention. Reproductive Health. Pregnancy Mortality Surveillance System. 2017. Available at https://www.cdc.gov/reproductivehealth/maternal-mortality/pregnancy-mortality-surveillance-system.htm.
63. Creanga AA, Syverson C, Seed K, Callaghan WM. Pregnancy-related mortality in the United States, 2011-2013. *Obstet Gynecol*. 2017;130(2):366-373.
64. MacKay AP, Berg CJ, Atrash HK. Pregnancy-related mortality from preeclampsia and eclampsia. *Obstet Gynecol*. 2001;97(4):533-538.
65. Maia SB, Katz L, Neto CN, et al. Abbreviated (12-hour) versus traditional (24-hour) postpartum magnesium sulfate therapy in severe pre-eclampsia. *Int J Gynaecol Obstet*. 2014;126(3):260-264.
66. Vigil-DeGracia P, Ludmir J, Ng J, et al. Is there benefit to continue magnesium sulphate postpartum in women receiving magnesium sulphate before delivery? A randomised controlled study. *BJOG*. 2018;125(10):1304-1311.
67. Viteri OA, England JA, Alrais MA, et al. Association of nonsteroidal antiinflammatory drugs and postpartum hypertension in women with preeclampsia with severe features. *Obstet Gynecol*. 2017;130(4):830-835.
68. Blue NR, Murray-Krezan C, Drake-Lavelle S, et al. Effect of ibuprofen vs acetaminophen on postpartum hypertension in preeclampsia with severe features: a double-masked, randomized controlled trial. *Am J Obstet Gynecol*. 2018;218(6):616.e1-616.e8.
69. Smith AM, Young P, Blosser CC, Poole AT. Multimodal stepwise approach to reducing in-hospital opioid use after cesarean delivery: a quality improvement initiative. *Obstet Gynecol*. 2019;133(4):700-706.
70. Magee L, von Dadelszen P. Prevention and treatment of postpartum hypertension. *Cochrane Database Syst Rev*. 2013;(4):CD004351.
71. Weinstein L. Syndrome of hemolysis, elevated liver enzymes, and low platelet count: a severe consequence of hypertension in pregnancy. *Am J Obstet Gynecol*. 1982;142(2):159-167.
72. Sibai BM, Ramadan MK, Usta I, et al. Maternal morbidity and mortality in 442 pregnancies with hemolysis, elevated liver enzymes, and low platelets (HELLP syndrome). *Am J Obstet Gynecol*. 1993;169(4):1000-1006.
73. Sibai BM. Diagnosis, controversies, and management of the syndrome of hemolysis, elevated liver enzymes, and low platelet count. *Obstet Gynecol*. 2004;103(5 Pt 1): 981-991.
74. American College of Obstetricians and Gynecologists. ACOG Practice Bulletin no. 207: thrombocytopenia in pregnancy. *Obstet Gynecol*. 2019;133(3):e181-e193.
75. Barton JR, Sibai BM. Diagnosis and management of hemolysis, elevated liver enzymes, and low platelets syndrome. *Clin Perinatol*. 2004;31(4):807-833..
76. Woudstra DM, Chandra S, Hofmeyr GJ, Dowswell T. Corticosteroids for HELLP (hemolysis, elevated liver enzymes, low platelets) syndrome in pregnancy. *Cochrane Database Syst Rev*. 2010;(9):CD008148.
77. Fonseca JE, Méndez F, Cataño C, Arias F. Dexamethasone treatment does not improve the outcome of women with HELLP syndrome: a double-blind, placebo-controlled, randomized clinical trial. *Am J Obstet Gynecol*. 2005;193(5):1591-1598.
78. O'Brien JM, Shumate SA, Satchwell SL, et al. Maternal benefit of corticosteroid therapy in patients with HELLP (hemolysis, elevated liver enzymes, and low platelet count) syndrome: impact on the rate of regional anesthesia. *Am J Obstet Gynecol*. 2002;186(3):475-479.
79. Martin JN Jr, Blake PG, Perry KG Jr, et al. The natural history of HELLP syndrome: patterns of disease progression and regression. *Am J Obstet Gynecol*. 1991; 164(6 Pt 1):1500-1509, discussion 1509-1513.
80. Belfort MA, Anthony J, Saade GR, Allen JC Jr; Nimodipine Study Group. A comparison of magnesium sulfate and nimodipine for the prevention of eclampsia. *N Engl J Med*. 2003;348(4):304-311.
81. Lucas MJ, Leveno KJ, Cunningham FG. A comparison of magnesium sulfate with phenytoin for the prevention of eclampsia. *N Engl J Med*. 1995;333(4):201-205.
82. Altman D, Carroli G, Duley L, et al; Magpie Trial Collaboration Group. Do women with pre-eclampsia, and their babies, benefit from magnesium sulphate? The Magpie Trial: a randomised placebo-controlled trial. *Lancet*. 2002;359(9321):1877-1890.
83. Duley L, Gülmezoglu AM, Chou D. Magnesium sulphate versus lytic cocktail for eclampsia. *Cochrane Database Syst Rev*. 2010;(9):CD002960.
84. Duley L, Henderson-Smart DJ, Chou D. Magnesium sulphate versus phenytoin for eclampsia. *Cochrane Database Syst Rev*. 2010;(10):CD000128.
85. Duley L, Henderson-Smart DJ, Walker GJ, Chou D. Magnesium sulphate versus diazepam for eclampsia. *Cochrane Database Syst Rev*. 2010;(12):CD000127.
86. Duley L, Gülmezoglu AM, Henderson-Smart DJ, Chou D. Magnesium sulphate and other anticonvulsants for women with pre-eclampsia. *Cochrane Database Syst Rev*. 2010;(11):CD000025.
87. Sibai BM. Magnesium sulfate prophylaxis in preeclampsia: evidence from randomized trials. *Clin Obstet Gynecol*. 2005;48(2):478-488.

88. Lu JF, Nightingale CH. Magnesium sulfate in eclampsia and pre-eclampsia: pharmacokinetic principles. *Clin Pharmacokinet*. 2000;38(4):305-314.

89. Cunningham F, Leveno KJ, Bloom SL, et al. *Williams Obstetrics*. 23rd ed. New York: McGraw-Hill Medical; 2009:737.

90. Sibai BM. Magnesium sulfate prophylaxis in preeclampsia: lessons learned from recent trials. *Am J Obstet Gynecol*. 2004;190(6):1520-1526.

91. von Dadelszen P, Magee LA. Antihypertensive medications in management of gestational hypertension-preeclampsia. *Clin Obstet Gynecol*. 2005;48(2):441-459.

92. Martin JN Jr, Thigpen BD, Moore RC, et al. Stroke and severe preeclampsia and eclampsia: a paradigm shift focusing on systolic blood pressure. *Obstet Gynecol*. 2005;105(2):246-254.

93. Duley L, Meher S, Jones L. Drugs for treatment of very high blood pressure during pregnancy. *Cochrane Database Syst Rev*. 2013;(7):CD001449.

94. Vermillion ST, Scardo JA, Newman RB, Chauhan SP. A randomized, double-blind trial of oral nifedipine and intravenous labetalol in hypertensive emergencies of pregnancy. *Am J Obstet Gynecol*. 1999;181(4):858-861.

95. Scardo JA, Vermillion ST, Newman RB, et al. A randomized, double-blind, hemodynamic evaluation of nifedipine and labetalol in preeclamptic hypertensive emergencies. *Am J Obstet Gynecol*. 1999;181(4):862-866.

96. Shields LE, Wiesner S, Klein C, et al. Early standardized treatment of critical blood pressure elevations is associated with a reduction in eclampsia and severe maternal morbidity. *Am J Obstet Gynecol*. 2017;216(4):415.e1-415.e5.

97. Ibdah JA, Bennett MJ, Rinaldo P, et al. A fetal fatty-acid oxidation disorder as a cause of liver disease in pregnant women. *N Engl J Med*. 1999;340(22):1723-1731.

98. Castro MA, Fassett MJ, Reynolds TB, et al. Reversible peripartum liver failure: a new perspective on the diagnosis, treatment, and cause of acute fatty liver of pregnancy, based on 28 consecutive cases. *Am J Obstet Gynecol*. 1999;181(2):389-395.

99. Fesenmeier MF, Coppage KH, Lambers DS, et al. Acute fatty liver of pregnancy in 3 tertiary care centers. *Am J Obstet Gynecol*. 2005;192(5):1416-1419.

100. Steingrub JS. Pregnancy-associated severe liver dysfunction. *Crit Care Clin*. 2004;20(4):763-776.

101. Nelson DB, Yost NP, Cunningham FG. Acute fatty liver of pregnancy: clinical outcomes and expected duration of recovery. *Am J Obstet Gynecol*. 2013;209(5):456.e1-456.e7.

102. Amon E, Allen SR, Petrie RH, Belew JE. Acute fatty liver of pregnancy associated with preeclampsia: management of hepatic failure with postpartum liver transplantation. *Am J Perinatol*. 1991;8(4):278-279.

102. Jin F, Cao M, Bai Y, et al. Therapeutic effects of plasma exchange for the treatment of 39 patients with acute fatty liver of pregnancy. *Discov Med*. 2012;13(72):369-373.

Late Pregnancy Bleeding

Learning Objectives

1. Identify major causes of vaginal bleeding in the second half of pregnancy.
2. Describe a systematic approach to identifying the cause of bleeding.
3. Describe treatment options based on diagnosis.

Introduction

Vaginal bleeding after midpregnancy is associated with maternal and fetal risks. In addition to maternal morbidity secondary to acute hemorrhage and operative delivery, the fetus may be compromised by uteroplacental insufficiency, preterm birth, and perinatal mortality.[1,2] Optimal management of late pregnancy bleeding depends on accurate identification of the cause and timely intervention.

Causes of Late Pregnancy Bleeding

The four conditions that account for most cases of serious or life-threatening hemorrhage are placenta previa, placental abruption, uterine rupture (uterine scar disruption), and vasa previa.[1,2] Trauma is a major cause of upper and lower genital tract bleeding in pregnancy in the setting of the aforementioned conditions. Nonemergent causes of bleeding include cervical dilatation during normal labor, which is commonly accompanied by a small amount of blood or blood-tinged mucus (*bloody show*). Many pregnant women experience spotting or minor bleeding after sexual intercourse or after digital examination. Cervicitis, cervical ectropion, cervical polyps, and cervical cancer are other possible causes of minor vaginal bleeding.[1,2] Other nongenital tract causes of bleeding, such as bladder or kidney stones, urinary tract infection, internal or external hemorrhoids, or lower gastrointestinal bleeding also should be considered.[3] Risk factors for major causes of late pregnancy bleeding in pregnancy are listed in *Table 1*.

Management of Antepartum Hemorrhage

The initial management of significant bleeding in late pregnancy is similar regardless of the etiology and is focused on assessing maternal and fetal well-being. The history, physical examination, ultrasonography for placental location, and a brief period of observation typically differentiate minor from serious causes of antepartum bleeding and will determine the need for urgent delivery versus expectant management. Evaluation with a sterile speculum examination may be performed safely before ultrasonographic evaluation; however, digital examination should not be performed until ultrasonography excludes placenta previa.[2]

Maternal vital signs and circulatory stability are the first steps along with establishing intravenous (IV) access and beginning prompt fluid resuscitation, as indicated. Although normal heart rate increases by 25% in pregnancy,[4] tachycardia and hypotension should be considered serious in the setting of antepartum bleeding. Signs and symptoms of shock are late findings in pregnant women, and the earliest signs of hypovolemic shock may not appear until after more than 1,000 mL of maternal blood loss.[5] Women with hypertensive disorders of pregnancy may have decreased intravascular volume and may exhibit signs of hemodynamic changes at lower blood losses. Hypotension, tachycardia, and maternal symptoms of hemodynamic instability are ominous indicators, and women with these signs require immediate fluid resuscitation, activation of a massive transfusion protocol, and preparation for a potential emergent cesarean delivery.[6]

The history should guide the physical examination and survey for trauma. Examination of the maternal abdomen should include assessment of the fetal heart rate (FHR), fundal height, estimated fetal weight, fetal presentation, location of tenderness (if present), and palpation for uterine contractions. Visual estimates of blood loss should be recorded but may be inaccurate or fail to account for concealed hemorrhage. Continuous fetal monitoring is recommended to determine if there is a fetal indication for urgent operative delivery.[7] FHR decelerations, tachycardia, or loss of variability may resolve with adequate maternal resuscitation. However, a persistently concerning FHR tracing (Category III or Category II without variability) may require delivery before the etiology of the hemorrhage is established.

Patients who present with vaginal bleeding should, at a minimum, have a complete blood count (CBC) and a blood type and antibody screen (type and screen) completed as soon as possible. With major hemorrhage, the previous type and screen results should be cross-matched to assist in timely transfusion in addition to the completion of coagulation studies, blood urea nitrogen, creatinine, and liver function studies.[2] The interpretation of laboratory results requires knowledge of how these values may differ in pregnancy.[8] Initial laboratory testing may not indicate coagulopathy; therefore, testing should be repeated if clinical suspicion remains.

Table 1. Risk Factors for Major Causes of Late Pregnancy Bleeding

Placenta Previa
- Advanced maternal age (>40 years)
- Chronic hypertension
- Multiparity
- Multiple gestation
- Previous cesarean delivery
- Tobacco use
- Previous uterine curettage
- Previous uterine surgery
- Cocaine use
- History of placenta previa
- IVF
- Male fetal sex

Placental Abruption
- Hypertensive disorders of pregnancy
- Multiparity
- Previous abruption
- Iron deficiency anemia
- Short umbilical cord
- Sudden decompression of an overdistended uterus
- Thrombophilias
- Chorioamnionitis
- Tobacco, cocaine, or methamphetamine use
- Trauma: blunt abdominal or sudden deceleration
- Unexplained elevated maternal alpha fetoprotein level
- Uterine fibroids

Uterine Rupture
- Abnormal placentation
- History of uterine surgery
- Labor induction (especially prostaglandins)
- Maternal connective tissue disease
- Obstructed labor
- Trauma
- Labor after cesarean delivery
- Uterine anomalies or uterine overdistension
- Inappropriate oxytocin administration
- Adenomyosis

Vasa Previa
- IVF
- Low-lying and second trimester placenta previa
- Multiple gestation
- Succenturiate-lobed and bilobed placenta
- Velamentous cord insertion

IVF = in vitro fertilization.

Information from Sakornbut E, Leeman L, Fontaine P. Late pregnancy bleeding. Am Fam Physician. *2007;75:119-206; Sheiner E, Shoham-Vardi I, Hallak M, et al. Placenta previa: obstetric risk factors and pregnancy outcome.* J Matern Fetal Med. *2001;10(6):414-419.*

Thrombocytopenia is the most common laboratory abnormality in disseminated intravascular coagulation (DIC),[9] but when isolated thrombocytopenia is present on admission it presents a diagnostic dilemma. In the presence of bleeding, DIC should be strongly considered. However, the differential diagnosis of isolated thrombocytopenia includes gestational thrombocytopenia, which typically manifests as a mild decrease in platelet count (greater than 100,000 to 150,000/µL), although in rare cases it can manifest in ranges between 75,000 to 100,000/µL.[10,11] Additional causes of isolated thrombocytopenia include primary and secondary immune thrombocytopenia, drug-induced thrombocytopenia, type IIb von Willebrand disease, and congenital thrombocytopenia. If these causes are suspected, evaluation should be performed with history and additional laboratory testing.[10] Initial admission laboratory test results should be compared with historical values if available.

Fibrinogen levels are higher in pregnancy, often greater than 400 mg/dL, therefore, values of 250 to 350 mg/dL, which are normal for nonpregnant women, may be abnormal in pregnancy; fibrinogen levels less than 200 mg/dL may indicate DIC.[12] Normal plasma thromboplastin and partial thromboplastin time values are shorter in pregnancy. D-dimer and other markers of fibrinolysis may be used for the evaluation of DIC in pregnancy but have a low specificity because moderate fibrinolysis may occur normally in pregnancy and thus the test lacks reliability to predict DIC in this population, except at extremely elevated values.[13]

The laboratory findings consistent with a diagnosis of DIC in order of importance are platelets (decreasing value), prothrombin time (prolongation), fibrin degradation products (increasing), and fibrinogen (decreasing).[9] If coagulation studies are not readily available, a serum sample may be obtained using a plain red top tube taped to the wall for a simple and inexpensive *wall test*. If no clot or a poor-quality clot is present after 6 minutes, then coagulopathy is present.[14] Women who are Rh negative should receive Rh_o (D)-immune globulin G; a Kleihauer-Betke test can be performed to determine the appropriate dose.[12,15]

Placenta Previa

Definitions and Pathophysiology

Placenta previa occurs when the placenta implants in a location overlying the internal cervical os.[16] The pathophysiology of placenta previa is not fully understood. Normally, placental implantation favors a fundal location. As pregnancy progresses, the apparent *migration* of the placenta away from the lower uterine segment is caused by the growth of placental trophoblasts toward the fundus (with its richer blood supply) and by the development or elongation of the lower uterine segment. Abnormal implantation of the placenta may occur when there is damage or disruption of the uterine endometrium, most commonly due to a previous cesarean delivery, but also in the setting of previous uterine curettage, myomectomy, endometrial ablation, or pelvic radiation.[17]

Transvaginal ultrasonography allows precise assessment of the distance between the internal os and the placental edge. Prior terminology, including *complete* and *marginal* previa, are no longer used and all placentas overlying the os to any degree are called previas. When the placental edge lies within 2 cm of the os but does not overlie the internal os, it is called low-lying.[17,18]

Epidemiology

Placenta previa is a common incidental finding on second-trimester ultrasonography, present on approximately 4% of ultrasound studies performed at 20 to 25 weeks' gestation but only 0.5% of pregnancies at term.[17] The likelihood of a previa persisting until term increases if the placenta is *completely* covering the internal os, if it is present at a later gestational age, or if there is a history of cesarean delivery.[19,20] The extent to which the placenta overlaps the internal os at 18 to 23 weeks' gestation is highly predictive of the persistence of placenta previa.[21,22] One study concluded that placenta overlap of less than 1.5 cm at 18 to 23 weeks' gestation was less consistent with retention of placenta previa diagnosis at term.[22] If the overlap is 2.5 cm or greater at 20 to 23 weeks' gestation, persistence to term is more likely.[21]

When a placenta previa or low-lying placenta is identified during the second trimester, ultrasound should be repeated at approximately 32 weeks' gestation to determine if it has regressed. One study recommended repeating the scan at 26 to 30 weeks' gestation so that providers can recommend removal of physical precautions at an earlier date, if the previa has resolved.[22] Consider a final scan at 36 weeks' gestation in women with a previa that has persisted so as to determine the optimal route and timing of delivery.[17] Routine late pregnancy ultrasound in low-risk or unselected populations does not confer benefit to the woman or fetus and may increase cesarean delivery rates.[23]

Risk factors associated with placenta previa include chronic hypertension, multiparity, multiple gestations, increasing maternal age, previous cesarean delivery, cocaine use, tobacco use, uterine curettage, previous placenta previa, in vitro fertilization (IVF), and male fetal sex.[2,17,20,24] Increasing numbers of cesarean deliveries are associated with increasing risk of placenta previa.[25]

Morbidity

Maternal morbidity associated with placenta previa can result from maternal hemorrhage, cesarean delivery, or abnormally invasive placenta (placenta accreta, increta, or percreta). Placenta previa is associated with higher morbidity compared with a low-lying placenta.[26,27]

Women who have had a prior cesarean delivery or uterine surgery and have a placenta previa or low anterior placenta in a subsequent pregnancy are at increased risk of abnormally invasive placenta.[2,28,29] The risk of abnormal placentation (placenta accreta) is approximately 10% with placenta previa in a woman who underwent a prior cesarean delivery, and increases with the number of previous cesarean deliveries, surpassing 60% at three or more prior cesarean deliveries.[25,30,31]

Perinatal morbidity and mortality associated with placenta previa are mainly related to the complications of prematurity because the blood loss comes from the maternal circulation.[32] Therefore the management of placenta previa and timing of delivery is influenced by gestational age and fetal lung maturity balanced with the degree of hemorrhage and urgency of the maternal condition. A cohort study of American women with placenta previa showed that greater than half delivered at term (more than 37 weeks' gestation), approximately 28% delivered between 34 and 37 weeks' gestation, and approximately 17% delivered before 34 weeks' gestation.[17]

Clinical Presentation

Symptomatic placenta previa typically manifests as vaginal bleeding in the late second or third

trimester, sometimes after sexual intercourse. The bleeding is painless unless contractions or placental abruption occurs. A large central previa may manifest with bleeding prior to 29 weeks' gestation—the so-called *sentinel bleed*. In patients who have undergone cervical instrumentation or a digital cervical examinaton, sentinel (ie, initial) bleeding does not typically cause hemodynamic instability or risk fetal well-being.

Diagnosis

Placenta previa should be suspected in patients who have a persistent malpresentation. A cephalic presentation may be impossible because of the presence of a large placenta filling the pelvis. Regardless of previous imaging results, vaginal bleeding, particularly if it is painless or provoked by intercourse, in the setting of a high presenting part or abnormal lie should prompt clinical suspicion for placenta previa.[2] The diagnosis of placenta previa is confirmed by ultrasound localization of the placenta. On abdominal ultrasound, a full bladder can create the false appearance of an anterior placenta previa or the presenting part may overshadow a posterior placenta previa.

When placenta previa is suspected on transabdominal ultrasonography, transvaginal ultrasonography should be performed.[2] Transvaginal ultrasonography is safe and more accurate than transabdominal ultrasonography for localizing the placental edge and the internal os. Between 26% and 60% of placentas determined to be low-lying on transabdominal scan are not low-lying on transvaginal ultrasonography and do not require further monitoring.[2,33]

Preoperative ultrasound in the setting of planned cesarean delivery can give information on fetal lie and placental location to determine the desired location of the uterine incision.[17] Gentle insertion of a speculum to view the vaginal vault and cervix and quantify bleeding should not cause disruption of a placenta previa. Placenta accreta should be suspected in any woman with a history of cesarean delivery who presents with placenta previa or a placenta located at the site of the previous cesarean incision. The placenta should be evaluated for potential placenta accreta via color flow Doppler ultrasound by an experienced ultrasonographer.[34,35] Ultrasound alone has sensitivities and specificities as high as 80% to 90% for placenta accreta, especially in clinical circumstances where accreta is suspected.[36] A typical sonographic sign of placenta accreta is the presence of large vascular placental lacunae, especially in the lower uterine segment, giving a *moth-eaten* or *swiss cheese* appearance to the placenta. Also, there may be a loss of the distinct echolucent zone between the placenta and the bladder wall.[37]

Magnetic resonance imaging (MRI) of the pelvis may help confirm the diagnosis of an invasive placenta and delineate other pelvic organ involvement in women with a placenta percreta.[28] MRI also has reportedly high sensitivity and specificity in the range of 80 to 90%, however, its use is controversial. MRI does not improve diagnosis or outcomes compared with ultrasound alone as found in two small studies conducting direct comparison of the modalities.[38,39] A 2018 retrospective study of the likelihood of MRI altering ultrasound diagnosis of placenta accreta spectrum disorders found that MRI often incorrectly changed the diagnosis and the planned management of accreta. Therefore, MRI is not routinely recommended as an adjunct imaging technique over ultrasound performed by an ultrasonographer experienced in diagnosing invasive placentation.[40] MRI has the additional disadvantages of expense and need for specific interpretation expertise for placenta accreta.[36]

Management

A Cochrane review showed few randomized trials of interventions for placenta previa.[41] Outpatient management of placenta previa in stable women who do not have active bleeding can be considered, but the data is limited for making a firm recommendation.[2] Outpatient management, if selected, requires close proximity to the hospital planned for delivery during the third trimester and having someone available to assist in transport to the hospital in the event of bleeding or onset of labor.

Women with asymptomatic previa in the second trimester can continue normal activities until follow-up ultrasonography is performed. Women with persistent placenta previa in the third trimester should report any bleeding and abstain from intercourse and use of tampons, although this recommendation is based on expert opinion, not evidence. When bleeding occurs, women with placenta previa should be evaluated in the hospital.[2] No evidence-based guidelines exist for the management of a small amount of third-trimester bleeding that has resolved by the time of presentation for

care, in the presence or absence of a placenta previa. However, in the setting of reassuring fetal and maternal testing, a brief period of observation and expectant management is reasonable if it is the first bleeding episode. With more significant bleeding a period of inpatient observation of 24 to 48 hours is reasonable to better characterize the bleeding and determine etiology.[3] Subsequent bleeding episodes typically are managed with inpatient admission for the remainder of the pregnancy.

Because most neonatal morbidity and mortality associated with placenta previa results from complications of prematurity, the main therapeutic strategy is to prolong pregnancy until fetal lung maturity is achieved whenever possible. Tocolytic drugs may be beneficial to prolong gestation, specifically to administer corticosteroids if vaginal bleeding occurs with preterm contractions, but tocolytics are considered controversial.[17,42,43] Corticosteroids should be administered to patients with bleeding from the placenta previa at less than 34 weeks' estimated gestation to promote fetal lung maturity.[44] In the late preterm period, a randomized trial showed benefit of antenatal steroid administration between 34 and 36 5/7 weeks' gestational age in reducing the risk of respiratory complications compared with no treatment.[45] However, delivery due to life-threatening bleeding should not be delayed for corticosteroid administration.[3]

Experts have proposed cerclage as a means of prolonging pregnancies with placenta previa, as cerclage may prevent late pregnancy bleeding caused by thinning of the lower uterine segment and/or dilation of the cervix.[46] Although a Cochrane meta-analysis showed that cerclage decreased the risk of preterm birth before 34 weeks' gestation, the data is not definitive, and thus, cerclage is not recommended for previa.[2,17,41]

Women with placenta previa should undergo cesarean delivery.[17] For women with a low-lying placenta, it is recommended that the decision for mode of delivery be deferred until ultrasonography is performed at 35 to 36 weeks' gestation.[47] Women with a placental edge of 2 cm or more from the internal os at term can expect to deliver vaginally unless heavy bleeding ensues. Women with a placenta located 1 to 2 cm from the os may attempt vaginal delivery in a facility capable of moving rapidly to cesarean delivery if necessary, with most women achieving vaginal delivery without increased hemorrhage.[16,48] Women with a placental edge less than 1 cm from the os should undergo planned cesarean delivery.[17]

The need for emergency cesarean delivery is more common in women with three or more episodes of antepartum bleeding and a first episode of bleeding before 29 weeks' gestation.[49] In addition, several small studies suggest that short cervical length in women with placenta previa may be associated with increased risk of emergent delivery. A study of 89 women found that women with placenta previa and a short cervix (less than 3 cm) were significantly more likely to require emergent delivery for hemorrhage (79% versus 28%) and to deliver preterm (69% [$P<0.001$] versus 21% [$P<0.001$]).[50] In another study of 56 women with a complete placenta previa, a cervical length less than 2.94 cm measured on transvaginal ultrasound predicted the risk of preterm emergency cesarean delivery at less than 34 weeks' gestation secondary to massive hemorrhage.[51] In addition, a study of 93 women examined change in cervical length as a predictor for emergent cesarean delivery due to hemorrhage and found an increased risk of emergent delivery in women with a decrease of more than 0.6 cm in cervical length between the second and third trimesters.[52] This information may be used to guide decisions on inpatient versus outpatient expectant management in the early preterm period, but routine cervical length screening in the late preterm period is not recommended.[3]

The optimal timing of delivery for the woman with placenta previa who is asymptomatic and stable is not firmly established, but delivery between 36 and 37 6/7 weeks' gestation has been recommended to maximize maternal and neonatal outcomes.[3,53-55] For the woman with a stable placenta accreta, delivery between 34 and 37 weeks' gestation is recommended.[3] Amniocentesis for assessment of fetal lung maturity is not recommended for determining the optimal timing of delivery.[55] In a retrospective study of the optimal timing of delivery, infants born at 35 to 37 weeks' gestation were no more likely to have fetal anemia, fetal distress, neonatal seizures, increased ventilator needs, or infant mortality compared with infants born at 38 weeks' gestation. Infants born at 35 to 36 weeks' gestation were at increased risk of 5-minute Apgar scores less than 7 and neonatal intensive care unit admission.[56] However, because the risk of recurrent bleeding increases with an increasing number of prior bleeding episodes and gestational

age, late preterm delivery (34 to 36 6/7 weeks' gestation) may be considered in women with mild bleeding who have had one or more prior episodes of bleeding before 34 weeks' gestation.[3]

Indications for operative delivery include the presence of persistent, brisk vaginal bleeding, which poses a threat to the stability of the maternal-fetal dyad, or any vaginal bleeding where the fetus is sufficiently mature to be delivered safely. General anesthesia has been associated with increased intraoperative blood loss and need for blood transfusion. Regional anesthesia appears to be a safe alternative, but it may need to be converted to general anesthesia if surgery is prolonged.[35]

Because a suspected placenta accreta necessitates preparation for a cesarean hysterectomy, including appropriate surgical expertise and availability of a large volume of blood products, transfer to a hospital with providers experienced in accreta management is recommended.[17] Delivery in a tertiary care center experienced in the management of placenta accreta demonstrated improved maternal outcomes compared with standard obstetric care including decreased rates of hemorrhage and surgical complications including lower rates of reoperation, ureteral injury, large volume blood loss, coagulopathy, and prolonged intensive care unit admission. Involvement of multidisciplinary teams including obstetric-gynecologic subspecialties (maternal-fetal medicine, gynecologic surgery, gynecologic oncology), anesthesia, intensivists, transfusion medicine, interventional radiology, and surgical subspecialties (trauma, vascular surgery, and urology) are essential in optimal outcomes for the woman.

In addition, to optimize fetal outcomes in a suspected placenta accreta, these multidisciplinary care teams should incorporate pediatricians, neonatologists, and specialized nursing staff.[36,57-59] Due to the potential need for blood products and surgical and ancillary services, it is recommended that all women with a placenta previa and prior uterine surgery deliver in centers experienced in the management of accreta spectrum disorders even if their placental ultrasound is not concerning for accreta.[60] Delivery is recommended for asymptomatic women with a suspected accreta at approximately 34 weeks' gestation. Earlier delivery may be required for women with bleeding or labor. Delay of delivery to 35 to 36 weeks' gestation in patients with a placenta previa at low risk of placenta accreta is reasonable.[36]

Placental Abruption

Epidemiology

Placental abruption is the separation of the placenta from the uterine wall before delivery. It can be partial or complete and can vary in degree. Abruption is the most common cause of serious vaginal bleeding, occurring in 1% of pregnancies.[61] The incidence of abruption increased between 1979 and 2001, possibly because of rising rates of hypertension, increased stimulant abuse, and increased surveillance bias by diagnosis with ultrasonography.[62]

Risk factors associated with abruption include abdominal trauma, stimulant use (cocaine, amphetamine, or tobacco), chronic hypertension, preeclampsia, thrombophilias, chorioamnionitis, oligohydramnios, iron deficiency anemia, prelabor rupture of membranes, uterine myomas, and abruption in a previous pregnancy.[61,63-67]

Pathophysiology

Placental abruption can result from several different pathophysiologic processes. In some cases, abnormalities in placental development and implantation that start in the first trimester lead to specific pathologic changes that in turn lead to abruption.[61] With blunt trauma to the abdomen, shearing of the uterine-placenta interface leads to placental detachment and hemorrhage that can be overt or retroplacental, and therefore, potentially concealed.[68] Depending on the degree of maternal injury, placental abruption is estimated to complicate 5% to 50% of traumas in pregnancy and is the most common cause of fetal death in blunt trauma.[12] In one large retrospective study of all injured pregnant women at Level I and Level II trauma centers, 84% experienced blunt trauma and 16% had penetrating injuries. Placental abruption was the most common complication, occurring in 3.5% of injured pregnant women and resulting in a rate of intrauterine demise greater than 50%.[69] Other etiologies of abruption include vasoconstriction associated with cocaine use and sudden uterine decompression after rupture of membranes or delivery of a first twin.[61,70]

Prevention

The incidence of placental abruption may be decreased by cessation of tobacco, cocaine, or amphetamine use and appropriate care for hypertensive disorders of pregnancy.[61] In patients with

preeclampsia, treatment with magnesium sulfate is associated with a reduced risk of placental abruption (relative risk 0.64; 95% confidence interval [CI] = 0.5-0.83).[71]

Pregnant women involved in severe motor vehicle crashes have an increased risk of abruption; proper restraints are frequently not used because of discomfort.[72] Appropriate use of seat belts during pregnancy should be routinely encouraged during prenatal care. See the *Maternal Resuscitation and Trauma* chapter for further recommendations.

Clinical Presentation

Placental abruption typically manifests as vaginal bleeding associated with abdominal pain, which can vary from mild cramps to severe pain. Women with posterior placental abruption may report back pain rather than abdominal pain, and patients with abruption may experience pain without bleeding (concealed hemorrhage).[73]

The history should include questions regarding trauma (including falls and domestic violence), presence of pain and contractions, rupture of membranes, and assessment of risk factors, including a history of hypertension (or symptoms of preeclampsia) and stimulant use (cocaine, amphetamine, or tobacco) .

The blood may be bright, dark, or intermixed with amniotic fluid. Blood from concealed hemorrhage is typically dark, having been sequestered behind the membranes. The amount of vaginal bleeding is not indicative of the severity of abruption.[61] It may be difficult to determine whether bleeding represents exuberant bloody show or abruption. If bleeding is noted at the time of rupture of membranes, vasa previa should also be considered.

Physical Examination and Diagnostic Testing

Fetal heart tones and uterine activity should be documented by continuous monitoring.[7] Tetanic contractions (ie, one uterine contraction lasting longer than 2 minutes) may be present and, if measured by an intrauterine pressure catheter, are typically recorded as a high resting tone with superimposed small frequent contractions. The presence of this finding is significant because it will often be accompanied by a concerning (Category II or III) fetal tracing. Based on the risk of abruption in the setting of trauma, it is recommended that pregnant patients (23 weeks' gestation or greater) who experience trauma with adverse factors be hospitalized for 24-hour observation. These factors include uterine tenderness, significant abdominal pain, vaginal bleeding, sustained contractions (more than one every 10 minutes), rupture of membranes, atypical or abnormal FHR pattern, high-risk mechanism of injury, or serum fibrinogen less than 200 mg/dL.[12]

Ultrasound findings, if available, may include a retroplacental echolucency, abnormal thickening of the placenta, or an abnormally round, *torn up* edge of the placenta. Unfortunately, acute blood clots and the placenta are both hyperechoic on ultrasonography and can be difficult to distinguish from one another.[74] The diagnosis of abruption is largely a clinical diagnosis, and urgent management should never be delayed for ultrasound confirmation. If the woman and fetus are stable, the placental location and appearance, fetal lie, and fetal weight estimation may be helpful in planning care. Computerized tomography (CT) scan is also able to identify placental abruption, and CT scan performed after trauma should include careful evaluation of the placenta.[75]

Management

Because the unpredictable nature of abruption does not allow for controlled trials, management remains empiric. A Cochrane review found no randomized controlled trials assessing interventions for placental abruption that met inclusion criteria.[76] However, a large cohort study found the risk of placental abruption is significantly increased (adjusted odds ratio [OR] = 93) in a subsequent pregnancy in women with a history of abruption in a first pregnancy. Based on that study and expert opinion, medical induction of labor is appropriate at 37 weeks' gestation in women with a history of placental abruption.[2,64]

Mild Abruption

A stable woman with a small partial abruption and a stable preterm fetus may be treated successfully in a conservative manner. Tocolysis is typically contraindicated[2] except in mild abruption before 34 weeks' gestation when it may be used to allow time for administration of corticosteroids.[77] It is important to note that IV magnesium sulfate may be indicated before 32 weeks' gestation not as a tocolytic, but rather for fetal neuroprotection to reduce the incidence of cerebral palsy during preterm delivery.[78]

Women experiencing recurrent bleeding attributed to placental separation may be diagnosed as having a chronic abruption. Management will be based on the degree of bleeding and gestational age. When expectant management of chronic abruption occurs, serial ultrasonography for fetal growth and antepartum surveillance are indicated in the third trimester because of the potential for uteroplacental insufficiency.[62]

Severe Abruption

Initial management of severe abruption includes rapid stabilization of maternal cardiopulmonary status and assessment of fetal well-being. Delay can be fatal to the fetus: 30% of perinatal mortalities in one case series occurred within 2 hours of admission.[79] In a study of placental abruption in the United States, perinatal mortality was 119 per 1,000 births in pregnancies complicated by placental abruption compared with 8.2 per 1,000 births in other births; the majority of perinatal mortality was associated with preterm delivery.[80]

Maternal stabilization requires monitoring of vital signs and urine output along with serial evaluation of the hematocrit and coagulation studies to determine whether DIC is present.[63] The circulatory status of the patient with abruption should be maintained to permit a margin of reserve. Hourly urine output should be maintained at 30 mL/hour or greater. Hematocrit should be maintained above 30%. In patients with preeclampsia or other confounding factors, central blood pressure monitoring may assist in fluid management.[14,81]

A concerning FHR tracing (eg, persistent Category III or Category II without variability) in the setting of placental abruption necessitates rapid delivery, typically cesarean delivery.[82,83] A decision-to-delivery interval of 20 minutes or less resulted in improved neonatal outcomes in a case-control study of severe abruption.[82] Occasionally, abruption occurs during the second stage and an operative vaginal delivery may be attempted. Neonatal resuscitation should be available for all deliveries, vaginal and operative.

When fetal mortality occurs secondary to abruption, vaginal delivery is recommended.[84] Labor should be permitted if adequate progress is made and maternal status can be supported. Although labor is often hypertonic with abruption, it may also be hypotonic. Oxytocin augmentation is not contraindicated but should be used judiciously with intrauterine pressure monitoring. Indications for operative delivery with fetal demise include other maternal indications for cesarean delivery, failure of labor progression, and brisk hemorrhage that cannot be compensated for by transfusion.

Approximately one-third of patients with placental abruption with fetal demise will develop coagulopathy. Coagulopathy is typically not seen in the patient presenting with abruption and a live fetus. Coagulopathy occurring with abruption may be related to two etiologies: consumptive coagulopathy and DIC. Replacement of platelets and fresh-frozen plasma should be administered just before operative delivery to provide maximum effectiveness. In addition, cryoprecipitate or factor VIII may be of specific benefit in severe coagulopathy.

Consideration of maternal transfer from a rural site with a Level I hospital is based on many factors.[60] Patients presenting with abruption and a live fetus are typically not stable for transfer because immediate operative delivery may be needed at any time during labor. However, neonatal transfer (rather than maternal-fetal) may be necessary for the premature or ill newborn. If fetal demise has occurred, a woman who does not have coagulopathy and is hemodynamically stable may be cared for if appropriate resources are available. Blood bank supply may determine whether a woman requires transport to a different facility.

Fetomaternal hemorrhage may occur with rupture of fetal vessels in the placenta. The Kleihauer-Betke test is useful to determine dosage of Rh_o (D) immune globulin in Rh-negative patients but is not useful for the diagnosis of abruption.[85,86]

Uterine Rupture

Epidemiology and Pathophysiology

Uterine rupture spans a spectrum from occult scar dehiscence discovered at repeat cesarean delivery to complete uterine rupture requiring emergency cesarean delivery (or if delivery has already occurred, subsequent emergency laparotomy.) Uncommonly, uterine rupture can be spontaneous and occur in the absence of risk factors. In complete rupture, the fetus or placenta may be partially or completely extruded from the uterus. This chapter focuses only on uterine rupture presenting with mid- and late pregnancy bleeding.

Spontaneous uterine rupture occurs rarely in pregnant women (0.006% to 0.0125%),[87] but in

approximately 0.5% to 0.9% of women with a uterine scar from a previous surgery.[88] Previous cesarean incision is the most common etiology for uterine rupture. Classic or T-shaped uterine incisions are associated with a higher likelihood of uterine rupture compared with a low transverse incision.[89] Other predisposing factors for uterine rupture include previous myomectomy, trauma, congenital uterine anomaly, uterine overdistension, intra-amniotic installation, gestational trophoblastic neoplasia, previous uterine rupture, inappropriate oxytocin use, maternal obesity, and adenomyosis.[89,90] Other risk factors include obstructed labor,[91] labor induction (OR 12.60; 95% CI = 4.4-36.4), labor induction with prostaglandins (OR 2.72; 95% CI = 1.6-4.7), and non-European ethnicity (OR 2.87; 95% CI = 1.8-4.7).[89] Conditions present during delivery that predispose to uterine rupture include fetal anomaly, vigorous uterine pressure, difficult manual removal of the placenta, or abnormalities of placental implantation.[90]

The most common maternal morbidity associated with uterine rupture is hemorrhage and subsequent anemia requiring blood transfusion.[92] Other morbidities include bladder injury, ureteral injury, parametrial vessel disruption, and hysterectomy, complications which occur in 14% to 33% of uterine ruptures.[93-95] Although rare, maternal mortality from DIC and sepsis has been reported.[92] The incidence of perinatal mortality associated with uterine rupture is variably reported from 0% to 60%.[96] Fetal and maternal morbidity are higher in cases of uterine rupture of an unscarred uterus.[93]

Clinical Presentation

The classic presentation for symptomatic, significant uterine rupture includes vaginal bleeding, pain, cessation of contractions, absence of fetal heart tones, loss of station, easily palpable fetal parts through the maternal abdomen, and profound maternal tachycardia and hypotension. However, in up to 70% of uterine ruptures the initial indication is abnormal fetal monitoring results.[88,93] In a review of 159,456 deliveries, the most frequent finding associated with uterine rupture was a sudden deterioration of the FHR pattern.[97] In one case-control study, markedly abnormal FHR tracings starting 1 hour before birth were significantly associated with uterine rupture in women undergoing labor after cesarean (LAC) delivery.[98] There may be a progression of signs from nonspecific, severe variable decelerations to the characteristic recession of the fetal head or suprapubic bulging. Contractions may show a *stair-step* appearance of gradually decreasing amplitude on tocodynamometry.[99]

Thirteen percent of uterine ruptures occur outside the hospital setting. Women with a prior uterine scar should be advised to present to the hospital for evaluation of new-onset regular contractions, abdominal pain, or vaginal bleeding as soon as possible.[97] Spontaneous antepartum rupture in nonlaboring women is rare and is typically associated with identifiable risk factors. In a case series of women experiencing spontaneous uterine rupture in the second or third trimester, six of seven events (during 13 years) involved placenta previa or percreta, and five of the seven uterine ruptures occurred in women with prior cesarean deliveries.[100] These findings suggest that a prior uterine scar and an abnormal placenta are important risk factors in uterine rupture.[101]

Management

Due to the association of FHR abnormalities with uterine rupture in the setting of a LAC, continuous fetal monitoring is recommended in women undergoing a trial of labor.[88] With a sudden change in fetal baseline or the onset of repetitive FHR decelerations, the provider should institute intrauterine resuscitation with maternal position change, administration of IV fluids and oxygen, discontinuation of oxytocin, and consideration for subcutaneous terbutaline. If these interventions are not effective, emergent cesarean or operative vaginal delivery may be indicated.

Depending on the clinical presentation, hemodynamic stability, fertility desires of the patient, degree of uterine rupture, and involvement of surrounding structures, surgical intervention for uterine rupture may range from revision and repair of the uterine scar dehiscence to hysterectomy with massive transfusion. Surgical intervention may be life-saving.[95] Asymptomatic or occult uterine rupture may be found at the time of cesarean delivery or palpation of the uterine cavity after vaginal delivery, and should be suspected in postpartum hemorrhage after LAC.[95] Routine inspection of the uterine scar after a successful LAC is not recommended.[95]

Because uterine rupture predisposes the woman to recurrent uterine rupture (from 6% if prior rupture was confined to the lower segment to 32% if it involves the contractile uterine body); subsequent pregnancies in women with a previous uterine rupture should be delivered via cesarean delivery before the onset of labor, preferably between 36 and 38 weeks' gestation.[88]

Vasa Previa

Vasa previa occurs when fetal blood vessels, unprotected by the umbilical cord or placenta, run through the membranes and across or within 2 cm of the cervix. Although uncommon, with an incidence of 1 per 2,500 deliveries, it is important to be familiar with vasa previa because rapid intervention is essential for fetal survival.[17,102]

Epidemiology and Pathophysiology

Vasa previa typically occurs in pregnancies with a low-lying placenta and velamentous insertion, bipartite placenta, or a placenta with a succenturiate lobe.[103] During rupture of the membranes, the fetal vessels are at risk of rupture, which can lead to significant fetal blood loss. Historical studies showed a significant rate of perinatal mortality secondary to vasa previa,[104] but antenatal diagnosis is associated with a reduction in the rate of neonatal morbidity and mortality.[103] Risk factors for vasa previa include IVF, multiple gestation, resolved placenta previa, and bilobed or succenturiate-lobed placentas.[3,104-106]

Clinical Presentation and Diagnosis

The goal of diagnosis is antenatal detection and delivery before membrane rupture.[107] Prenatal diagnosis using color flow Doppler ultrasound significantly affects fetal survival. Prenatal diagnosis is associated with a 98% survival rate compared with a 44% survival rate with intrapartum or postpartum diagnosis.[102] Because of increased prenatal diagnosis, contemporary case series show perinatal mortality rates of less than 10%.[102] Prenatal ultrasound has a 93% detection rate and a specificity of 99% for vasa previa with an optimal detection window between 18 and 26 weeks' gestation; detection is less likely on ultrasound performed in the third trimester.[102] Prenatally, combination transabdominal transvaginal ultrasound may improve diagnostic accuracy. Power Doppler and three-dimensional ultrasonography are reported to assist with diagnosis, but superiority to two-dimensional ultrasound has not been shown.[17]

Clinically, vasa previa is suspected when vaginal bleeding occurs with membrane rupture, classically in the setting of a FHR tracing showing an initial tachycardia, followed by variable decelerations, bradycardia, or a sinusoidal pattern.[102,108] The hemorrhage is fetal blood, and exsanguination can occur rapidly because the average blood volume of a term fetus is approximately 250 mL. Vessels are rarely palpated in the presenting membranes, but if they are palpable this prohibits artificial rupture and vaginal delivery. In addition to diagnosis by ultrasound or classic clinical presentation, vasa previa may be diagnosed by MRI. Intrapartum identification of fetal blood intermixed with vaginal blood, using tests such as the Apt test, Wright stain, Kleihauer-Betke test and hemoglobin electrophoresis, is too slow to use clinically. If fetal blood is suspected vaginally, immediate delivery should be performed.[103,108]

Management

In a woman with antenatally detected vasa previa who presents with prelabor rupture of membranes or labor, cesarean delivery should be performed.[17,102] Delivery should not be deferred for confirmation of fetal blood in women with severe hemorrhage or when fetal heart tones are concerning. If the onset of vaginal bleeding occurred with rupture of membranes and the FHR is concerning, cesarean delivery should be performed immediately. Because fetal exsanguination is the cause of neonatal mortality in this condition, preparation for resuscitation at delivery includes availability of normal saline for a 10 to 20 mL/kg bolus or aggressive postnatal transfusion.[108]

In the presence of an antenatal diagnosis of vasa previa, serial ultrasounds are recommended to evaluate for regression of vessels, which can occur in approximately 20% of women.[102] With persistent vasa previa, antenatal steroids should be administered between 28 and 32 weeks' gestation. Hospitalization at 30 to 34 weeks' gestation should be considered, allowing for closer observation for signs of labor onset and time to operative delivery if membranes rupture. However, data to support this approach compared with outpatient management is lacking.[102] Outpatient management can be considered for asymptomatic women with no uterine activity and a long closed cervix

on transvaginal ultrasound.[107] More than half of women treated in the outpatient setting will eventually require hospitalization for a complication.[102] The optimal gestational age for delivery is not known, but cesarean delivery between 34 and 37 weeks' gestation has been recommended to balance the risk of respiratory distress syndrome with catastrophic risk of membrane rupture and fetal exsanguination.[3,17,102,107] Amniocentesis to assess for fetal lung maturity is not indicated because timing of delivery would not be influenced by the result.[102] Delaying delivery beyond 37 weeks' gestation is not recommended.

Prevention

There are no strategies for primary prevention of vasa previa. However, hemorrhage theoretically is preventable with antenatal screening of women at high risk, and cesarean delivery at 34 to 37 weeks' gestation when vasa previa is present. Screening is conducted with transvaginal color flow Doppler ultrasound to identify the presence of fetal arterial or venous blood flow in the fetal membranes.[3] Screening in the general population has not been recommended because the condition is rare (1 diagnosis per 2,000 to 5,000 screenings).[109] Screening is recommended in women at increased risk of vasa previa, which includes women after the detection of a low-lying, bilobed, multilobed, or succenturiate-lobed placenta, in women with velamentous cord insertion on routine ultrasound, or women who are pregnant via IVF.[17,103] Careful evaluation of the placenta including site of cord insertion on routine ultrasound may facilitate identifying women at greater risk of vasa previa. A transvaginal ultrasound with color and pulsed Doppler ultrasound at 32 weeks' gestation has been recommended for women who had a placenta previa or low-lying placenta during a second-trimester ultrasound with subsequent resolution.[102]

Summary

Vaginal bleeding in late pregnancy may occur because of potentially life-threatening conditions for the woman and fetus. Providers must be able to distinguish emergent causes of bleeding from those that are less urgent and be prepared to act quickly and decisively when severe maternal hemorrhage or suspected vasa previa are present. Vaginal examination should be avoided until placental location is known. The timely diagnosis of vaginal bleeding in late pregnancy, including antenatal diagnosis with color flow Doppler ultrasound, can reduce perinatal mortality. Institutional policies should be in place to ensure adequate blood bank response to massive hemorrhage and to mobilize resources for emergent cesarean delivery.

Nursing Considerations: Late Pregnancy Bleeding

- Nurses are usually the first obstetric providers to assess patients presenting with vaginal bleeding in late pregnancy; early detection and prompt action may improve outcomes
- Understand maternal and fetal clinical presentations for placenta previa, placental abruption, and uterine rupture and identify the system in your institution to immediately notify physicians, midwives, and other team members when these conditions are suspected
- Obtain intravenous access (two sites if considering administration of blood products) and laboratory tests
- Establish a policy of not performing vaginal examination if placental location is unknown
- Prepare for possible emergent cesarean delivery when concerning late pregnancy bleeding is present

References

1. McCormack RA, Doherty DA, Magann EF, et al. Antepartum bleeding of unknown origin in the second half of pregnancy and pregnancy outcomes. *BJOG*. 2008; 115(11):1451-1457.
2. Royal College of Obstetricians and Gynaecologists. Green-top Guideline no. 63. Antepartum haemorrhage. London, England: Royal College of Obstetricians and Gynaecologists; 2011.
3. Gyamfi-Bannerman C; Society for Maternal-Fetal Medicine (SMFM). Electronic address: pubs@smfm.org. Society for Maternal-Fetal Medicine (SMFM) Consult Series #44: Management of bleeding in the late preterm period. *Am J Obstet Gynecol*. 2018;218(1):B2-B8.
4. Adamson DL, Nelson-Piercy C. Managing palpitations and arrhythmias during pregnancy. *Heart*. 2007;93(12): 1630-1636.
5. Pacagnella RC, Souza JP, Durocher J, et al. A systematic review of the relationship between blood loss and clinical signs. *PLoS One*. 2013;8(3):e57594.
6. De Kock J, Heyns T, Van Rensburg GH. The ABC of haemorrhagic shock in the pregnant woman. *Professional Nursing Today*. 2008;12(5):54-57.
7. American College of Obstetricians and Gynecologists. ACOG Practice Bulletin no. 106: intrapartum fetal heart rate monitoring: nomenclature, interpretation, and general management principles. *Obstet Gynecol*. 2009; 114(1):192-202.
8. James D, Steer P, Weiner C, et al. Pregnancy and laboratory studies: a reference table for clinicians. *Obstet Gynecol*. 2010;115(4):868, author reply 868-869.
9. Erez O, Mastrolia SA, Thachil J. Disseminated intravascular coagulation in pregnancy: insights in pathophysiology, diagnosis and management. *Am J Obstet Gynecol*. 2015;213(4):452-463.
10. Rajasekhar A, Gernsheimer T, Stasi R, James AH. 2013 Clinical Practice Guide on thrombocytopenia in pregnancy. 2013. Available at www.hematology.org/Clinicians/Guidelines-Quality/Quick-Ref/530.aspx.
11. Cines DB, Levine LD. Thrombocytopenia in pregnancy. *Blood*. 2017;130(21):2271-2277.
12. Jain V, Chari R, Maslovitz S, et al; Maternal Fetal Medicine Committee. Guidelines for the management of a pregnant trauma patient. *J Obstet Gynaecol Can*. 2015; 37(6):553-574.
13. Jonard M, Ducloy-Bouthors AS, Fourrier F. Comparison of two diagnostic scores of disseminated intravascular coagulation in pregnant women admitted to the ICU. *PLoS One*. 2016;11(11):e0166471.
14. Hull AD, Resnik R. Placenta previa, placenta accreta, abruptio placentae, and vasa previa. In: Creasy RKRR, Iams JD, Lockwood CJ, Moore TR, Greene MF, eds. *Creasy and Resnik's Maternal-Fetal Medicine*. Philadelphia: Elsevier; 2014.
15. American College of Obstetrics and Gynecology. ACOG Practice Bulletin. Prevention of Rh D alloimmunization. Number 4, May 1999 (replaces educational bulletin Number 147, October 1990). Clinical management guidelines for obstetrician-gynecologists. *Int J Gynaecol Obstet*. 1999;66(1):63-70.
16. Vergani P, Ornaghi S, Pozzi I, et al. Placenta previa: distance to internal os and mode of delivery. *Am J Obstet Gynecol*. 2009;201(3):266.e1-266.e5.
17. Silver RM. Abnormal placentation: placenta previa, vasa previa, and placenta accreta. *Obstet Gynecol*. 2015; 126(3):654-668.
18. Reddy UM, Abuhamad AZ, Levine D, Saade GR; Fetal Imaging Workshop Invited Participants. Fetal imaging: executive summary of a joint Eunice Kennedy Shriver National Institute of Child Health and Human Development, Society for Maternal-Fetal Medicine, American Institute of Ultrasound in Medicine, American College of Obstetricians and Gynecologists, American College of Radiology, Society for Pediatric Radiology, and Society of Radiologists in Ultrasound Fetal Imaging Workshop. *J Ultrasound Med*. 2014;33(5):745-757.
19. Mustafá SA, Brizot ML, Carvalho MH, et al. Transvaginal ultrasonography in predicting placenta previa at delivery: a longitudinal study. *Ultrasound Obstet Gynecol*. 2002;20(4):356-359.
20. Dashe JS, McIntire DD, Ramus RM, et al. Persistence of placenta previa according to gestational age at ultrasound detection. *Obstet Gynecol*. 2002;99(5 Pt 1): 692-697.
21. Becker RH, Vonk R, Mende BC, et al. The relevance of placental location at 20-23 gestational weeks for prediction of placenta previa at delivery: evaluation of 8650 cases. *Ultrasound Obstet Gynecol*. 2001;17(6):496-501.
22. Taipale P, Hiilesmaa V, Ylöstalo P. Transvaginal ultrasonography at 18-23 weeks in predicting placenta previa at delivery. *Ultrasound Obstet Gynecol*. 1998;12(6): 422-425.
23. Bricker L, Neilson JP, Dowswell T. Routine ultrasound in late pregnancy (after 24 weeks' gestation). *Cochrane Database Syst Rev*. 2008;(4):CD001451.
24. Yang Q, Wen SW, Phillips K, et al. Comparison of maternal risk factors between placental abruption and placenta previa. *Am J Perinatol*. 2009;26(4):279-286.
25. Silver RM, Landon MB, Rouse DJ, et al; National Institute of Child Health and Human Development Maternal-Fetal Medicine Units Network. Maternal morbidity associated with multiple repeat cesarean deliveries. *Obstet Gynecol*. 2006;107(6):1226-1232.
26. Bahar A, Abusham A, Eskandar M, et al. Risk factors and pregnancy outcome in different types of placenta previa. *J Obstet Gynaecol Can*. 2009;31(2):126-131.
27. Tuzovic L. Complete versus incomplete placenta previa and obstetric outcome. *Int J Gynaecol Obstet*. 2006; 93(2):110-117.
28. Committee on Obstetric Practice. Committee Opinion no. 529: placenta accreta. *Obstet Gynecol*. 2012;120(1): 207-211.
29. Baldwin HJ, Patterson JA, Nippita TA, et al. Antecedents of abnormally invasive placenta in primiparous women: risk associated with gynecologic procedures. *Obstet Gynecol*. 2018;131(2):227-233.
30. Miller DA, Chollet JA, Goodwin TM. Clinical risk factors for placenta previa-placenta accreta. *Am J Obstet Gynecol*. 1997;177(1):210-214.

31. Zaki ZM, Bahar AM, Ali ME, et al. Risk factors and morbidity in patients with placenta previa accreta compared to placenta previa non-accreta. *Acta Obstet Gynecol Scand*. 1998;77(4):391-394.
32. Zlatnik MG, Cheng YW, Norton ME, et al. Placenta previa and the risk of preterm delivery. *J Matern Fetal Neonatal Med*. 2007;20(10):719-723.
33. Smith RS, Lauria MR, Comstock CH, et al. Transvaginal ultrasonography for all placentas that appear to be low-lying or over the internal cervical os. *Ultrasound Obstet Gynecol*. 1997;9(1):22-24.
34. Comstock CH, Love JJ Jr, Bronsteen RA, et al. Sonographic detection of placenta accreta in the second and third trimesters of pregnancy. *Am J Obstet Gynecol*. 2004;190(4):1135-1140.
35. Oppenheimer L; MATERNAL FETAL MEDICINE COMMITTEE. Diagnosis and management of placenta previa. *J Obstet Gynaecol Can*. 2007;29(3):261-266.
36. Silver RM, Branch DW. Placenta Accreta Spectrum. *N Engl J Med*. 2018;378(16):1529-1536.
37. Silver RM. Placenta Accreta. In: Queenan JT, Spong CY, Lockwood CJ, eds. *Protocols for High-Risk Pregnancies: An Evidence-Based Approach*. 6th ed. Oxford, UK: Wiley-Blackwell; 2015:435-444.
38. Riteau AS, Tassin M, Chambon G, et al. Accuracy of ultrasonography and magnetic resonance imaging in the diagnosis of placenta accreta. *PLoS One*. 2014;9(4):e94866.
39. Balcacer P, Pahade J, Spektor M, et al. Magnetic resonance imaging and sonography in the diagnosis of placental invasion. *J Ultrasound Med*. 2016;35(7):1445-1456.
40. Einerson BD, Rodriguez CE, Kennedy AM, et al. Magnetic resonance imaging is often misleading when used as an adjunct to ultrasound in the management of placenta accreta spectrum disorders. *Am J Obstet Gynecol*. 2018;218(6):618.e1-618.e7.
41. Neilson JP. Interventions for suspected placenta praevia. *Cochrane Database Syst Rev*. 2003;(2):CD001998.
42. Sharma A, Suri V, Gupta I. Tocolytic therapy in conservative management of symptomatic placenta previa. *Int J Gynaecol Obstet*. 2004;84(2):109-113.
43. Verspyck E, de Vienne C, Muszynski C, et al. Maintenance nifedipine therapy for preterm symptomatic placenta previa: A randomized, multicenter, double-blind, placebo-controlled trial. *PLoS One*. 2017;12(3):e0173717.
44. Roberts D, Dalziel S. Antenatal corticosteroids for accelerating fetal lung maturation for women at risk of preterm birth. *Cochrane Database Syst Rev*. 2006;(3):CD004454.
45. Gyamfi-Bannerman C, Thom EA, Blackwell SC, et al; NICHD Maternal–Fetal Medicine Units Network. Antenatal betamethasone for women at risk for late preterm delivery. *N Engl J Med*. 2016;374(14):1311-1320.
46. Cobo E, Conde-Agudelo A, Delgado J, et al. Cervical cerclage: an alternative for the management of placenta previa? *Am J Obstet Gynecol*. 1998;179(1):122-125.
47. Jauniaux E, Alfirevic Z, Bhide AG, et al; Royal College of Obstetricians and Gynaecologists. Placenta praevia and placenta accreta: diagnosis and management: Green-top Guideline no. 27a. *BJOG*. 2019;126(1):e1-e48.
48. Al Wadi K, Schneider C, Burym C, et al. Evaluating the safety of labour in women with a placental edge 11 to 20 mm from the internal cervical Os. *J Obstet Gynaecol Can*. 2014;36(8):674-677.
49. Pivano A, Alessandrini M, Desbriere R, et al. A score to predict the risk of emergency caesarean delivery in women with antepartum bleeding and placenta praevia. *Eur J Obstet Gynecol Reprod Biol*. 2015;195:173-176.
50. Stafford IA, Dashe JS, Shivvers SA, et al. Ultrasonographic cervical length and risk of hemorrhage in pregnancies with placenta previa. *Obstet Gynecol*. 2010;116(3):595-600.
51. Ghi T, Contro E, Martina T, et al. Cervical length and risk of antepartum bleeding in women with complete placenta previa. *Ultrasound Obstet Gynecol*. 2009;33(2):209-212.
52. Shin JE, Shin JC, Lee Y, Kim SJ. Serial change in cervical length for the prediction of emergency cesarean section in placenta previa. *PLoS One*. 2016;11(2):e0149036.
53. Blackwell SC. Timing of delivery for women with stable placenta previa. *Semin Perinatol*. 2011;35(5):249-251.
54. Zlatnik MG, Little SE, Kohli P, et al. When should women with placenta previa be delivered? A decision analysis. *J Reprod Med*. 2010;55(9-10):373-381.
55. American College of Obstetricians and Gynecologists. ACOG Committee Opinion no. 560: medically indicated late-preterm and early-term deliveries. *Obstet Gynecol*. 2013;121(4):908-910.
56. Balayla J, Wo BL, Bédard MJ. A late-preterm, early-term stratified analysis of neonatal outcomes by gestational age in placenta previa: defining the optimal timing for delivery. *J Matern Fetal Neonatal Med*. 2015;28(15):1756-1761.
57. Eller AG, Bennett MA, Sharshiner M, et al. Maternal morbidity in cases of placenta accreta managed by a multidisciplinary care team compared with standard obstetric care. *Obstet Gynecol*. 2011;117(2 Pt 1):331-337.
58. Shamshirsaz AA, Fox KA, Salmanian B, et al. Maternal morbidity in patients with morbidly adherent placenta treated with and without a standardized multidisciplinary approach. *Am J Obstet Gynecol*. 2015;212(2):218.e1-218.e9.
59. Wright JD, Herzog TJ, Shah M, et al. Regionalization of care for obstetric hemorrhage and its effect on maternal mortality. *Obstet Gynecol*. 2010;115(6):1194-1200.
60. Obstetric Care Consensus no. 2: levels of maternal care. *Obstet Gynecol*. 2015;125(2):502-515.
61. Oyelese Y, Ananth CV. Placental abruption. *Obstet Gynecol*. 2006;108(4):1005-1016.
62. Ananth CV, Oyelese Y, Yeo L, et al. Placental abruption in the United States, 1979 through 2001: temporal trends and potential determinants. *Am J Obstet Gynecol*. 2005;192(1):191-198.

63. Hladky K, Yankowitz J, Hansen WF. Placental abruption. *Obstet Gynecol Surv*. 2002;57(5):299-305.
64. Ruiter L, Ravelli ACJ, de Graaf IM, et al. Incidence and recurrence rate of placental abruption: a longitudinal linked national cohort study in the Netherlands. *Am J Obstet Gynecol*. 2015;213(4):573.e1-573.e8.
65. Arnold DL, Williams MA, Miller RS, et al. Iron deficiency anemia, cigarette smoking and risk of abruptio placentae. *J Obstet Gynaecol Res*. 2009;35(3):446-452.
66. Brenner B, Aharon A. Thrombophilia and adverse pregnancy outcome. *Clin Perinatol*. 2007;34(4):527-541, v.
67. Ananth CV, Savitz DA, Williams MA. Placental abruption and its association with hypertension and prolonged rupture of membranes: a methodologic review and meta-analysis. *Obstet Gynecol*. 1996;88(2):309-318.
68. Brown HL. Trauma in pregnancy. *Obstet Gynecol*. 2009;114(1):147-160.
69. Rogers FB, Rozycki GS, Osler TM, et al. A multi-institutional study of factors associated with fetal death in injured pregnant patients. *Arch Surg*. 1999;134(11): 1274-1277.
70. Fleming AD. Abruptio placentae. *Crit Care Clin*. 1991; 7(4):865-875.
71. Duley L, Gülmezoglu AM, Henderson-Smart DJ, Chou D. Magnesium sulphate and other anticonvulsants for women with pre-eclampsia. *Cochrane Database Syst Rev*. 2010;(11):CD000025.
72. Reis PM, Sander CM, Pearlman MD. Abruptio placentae after auto accidents. A case-control study. *J Reprod Med*. 2000;45(1):6-10.
73. Golan A, Sandbank O, Teare AJ. Trauma in late pregnancy. A report of 15 cases. *S Afr Med J*. 1980;57(5): 161-165.
74. Glantz C, Purnell L. Clinical utility of sonography in the diagnosis and treatment of placental abruption. *J Ultrasound Med*. 2002;21(8):837-840.
75. Manriquez M, Srinivas G, Bollepalli S, et al. Is computed tomography a reliable diagnostic modality in detecting placental injuries in the setting of acute trauma? *Am J Obstet Gynecol*. 2010;202(6):611.e1-611.e5.
76. Neilson JP. Interventions for treating placental abruption. *Cochrane Database Syst Rev*. 2003;(1):CD003247.
77. Towers CV, Pircon RA, Heppard M. Is tocolysis safe in the management of third-trimester bleeding? *Am J Obstet Gynecol*. 1999;180(6 Pt 1):1572-1578.
78. Rouse DJ, Hirtz DG, Thom E, et al; Eunice Kennedy Shriver NICHD Maternal-Fetal Medicine Units Network. A randomized, controlled trial of magnesium sulfate for the prevention of cerebral palsy. *N Engl J Med*. 2008; 359(9):895-905.
79. Knab DR. Abruptio placentae. An assessment of the time and method of delivery. *Obstet Gynecol*. 1978; 52(5):625-629.
80. Ananth CV, Wilcox AJ. Placental abruption and perinatal mortality in the United States. *Am J Epidemiol*. 2001; 153(4):332-337.
81. Neligan PJ, Laffey JG. Clinical review: Special populations—critical illness and pregnancy. *Crit Care*. 2011; 15(4):227.
82. Kayani SI, Walkinshaw SA, Preston C. Pregnancy outcome in severe placental abruption. *BJOG*. 2003;110(7): 679-683.
83. Witlin AG, Sibai BM. Perinatal and maternal outcome following abruptio placentae. *Hypertens Pregnancy*. 2001;20(2):195-203.
84. Boisramé T, Sananès N, Fritz G, et al. Placental abruption: risk factors, management and maternal-fetal prognosis. Cohort study over 10 years. *Eur J Obstet Gynecol Reprod Biol*. 2014;179:100-104.
85. Emery CL, Morway LF, Chung-Park M, et al. The Kleihauer-Betke test. Clinical utility, indication, and correlation in patients with placental abruption and cocaine use. *Arch Pathol Lab Med*. 1995;119(11):1032-1037.
86. Mendez-Figueroa H, Dahlke JD, Vrees RA, Rouse DJ. Trauma in pregnancy: an updated systematic review. *Am J Obstet Gynecol*. 2013;209(1):1-10.
87. Ofir K, Sheiner E, Levy A, et al. Uterine rupture: differences between a scarred and an unscarred uterus. *Am J Obstet Gynecol*. 2004;191(2):425-429.
88. Committee on Practice Bulletins-Obstetrics. Practice Bulletin no. 184: vaginal birth after previous cesarean delivery. *Obstet Gynecol*. 2017;130(5):e217-e233.
89. Mirza FG, Gaddipati S. Obstetric emergencies. *Semin Perinatol*. 2009;33(2):97-103.
90. Smith JG, Mertz HL, Merrill DC. Identifying risk factors for uterine rupture. *Clin Perinatol*. 2008;35(1):85-99, viii.
91. Neilson JP, Lavender T, Quenby S, Wray S. Obstructed labour. *Br Med Bull*. 2003;67(1):191-204.
92. Aziz N, Yousfani S. Analysis of uterine rupture at university teaching hospital Pakistan. *Pak J Med Sci*. 2015; 31(4):920-924.
93. Zwart JJ, Richters JM, Ory F, et al. Uterine rupture in The Netherlands: a nationwide population-based cohort study. *BJOG*. 2009;116(8):1069-1078, discussion 1078-1080.
94. National Institutes of Health Consensus Development Conference Panel. National Institutes of Health Consensus Development conference statement: vaginal birth after cesarean: new insights March 8-10, 2010. *Obstet Gynecol*. 2010;115(6):1279-1295.
95. Committee on Practice Bulletins-Obstetrics. Practice Bulletin no. 183: postpartum hemorrhage. *Obstet Gynecol*. 2017;130(4):e168-e186.
96. Guise JM, McDonagh MS, Osterweil P, et al. Systematic review of the incidence and consequences of uterine rupture in women with previous caesarean section. *BMJ*. 2004;329(7456):19-25.
97. Leung AS, Leung EK, Paul RH. Uterine rupture after previous cesarean delivery: maternal and fetal consequences. *Am J Obstet Gynecol*. 1993;169(4):945-950.
98. Desseauve D, Bonifazi-Grenouilleau M, Fritel X, et al. Fetal heart rate abnormalities associated with uterine rupture: a case-control study: a new time-lapse approach using a standardized classification. *Eur J Obstet Gynecol Reprod Biol*. 2016;197:16-21.

99. Matsuo K, Scanlon JT, Atlas RO, Kopelman JN. Staircase sign: a newly described uterine contraction pattern seen in rupture of unscarred gravid uterus. *J Obstet Gynaecol Res*. 2008;34(1):100-104.

100. Vaknin Z, Maymon R, Mendlovic S, et al. Clinical, sonographic, and epidemiologic features of second- and early third-trimester spontaneous antepartum uterine rupture: a cohort study. *Prenat Diagn*. 2008;28(6):478-484.

101. Jauregui I, Kirkendall C, Ahn MO, Phelan J. Uterine rupture: a placentally mediated event? *Obstet Gynecol*. 2000;95(4)(Suppl 1):S75.

102. Sinkey RG, Odibo AO, Dashe JS; Society of Maternal-Fetal (SMFM) Publications Committee. #37: Diagnosis and management of vasa previa. *Am J Obstet Gynecol*. 2015;213(5):615-619.

103. Gagnon R, Morin L, Bly S, et al; DIAGNOSTIC IMAGING COMMITTEE; MATERNAL FETAL MEDICINE COMMITTEE. Guidelines for the management of vasa previa. *J Obstet Gynaecol Can*. 2009;31(8):748-753.

104. Oyelese KO, Turner M, Lees C, Campbell S. Vasa previa: an avoidable obstetric tragedy. *Obstet Gynecol Surv*. 1999;54(2):138-145.

105. Baulies S, Maiz N, Muñoz A, et al. Prenatal ultrasound diagnosis of vasa praevia and analysis of risk factors. *Prenat Diagn*. 2007;27(7):595-599.

106. Bronsteen R, Whitten A, Balasubramanian M, et al. Vasa previa: clinical presentations, outcomes, and implications for management. *Obstet Gynecol*. 2013;122(2 Pt 1):352-357.

107. Hasegawa J, Arakaki T, Ichizuka K, Sekizawa A. Management of vasa previa during pregnancy. *J Perinat Med*. 2015;43(6):783-784.

108. Rao KP, Belogolovkin V, Yankowitz J, Spinnato JA II. Abnormal placentation: evidence-based diagnosis and management of placenta previa, placenta accreta, and vasa previa. *Obstet Gynecol Surv*. 2012;67(8):503-519.

109. Lee W, Lee VL, Kirk JS, et al. Vasa previa: prenatal diagnosis, natural evolution, and clinical outcome. *Obstet Gynecol*. 2000;95(4):572-576.

Labor Dystocia

Learning Objectives

1. Understand the difference between normal labor and labor dystocia.
2. Describe how to prevent, diagnose, and treat labor dystocia.
3. Describe how obesity and induction may affect labor dystocia.

Introduction

Caring for women with labor dystocia, literally meaning *difficult labor*, is one of the greatest challenges of maternity care. Although this condition may not need the same emergent treatment as other clinical scenarios in the ALSO Provider Course, labor dystocia is common, and appropriate evidence-based care can improve clinical outcomes. This chapter reviews important concepts in the diagnosis, treatment, and prevention of dystocia.

Dystocia refers to prolonged or slowly progressing labor, which is common in nulliparous women as gauged by the number who require augmentation, operative vaginal delivery, or cesarean delivery. In 2017, 21.3% of women in the United States received augmentation during labor,[1] and the primary cesarean delivery rate (cesarean delivery in women without a prior cesarean) in reporting states was 21.9%.[1] In 30% to 50% of these deliveries, labor dystocia is cited as the indication for the procedure.[2,3] Although the overall cesarean delivery rate in the United States has declined slightly in recent years, it remains high at approximately 32% (*Figure 1*),[1] and thus attention is focusing on the two reasons for the primary cesarean delivery rate — labor dystocia and fetal heart rate (FHR) tracing interpretation.[2] The wide variation in cesarean delivery rates for labor dystocia suggests a need for more thoughtful, evidence-based care.[2,4-6] All maternity care clinicians need expertise in the care of women with dystocia.

Rethinking Latent Labor

Understanding normal and abnormal labor progress requires understanding latent or prodromal labor, to avoid performing cesarean delivery during a prolonged latent phase that is misdiagnosed as active labor. During latent labor, regular, painful contractions result in minimal or slow cervical change. The latent phase of labor starts with maternal perception of regular, painful contractions and ends when the rate of dilation starts to accelerate.[7]

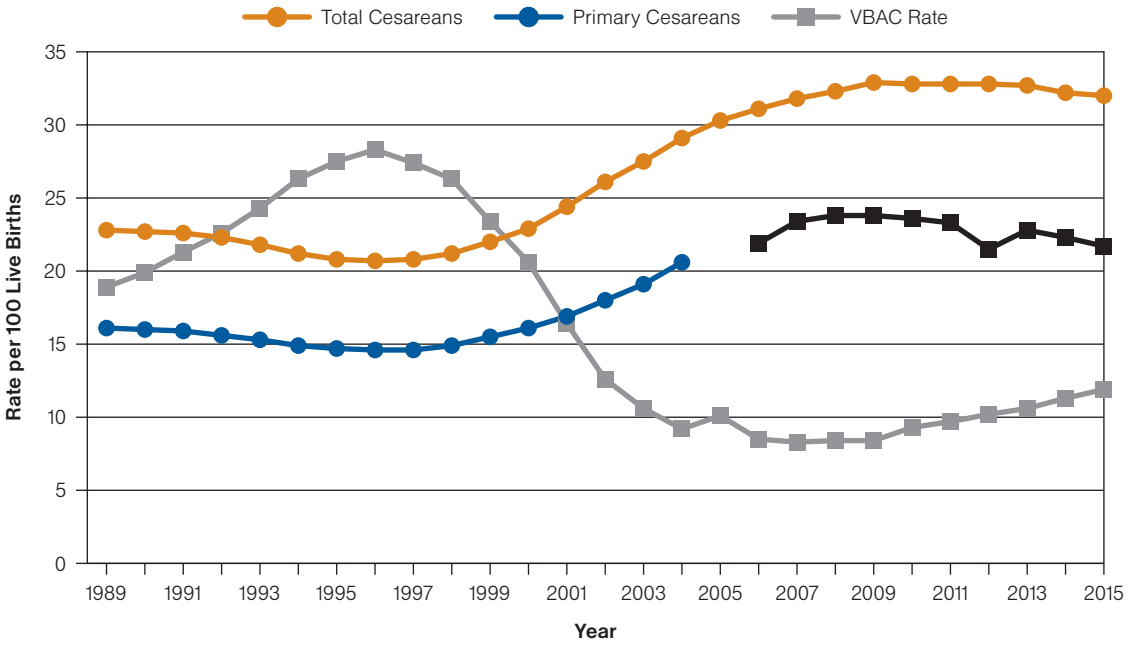
Recent studies show that normal labor may include a longer latent phase and thus a longer time frame and a less clear-cut transition to active labor than previously thought.[8] Reanalysis of data from the National Collaborative Perinatal Project from the 1960s, when fewer obstetric interventions were practiced, has allowed the normal course of natural spontaneous labor to be more accurately defined. This reanalysis showed that the active phase of labor, the onset of rapid dilation, may not start for multiparous women until at least 5-cm dilation, and may not start for nulliparous women until an even greater dilation that is more difficult to define.[9] Similar data from contemporary studies (Consortium on Safe Labor) suggest that the active phase of labor may not occur in multiparous women until 6-cm dilation.[9,10]

Care of Women in Latent Labor

For women in latent labor, clinicians can encourage adequate hydration, rest, emotional and physical support, and pharmacotherapy with antihistamines or short-term pharmacotherapy with opiates (eg, IV morphine) if needed.[11] Clinicians should work with women and their support systems to "create a plan for self-care activities and coping techniques" and schedule follow-up appointments to assess the plan.[12]

To prevent the misdiagnosis of labor dystocia in women in the latent phase of labor, clinicians can avoid admitting women too early.[13] This practice significantly reduces the risk of needing augmentation of labor or epidural analgesia.[14] Although cohort studies have not shown that whether women who are admitted early are at an increased risk of difficult labor and increased cesarean delivery rates, an analysis of Consortium on Safe Labor data in which more than two-thirds of low-risk nulliparous women who were admitted before the onset of active labor showed that it is "unlikely that early admission decisions are reserved only for women with inherent labor abnormalities. This supports the notion that systems factors that may affect patient outcomes should be examined, rather than exclusively

Figure 1. Total and Primary Cesarean Rate and Vaginal Birth After Previous Cesarean: United States, 1989-2015



Note: The black line indicates a primary cesarean delivery rate based on incomplete national data for US births in 2007 (53%) to 2015 (97%).

Information from National Partnership for Women & Families. Cesarean Birth Trends in the United States, 1989–2015. 2017. Available at http://www.nationalpartnership.org/research-library/maternal-health/cesarean-section-trends-1989-2014.pdf; Martin JA, Hamilton BE, Osterman MJK, Driscoll AK, Drake P. Births: Final data for 2016. Natl Vital Stat Rep. 2018;67(1):1-55; National Center for Health Statistics. The public use natality file – 2015 update. 2015. Available at ftp://ftp.cdc.gov/pub/Health_Statistics/NCHS/Dataset_Documentation/DVS/natality/UserGuide2015.pdf.

examining patient factors."[15] In another recent cohort study, term nulliparous women in spontaneous labor admitted before 4-cm dilation had a significantly higher risk of cesarean delivery compared with women admitted with more advanced dilation (21.8% versus 14.5%; relative risk [RR] 1.50; 95% confidence interval [CI] =1.32-1.70), and cesarean delivery rates decreased significantly (from 10.5% to 7.9%) when later admission became the standard.[16]

Although admission before the onset of advanced dilation of active labor is sometimes indicated, patience remains critical in treating such women. Care options for women admitted between 3- and 6-cm dilation include an antepartum department for rest and support,[17] including the availability of pharmacotherapy for maternal exhaustion or pain, as well as nonpharmacotherapies (eg, massage, water immersion).[12] Hospitals and providers can institute systems and policies such as the American College of Nurse-Midwives Promoting Spontaneous Progress in Labor bundle, which is based on the Five Rs mnemonic:[18]

- **R**eadiness (including early labor support lounges)
- **R**isk and appropriate assessment (including shared decision making)
- **R**eliable delivery of appropriate care (clear criteria for active labor diagnosis)
- **R**ecognition and response (shared information)
- **R**eporting/systems learning (tracking results)

Quality evidence about the effectiveness of these early labor strategies is limited.[19]

There is no contemporary definition of a prolonged latent phase of labor; a traditional definition used 14 hours for multiparous women and 20 hours for nulliparous women after regular contractions occurred.[20] Thus, there should be clear maternal or fetal indications to augment labor during the latent phase to justify the risks associated with oxytocin augmentation such as

uterine tachysystole, fetal intolerance of labor, and increased rates of operative intervention.[7,21] Cesarean delivery for labor dystocia should not be performed in latent labor unless certain criteria are met (*Table 1*).[2]

Factors Prolonging Latent Labor

The latent phase of labor may last longer in women undergoing labor induction than in women with spontaneous labor.[22] Several studies confirm the clinical safety of allowing women undergoing induction to have a latent phase of at least 12 hours after rupture of membranes (ROM).[23] Induced labor lasts longer than spontaneous labor, particularly for nulliparous women with less than 6-cm dilation. One retrospective study showed each centimeter of dilation before 6 cm could take 2 to 5.5 hours longer in women with induced rather than spontaneous labor.[22] Studies also show that women with obesity take longer to attain active labor, regardless of whether labor is spontaneous or induced.[24,25]

Diagnosis of Labor Dystocia

Contemporary studies have led to new definitions of arrested labor.[4,9,15] Thus, the key issue in diagnosing labor dystocia is not to apply active labor criteria for rate of dilation before 6-cm dilation. In addition, providers need to understand that cervical dilation is not linear, particularly in nulliparous women with early labor.[9] The clinical implication of using an inaccurate labor curve is that many women are admitted before the onset of active labor yet held to traditional expectations of the rate of progress of active labor.[26] This results in misdiagnosis of labor dystocia followed by a cascade of interventions that increase the risk of cesarean delivery. A 2013 study showed that more than 40% of primiparous women and more than 30% of multiparous women who undergo cesarean delivery for dystocia are less than 5-cm dilated and not in active labor.[4] An analysis of Consortium on Safe Labor data showed that approximately half of cesarean deliveries performed for dystocia in women admitted in active labor occurred when cervical dilation was physiologic.[15]

Not only is the start of active labor later and more difficult to define than previously thought, the normal rate of cervical change in active labor is slower. *Table 2* lists more accurate labor stage durations. A retrospective cohort study showed that, after 6-cm dilation, nulliparous women required an average of 2.2 hours to attain full dilation (95th percentile standard deviation = 10 hours, less than 0.5 cm/hour).[10]

After a woman has started active labor (6-cm dilation), the rate of cervical change may accelerate, but patience regarding the rate of dilation remains important (*Table 3*).[2,9] In the first stage of labor, arrest occurs when a woman with at least

Table 1. Definitions of Failed Induction and Arrest Disorders

Failed Induction of Labor

Failure to generate regular contractions and cervical change after at least 24 hours of oxytocin administration, with artificial membrane rupture if feasible

First-Stage Arrest

Six centimeters or greater dilation with membrane rupture and no cervical change for 4 hours or more of adequate contractions (eg, >200 Montevideo units) or 6 hours or more of oxytocin administration if contractions are inadequate

Second-Stage Arrest

No progress (descent or rotation) for:

≥4 hours in nulliparous women with epidural analgesia

≥3 hours in nulliparous women without epidural analgesia

≥3 hours in multiparous women with epidural analgesia

≥2 hours in multiparous women without epidural analgesia

Information from American College of Obstetricians and Gynecologists; Society for Maternal-Fetal Medicine. Obstetric care consensus no. 1: safe prevention of the primary cesarean delivery. Obstet Gynecol. *2014;123(3):693-711.*

Table 2. Labor Progress in Nulliparous and Multiparous Women

	Nulliparous	Multiparous
Active labor starts	>6 cm dilation	>6 cm dilation
Rate of dilation in active labor	Median 1.8 cm/hour (95% SD 0.4 cm/hr)	Median 2.5 cm/hour (95% SD 0.4 cm/hr)
Second stage duration	Median 0.9 hour (95% SD 3.1 hour)	Median 0.3 hour (95% SD 1.7 hour)

SD = standard deviation.

Information from Laughon SK, Branch DW, Beaver J, Zhang J. Changes in labor patterns over 50 years. Am J Obstet Gynecol. 2012;206(5):419.e1-419.e9; Spong CY, Berghella V, Wenstrom KD, Mercer BM, Saade GR. Preventing the first cesarean delivery: summary of a joint Eunice Kennedy Shriver National Institute of Child Health and Human Development, Society for Maternal-Fetal Medicine, and American College of Obstetricians and Gynecologists Workshop. Obstet Gynecol. 2012;120(5):1181-1193.

Table 3. Spontaneous Labor Progress Stratified by Cervical Dilation and Parity

Cervical Dilation (cm)	Parity 0 (95th percentile)	Parity 1 (95th percentile)	Parity 2 or Greater (95th percentile)
3-4	1.8 (8.1)	—	—
4-5	1.3(6.4)	1.4 (7.3)	1.4 (7.0)
5-6	0.8 (3.2)	0.8 (3.4)	0.8 (3.4)
6-7	0.6 (2.2)	0.5 (1.9)	0.5 (1.8)
7-8	0.5 (1.6)	0.4 (1.3)	0.4 (1.2)
8-9	0.5 (1.4)	0.3 (1.0)	0.3 (0.9)
9-10	0.5 (1.8)	0.3 (0.9)	0.3 (0.8)

Reprinted from American College of Obstetricians and Gynecologists; Society for Maternal-Fetal Medicine. Obstetric care consensus no. 1: safe prevention of the primary cesarean delivery. Obstet Gynecol. *2014;123(3):693-711.*

6-cm dilation and ROM has no cervical change for 4 hours or more of adequate contractions (eg, greater than 200 Montevideo units [MVUs]) or 6 hours or more of oxytocin administration if contractions are inadequate.[2]

Dystocia should be clinically defined by the 95th percentile of the current dilation rate. For example, if a cervix is 5-cm dilated, dystocia occurs if more than 3.2 hours is needed to attain 6-cm dilation, a progression of less than 0.3 to 0.4 cm/hour. Similarly, if a cervix is 8-cm dilated, dystocia occurs if the dilation rate is less than 0.7 cm/hour.[2] Given the limited accuracy of cervical examinations for minor degrees of dilation and the desire to avoid increased risk of infection, examinations to assess progress are commonly performed at 2- to 4-hour intervals.[2] As part of making this diagnosis, the clinician needs to address the following questions, using the Six Ps mnemonic as a guide:

- **P**assenger: Is there a malposition or malpresentation or suspected macrosomia?
- **P**ower: Are contractions adequate in frequency, duration, and strength?
- **P**elvis: Is there cephalopelvic disproportion because of a contracted pelvis?
- **P**atient: Are there other coexisting clinical issues such as chorioamnionitis or nonreassuring fetal monitoring that affect the treatment choices?
- **P**syche: How are the woman and her support individuals coping with the labor?
- **P**rovider: Is there a consistent examiner to evaluate subtle change? Are there any indications for consultation or second opinion?

Treatment in the Active Phase of Labor

First Stage Labor Dystocia

Treatment strategies in women with a slow progression of the active phase of labor include amniotomy, pharmacologic augmentation with oxytocin, or both.

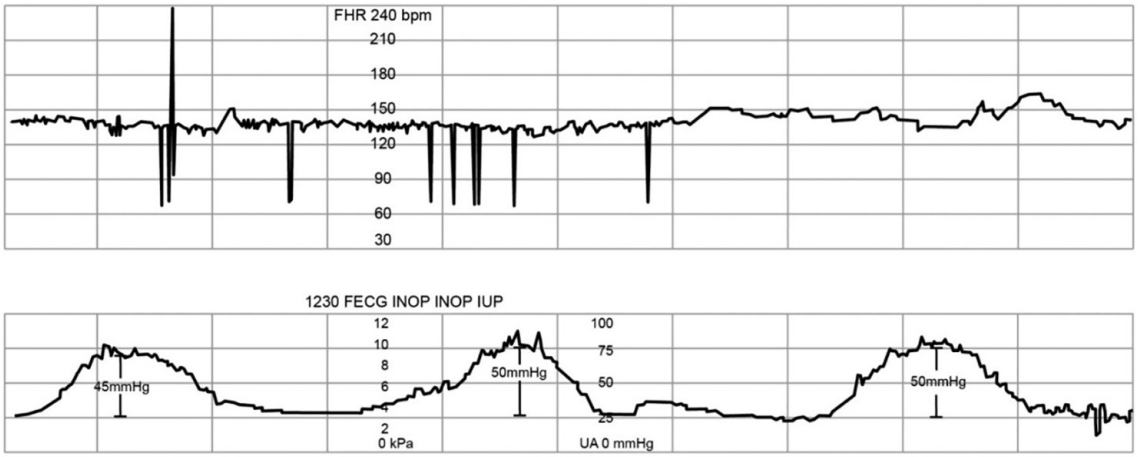
Amniotomy alone. A meta-analysis showed that women of any parity undergoing amniotomy alone were less likely to have nonprogressing labor than women who did not (RR 0.75; 95% CI = 0.64-0.88); similarly, oxytocin augmentation was less likely to be indicated in women who routinely underwent amniotomy (RR 0.73; 95% CI = 0.57-0.95).[27] Nulliparous women who underwent amniotomy had a slightly shorter second stage of labor (1 to 10 minutes) than those who did not. However, routine amniotomy alone in spontaneous labor had no effect on the overall duration of the first stage of labor or cesarean delivery rates.[27]

Pharmacologic augmentation. Oxytocin is the mainstay of pharmacotherapy for slow progression of the first stage of labor and will shorten the overall duration of labor. However, in randomized studies and meta-analyses addressing cesarean delivery rates as an outcome, oxytocin alone did not improve vaginal delivery rates.[28]

Combined amniotomy and pharmacologic augmentation. Although combining amniotomy with oxytocin may be the most effective augmentation strategy for *preventing* labor dystocia, data does not support their combined use in the *treatment* of labor dystocia. In trials of *prevention* strategies for labor dystocia, early augmentation with amniotomy and oxytocin was associated with a slight reduction in the number of cesarean deliveries (RR 0.87; 95% CI = 0.77-0.99) and a reduced duration of labor (average mean difference = -1.28 hours; 95% CI = -1.97 – -0.59) but no change in neonatal outcomes or maternal morbidity or satisfaction (number needed to treat [NNT] = 65).[29] Only three studies in this meta-analysis were for *treatment* of dystocia, with the combination of amniotomy and oxytocin use showing no reduction in cesarean delivery rates in women with slow progress in labor.

For women with protracted or arrested active-phase labor, the clinician can evaluate the strength

Figure 2. Measurement of Uterine Contraction Strength by Montevideo Units



and frequency of uterine contractions by abdominal palpation or an intrauterine pressure catheter (IUPC), which allows for calculation of MVUs (*Figure 2*). Two hundred MVUs or more in 10 minutes is considered evidence of adequate contractions.[30] IUPC use may be most beneficial if contractions seem to be of sufficient frequency and duration but are not producing the expected cervical change. However, a meta-analysis showed that IUPC use does not seem to affect labor duration or cesarean delivery rates.[31] IUPC use may increase the risk of maternal fever and should not be used routinely.[32]

If contractions are inadequate, administering intravenous (IV) oxytocin increases contraction frequency, duration, and strength.[33] There are numerous approaches to dosage selection, dosing interval, and duration of treatment. Low-dose regimens, which attempt to mimic the known steady-state pharmacodynamics of oxytocin, start at 1 to 2 mU/minute and increase by 1 to 2 mU/minute every 30 minutes to maximum doses of 36 mU/minute.[33] High-dose regimens, which should be routinely used only in nulliparous women, have starting doses of 4 to 6 mU/minute and incremental increases of 1 to 6 mU/minute up to a maximum of 36 mU/minute.[33] High-dose oxytocin for labor augmentation appears to result in shorter labors and to reduce the likelihood of cesarean delivery, but further trials are needed to assess important potential adverse outcomes and address women's experience of this treatment strategy.[34]

Appendix A lists sample oxytocin orders. Oxytocin is a drug with potential adverse effects so its use should be reserved for clearly defined indications.[21] Any use of oxytocin is typically combined with the routine use of continuous electronic fetal monitoring or the use of structured intermittent fetal auscultation every 15 minutes in stage one of labor and every 5 minutes in stage two (see the *Intrapartum Fetal Surveillance* chapter).[35]

After oxytocin has been administered to augment slow labor, patience in monitoring labor progress is once again critical.[2] Assuming fetal well-being, the clinician and the women should wait for at least 4 hours of adequate contractions after oxytocin augmentation before operative intervention for arrested dilation is considered. A study showed that not performing a cesarean delivery for labor dystocia until at least 4 hours of no cervical change during active labor rather than the traditional 2 hours lowered the cesarean delivery rate for arrest of labor from 26% to 8% without increasing maternal or fetal morbidity.[36,37]

During prolonged active phase labor, clinicians must remain vigilant to other clinical factors affecting the woman and fetus, such as fever portending chorioamnionitis or Category II and III FHR tracings.[38] Indeed, in clinical practice, it is sometimes challenging to determine whether a cesarean delivery is being performed because of a lack of labor progress or because of fetal intolerance to the oxytocin needed to affect labor progress. Clinicians should follow the Eunice Kennedy Shriver National Institute of Child Health and Human Development (NICHD) categories of FHR monitoring to classify abnormalities (see the *Intrapartum Fetal Surveillance* chapter).[39]

Second Stage Labor Dystocia

Labor dystocia can also occur in the second stage of labor and is characterized by prolonged duration or arrest of fetal descent (*Table 1*).[30] The median second-stage duration lasts longer for nulliparous women than traditionally defined with the 95th percentile at 2.8 hours without regional anesthesia and 3.6 hours with regional anesthesia.[2] For multiparous women, the 95th percentiles for second-stage duration with and without regional anesthesia remained at approximately 2 hours and 1 hour, respectively.[9] Studies on the neonatal and maternal effects of a prolonged second stage of labor yield mixed results,[38,40,41] but the likelihood of vaginal delivery diminishes when the second stage lasts longer than the time frames noted in *Table 1*.[30] Prolongation of the second stage beyond an arbitrary time limit is no longer an indication for operative vaginal or cesarean delivery. [42,43] Again, during a prolonged second stage of labor, clinicians must remain vigilant in evaluating labor progress regularly, if chorioamnionitis has developed, and assessing how the fetus is tolerating this stage of labor.

Second stage labor dystocia may occur secondary to fetal malposition, inadequate contractions, maternal exhaustion, or cephalopelvic disproportion. Each of these etiologies has potential management options.

Fetal malposition. The most common fetal malposition is occiput posterior (OP), where the fetus lies with its occiput toward the woman's spine and its face toward the woman's pubic symphysis. In the second stage of labor, this malposition occurs more frequently in women with epidural analgesia (12%) compared with women without epidural analgesia (3%) and is associated with a marked increase in operative intervention and increased maternal and neonatal morbidity.[44] Clinical diagnosis of OP is difficult during the second stage of labor, and the use of intrapartum ultrasound improves the accuracy of the diagnosis.[45] Manual rotation, as described in the *Malpresentations, Malpositions and Multiple Gestations* chapter, can result in vaginal delivery if performed successfully.[46]

Maternal position. The position of a woman with no epidural analgesia during the second stage of labor may affect labor duration by a few minutes, but not cesarean delivery rates.[47] Positioning the woman in an upright position may reduce the risk of abnormal FHR tracings (RR 0.46; 95% CI = 0.22-0.93) and assisted vaginal delivery compared with the supine position (RR 0.78; 95% CI = 0.68-0.90), but may increase the risk of postpartum hemorrhage (RR 1.65; 95% CI = 1.32-2.60).[47] Studies of the effects on labor of upright positioning in women with epidural analgesia are inconclusive.[48] Women in the second stage of labor with or without epidural analgesia should be encouraged to assume whatever position is most comfortable for them.

Inadequate contractions. During the second stage of labor, as in the first, if contractions have decreased in strength or frequency pharmacotherapy with oxytocin may be necessary. Thus, when fetal descent has slowed in the second stage, the provider may need to initiate or increase IV oxytocin, again remaining aware of how the fetus tolerates this augmentation.[49-51]

Maternal exhaustion. Maternal exhaustion can affect the duration of the second stage of labor. In women receiving epidural analgesia, an alternative to initiating active pushing at full cervical dilation is allowing the fetus to *labor down* to a lower station, or delaying pushing for 60 to 90 minutes until the woman develops an urge to push or the fetus is at the introitus.[50] A recent multicenter randomized controlled trial (RCT) did not show an increase in spontaneous vaginal delivery with a 60-minute period of passive descent.[52] Initiation of active pushing efforts without a period of passive descent was associated with lower rates of chorioamnionitis (RR 0.7; 95% CI = 0.6-0.9), postpartum hemorrhage (RR 0.6; 95% CI = 0.3-0.9), and neonatal acidemia (0.8% versus 1.2%, RR 0.7; 95% CI = 0.6-0.9). If women choose passive descent, they should be informed of the possible increased risk of morbidity.[52] If contractions occur more than 3 minutes apart, the addition of oxytocin should be considered during the laboring down process.[50]

During a prolonged second stage of labor, ongoing assessment of fetal well-being remains critical. Two basic strategies to prevent fetal intolerance of second stage labor are preventing vena cava compression by moving the woman out of the dorsal lithotomy position and allowing adequate periods of rest between pushing. Significant variable decelerations in the second stage of labor do not necessarily indicate fetal acidosis and may respond to amnioinfusion if an IUPC is already in place.[53] If

the FHR tracing continues to deteriorate with the combination of minimal/absent variability and late or variable decelerations, the provider should institute measures of fetal resuscitation while expediting delivery via assisted vaginal or cesarean delivery (see the *Intrapartum Fetal Surveillance* chapter).[35]

Prevention

Maternity care providers can attempt to decrease the risk of dystocia with the following antepartum and intrapartum strategies: undertaking prenatal interventions to decrease the incidence of fetal macrosomia, providing labor support and hydration, avoiding elective labor induction with an unripe cervix, using epidural analgesia judiciously, and preventing chorioamnionitis. The American College of Obstetricians and Gynecologists (ACOG) Committee on Obstetric Practice published additional methods for limiting interventions in normal birth.[12]

Obesity

Maternal obesity, particularly in association with excessive maternal weight gain during pregnancy or gestational diabetes, increases the risk of fetal macrosomia, which may predispose women to prolonged labor and operative delivery.[54,55] Dietary counseling during preconception, interconception, and prenatal care can help women with obesity and woman who are overweight to limit their weight gain, which has been shown to decrease the risk of shoulder dystocia.[56] A meta-analysis that also included women with normal weight showed that these lifestyle interventions, including physical activity counseling, reduce the risk of cesarean delivery (RR 0.91; 95% CI = 0.83-0.9).[57,58]

Managing suspected macrosomia remains controversial. A RCT showed a lower risk of cesarean delivery and birth injury in women induced between 37 and 39 weeks' gestation for ultrasound-confirmed estimated fetal weight greater than the 95th percentile,[59] whereas a meta-analysis showed no overall reduction in cesarean delivery and a reduced risk of shoulder dystocia and birth injury.[60]

In women with obesity, providers should follow the same initial guidelines for recognition and treatment of dysfunctional labor as for women without obesity. However, patience with slower labor progress in women with obesity is important in latent labor, as discussed previously, and in active labor. One study showed nulliparous women who are overweight or obese had significantly longer active phases of labor compared with women with normal weight.[61] Induction with prostaglandins appears to take longer in women with obesity;[62] one retrospective study also suggests that misoprostol rather than dinoprostone may improve the likelihood of vaginal delivery in women with obesity.[63] Finally, women with obesity may need higher doses of oxytocin than women with normal weight to attain adequate contractions.[64]

Labor Support

A meta-analysis of studies on providing trained labor support companions (doulas) showed a decreased incidence of labor dystocia, operative vaginal deliveries, and cesarean deliveries, particularly in nulliparous women.[65] The greatest effect on labor occurs when trained lay individuals rather than clinicians are used, when epidural analgesia is not routinely used, and when support starts in early labor.[65] A potential low-cost alternative is for a woman to select a friend or family member to receive specific brief labor support training as part of prenatal care and to accompany her during labor; in one trial, this strategy led to an overall reduction in labor durations but no difference in cesarean delivery rates.[66] See *Appendix B* for additional information about doula programs.[67]

Hydration

Hydration during active labor may prevent prolonged labor and reduce the risk of cesarean delivery for dystocia. A meta-analysis of studies of nulliparous women in spontaneous labor at term found that administering 250 mL/hour of IV fluids during labor instead of 125 mL/hour reduces the risk of cesarean delivery for any indication (12.5 versus 18.1%; RR 0.70, 95% CI = 0.53-0.92) and labor dystocia (4.9 versus 7.7%; RR 0.60, 95% CI = 0.38-0.97); the higher rate is also associated with a shorter first stage of labor (mean difference -64.38 minutes; 95% CI = -121.88 – -6.88).[68] However, in the few trials where women had unrestricted oral intake, the rates of IV fluid administration did not affect labor outcomes.[68,69]

Labor Induction

Labor induction rates in the United States, after a decline,[70] increased to 25.7% in 2017.[1] Recent study results show that, contrary to previous

thought, labor induction does not increase and may even decrease cesarean delivery risk compared with expectant management, although methodological questions remain.[71-74] Cochrane reviews of mechanical methods for cervical ripening, vaginal misoprostol and vaginal prostaglandin, showed that these agents decrease the duration of labor but do not change the overall cesarean delivery rate for labor dystocia;[71-73] however, oral misoprostol for induction compared with vaginal dinoprostone or oxytocin may lead to fewer cesarean deliveries.[74]

In women receiving oxytocin for labor induction before 6-cm dilation, some study results show that cesarean delivery rates may be reduced by discontinuing the infusion at approximately 5-cm dilation,[75] whereas a meta-analysis found no benefit to this practice.[76] A meta-analysis showed that an approach to patient treatment that uses selective induction in women who are at or near term with specific risk factors may reduce cesarean delivery rates.[77] A retrospective analysis of cross-sectional data from the Consortium on Safe Labor showed that elective induction at term had a lower risk of cesarean delivery than expectant management, regardless of parity or cervical readiness.[78]

A 2016 RCT of induction at 39 weeks' gestation in women older than 35 years showed high baseline operative delivery rates but no difference in cesarean delivery rates when comparing induced labor with expectant management.[79] A meta-analysis of six cohort studies comparing induction at 39 weeks' gestation with expectant management showed that induction was associated with a significantly lower risk of cesarean delivery, maternal peripartum infection, and perinatal adverse outcomes (eg, respiratory morbidity, intensive care unit admission, mortality).[80]

Finally, the 2018 A Randomized Trial of Induction Versus Expectant Management (ARRIVE) study showed that in low-risk nulliparous women, induction at 39 weeks' gestation reduced the cesarean delivery rate from 22.2% to 18.6% (NNT = 28).[81] What remains unclear is how applicable these results are beyond the low-risk women in the trial and hospitals with baseline nulliparous, term, singleton, vertex cesarean delivery rates less than 18%.[81] Although the study included women receiving care in community hospitals as well as academic centers, it also remains uncertain whether such results can be replicated in real-world practice where pressures around duration of induction and hospital/staff capacity may hinder the patience needed for induction.[81]

Offering labor induction at 39 weeks' gestation should be conducted in the context of shared decision making.[82] Training may be needed to better implement shared decision making in maternity care. Recent study results show that true shared decision making principles are not used reliably[83,84] and women from marginalized social groups particularly may be at increased risk of being excluded from the decision making process.[85] In addition, providers and hospitals offering elective labor induction should also offer labor support (doula care), which has also been shown to be effective in reducing cesarean delivery rates in nulliparous women.[65] Following contemporary labor progress guidelines, including the understanding that induced labor takes longer than spontaneous labor, is also critical when implementing a labor induction protocol (see the Quality Improvement section of this chapter). Elective induction without medical indication should still only be performed after 39 weeks' gestation.[2]

Labor induction with the goal of vaginal delivery requires patience in allowing adequate time for progress in latent and active labor. A failed induction should be diagnosed only when a woman has not had regular contractions (every 3 minutes) with cervical change after at least 24 hours of oxytocin administration, assuming fetal well-being during that time. If a woman has intact membranes, fetal monitoring is not concerning, and there are no other medical concerns (eg, hypertension), it may be possible to discontinue labor induction efforts and discharge the woman instead of performing a cesarean delivery when cervical change is not occurring.[2]

Epidural Anesthesia

Although meta-analyses consistently show no difference in cesarean delivery rates among women receiving low-dose epidural analgesia compared with parenteral opioids,[86] epidural analgesia does affect labor progress and other outcomes. Women receiving epidural analgesia are more likely to require oxytocin augmentation in the first stage of labor, have longer second stages of labor,[87] have a sixfold increase in the incidence of maternal fever, have increased incidence of persistent OP

malposition, and undergo more operative vaginal deliveries.[88]

Whether administering epidural analgesia early in labor (before 4- to 5-cm dilation) increases the risk of cesarean delivery has been controversial.[89] Epidural analgesia is not a single entity, and RCTs that have specifically investigated early versus standard placement are small or do not use contemporary low-dose techniques.[89] The study most commonly cited to support the use of epidural analgesia early in labor actually compared a combined spinal epidural analgesia technique (intrathecal opioid administered at 2-cm dilation) to epidural analgesia administered at 4 cm or later. This study showed no significant differences in labor duration or rates of cesarean delivery.[90]

Maternal request is sufficient indication for pain relief during labor,[91] and epidural analgesia is associated with significantly lower pain scores compared with systemic opioids.[86] Providers should individualize treatment when deciding whether and when to administer epidural analgesia. Women with significant pain early in labor should not be required to attain 4- to 5-cm dilation before receiving epidural analgesia.[91] Conversely, a woman who is informed and prepared to handle labor pain with lesser interventions should not be subjected to the expectation of routine epidural analgesia administration. Providers should support the availability of other pain-relief options that may have a lesser effect on labor duration such as water immersion, nitrous oxide, and sterile water injections.[92-95]

If a woman is experiencing severe *back labor* (ie, experiencing contractions most intensely in the lower back), this may indicate a persistent OP malposition. If changing the woman's position does not relieve this discomfort, another option for pain relief before attempting regional anesthesia is a trial of sterile water injections (*Appendix C*). The effectiveness of these injections in reducing pain or cesarean delivery rates is debatable. One review showed studies to be inconclusive.[96] Another review, which included slightly different trials, showed that sterile water injections for low back pain in labor reduced pain for up to 2 hours and reduced the risk of cesarean delivery (RR 0.51; 95% CI = 0.30-0.87) compared with alternative therapies; however, the overall cesarean delivery rate was less than 10% in the comparison therapy group.[94]

Infection

Chorioamnionitis is associated with an increased incidence of labor dystocia (adjusted odds ratio [OR] 2.3, 95% CI = 2.0-2.7 in the first stage of labor, and OR 1.8, 95% CI = 1.5-2.2 in the second stage of labor) and cesarean delivery (OR 1.8; 95% CI = 1.5-2.1).[97] Clinicians should delay the initial digital vaginal examination and substitute a sterile speculum examination in any patient with term ROM who is not in labor. Attempts should be made to limit the total number of vaginal examinations after ROM to five or less,[98] although more recent studies show less risk than previously thought.[99]

Ambulation and Position

A recent meta-analysis concludes that women who walk or remain upright during the first stage of labor have reduced labor durations and reduced risk of cesarean delivery compared with women who remain recumbent or supine during labor.[100] However, as mentioned previously, low-risk women should be encouraged to assume whatever position is most comfortable for them during labor.

Quality Improvement

Certain aspects of clinician care styles and health care systems may prevent labor dystocia and resultant cesarean delivery. These include caregiver continuity during the assessment of early labor, encouraging a pronatalist cultural attitude toward natural childbirth,[101,102] requiring consultation with a second clinician before nonemergent cesarean deliveries for labor dystocia,[101] and providing regular feedback to clinicians regarding cesarean delivery rates.[103,104]

One small study showed a decreased cesarean delivery rate in the first stage of labor when women receive care from their primary prenatal health care provider rather than an on-call hospitalist.[105] Improving quality of care as part of overall maternity safety has led to a plateauing of cesarean delivery rates.[106,107] Finally, reductions in cesarean delivery rates were shown in recent studies of several hospital systems that adopted the Council on Patient Safety in Women's Health Care Safe Reduction of Primary Cesarean Births patient safety bundle (see page 10) and ACOG and Society for Maternal-Fetal Medicine consensus guidelines to safely prevent cesarean deliv-

ery.[106,107] Whether a laborist model of care or a midwifery model of care affects cesarean delivery rates remains unclear.[108-110] The recent ACOG Committee Opinion on strategies to limit intervention in labor and support vaginal birth aligns with other national recommendations about avoiding iatrogenic labor dystocia and unnecessary cesarean deliveries. [6,12]

Summary

Labor dystocia is common and requires the maternity care clinician to have excellent clinical assessment skills and thorough knowledge of nonpharmacologic and pharmacologic strategies to prevent and treat nonprogressing labor.

Nursing Considerations: Labor Dystocia

- Nurses can directly affect cesarean delivery rates. Consider the effect of care on patients.
- Advocate: triage prior to admission, hydration, rest, emotional and physical support, and judicious use of oxytocin
- Encourage and model shared decision making and family centered care
- Champion a quality improvement project at your institution
- Remember that oxytocin is a high-alert drug with an onset of action of 3 to 5 minutes and a steady state of 30 to 60 minutes[1]

1. Page K, McCool WF, Guidera M. Examination of the pharmacology of oxytocin and clinical guidelines for use in labor. J Midwifery Women's Health.*2017; 62(4):425-433.*

COUNCIL ON PATIENT SAFETY
IN WOMEN'S HEALTH CARE
safe health care for every woman

PATIENT SAFETY BUNDLE

Safe Reduction of Primary Cesarean Births

SAFE REDUCTION OF PRIMARY CESAREAN BIRTHS: SUPPORTING INTENDED VAGINAL BIRTHS

READINESS

Every Patient, Provider and Facility

- Build a provider and maternity unit culture that values, promotes, and supports spontaneous onset and progress of labor and vaginal birth and understands the risks for current and future pregnancies of cesarean birth without medical indication.
- Optimize patient and family engagement in education, informed consent, and shared decision making about normal healthy labor and birth throughout the maternity care cycle.
- Adopt provider education and training techniques that develop knowledge and skills on approaches which maximize the likelihood of vaginal birth, including assessment of labor, methods to promote labor progress, labor support, pain management (both pharmacologic and non-pharmacologic), and shared decision making.

RECOGNITION AND PREVENTION

Every patient

- Implement standardized admission criteria, triage management, education, and support for women presenting in spontaneous labor.
- Offer standardized techniques of pain management and comfort measures that promote labor progress and prevent dysfunctional labor.
- Use standardized methods in the assessment of the fetal heart rate status, including interpretation, documentation using NICHD terminology, and encourage methods that promote freedom of movement.
- Adopt protocols for timely identification of specific problems, such as herpes and breech presentation, for patients who can benefit from proactive intervention before labor to reduce the risk for cesarean birth.

October 2015

COUNCIL ON PATIENT SAFETY
IN WOMEN'S HEALTH CARE
safe health care for every woman

PATIENT SAFETY BUNDLE

Safe Reduction of Primary Cesarean Births

RESPONSE

To Every Labor Challenge

- Have available an in-house maternity care provider or alternative coverage which guarantees timely and effective responses to labor problems.
- Uphold standardized induction scheduling to ensure proper selection and preparation of women undergoing induction.
- Utilize standardized evidence-based labor algorithms, policies, and techniques, which allow for prompt recognition and treatment of dystocia.
- Adopt policies that outline standard responses to abnormal fetal heart rate patterns and uterine activity.
- Make available special expertise and techniques to lessen the need for abdominal delivery, such as breech version, instrumented delivery, and twin delivery protocols.

REPORTING/SYSTEMS LEARNING

Every birth facility

- Track and report labor and cesarean measures in sufficient detail to: 1) compare to similar institutions, 2) conduct case review and system analysis to drive care improvement, and 3) assess individual provider performance.
- Track appropriate metrics and balancing measures, which assess maternal and newborn outcomes resulting from changes in labor management strategies to ensure safety.

Standardization of health care processes and reduced variation has been shown to improve outcomes and quality of care. The Council on Patient Safety in Women's Health Care disseminates patient safety bundles to help facilitate the standardization process. This bundle reflects emerging clinical, scientific, and patient safety advances as of the date issued and is subject to change. The information should not be construed as dictating an exclusive course of treatment or procedure to be followed. Although the components of a particular bundle may be adapted to local resources, standardization within an institution is strongly encouraged.

The Council on Patient Safety in Women's Health Care is a broad consortium of organizations across the spectrum of women's health for the promotion of safe health care for every woman.

October 2015

Reprinted from Council on Patient Safety in Women's Health Care. AIM-Supported Patient Safety Bundles. Safe Reduction of Primary Cesarean Births. Available at https://safehealthcareforeverywoman.org/.

Appendix A
Sample Labor Orders for Oxytocin

1. Consider oxytocin augmentation if:
   a) Cervical dilation rate is less than 0.4 cm/hour in a woman up to 6-cm dilation or less than 1.0 cm/hour if 6-cm dilation or more and any of the following:
   b) Contractions occur less frequently than every 3 minutes
   c) Contractions last less than 30 seconds
   d) Contractions are not palpable
   e) Intrauterine pressure catheter documents less than 200 Montevideo units (MVUs) in a 30-minute period
   f) Amniotomy does not cause labor progress in 1 to 3 hours
2. Document the decision to augment in the medical record; consider using a formal oxytocin checklist or bundle
3. Electronically monitor fetal heart rate and uterine activity for a minimum of 15 minutes before initiation of oxytocin
4. Obtain blood pressure measurements every 15 to 30 minutes
5. Start primary intravenous (IV) infusion of 1,000 mL lactated Ringer's solution at a keep vein open rate
6. Through the infusion pump, add a secondary IV dose of 1,000 mL 5% lactated Ringer's solution with 10 to 20 units of oxytocin (20 units preferred to decrease the total amount of fluid administered). An alternative could be 500 mL with 30 units of oxytocin
7. Start oxytocin infusion at 0.5 to 2 mU/minute
8. Increase oxytocin dose by 1 to 2 mU/minute every 30 minutes, until an adequate contraction pattern has been attained. After 10 mU/minute has been attained, the rate of increase may be 1 to 4 mU/minute
9. Notify the clinician before exceeding 20 mU/minute. The clinician should document any decision to exceed this dosage in the medical record. The maximum infusion rate is 36 mU/minute
10. Discontinue or decrease oxytocin dose by 50% if any of the following occur and notify provider:
    a) Tachysystole (more than 5 contractions within 10 minutes averaged over 30 minutes)
    b) Uterine tone between contractions exceeds 15 to 20 mm Hg
    c) Tetanic contractions occur (contractions lasting longer than 2 minutes)
    d) Severe variable or late decelerations, bradycardia, or tachycardia occur
    e) Less severe fetal heart rate patterns may be managed as if the patient has spontaneous labor by administration of a fluid bolus, position change, and oxygen at 10 L/minute via non-rebreather mask
    f) Interventions such as these should be documented in the medical record
11. Consider augmentation to have failed if:
    a) The woman is at least 6 cm dilated with ruptured membranes and has had no cervical change after 4 hours of adequate contractions (defined as more than 200 MVUs in 30 minutes) by intrauterine pressure catheter or 6 hours of inadequate contractions without intrauterine pressure catheter
    b) No regular contractions (every 3 minutes) after 24 hours of oxytocin administration, preferably with rupture of membranes

Information from various sources.

Appendix B
Doula Organizations and Resources

This list is not meant to be complete but to provide suggested resources. Most of the doula certification programs require trainees to provide labor support for a certain number of births to obtain certification. Additional resources, including lists of doula professionals, are available at http://www.doulas.com/.

Doulas of North America International (DONA): http://www.dona.org

Childbirth and Postpartum Professional Association (CAPPA): http://www.cappa.net

The International Childbirth Education Association (ICEA): http://www.icea.org

Appendix C
Intradermal Sterile Water Injections

Intradermal sterile water injections can be used to treat *back labor* pain in the first stage of labor. Four 0.1 mL intradermal injections of sterile water with a 25- or 27-gauge needle are administered to form blebs in the skin. Two injection sites are over the posterior superior iliac spines; two are 2 to 3 cm below and 1 to 2 cm medial to the first points. The injections can cause pain for 15 to 30 seconds but are followed within 2 minutes by partial to complete relief of back pain that lasts 45 to 90 minutes. These injections can be repeated if necessary. Using two clinicians, these injections can be injected simultaneously to decrease the duration of discomfort.

References

1. Martin JA, Hamilton BE, Osterman MJK, et al. Births: Final Data for 2017. *Natl Vital Stat Rep*. 2018;67(8):1-50.
2. Spong CY, Berghella V, Wenstrom KD, et al. Preventing the first cesarean delivery: summary of a joint Eunice Kennedy Shriver National Institute of Child Health and Human Development, Society for Maternal-Fetal Medicine, and American College of Obstetricians and Gynecologists Workshop. *Obstet Gynecol*. 2012;120(5): 1181-1193.
3. Barber EL, Lundsberg LS, Belanger K, et al. Indications contributing to the increasing cesarean delivery rate. *Obstet Gynecol*. 2011; 118(1):29-38.
4. Boyle A, Reddy UM, Landy HJ, et al. Primary cesarean delivery in the United States. *Obstet Gynecol*. 2013;122(1):33-40.
5. King TL. Preventing primary cesarean sections: intrapartum care. *Semin Perinatol*. 2012;36(5):357-364.
6. Smith H, Peterson N, Lagrew D, Main E; California Maternal Quality Care Collaborative. Toolkit to support vaginal birth and reduce primary cesareans. 2017. Available at https://www.cmqcc.org/VBirthToolkitResource.
7. Greulich B, Tarrant B. The latent phase of labor: diagnosis and management. *J Midwifery Womens Health*. 2007;52(3):190-198.
8. Friedman EA. Primigravid labor; a graphicostatistical analysis. *Obstet Gynecol*. 1955;6(6):567-589.
9. Zhang J, Landy HJ, Branch DW, et al; Consortium on Safe Labor. Contemporary patterns of spontaneous labor with normal neonatal outcomes. *Obstet Gynecol*. 2010;116(6):1281-1287.
10. Laughon SK, Branch DW, Beaver J, Zhang J. Changes in labor patterns over 50 years. *Am J Obstet Gynecol*. 2012;206(5):419.e1-419.e9.
11. Simkin P, Hanson L, Ancheta RS. *The Labor Progress Handbook: Early Interventions to Prevent and Treat Dystocia*, 4th ed. Hoboken, NJ: Wiley-Blackwell; 2017.
12. Committee on Obstetric Practice. Committee opinion no. 687: Approaches to limit intervention during labor and birth. *Obstet Gynecol*. 2017;129(2):e20-e28.
13. Neal JL, Lamp JM, Buck JS, et al. Outcomes of nulliparous women with spontaneous labor onset admitted to hospitals in preactive versus active labor. *J Midwifery Womens Health*. 2014;59(1):28-34.
14. McNiven PS, Williams JI, Hodnett E, et al. An early labor assessment program: a randomized, controlled trial. *Birth*. 1998;25(1):5-10.
15. Neal JL, Lowe NK, Caughey AB, et al. Applying a physiologic partograph to Consortium on Safe Labor data to identify opportunities for safely decreasing cesarean births among nulliparous women. *Birth*. 2018;45(4):358-367.
16. Kauffman E, Souter VL, Katon JG, Sitcov K. Cervical dilation on admission in term spontaneous labor and maternal and newborn outcomes. *Obstet Gynecol*. 2016;127(3):481-488.
17. Paul JA, Yount SM, Breman RB, et al. Use of an early labor lounge to promote admission in active labor. *J Midwifery Womens Health*. 2017;62(2):204-209.
18. The American College of Nurse-Midwives. Promoting spontaneous progress in labor. 2015. Available at http://birthtools.org/birthtools/files/BirthToolFiles/FILENAME/000000000091/BundlePromotingLaborProgress-Final-091515.pdf.
19. Kobayashi S, Hanada N, Matsuzaki M, et al. Assessment and support during early labour for improving birth outcomes. *Cochrane Database Syst Rev*. 2017;4:CD011516. .
20. Cohen WR, Friedman EA. Diagnosing and treating dysfunctional labor. In: Cohen WR, Friedman EA. *Labor and delivery care: a practical guide*. Hoboken, NJ: Wiley-Blackwell; 2011.
21. Clark SL, Simpson KR, Knox GE, Garite TJ. Oxytocin: new perspectives on an old drug. *Am J Obstet Gynecol*. 2009;200(1):35.e1-35.e6.
22. Harper LM, Caughey AB, Odibo AO, et al. Normal progress of induced labor. *Obstet Gynecol*. 2012;119(6):1113-1118.
23. Rouse DJ, Weiner SJ, Bloom SL, et al; Eunice Kennedy Shriver National Institute of Child Health and Human Development (NICHD) Maternal-Fetal Medicine Units Network (MFMU). Failed labor induction: toward an objective diagnosis. *Obstet Gynecol*. 2011;117(2 Pt 1): 267-272.
24. Norman SM, Tuuli MG, Odibo AO, et al. The effects of obesity on the first stage of labor. *Obstet Gynecol*. 2012;120(1):130-135.
25. Kominiarek MA, Zhang J, Vanveldhuisen P, et al. Contemporary labor patterns: the impact of maternal body mass index. *Am J Obstet Gynecol*. 2011;205(3):244.e1-244.e8.
26. Neal JL, Lowe NK, Ahijevych KL, et al. "Active labor" duration and dilation rates among low-risk, nulliparous women with spontaneous labor onset: a systematic review. *J Midwifery Womens Health*. 2010; 55(4):308-318.
27. Smyth RMD, Alldred SK, Markham C. Amniotomy for shortening spontaneous labour. *Cochrane Database Syst Rev*. 2013;(1): CD006167.
28. Bugg GJ, Siddiqui F, Thornton JG. Oxytocin versus no treatment or delayed treatment for slow progress in the first stage of spontaneous labour. *Cochrane Database Syst Rev*. 2013;6(6):CD007123.
29. Wei S, Wo BL, Qi HP, et al. Early amniotomy and early oxytocin for prevention of, or therapy for, delay in first stage spontaneous labour compared with routine care. *Cochrane Database Syst Rev*. 2013; 8(8):CD006794.
30. American College of Obstetricians and Gynecologists; Society for Maternal-Fetal Medicine. Obstetric care consensus no. 1: safe prevention of the primary cesarean delivery. *Obstet Gynecol*. 2014; 123(3):693-711.
31. Bakker JJH, Janssen PF, van Halem K, et al. Internal versus external tocodynamometry during induced or augmented labour. *Cochrane Database Syst Rev*. 2013;8(8):CD006947.
32. Harper LM, Shanks AL, Tuuli MG, et al. The risks and benefits of internal monitors in laboring patients. *Am J Obstet Gynecol*. 2013; 209(1):38.e1-38.e6.
33. American College of Obstetrics and Gynecology Committee on Practice Bulletins-Obstetrics. ACOG Practice Bulletin number 49, December 2003: Dystocia and augmentation of labor. *Obstet Gynecol*. 2003;102(6):1445-1454.
34. Kenyon S, Tokumasu H, Dowswell T, et al. High-dose versus low-dose oxytocin for augmentation of delayed labour. *Cochrane Database Syst Rev*. 2013;7(7):CD007201.
35. American College of Obstetricians and Gynecologists. ACOG Practice Bulletin no. 106: Intrapartum fetal heart rate monitoring: nomenclature, interpretation, and general management principles. *Obstet Gynecol*. 2009;114(1):192-202.
36. Rouse DJ, Owen J, Hauth JC. Active-phase labor arrest: oxytocin augmentation for at least 4 hours. *Obstet Gynecol*. 1999;93(3): 323-328.
37. Rouse DJ, Owen J, Savage KG, Hauth JC. Active phase labor arrest: revisiting the 2-hour minimum. *Obstet Gynecol*. 2001;98(4): 550-554.

38. Laughon SK, Berghella V, Reddy UM, et al. Neonatal and maternal outcomes with prolonged second stage of labor. *Obstet Gynecol*. 2014;124(1):57-67.
39. Macones GA, Hankins GD, Spong CY, et al. The 2008 National Institute of Child Health and Human Development workshop report on electronic fetal monitoring: update on definitions, interpretation, and research guidelines. *Obstet Gynecol*. 2008;112(3):661-666.
40. Gimovsky AC, Berghella V. Randomized controlled trial of prolonged second stage: extending the time limit vs usual guidelines. *Am J Obstet Gynecol*. 2016;214(3):361.e1-361.e6.
41. Zipori Y, Grunwald O, Ginsberg Y, et al. The impact of extending the second stage of labor to prevent primary cesarean delivery on maternal and neonatal outcomes. *Am J Obstet Gynecol*. 2019; 220(2):191.e1-191.e7.
42. El-Sayed YY. Diagnosis and management of arrest disorders: duration to wait. *Semin Perinatol*. 2012;36(5):374-378.
43. Rouse DJ, Weiner SJ, Bloom SL, et al; Eunice Kennedy Shriver National Institute of Child Health and Human Development Maternal-Fetal Medicine Units Network. Second-stage labor duration in nulliparous women: relationship to maternal and perinatal outcomes. *Am J Obstet Gynecol*. 2009;201(4):357.e1-357.e7.
44. Caughey AB, Sharshiner R, Cheng YW. Fetal malposition: impact and management. *Clin Obstet Gynecol*. 2015;58(2):241-245.
45. Malvasi A, Tinelli A, Barbera A, et al. Occiput posterior position diagnosis: vaginal examination or intrapartum sonography? A clinical review. *J Matern Fetal Neonatal Med*. 2014;27(5):520-526.
46. Shaffer BL, Cheng YW, Vargas JE, Caughey AB. Manual rotation to reduce caesarean delivery in persistent occiput posterior or transverse position. *J Matern Fetal Neonatal Med*. 2011;24(1):65-72.
47. Gupta JK, Hofmeyr GJ, Shehmar M. Position in the second stage of labour for women without epidural anaesthesia. *Cochrane Database Syst Rev*. 2012;5(5):CD002006.
48. Kibuka M, Thornton JG. Position in the second stage of labour for women with epidural anaesthesia. *Cochrane Database Syst Rev*. 2017;2:CD008070. .
49. O'Driscoll K, Meagher D, Robson M. *Active Management of Labour: the Dublin Experience.* 4th ed. Edinburgh; New York: Mosby; 2003.
50. Sprague AE, Oppenheimer L, McCabe L, et al. The Ottawa Hospital's Clinical Practice Guideline for the Second Stage of Labour. *J Obstet Gynaecol Can*. 2006;28(9):769-779.
51. Kopas ML. A review of evidence-based practices for management of the second stage of labor. *J Midwifery Womens Health*. 2014; 59(3):264-276.
52. ACOG Committee Opinion no. 766: approaches to limit intervention during labor and birth. *Obstet Gynecol*. 2019;133(2):e164-e173.
53. Hofmeyr GJ, Lawrie TA. Amnioinfusion for potential or suspected umbilical cord compression in labour. *Cochrane Database Syst Rev*. 2012;1(1):CD000013.
54. American College of Obstetricians and Gynecologists. ACOG Practice Bulletin no, 156: Obesity in Pregnancy. *Obstet Gynecol*. 2015; 126(6):e112-e126.
55. Ehrenberg HMMB, Mercer BM, Catalano PM. The influence of obesity and diabetes on the prevalence of macrosomia. *Am J Obstet Gynecol*. 2004;191(3):964-968.
56. Thangaratinam S, Rogozinska E, Jolly K, et al. Effects of interventions in pregnancy on maternal weight and obstetric outcomes: meta-analysis of randomised evidence. *BMJ*. 2012;344:e2088. .
57. International Weight Management in Pregnancy (i-WIP) Collaborative Group. Effect of diet and physical activity based interventions in pregnancy on gestational weight gain and pregnancy outcomes: meta-analysis of individual participant data from randomised trials. *BMJ*. 2017;358:j3119.
58. Muktabhant B, Lawrie TA, Lumbiganon P, Laopaiboon M. Diet or exercise, or both, for preventing excessive weight gain in pregnancy. *Cochrane Database Syst Rev*. 2015;6(6):CD007145.
59. Boulvain M, Senat MV, Perrotin F, et al; Groupe de Recherche en Obstétrique et Gynécologie (GROG). Induction of labour versus expectant management for large-for-date fetuses: a randomised controlled trial. *Lancet*. 2015;385(9987):2600-2605.
60. Boulvain M, Irion O, Dowswell T, Thornton JG. Induction of labour at or near term for suspected fetal macrosomia. *Cochrane Database Syst Rev*. 2016;(5):CD000938.
61. Vahratian A, Zhang J, Troendle JF, et al. Maternal prepregnancy overweight and obesity and the pattern of labor progression in term nulliparous women. *Obstet Gynecol*. 2004;104(5 Pt 1):943-951.
62. Pevzner L, Powers BL, Rayburn WF, et al. Effects of maternal obesity on duration and outcomes of prostaglandin cervical ripening and labor induction. *Obstet Gynecol*. 2009;114(6):1315-1321.
63. Suidan RS, Rondon KC, Apuzzio JJ, Williams SF. Labor outcomes of obese patients undergoing induction of labor with misoprostol compared to dinoprostone. *Am J Perinatol*. 2015;30(2):187-192.
64. Hill M, Reed KL, Cohen WR. Oxytocin utilization for labor induction in obese and lean women. *J Perinat Med*. 2015;43(6):703-706.
65. Bohren MA, Hofmeyr GJ, Sakala C, et al. Continuous support for women during childbirth. *Cochrane Database Syst Rev*. 2017;7(7): CD003766.
66. Campbell DA, Lake MF, Falk M, Backstrand JR. A randomized control trial of continuous support in labor by a lay doula. *J Obstet Gynecol Neonatal Nurs*. 2006;35(4):456-464.
67. Doulas of North America. Available at www.dona.org.
68. Ehsanipoor RM, Saccone G, Seligman NS, et al. Intravenous fluid rate for reduction of cesarean delivery rate in nulliparous women: a systematic review and meta-analysis. *Acta Obstet Gynecol Scand*. 2017;96(7):804-811.
69. Coco A, Derksen-Schrock A, Coco K, et al. A randomized trial of increased intravenous hydration in labor when oral fluid is unrestricted. *Fam Med*. 2010;42(1):52-56.
70. Osterman MJK, Martin JA. Recent declines in induction of labor by gestational age. *NCHS Data Brief*. 2014;(155):1-8.
71. Jozwiak M, Bloemenkamp KWM, Kelly AJ, et al. Mechanical methods for induction of labour. *Cochrane Database Syst Rev*. 2012;3(3): CD001233.
72. Hofmeyr GJG, Gülmezoglu AM, Pileggi C. Vaginal misoprostol for cervical ripening and induction of labour. *Cochrane Database Syst Rev*. 2010;(10):CD000941.
73. Thomas J, Fairclough A, Kavanagh J, Kelly AJ. Vaginal prostaglandin (PGE2 and PGF2a) for induction of labour at term. *Cochrane Database Syst Rev*. 2014;6(6):CD003101.
74. Alfirevic Z, Aflaifel N, Weeks A. Oral misoprostol for induction of labour. *Cochrane Database Syst Rev*. 2014;6(6):CD001338.
75. Saccone G, Ciardulli A, Baxter JK, et al. Discontinuing oxytocin infusion in the active phase of labor: A systematic review and meta-analysis. *Obstet Gynecol*. 2017;130(5):1090-1096.

76. Boie S, Glavind J, Velu AV, et al. Discontinuation of intravenous oxytocin in the active phase of induced labour. *Cochrane Database Syst Rev*. 2018;8:CD012274.
77. Nicholson JM, Kellar LC, Henning GF, et al. The association between the regular use of preventive labour induction and improved term birth outcomes: findings of a systematic review and meta-analysis. *BJOG*. 2015;122(6):773-784.
78. Gibson KS, Waters TP, Bailit JL. Maternal and neonatal outcomes in electively induced low-risk term pregnancies. *Am J Obstet Gynecol*. 2014;211(3):249.e1-249.e16.
79. Walker KF, Bugg GJ, Macpherson M, et al; 35/39 Trial Group. Randomized Trial of Labor Induction in Women 35 Years of Age or Older. *N Engl J Med*. 2016;374(9):813-822.
80. Grobman WA, Caughey AB. Elective induction of labor at 39 weeks compared with expectant management: a meta-analysis of cohort studies. *Am J Obstet Gynecol*. 2019;221(4):304-310.
81. Grobman WA, Rice MM, Reddy UM, et al; Eunice Kennedy Shriver National Institute of Child Health and Human Development Maternal–Fetal Medicine Units Network. Labor Induction versus Expectant Management in Low-Risk Nulliparous Women. *N Engl J Med*. 2018;379(6):513-523.
82. American College of Obstetricians and Gynecologists. Practice Advisory: clinical guidance for integration of the findings of The ARRIVE Trial: labor induction versus expectant management in low-risk nulliparous women. Available at https://www.acog.org/Clinical-Guidance-and-Publications/Practice-Advisories/Practice-Advisory-Clinical-guidance-for-integration-of-the-findings-of-The-ARRIVE-Trial?IsMobileSet=false.
83. Declercq ER, Cheng ER, Sakala C. Does maternity care decision-making conform to shared decision-making standards for repeat cesarean and labor induction after suspected macrosomia? *Birth*. 2018;45(3):236-244.
84. Molenaar J, Korstjens I, Hendrix M, et al. Needs of parents and professionals to improve shared decision-making in interprofessional maternity care practice: A qualitative study. *Birth*. 2018;45(3):245-254.
85. Attanasio LB, Kozhimannil KB, Kjerulff KH. Factors influencing women's perceptions of shared decision making during labor and delivery: Results from a large-scale cohort study of first childbirth. *Patient Educ Couns*. 2018;101(6):1130-1136.
86. Anim-Somuah M, Smyth RMD, Jones L. Epidural versus non-epidural or no analgesia in labour. *Cochrane Database Syst Rev*. 2011;(12):CD000331.
87. Cheng YW, Shaffer BL, Nicholson JM, Caughey AB. Second stage of labor and epidural use: a larger effect than previously suggested. *Obstet Gynecol*. 2014;123(3):527-535.
88. Lieberman E, Davidson K, Lee-Parritz A, Shearer E. Changes in fetal position during labor and their association with epidural analgesia. *Obstet Gynecol*. 2005;105(5 Pt 1):974-982.
89. Klein MC. Does epidural analgesia increase rate of cesarean section? *Can Fam Physician*. 2006;52(4):419-421, 426-428.
90. Wong CA, Scavone BM, Peaceman AM, et al. The risk of cesarean delivery with neuraxial analgesia given early versus late in labor. *N Engl J Med*. 2005;352(7):655-665.
91. Committee on Practice Bulletins-Obstetrics. Practice Bulletin no. 177: obstetric analgesia and anesthesia. *Obstet Gynecol*. 2017;129(4):e89.
92. Cluett ER, Burns E. Immersion in water in labour and birth. *Cochrane Database Syst Rev*. 2009;(2):CD000111.
93. Rooks JP. Safety and risks of nitrous oxide labor analgesia: a review. *J Midwifery Womens Health*. 2011;56(6):557-565.
94. Collins MR, Starr SA, Bishop JT, Baysinger CL. Nitrous oxide for labor analgesia: expanding analgesic options for women in the United States. *Rev Obstet Gynecol*. 2012;5(3-4):e126-e131.
95. Hutton EK, Kasperink M, Rutten M, et al. Sterile water injection for labour pain: a systematic review and meta-analysis of randomised controlled trials. *BJOG*. 2009;116(9):1158-1166.
96. Derry S, Straube S, Moore RA, et al. Intracutaneous or subcutaneous sterile water injection compared with blinded controls for pain management in labour. *Cochrane Database Syst Rev*. 2012;1(1):CD009107.
97. Mark SP, Croughan-Minihane MS, Kilpatrick SJ. Chorioamnionitis and uterine function. *Obstet Gynecol*. 2000;95(6 Pt 1):909-912.
98. Seaward PG, Hannah ME, Myhr TL, et al. International Multicentre Term Prelabor Rupture of Membranes Study: evaluation of predictors of clinical chorioamnionitis and postpartum fever in patients with prelabor rupture of membranes at term. *Am J Obstet Gynecol*. 1997;177(5):1024-1029.
99. Cahill AG, Duffy CR, Odibo AO, et al. Number of cervical examinations and risk of intrapartum maternal fever. *Obstet Gynecol*. 2012;119(6):1096-1101.
100. Lawrence A, Lewis L, Hofmeyr GJ, Styles C. Maternal positions and mobility during first stage labour. *Cochrane Database Syst Rev*. 2013;(10):CD003934.
101. Leeman L, Leeman R. A Native American community with a 7% cesarean delivery rate: does case mix, ethnicity, or labor management explain the low rate? *Ann Fam Med*. 2003;1(1):36-43.
102. Deline J, Varnes-Epstein L, Dresang LT, et al. Low primary cesarean rate and high VBAC rate with good outcomes in an Amish birthing center. *Ann Fam Med*. 2012;10(6):530-537.
103. Khunpradit S, Tavender E, Lumbiganon P, et al. Non-clinical interventions for reducing unnecessary caesarean section. *Cochrane Database Syst Rev*. 2011;6(6):CD005528.
104. Skeith AE, Valent AM, Marshall NE, et al. Association of a health care provider review meeting with cesarean delivery rates: a quality improvement program. *Obstet Gynecol*. 2018;132(3):637-642.
105. Abenhaim HA, Benjamin A, Koby RD, et al. Comparison of obstetric outcomes between on-call and patients' own obstetricians. *CMAJ*. 2007;177(4):352-356.
106. Bell AD, Joy S, Gullo S, et al. Implementing a systematic approach to reduce cesarean birth rates in nulliparous women. *Obstet Gynecol*. 2017;130(5):1082-1089.
107. Thuillier C, Roy S, Peyronnet V, et al. Impact of recommended changes in labor management for prevention of the primary cesarean delivery. *Am J Obstet Gynecol*. 2018;218(3):341.e1-341.e9.
108. Feldman DS, Bollman DL, Fridman M, et al. Do laborists improve delivery outcomes for laboring women in California community hospitals? *Am J Obstet Gynecol*. 2015;213(4):587.e1-587.e13.
109. Iriye BK, Huang WH, Condon J, et al. Implementation of a laborist program and evaluation of the effect upon cesarean delivery. *Am J Obstet Gynecol*. 2013;209(3):251.e1-251.e6.
110. Nijagal MA, Kuppermann M, Nakagawa S, Cheng Y. Two practice models in one labor and delivery unit: association with cesarean delivery rates. *Am J Obstet Gynecol*. 2015;212(4):491.e1-491.e8.

Malpresentations, Malpositions, and Multiple Gestation

Learning Objectives

1. List complications associated with various malpresentations.
2. Discuss delivery management of multiple gestation.
3. List the steps to safely perform a breech delivery utilizing the CAREFUL mnemonic.

Definitions

Definitions are important to a discussion of malpresentations. *Lie* is the relationship of the long axis of the fetus to that of the woman, specified as longitudinal, transverse, or oblique (also called *unstable*). *Presentation* is the portion of the fetus that is foremost, or presenting, in the birth canal. The fetus may present by vertex, breech, face, brow, or shoulder. *Position* is a reference point on the presenting part and how it relates to the woman's pelvis. For example, the reference point on the vertex is the occiput. When the fetal occiput is directed toward the woman's symphysis, or anteriorly, the fetus is in *occiput anterior* (OA) position. When the occiput is directed toward the maternal spine, the fetus is in *occiput posterior* (OP) position. Intermediate positions around the compass are *left occiput anterior (LOA)* and *right occiput anterior* (ROA), *left occiput transverse* (LOT) and *right occiput transverse (*ROT), and *left occiput posterior* (LOP) and *right occiput posterior* (ROP). In breech presentation, the reference point is the sacrum and the desired position during delivery is sacrum anterior.

Methods of Diagnosis

There are three principal methods of determining fetal lie, presentation, and position: (1) Leopold maneuvers, or abdominal palpation, (2) vaginal examination, and (3) imaging with ultrasound. Ultrasound examination is performed in the labor department and is commonly used in hospitals of all sizes. All maternity care clinicians should have ultrasound skills to determine fetal lie, presentation, and position. Handheld ultrasound devices are available.

Vaginal Examination

In *vertex* presentation, the scalp (sometimes with hair) can be felt, and the sagittal suture, the Y-shaped posterior fontanel, and the larger diamond-shaped anterior fontanel can be palpated. Following the sagittal suture to determine the location of the posterior fontanel allows determination of OA and OP position.

In *breech* presentation, the buttocks (smooth skin, no hair) can be felt, and an orifice (meconium may be present on the glove if a finger is introduced into the orifice), and/or the ischial tuberosities (in a line with the anus) also may be felt.

In *face* presentation, the face can be felt (smooth skin, no hair). An orifice may be felt (introducing a finger into the mouth may elicit a sucking response from the fetus), and/or the malar prominences, which form a triangle with the mouth. The fetus in this presentation may deliver vaginally if the mentum (chin) is anterior, allowing flexion of the head around the symphysis pubis. The fetus with the mentum located posteriorly cannot be delivered vaginally unless spontaneous rotation to the anterior occurs, because the fetal head will not flex but rather must extend. A cesarean delivery will be required.

In *brow* presentation, the anterior fontanel, orbital ridges, eyes, and the base of the nose can be felt. This presentation is unstable and will typically convert to a face or vertex presentation. This can be a difficult presentation to detect by examination.

In a *transverse* lie, the pelvis will be empty on vaginal examination, and the diagnosis is typically made easily by palpation or ultrasound examination.

In a *cord prolapse or other fetal part* malpresentation (eg, arm), the diagnosis is typically made by visual inspection or palpation on immediate vaginal examination.

The incidence of malpresentations and malpositions at term are listed in *Table 1*.

The Fetal Head and the Maternal Pelvis

Most fetal malpresentations (ie, OP, breech, transverse, face, brow) are clinically significant because the fetal head is not round, but rather ovoid (ie, egg-shaped). The smallest of the fetal diameters is the suboccipitobregmatic; the largest is the occipitomental (*Figure 1*). The difference between them is 3 cm on average, or approximately 24%.[1] When the head is in full flexion, the suboccipitobregmatic diameter presents to the pelvis. When the head is in full extension (or deflexion), the occipitomental diameter presents. Delivery is more likely to occur and be easier if a smaller diameter presents. Therefore, the attitude of the fetal head (flexion versus extension) as it presents to the pelvis is of paramount importance. A degree of fetal extension of the head occurs with OP, face, and brow presentations, and some breech presentations.[2]

Asynclitism also affects the mechanics of labor. Asynclitism is lateral flexion of the head that causes the sagittal suture not to be in the middle of the birth canal. Some degree of asynclitism is normal, and the fetal head may even shift back and forth from anterior to posterior asynclitism as it moves further into the pelvis. Extreme degrees of asynclitism may prevent labor from progressing. Asynclitism becomes a factor in achieving correct application of instruments for assisted vaginal delivery (eg, forceps, vacuum devices).

The maternal pelvis type also affects the cause of various malpresentations and prognosis for delivery. There are four pure types of pelvises (*Figure 2*):

- Gynecoid (round)
- Anthropoid (oval with the long axis in the anteroposterior [AP] plane)
- Platypelloid (oval with the long axis in the transverse plane)
- Android (triangular or heart-shaped with the apex of the triangle anteriorly)

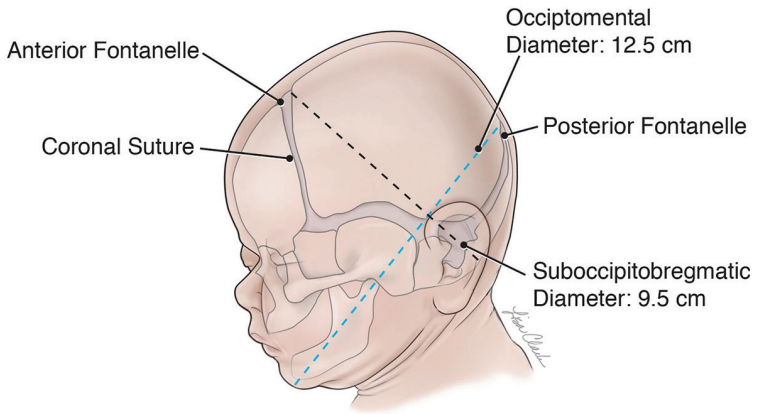
Most women have a gynecoid pelvis. A narrow pelvis, such as the anthropoid, can cause persistent OP position; the platypelloid pelvis can cause a transverse arrest; the android pelvis is prejudicial to delivery with all malpresentations; and an inadequate or small pelvis can be associated with most of the malpresentations secondary to the inability of the head to descend, engage, or rotate.[3]

Table 1. Incidence of Malpresentations and Malpositions at Term

Malpresentation	Incidence	Percentage
Occiput posterior (persistent)	1 in 8 to 20	5 to 12
Breech	1 in 25 to 33	3 to 4
Transverse lie or shoulder	1 in 300 to 400	0.33 to 0.25
Face	1 in 500 to 600	0.2 to 0.3
Brow	1 in 1,400	0.07

Information from Barth WH Jr. Persistent occiput posterior. Obstet Gynecol. *2015; 125(3):695-709; Ratcliffe SD, Baxley EG, Cline MK, Sakornbut EL.* Family Medicine Obstetrics. *3rd ed. Philadephia, PA: Mosby Elsevier; 2008; Vlemmix F, Bergenhenegouwen L, Schaaf JM, et al. Term breech deliveries in the Netherlands: did the increased cesarean rate affect neonatal outcome? A population-based cohort study.* Acta Obstet Gynecol Scand. *2014;93(9):888-896; Royal College of Obstetricians and Gynaecologists. The Management of Breech Presentation: Guideline No. 20b. 2006.*

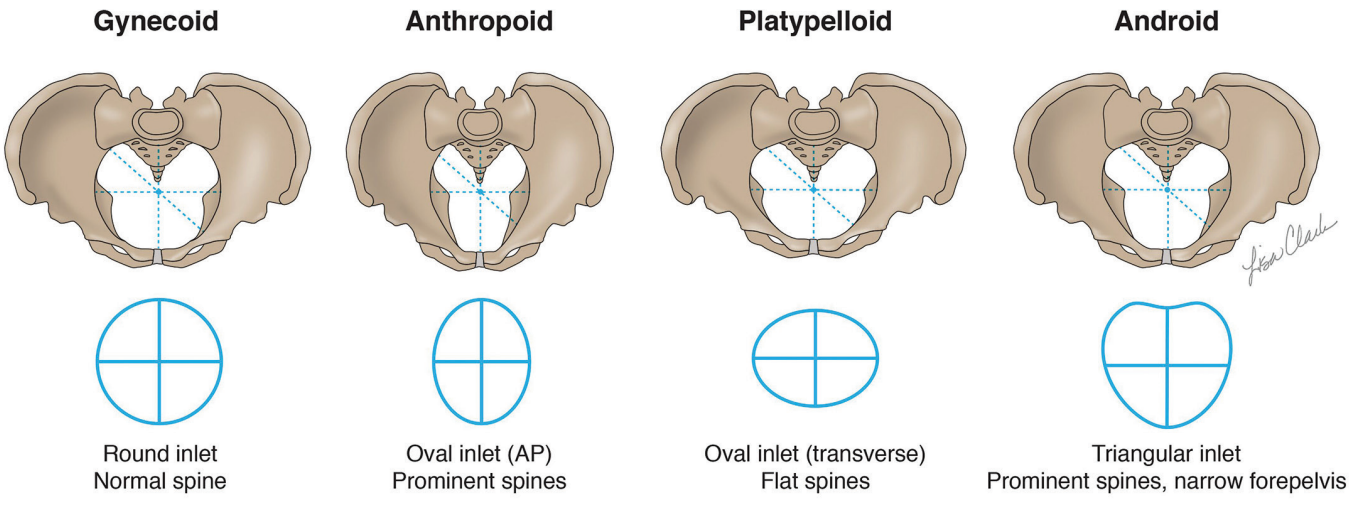
Figure 1. Fetal Head Diameters



Occiput Posterior Position

In OP position, the fetus lies with the occiput toward the woman's spine and the face toward the woman's symphysis and abdomen. The fetus is face up when the woman is supine or in lithotomy position. Typically, a fetus in OP position will rotate spontaneously to OA position and deliver spontaneously. Spontaneous rotation fails to occur in 5% to 12% of cases, and the fetus remains in persistent OP position.[4] The exact cause of persistent OP positioning is unknown, but transverse narrowing of the pelvis has an effect. All fetuses in OP position are somewhat deflexed because the vertex drops back to fill the hollow of the sacrum. The combination of deflexion and posterior presentation causes less favorable diameters of the fetal head to present to the pelvis than when the fetus is in OA position.[4]

Figure 2. Pelvic Types



The diagnosis of OP position is based on observation and examination of the patient. Back pain, or *back labor,* is a clinical hallmark of OP position. Easy palpation of the anterior fontanel on vaginal examination is a diagnostic aid in determining OP position because the anterior fontanel is most easily felt when the head is partially deflexed. If the anterior fontanel is palpated, the sagittal suture must be identified. This can be accomplished by following each suture with the examining finger until the posterior fontanel is felt. Occasionally, an ear can be palpated, revealing the fetal position. The examination findings can be confusing because of molding, overriding of sutures, edema, and asynclitism. Dilation is often asymmetric, and a persistent anterior lip is common. Ultrasound imaging can be helpful but occasionally confusing.[5] Abdominal, transvaginal, and transperineal ultrasound can be used depending on the station of the fetal head and sonographer experience. In addition to determining OP versus OA position, ultrasound can identify a deflexed head by noting the distance from the chin to the chest. Unfortunately, a deflexed OP position with deep engagement presents difficulty with manual rotation, vaginal delivery, and even cesarean delivery where a reverse breech extraction may be the safest delivery technique.[6]

Diagnosing OP position can be difficult. Maternity care clinicians often make a last-minute diagnosis when the fetal head seems to fill the posterior pelvis as it delivers, or even later as the fetal face becomes visible under the symphysis. Even skilled clinicians occasionally rotate fetuses the wrong direction, from OA to OP position.

Labor and delivery with a fetus in persistent OP position is not markedly different from the delivery of a fetus in OA position. The progress of labor can be monitored by cervical dilation and the descent of the vertex through the birth canal. Labor with a fetus in OP position is often prolonged, and there is an increased incidence of assisted vaginal delivery, cesarean delivery, and anal sphincter lacerations.[1] Perinatal mortality in OP position does not differ significantly from OA position, and there is no significant difference in Apgar scores except in fetuses requiring assisted vaginal delivery.[7] Bruising of the fetal face can occur. Perineal lacerations and extensions of episiotomies may be increased because the vertex sweeps through the posterior pelvis, larger diameters are presented to the pelvic outlet, and the occiput places maximal pressure on the perineum as it delivers. The five possibilities for vaginal delivery when persistent OP position occurs are spontaneous delivery, manual rotation, vacuum delivery, forceps delivery, and forceps rotation.[4]

Spontaneous Delivery

Spontaneous delivery using expectant management occurred in 45% of deliveries in one study.[8] Because the fetal head cannot stem upward until the face has cleared the symphysis, the fetal vertex must pass through the posterior pelvis, where it

places strain on the perineum. These fetuses look like they *want* to deliver through the rectum. However, the delivery is frequently easy.

Manual Rotation

Attempted turning of fetuses in OP position has been undertaken by placing the laboring woman in various positions, such as on the side, squatting, ambulating, on hands and knees, or with the back arched (to make the fetus so uncomfortable it turns itself). A randomized controlled trial (RCT) comparing modified Sims maternal position with freely adopted maternal positions in women with fetuses in OP position showed a higher proportion of fetuses rotating to OA position (50.8% versus 21.7%; $P = .001$) and increased rates of vaginal delivery (84.7% versus 68.3%, $P = .035$) with the modified Sims position.[9] If repositioning the woman fails to rotate the fetus, manual rotation becomes an alternative intervention during a long second stage of labor because it can be attempted during any vaginal examination. If successful, delivery may be greatly expedited; if unsuccessful, no harm is done.

The key to manual rotation is to enhance the natural and normal forces of rotation. Rotation normally occurs when the flexed fetal head strikes the muscles of the pelvic floor, called the levator sling. Therefore, the clinician must first flex the head. This is accomplished by placing a hand in the posterior pelvis behind the occiput. The clinician's hand replicates and enhances the levator sling effect, acting as a wedge to flex the head and apply rotatory force. Some clinicians also grasp the head with the thumb. The rotation should be attempted at the same time as a contraction when the woman is pushing to force the head down on the levator sling (and the hand), using the natural mechanism for flexion and rotation. An assistant may massage the fetal shoulder in the direction of the rotation with suprapubic or abdominal pressure. Manual rotation can be attempted with the patient in the lithotomy, the lateral Sims, or the hands-and-knees positions. In the hands-and-knees position, the abdominal assist is impractical.

If the fetus is in straight OP position, the dominant hand will be used to rotate the fetus, but rotation should go *the shortest distance* if the fetus is in ROP or LOP position. Therefore, the fetus in ROP position should be rotated clockwise, and the fetus in LOP position should be rotated counterclockwise. The hand that pronates during the rotation (like closing a book) should be used: left hand for ROP position and right hand for LOP position.[10]

Manual rotation is part of the art of maternity care. It is a neglected skill, but one with minimal risk that requires no technology or instrumentation. Successful manual rotation may shorten the second stage of labor and decrease the likelihood of an assisted vaginal or cesarean delivery. Additional information regarding manual rotation is in the *Labor Dystocia* chapter.

Vacuum Delivery

Vacuum delivery is an option for fetuses in persistent OP position when the vertex is not appropriately descending. However, OP position is not itself an indication for assisted vaginal delivery. The vacuum cup should be applied to the flexion point anterior to the posterior fontanel to facilitate flexion, which will increase the likelihood of a successful delivery. The clinician may be uncertain of the exact position of the head because of molding, edema, and overriding of sutures. Ultrasound should be considered to determine or confirm position because it is more accurate than digital examination.[11] The vacuum may successfully draw the head out in OP position. Alternatively, in flexing the head and drawing it down against the levator sling, the vacuum may promote rotation. Delivery will then occur in the OA position. The vacuum allows the fetal head to find its own optimal plane for delivery. It is not uncommon for the head to rotate 180 degrees as traction is applied, sometimes in the moment before delivery. However, no direct rotary force should be applied to the cup because this may cause a *cookie cutter* type injury to the scalp and cause the cup to disengage.

The vacuum cup typically needs to be placed as posteriorly on the head as possible to reach the flexion spot for the fetus in OP position. The mushroom bell cup is best suited for vacuum-assisted delivery of fetuses in OP position (see the *Assisted Vaginal Delivery* chapter). The mechanism of delivery for a fetus in OP position is the same with a vacuum as with forceps or spontaneous delivery: the fetal vertex takes a more posterior course through the pelvis. As with any vacuum delivery, the shaft of the extractor must be kept at right angles to the plane of the cup, or detachment will occur. OP position increases the incidence of

third- and fourth-degree lacerations because the forces are directed toward the rectum.[12]

Forceps Delivery

With OP presentation, the usual indications for forceps delivery apply (see the *Assisted Vaginal Delivery* chapter). Forceps fit the OP vertex equally as well as the OA vertex. An OP presentation is not itself a sufficient indication for forceps use. The mechanism of delivery is the same as for a spontaneous OP delivery. The head is delivered by flexion, not extension. The face must pass beneath the symphysis before the head can flex upward, so traction on the forceps must be in a more posterior direction for longer than in OA deliveries. Pressure on the perineum can be intense with resulting third- and fourth-degree lacerations, especially after episiotomy.[13]

Occasionally, with an OP delivery and a prolonged second stage of labor, severe molding and edema will occur. The fetal vertex will initially appear to be at +2 station or even on the perineum, but careful examination will reveal that the fetal head is elongated and the biparietal diameter is not engaged. Under such circumstances, attempts at assisted delivery are not likely to be successful and may be harmful. Cesarean delivery is indicated, and the lack of engagement may be confirmed by the ease with which the fetus is lifted out of the pelvis.

Forceps Rotation

Only skilled clinicians trained in the Scanzoni maneuver of rotation with Kielland forceps should consider using forceps for rotating a fetus in OP position. The application of forceps rotation is not included in the ALSO curriculum. In most US hospitals, these techniques are seldom practiced. However, a recent series of studies has shown a high likelihood of successful rotation with minimal morbidity,[14,15] leading to advocacy of expanded training and use of rotational forceps.[4] Cesarean delivery should always be the backup method of delivery for any OP presentation that cannot be safely delivered vaginally.

Breech Presentation

Breech presentation is defined as the fetal breech, or buttocks, presenting in the birth canal with the head aftercoming in the uterine fundus. Breech presentations may be classified as:

- *Frank breech:* the hips are flexed, and the legs are extended over the anterior surface of the body
- *Complete breech*: the hips and legs are flexed (tailor sitting or squatting)
- *Footling breech:* One or both hips are extended, with one or both feet presenting. One or both feet may commonly be palpable on vaginal examination of a complete breech presentation, but the presentation is not considered footling if the knees are flexed and the buttocks are lower than the feet.

Breech presentation has many predisposing factors. Prematurity is commonly associated with breech presentation. As the fetus approaches term, the incidence of breech presentation decreases to 3% to 4%.[16]

Other predisposing factors for breech presentation include high parity and relaxation of the uterine and abdominal walls; uterine anomalies (including fibroid tumors); pelvic tumors; polyhydramnios; oligohydramnios; various fetal anomalies including hydrocephalus, anencephaly, and Down syndrome; macrosomia; multiple pregnancy; placenta previa; absolute cephalopelvic disproportion; and previous breech delivery. An ultrasound with a fetal anatomical survey performed by a qualified clinician is indicated when the diagnosis of breech presentation is made in the mid-third trimester or later, but the cause is typically not found.[16]

Diagnosis

The diagnosis of breech presentation can often be made by abdominal palpation and vaginal examination. On Leopold maneuvers, the firm, ballotable, rounded head is felt in the fundus. However, it is common for a breech presentation to be misdiagnosed on Leopold maneuvers during prenatal visits with the diagnosis not made until the patient presents in labor or with rupture of membranes at term. A study of antenatal detection of breech presentation in a large maternity care department in the United Kingdom showed that 27.9% of breech presentations were not detected during prenatal care and that the percentage increased from 23.2% to 32.5% from 1999 to 2009.[17] A retrospective study of 251 women with fetuses in breech presentation from 2012 to 2015 also showed that 32% of fetuses in breech presentation were not identified until 38 weeks' gestation or later and that the cohort diagnosed prior to 38 weeks' gestation was more likely to have a vaginal delivery (31.1% versus 12.5%; $P<.01$).[18]

The Pregnancy Outcome Prediction (POP) study of 3,879 nulliparous women showed that routine ultrasound at 36 weeks' gestation virtually eliminated unanticipated breech presentation at term and that 40 scans were needed to identify each unanticipated breech presentation.[19]

When the examining clinician is unsure about the presentation at 35 weeks' or greater gestational age, it is recommended that a vaginal examination or limited ultrasound be performed. The fetal head may be low in the pelvis and difficult to palpate on Leopold maneuvers, yet sutures are palpable on vaginal examination.

On vaginal examination of a breech presentation, small parts or the breech may be detected. If small parts are palpated, it is essential to distinguish between a hand and a foot. The breech itself is smooth and rounded and may feel remarkably like a vertex. Most physicians and midwives who provide maternity care have misdiagnosed a breech on vaginal examination. The key is to seek fontanels and sutures with the examining finger, which always signifies a vertex.

Breech presentation can be confused with face presentation. In breech presentation, the anus and ischial tuberosities form a straight line, whereas the mouth and malar prominences form a triangle. In addition, the skin of the fetal buttock is smooth. An alert examiner can distinguish it from the hairy feel of the scalp. This subtle sign may raise an examiner's index of suspicion to perform a more definitive examination. If the examiner's finger encounters an orifice when membranes are ruptured, the finger can be gently inserted into the orifice. If it is the mouth (signifying a face presentation), the fetus will suck on the finger. If it is the anus (signifying a breech), the finger will be coated with meconium when withdrawn.

Figure 3. Breech Postural Exercise

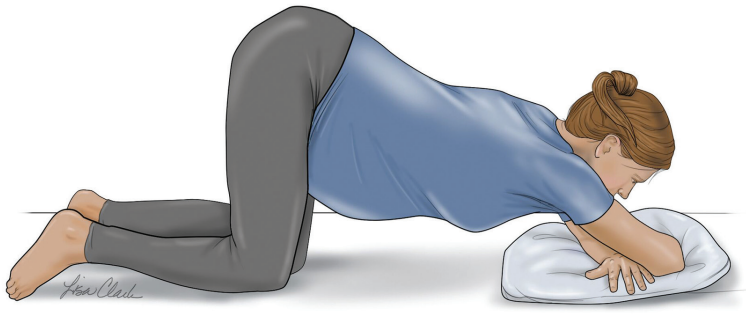
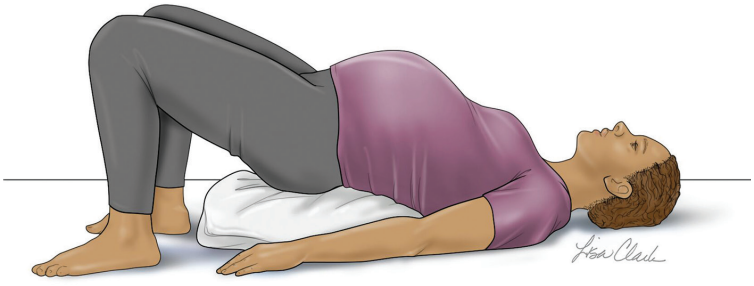


Figure 3a. Deep Trendelenberg Position



Prenatal Management of Breech Presentation

There are four elements to the prenatal management of breech presentation. First, a cause must be determined for the breech presentation, most of those that can be identified are detectable by ultrasound. Second, the woman may attempt certain exercises to turn the breech. Third, external cephalic version (ECV) should typically be offered and attempted. Fourth, failing a successful version, a decision must be reached regarding the most favorable mode of delivery.

Postural Management of Breech Presentation

Various exercises and positions have been tried to attempt to turn a fetus in breech presentation. No difference in outcome has been shown in a review of trials in which women were randomized to a postural management group or a control group.[20,21]

The exercises themselves are simple. One exercise method is for the woman to assume a knee-chest position for 15 minutes 3 times a day for 5 days after the diagnosis of the breech presentation. Another version is for the woman to assume a deep Trendelenburg position by elevating the hips 9 to 12 inches while lying supine with a pillow(s) under the hips for 10 minutes once or twice a day (*Figure 3*). Pelvic rocking while in these positions is often recommended.[20]

Although effectiveness cannot be proven, these exercises are not harmful, and they provide a focus of activity for the woman, who may be anxious regarding the fetus being in a breech position. There are no contraindications to these exercises.[20]

External Cephalic Version

External cephalic version, turning a fetus in breech presentation to vertex by manipulation through the woman's abdominal wall and uterus, has

become an accepted component of the prenatal management of breech presentation. This process is widely supported, including by national guidelines from the American College of Obstetricians and Gynecologists (ACOG) and the Royal College of Obstetricians and Gynaecologists (RCOG), and a 2015 Cochrane systematic review.[22-24] This procedure is low-tech, low-cost, and can decrease cesarean delivery rates, which prevents potential operative morbidity. The risk of an adverse event occurring because of ECV is low, and the cesarean delivery rate is significantly lower among women who have undergone a successful ECV. Women near term with fetuses in breech presentation and no contraindication to vaginal delivery should be offered an ECV attempt.[23]

The success rate of ECV was 53% in a 2008 meta-analysis of 53 articles.[25] In a comprehensive program of ECV, cesarean delivery for breech presentation can be reduced by approximately one half. A 2015 Cochrane review of eight RCTs showed a 43% decrease in cesarean deliveries without a significant increase in maternal or fetal complications (95% CI = 40-82).[23] The primary factors associated with a successful ECV are parity, gestational age and the amount of amniotic fluid, frank breech presentation, and a relaxed uterus. The clinician's skill and the woman's tolerance of the procedure affect the success. In one study, the introduction of a dedicated team of physicians and midwives for ECV increased the success rate of ECV from 39.8% to 69.5% ($P<0.001$) as well as the vaginal delivery rate (43% to 71%). The success rate in nulliparous women increased from 23.5% to 58.5% ($P=0.002$).[26]

Gestational age is also a factor in the success rate of ECV. ECV is not recommended before approximately 37 weeks' gestation, unless a patient with a fetus in breech presentation presents in preterm labor or for a medically indicated induction. A study of ECV at 34 to 35 weeks' gestation compared with 37 weeks' gestation showed a higher proportion of cephalic presentations at term, but the overall cesarean delivery rate was not decreased.[27,28] Performing ECV at 34 to 35 weeks' gestation presents the risk of delivering a premature infant if urgent cesarean delivery is indicated. After 37 weeks' gestation, the likelihood of successful ECV decreases as the breech presentation may become engaged in the pelvis. Deferring ECV beyond 37 weeks' gestation also incurs an increased risk of labor or rupture of membranes occurring while the fetus is in breech presentation. ECV may be attempted in early labor when membranes are intact.[22]

Although many contraindications to ECV are commonly listed in clinical guidelines or recommendations, there is limited evidence regarding many of these contraindications.[25] If vaginal delivery is contraindicated (eg, placenta previa, prior classical cesarean delivery), ECV should not be attempted. A systematic review of other potential contraindications to ECV evaluated five guidelines and showed 18 different contraindications with a range of five to 13 per guideline.[25] The review also analyzed 60 articles that described a total of 39 different contraindications, but evidence for the contraindications could only be assessed for six contraindications. The authors of the review concluded that there was only reasonable evidence to support three contraindications: history of placental abruption or current abruption, preeclampsia with severe features (or HELLP syndrome) and concerning fetal monitoring results, including abnormal Doppler ultrasound results.[25] All the guidelines included oligohydramnios, and four of the five included intrauterine growth restriction (IUGR), but the systematic review did not show evidence for these common recommendations.[25]

A commentary on the review recommended that in some of the clinical scenarios commonly described as contraindications, an ECV may be successfully and safely performed in the operating room with regional anesthesia. The author questioned whether severe preeclampsia should be considered a contraindication.[25] A prior cesarean delivery in a woman who is a candidate for labor after cesarean (LAC) is not a contraindication for ECV based on small studies and a 2017 ACOG guideline,[29-32] though data is not available regarding the rates of uterine rupture with a trial of labor (TOL) after ECV.

Various strategies have been used to increase the success of ECV. Routine tocolysis appears to reduce the failure rate of ECV at term.[33] Although promising, there is insufficient evidence to evaluate the use of fetal acoustic stimulation,[33] hypnosis, or moxibustion.[33,34] Regional anesthesia in combination with a tocolytic drug has been shown to be effective, especially in primiparous women, with no increased rate of complications.[33,35-37] A 2011 meta-analysis of six RCTs found that regional

anesthesia increased the success rate of ECV from 37.6% to 59.7% (odds ratio 1.58; 95% CI = 1.29-1.93; NNT =5). The use of regional anesthesia increases costs and requires the patient to spend a prolonged time in the labor and delivery department while waiting for the regional anesthesia to wear off; however, two studies have shown a beneficial cost analysis.[38,39] A small case series using gloves with built-in pressure sensors to measure the amount of pressure used for ECV with and without regional anesthesia showed that less pressure was applied when the patient received regional anesthesia, presumably because the abdominal skeletal muscles provided less resistance.[40]

Complications of ECV occur in less than 1% of attempts.[22] After ECV, rupture of membranes and labor has been noted. There have also been some reports of placental abruption, fetal hemorrhage, maternal hemorrhage, a knotted or entangled cord, and fetal mortality.[22,41]

Fetal bradycardia and decelerations are common, but they typically resolve spontaneously or with cessation of the procedure. A retrospective study in Japan of 390 patients who underwent ECV showed that 48.5% had a period of fetal bradycardia during or after the procedure. Of the bradycardic episodes, 43.4% lasted less than 1 minute, 88.9% lasted less than 5 minutes, and 98.4% lasted less than 10 minutes.[42] The patients who had fetuses with bradycardic episodes had good fetal outcomes; however, two of the three patients with episodes lasting greater than 10 minutes had Apgar scores less than 5 at 5 minutes and arterial cord blood pH levels less than 7.1. This suggests the importance of not proceeding to emergency cesarean delivery for brief periods of bradycardia as well as the importance of preparing for emergency cesarean delivery if the bradycardia does not resolve after approximately 10 minutes.[42] When performing ECV, facilities and personnel must be available for performing an immediate cesarean delivery.[17]

Procedure for External Cephalic Version

(This is a sample protocol similar to many published protocols. Other variations exist.)

Preparation

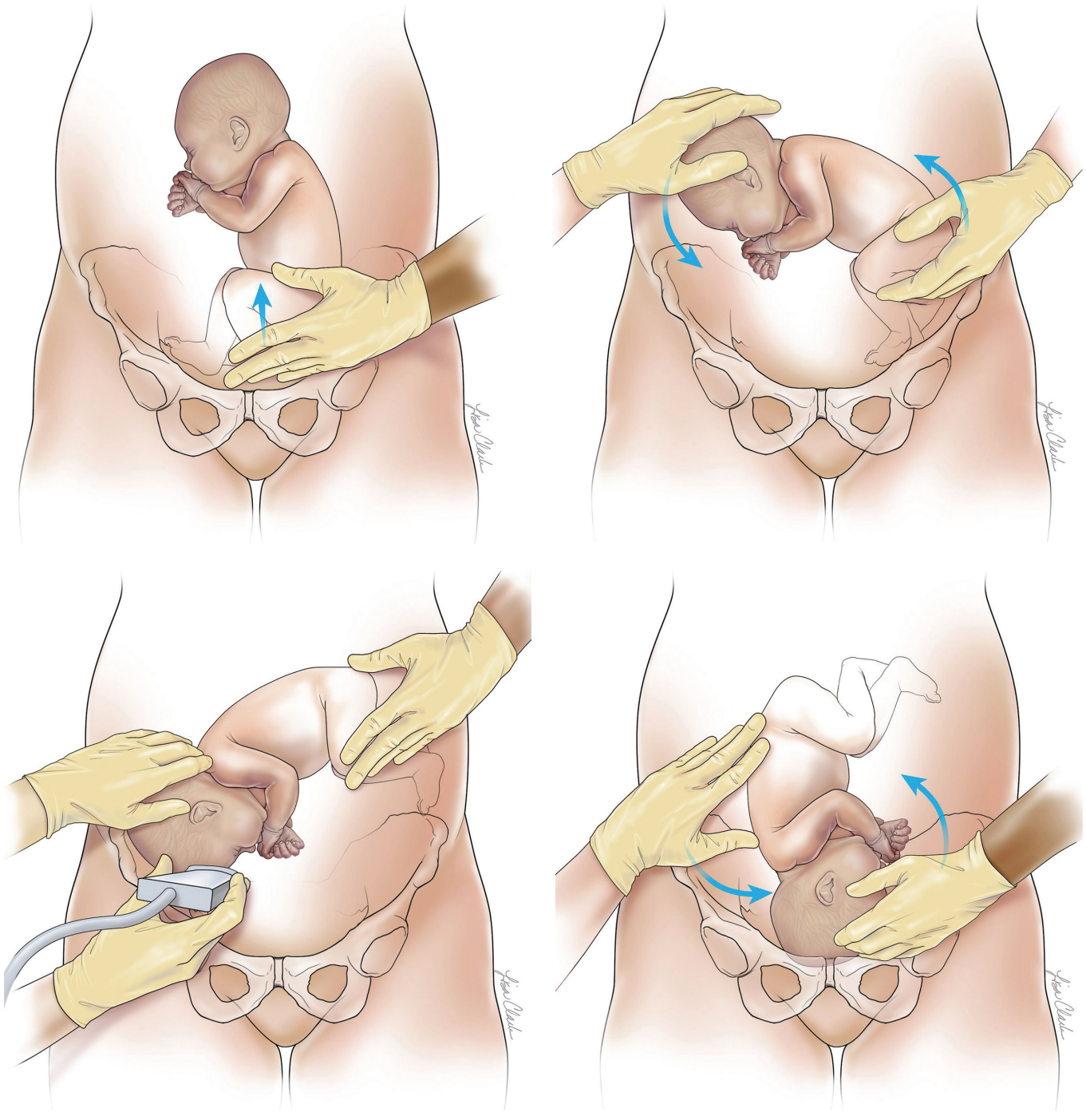
- Patient may be accompanied by a support individual
- Patient NPO for 6 to 8 hours prior to the procedure
- Patient gowned, and bladder emptied
- Confirm breech presentation by ultrasound and evaluate for fetal anomalies if not obtained via prior anatomic survey
- Perform nonstress test (NST)
- Obtain consent
- Cesarean delivery personnel and facilities available
- Intravenous (IV) access
- Tocolysis: administer 0.25 mg of terbutaline (subcutaneous) 15 minutes before starting ECV, or IV immediately before the procedure[43]
- Position: supine, slight left lateral tilt, Trendelenburg, knees slightly bent
- Abdomen coated with ultrasound gel (optional)

Procedure (for two clinicians)

- Clinician 1 elevates the fetus in breech presentation from pelvis by placing a hand suprapubically beneath the fetal buttocks (*Figure 4*)
- Clinician 1 pushes the fetus into the iliac fossa
- Clinician 2 flexes the head (for a forward roll) and rotates the fetus into an oblique lie
- Two-thirds of the force or pressure should be applied to the breech, and one-third of the force should be applied to the head. Avoid excessive force
- Both clinicians should rotate the fetus slowly around using just enough force to move the fetus. Progress will occur in stages, or *cogwheel* fashion. The fetus will rotate slightly, then resist, then rotate more. Allow the woman and fetus brief rest periods when resistance is felt, while attempting to maintain the progress already achieved
- Monitoring should be performed periodically during and after the attempt at ECV and may be performed via ultrasound, external fetal monitor, or a Doppler stethoscope
- When the fetus is just past the transverse, it may rotate the rest of the way without effort as it adjusts to the shape of the uterus
- The vertex may be guided gently over and into the pelvic inlet with suprapubic manipulation and fundal pressure
- Perform ultrasound to confirm status
- After successful version, monitor for a minimum of 20 to 40 minutes and until a reactive NST result is obtained[44]
- In patients who are Rh negative, administer Rh_o(D) immune globulin; may obtain Kleihauer-Betke test

- If the forward roll fails, try a backward flip, especially if the vertex and breech lie on the same side of the maternal midline
- If not successful after 15 to 20 minutes, discontinue the procedure[22]
- If the patient feels sharp pain or is unable to tolerate the procedure, discontinue until she is comfortable and then reassess whether to proceed or discontinue the procedure. Use of regional anesthesia can be considered
- If bradycardia occurs, discontinue the procedure. If it persists, revert the fetus to its original breech position. If bradycardia persists, prepare for cesarean delivery

Figure 4. Elevating Breech with Suprapubic Hand



- If using regional anesthesia, wait until the anesthesiologist is confident that blood pressure levels are stable because it can be difficult to distinguish fetal bradycardia from hypotension versus the ECV procedure itself

After a successful ECV, physicians and patients may consider elective induction of labor to prevent reversion to breech presentation while the woman is hospitalized, with IV access and possibly regional anesthesia. This is typically not recommended unless there is another obstetric indication, because the likelihood of reversion to breech presentation is approximately 5%.[44,45] If, however, a second ECV is required after a fetus has reverted to breech presentation, induction may be considered after 39 weeks' gestation. Induction is not indicated at an earlier gestational age.[46,47]

Choosing Delivery Route for Breech Presentation

The optimal delivery route for a fetus in breech presentation is controversial. In the United States, most fetuses in breech presentation are delivered by cesarean delivery. In 2003, 85% of fetuses in breech presentation were delivered via cesarean delivery, and the rate currently is greater than 95% in many areas.[48,49] The practice of routine cesarean delivery for breech presentation was adopted without high-level evidence supporting this intervention. Cesarean delivery does not prevent all infant morbidity, which in some instances may arise from the same problems that caused the breech presentation (eg, neuromuscular disease, oligohydramnios, polyhydramnios).[50]

A multicenter, international RCT (Term Breech Trial [TBT]) compared elective cesarean delivery with vaginal delivery for select breech presentations: greater than 37 weeks' gestation, frank or complete breech, and less than 4,000 g (approximately 8.8 lb) estimated fetal weight.[51] This trial was discontinued early in 2000 after analysis of short-term outcomes showed significant reductions in perinatal mortality and morbidity, and no increase in serious maternal complications in the elective cesarean delivery group.[51] The short-term outcomes from the TBT showed that the incidence of perinatal mortality, neonatal mortality, or serious neonatal morbidity was 1.6% in the planned cesarean delivery group compared with 5% in the planned vaginal delivery group (relative risk [RR] 0.33; 95% CI = 0.19-0.56; P<0.0001). National guidelines and commentaries after the TBT suggested that planned vaginal delivery of a breech fetus may no longer be an acceptable option except when a woman refuses the recommended cesarean delivery.[22,52,53]

After publication of the TBT, two additional studies were published that led to a reconsideration of the initial recommendation that planned vaginal delivery of a fetus in breech presentation may not be appropriate. The 2-year neonatal outcomes of the TBT were published in 2003 and showed no difference in neurodevelopmental outcomes in the 79.6% of the infants that were monitored for 2 years.[54] The 2-year outcomes were monitored only in settings that estimated a greater than 80% 2-year follow-up could be achieved. The long-term outcomes showed that the surrogate outcomes of morbidity in the short-term TBT outcomes, such as decreased neuromuscular tone at 2 hours, were poorly predictive of long-term developmental outcomes. In the subset monitored for 2 years, there was no difference in the combined perinatal mortality and abnormal neurologic outcome: 3.1% in the planned cesarean delivery group, and 2.8% in the TOL group. Seventeen of the 18 infants with serious neonatal morbidity were developmentally normal at 2 years.[54]

The Presentation et Mode d'Accouchement: presentation and mode of delivery (PREMODA) observational study took place at 174 centers in France and Belgium.[55] Strict protocols for patient selection for planned vaginal breech delivery and for labor management were used, and 8,105 women were monitored, representing 4 times the number monitored in the TBT. Thirty-one percent of women in the study planned for vaginal delivery. Of the 2,526 women with a TOL, 1,796 (71%) delivered vaginally for an overall vaginal birth rate of 22.5% in the entire cohort. There was no difference in fetal mortality (0.08% versus 0.15%; relative risk [RR] 0.64; 95% CI = 0.13-3.06) or combined fetal/neonatal mortality and serious neonatal morbidity (1.6% versus 1.4%; RR 1.10; 95% CI = 0.75-1.61) between the planned vaginal delivery and planned cesarean delivery groups. The PREMODA study differed from the TBT in requiring an obstetric ultrasound, having rapid access to emergency cesarean delivery, and converting to cesarean delivery sooner when labor was prolonged. An example is that a second stage of labor of up to 3.5 hours was permissible in the

TBT, but in the PREMODA study the TOL was typically converted to cesarean delivery when the active second stage of labor exceeded 1 hour. Only 0.2% of women in the PREMODA study had an active second stage of labor longer than 1 hour compared with 5% in the TBT.[51,55]

Current ACOG and RCOG guidelines state it is acceptable to offer vaginal breech delivery based on hospital-based protocols if an experienced physician is available and the patient chooses vaginal breech delivery after careful counseling about risks.[52,56]

The 2016 SOGC guidelines encourage retraining of obstetricians in vaginal breech delivery and recommend that women be offered options of vaginal breech delivery or cesarean delivery based on the individual clinical situation.[57] The SOGC guidelines on patient selection and labor management are adapted from the PREMODA study protocols. The SOGC guidelines estimated that perinatal mortality in appropriately selected patients is between 0.8 and 1.7 per 1,000 for planned vaginal breech delivery and between 0 and 0.8 per 1,000 for planned caesarean delivery.[57] RCOG describes perinatal mortality by comparing rates between planned vaginal breech delivery (2 per 1,000), planned vaginal cephalic delivery (1 per 1000), and planned cesarean delivery after 39 weeks' gestation (0.5 per 1,000).[52] RCOG guidelines include the perspective that the risks in vaginal breech delivery are due in part to intrapartum risks that are also present in vaginal cephalic delivery, risk of stillbirth due to continuing pregnancy beyond 39 weeks' gestation, and risks inherent in vaginal breech delivery.[52]

Planned vaginal breech delivery remains controversial. Epidemiologic studies in Scandinavia and Canada showed that vaginal breech delivery continues to be associated with an increased incidence of neonatal morbidity and mortality, the rates of which appear to be decreasing overall in association with increasing cesarean delivery rates for breech presentation.[49,58,59] A 2015 Cochrane review showed decreased short-term neonatal mortality and morbidity with increased short-term maternal mortality in settings with low perinatal mortality. The level of evidence was considered low, and the authors concluded that the benefits of elective cesarean delivery needed to be weighed against the preference of some women for vaginal delivery.[27] A 2015 meta-analysis of 27 studies, which included observational studies, of a total of 258,953 women, showed increased relative risk of neonatal morbidity and mortality in the range of 2 to 5 times with vaginal breech delivery versus cesarean delivery, yet low absolute delivery rates. The study results found that individualized decision making remains appropriate.[60]

Two considerations, not strictly medical, affect the decision about whether to perform a cesarean or vaginal delivery. First, the skills to perform a safe vaginal breech delivery are not taught in many residency programs, and clinicians who retain these skills are aging. Second, the medicolegal ramifications of vaginal delivery are unacceptable to many clinicians. Elective vaginal breech delivery is beyond the scope of the ALSO course, but the skills to perform an unplanned emergency vaginal breech delivery are essential for all maternity care clinicians. An understanding of the selection criteria and controversies regarding elective vaginal breech delivery may help maternity care clinicians decide if vaginal delivery is a reasonable option. In some situations, the physician or midwife will not have time to assess for appropriate candidacy or to convert to emergency cesarean delivery.

Certain contraindications exist for elective vaginal delivery of a fetus in breech presentation (*Table 2*):

- Unfavorable pelvis: if the pelvis is known to be small, or if it is android or platypelloid, vagi-

Table 2. Contraindications to Elective Vaginal Breech Delivery

Macrosomia (defined variously from 3,800 g [approximately 8.4 lb] upward)
Lack of physician experience with vaginal breech delivery
Footling breech presentation
Occult cord prolapse
Intrauterine growth restriction
Lack of facilities and personnel to switch rapidly to cesarean delivery
Fetal anomalies preventing vaginal delivery
Clinical or x-ray evidence of inadequate pelvis
Extended (*stargazing*) head on ultrasound examination

Information from Kotaska A, Menticoglou S, Gagnon R; MATERNAL FETAL MEDICINE COMMITTEE. Vaginal delivery of breech presentation. J Obstet Gynaecol Can. *2009;31(6):557-566.*

nal delivery should not be attempted. Magnetic resonance imaging (MRI) or computed tomography (CT) pelvimetry have been used in some studies.[55,61] However, radiologic pelvimetry has not been shown to improve outcomes in vaginal breech deliveries. The SOGC guidelines recommend clinical pelvimetry and state that radiologic pelvimetry is not necessary for a safe TOL with a fetus in breech presentation. MRI is the preferred study because of risks of maternal and fetal radiation exposure from pelvic CT.[57]

- Macrosomia (defined variously from 3,800 g [approximately 8.4 lb] upward)[62]
- Severe prematurity (defined variously)
- Intrauterine growth restriction or evidence of placental insufficiency
- Footling breech presentation: the feet may be palpable in many complete breech presentations, and a trial of vaginal breech delivery is acceptable. However, if the feet descend below the buttocks during the first stage of labor, a cesarean delivery is indicated
- Hyperextension of the fetal head (*stargazer*): delivery can be difficult, and labor can result in high incidence of spinal cord or other neurologic injuries as a result of a hyperextended head. An ultrasound should be performed at the start of labor to determine the attitude of the fetal head
- Fetal anomalies (eg, hydrocephalus)
- Absence of labor (eg, patients with prelabor rupture of membranes, nonprogressive labor): induction and augmentation of labor are controversial, and often avoided in favor of cesarean delivery.[56] However, in the PREMODA study, 74% of women received augmentation with oxytocin.[55] Augmentation may be reasonable if undertaken to increase infrequent contractions to every 2 to 4 minutes rather than to increase the strength of contractions that are already occurring at an acceptable frequency. RCOG and SOGC state that oxytocin augmentation is acceptable for treating weak or infrequent uterine contractions.[52,57] The 2009 SOGC guidelines recommended against labor induction; however, the 2019 SOGC guidelines state that, although evidence is limited, induction appears to be a safe option in well-selected patients.[57]
- Lack of a clinician with the experience and skill necessary for vaginal delivery and ability to maintain an operating room team (ie, nursing personnel, anesthesia personnel)

Various scoring systems have been developed to predict outcomes of vaginal breech delivery. The best known of these systems is the Zatuchni-Andros prognostic scoring index. It assigns points for parity, gestational age less than 37 weeks, estimated fetal weight less than 3.18 kg (7.01 lb), previous breech delivery, dilation at presentation, and station at presentation.[63] This system has several deficiencies including rewarding prematurity and the woman who labors at home who presents at a greater dilation and lower station. However, a better validated system of predicting breech outcomes has not been developed. The use of appropriate selection criteria and converting to cesarean delivery when labor progress is not adequate are the essential clinical components in the treatment of women who desire a trial of vaginal breech delivery.

In summary, the decision regarding the best mode of delivery of a fetus in breech presentation is complicated. Many factors must be considered, including the best conclusions from the medical literature, community and national standards as well as the individual case, the woman's wishes, and the clinician's skill.

Labor and Vaginal Delivery of a Fetus in Breech Presentation

A standard method of vaginal breech delivery is presented here, but acceptable variations exist. The ALSO program presents this method as one that is widely accepted and can be learned and practiced on a mannequin—but not necessarily as the only one, nor even the best. Although planned vaginal breech delivery remains an acceptable option for the skilled maternity care clinician under well-delineated hospital guidelines, the focus of the method presented here is the unplanned urgent vaginal breech delivery. Every maternity care clinician should know how to deliver a fetus in breech presentation.

Fundamental differences exist between delivery of infants in vertex and breech presentations. In vertex presentation, the fetus's largest part, the head, delivers first. Molding of the cranium can occur over several hours. In a breech delivery, the order of presentation is the buttocks, the shoulders, and the head with each part larger and less compressible than the one before. Molding of the

head does not occur because the fetal head is in the pelvis for only a few minutes, and because the head enters the pelvis with the base of the skull leading, which unlike the vertex, cannot mold. The challenge of the vaginal breech delivery is that the last part of the fetus to deliver is the largest part, and it might not fit through the pelvis.

Most clinical recommendations for vaginal delivery of the fetus in breech presentation have described delivery occurring in the dorsal lithotomy position, which was common when vaginal breech delivery was routine (pre-1980s). Based on this, in 2006, the RCOG advised that vaginal breech delivery occur in the dorsal lithotomy position because of a lack of studies or experience with alternative positions (eg, squatting, hands and knees, upright); however, the 2017 RCOG guidelines state that a recumbent or all-fours position may be used based on maternal preference and clinician experience.[52] A retrospective German study of 229 successful vaginal breech deliveries performed in the upright position compared with 40 deliveries performed in the dorsal lithotomy position between 2004 and 2011 found use of the upright position reduced the duration of the second stage of labor, the need for delivery maneuvers, and neonatal birth injuries.[64]

Labor with a fetus in breech presentation is similar to labor with a fetus in vertex position and may be allowed to continue spontaneously if the woman is a candidate for vaginal breech delivery and has been appropriately counseled, if progressive dilation and descent occur, and there is no fetal or maternal compromise. Most vaginal breech deliveries require a minimal number of maneuvers. Careful observation during each stage of the delivery and knowing when to assist the delivery and how to manage complications are essential for a safe vaginal breech delivery. The ALSO program has developed the CAREFUL mnemonic as a standardized technique for the vaginal delivery of a fetus in breech presentation.

Breech mnemonic: **CAREFUL**

Check presentation, dilation, and cord

Await umbilicus

Rotate for arms

Enter for the Mauriceau-Smellie-Veit (MSV) maneuver

Flex head

Back **U**p (sacrum anterior)

Lift baby onto mother

Check for Complete Dilation and Presenting Part, and Rule Out Cord Prolapse

The cervix must be completely dilated to avoid the potential for catastrophic cervical head entrapment. The determination of complete dilation with a fetus in breech presentation can be difficult because the clinician is feeling for the soft thin cervix against the soft buttocks rather than the hard skull. Because of the increased incidence of cord prolapse, it is essential to feel for an occult cord and to ensure that the buttocks are the leading part.

After the cervix is fully dilated, a period of passive descent should be considered. This gives additional assurance that no cervix remains, and it shortens the period of active pushing. A fetus in breech presentation can experience repetitive variable decelerations because of cord compression during the active second stage of labor. The SOGC guidelines recommend converting to cesarean delivery if delivery is not imminent after 1 hour of active pushing but permit up to 90 minutes of passive second stage.[57]

A frank breech presentation will distend the perineum and dilate the introitus similar to a vertex presentation. Episiotomy was traditionally recommended, but selective use is now recommended when additional room is required to enter the vagina to perform maneuvers (eg, Piper forceps application) to facilitate delivery.[65] Episiotomy will not create more room in the bony pelvis and can be difficult to perform after the body, except for the head, has been delivered.

Await Umbilicus

Typically, the fetus in frank breech position delivers with the axis of the hips in the AP plane, and the fetal sacrum will be to the left or the right. The anterior hip descends in to the introitus and passes below the symphysis in a manner analogous to the anterior shoulder. With lateral flexion of the fetal body, the posterior hip delivers over the perineum. External rotation follows delivery of the fetus, allowing the back to turn anteriorly.

Delivery should proceed spontaneously until the fetal umbilicus appears at the introitus. The woman should be making strong, controlled pushing efforts. Traction by the clinician before delivery of the umbilicus may promote extension of the fetal head or nuchal placement of the arms; therefore, the clinician should not pull on the fetus until the umbilicus is delivered, and

even then, traction is not necessary if the delivery continues to progress.

When the umbilicus delivers, a loop of several inches may be pulled down gently, but doing so is optional. If performed, it prevents tension on the cord as the body delivers and allows easy monitoring of the fetal pulse by palpation. The legs of a fetus in frank breech presentation may be delivered by inserting a finger behind the knee to flex the knee and abduct the thigh (Pinard maneuver [*Figure 5*]). Efforts to deliver the legs are not mandatory because the legs will deliver spontaneously, and the feet will spring free eventually.

After the umbilicus is delivered, gentle downward traction may be used to deliver the torso. The fetus may be grasped on the pelvis by the clinician's fingers, with thumbs on the sacroiliac regions. This avoids placing the hands too high on the fetus and injuring abdominal organs such as the spleen or liver. Traction should be in a 45-degree downward axis, toward the floor. The clinician may assume a position below the fetus (ie, on one knee in front of a delivery room table).

Rotate for Arms

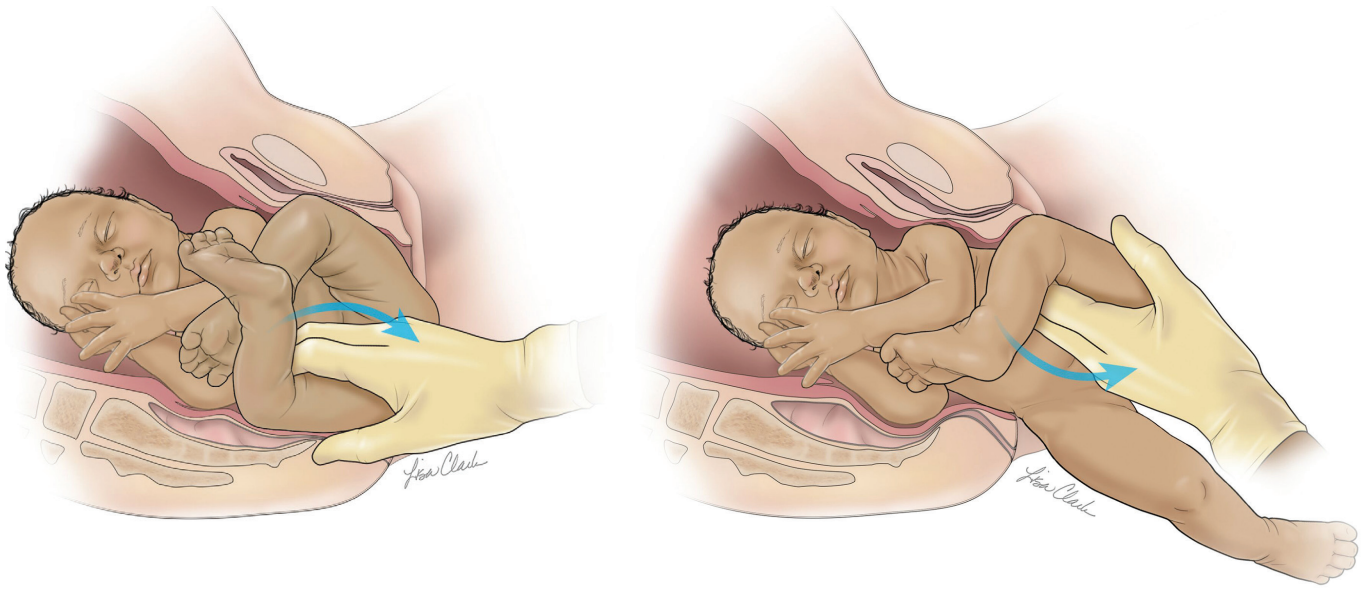
Delivery of the fetal trunk may be quick and may not require effort by the clinician, or delivery may require considerable effort. Rotation of the fetal back from one anterior oblique to the other may facilitate the extraction of the trunk and encourage the fetal arms to move to a flexed position across the chest. It is critical to keep the back up (sacrum anterior) during the delivery because it allows the fetal head to enter the pelvis OA. If the fetus rotates to the abdomen-up (sacrum posterior) position, the head will present unfavorable diameters to the maternal pelvis, jeopardizing a safe delivery.

Delivery of the arms occurs by rotating the fetal body into an oblique position. The tip of the fetal scapula will come into view, typically being easy to identify because it is *winged*. The anterior arm may then be swept down across the fetal chest and out of the introitus. If possible, the humerus should be splinted with two fingers rather than hooking the antecubital fossa, or elbow pit, with a finger. Rotation of the fetus into the opposite oblique lie allows delivery of the opposite arm in a similar fashion.

Enter for Mauriceau-Smellie-Veit Maneuver and Flex Head

Delivery of the head follows delivery of the trunk and is potentially the most difficult and hazardous part of the breech delivery. After the delivery of the arms, the head follows rapidly and spontaneously. Alternatively, the head may not be low enough in the pelvis to initiate assistive efforts. The clinician should attempt to see the nape of the neck. If not visible, the fetus can be allowed to

Figure 5. Pinard Maneuver



dangle with the head still inside the pelvis for up to 30 seconds while ensuring the fetus does not fall to the floor. The sacrum must be anterior.[1]

The head must be delivered by flexion through the pelvis. When the breech head is flexed and OA, and passes through the birth canal by further flexion, the same favorable diameters are presented to the pelvis as in a vertex OA delivery. A modification of the MSV maneuver that is designed to promote flexion is recommended to deliver the head. One of the clinician's hands should be placed inside the vagina superior to the fetus with one finger placed on the occiput, and one finger on each of the fetal shoulders. The other hand is placed beneath the fetus. The classic MSV maneuver describes placing a finger in the mouth, but this is no longer recommended because traction on the jaw can cause dislocation. As an alternative, two fingers may be placed on the maxillae. An assistant should follow the head abdominally and be prepared to apply suprapubic pressure to flex the head through the pelvis. The fetus may be wrapped in a sling that is held by the assistant or may be draped on the clinician's lower arm before delivery of the head.

After the head is in the appropriate position, the assisted delivery of the head commences. The head is flexed through the pelvis by four separate mechanisms: the occipital finger applies flexing pressure on the occiput, the assistant also applies suprapubic pressure on the occiput, the fingers on the maxillae apply pressure on the lower face to promote flexion, and the fetal body is raised upward by the sling in a large arc. Although strong controlled expulsive efforts by the woman are most helpful, some traction is also required for the delivery. This is accomplished by applying downward pressure from the fingers on the shoulders. The assistant who is holding the fetus by a sling may also hold the feet and pull gently as the body describes its arc. The fetal body should stay in a neutral position with regard to the head to avoid hyperextension. Ultimately, the body becomes inverted in the vertical plane, and at this point, an assistant must hold the feet to prevent the fetus from falling to the floor.

Back Up

The breech delivery is almost always accompanied by rotation into a sacrum anterior position as the trunk is delivering after the buttocks. In the unusual situation where the fetus attempts to move into a sacrum posterior position, the delivering clinician must gently guide and rotate the fetus into the sacrum anterior position *before* the delivery of the arms.

Lift Baby Onto Mother

As the mouth and nose appear over the perineum, they may be suctioned. The cranial vault then delivers by further flexion; the clinician may use the Ritgen maneuver. As the head emerges, the infant's body flips over past vertical onto the woman's abdomen. Delayed cord clamping is appropriate if the infant does not need resuscitation or additional treatment. It is more common for breech infants to be born with decreased tone and to require resuscitation, such as positive pressure ventilation, likely because of increased cord compression during the second stage of labor. In all vaginal breech deliveries, additional personnel must be present to perform neonatal resuscitation if needed.

Delivery in the Upright or All-Fours Positions

Vaginal breech delivery in an upright or all-fours position is now considered an acceptable option by RCOG and the Australian *Becoming a Breech Expert Course Manual* if the position is preferred by the woman and the clinician is experienced in its use.[52,65] Conversion to dorsal lithotomy position may be required to manage rare complications such as head entrapment. A 2017 case report demonstrated vaginal breech delivery in the upright position using a sequence of photographic images.[66] A 2017 study included descriptions of maneuvers that can be used in the upright position for shoulder dystocia (eg, the 180-degree torque maneuver) and to facilitate flexion to deliver the fetal head by pushing the fetus's shoulders against the pubic bone (the Frank nudge maneuver).[64]

Cesarean Delivery of a Fetus in Breech Presentation

Most planned breech deliveries in high-resource settings occur by cesarean delivery. Even when a vaginal breech delivery is planned, a substantial proportion will need to be converted to cesarean delivery as shown by the 29% cesarean delivery rate in the planned vagina breech group in the PREMODA study.[55] Some of these deliveries will need to be converted emergently because of

cord prolapse, fetal intolerance of second stage of labor, or rarely, abdominal rescue from a difficult vaginal breech delivery. The SOGC recommends the active second stage of labor occur near an operating room with personnel present to convert to emergent cesarean delivery, and RCOG guidelines state that ready access to cesarean delivery is important.[22,57]

Extraction of a fetus in breech presentation during cesarean delivery requires maneuvers like those used in vaginal delivery. Therefore, cesarean delivery of a fetus in breech presentation is an opportunity for a surgeon to practice the mechanics of vaginal breech delivery. The goal of cesarean delivery of a fetus in breech presentation is a gentle delivery. If the uterine or abdominal incisions are too small for easy delivery, they can be enlarged. This is not an option during vaginal delivery.

Piper Forceps

Piper forceps were designed to deliver the aftercoming head of a fetus in breech presentation. They are long and have an axis-traction curve. It is impossible to determine if Piper forceps application is appropriate by visualizing the placement of the Piper forceps on the fetal head, so they are always applied the same way—straight to the maternal pelvis as if the fetal position were OA. The blades are springy and grasp the fetal head in a nonspecific *basket catch* that has proved safe and effective.[1]

Forceps are indicated when the MSV maneuver fails. Although strict guidelines are lacking, Piper forceps should be considered if 2 or 3 minutes have passed without progress while attempting the MSV maneuver. Piper forceps may also be applied prophylactically if a fetus is thought to be fragile, such as a premature fetus. It is prudent to have Piper forceps readily available for any vaginal breech delivery, but Simpson forceps or other forceps can be used in an emergency.

To apply Piper forceps, the fetus (including the arms) is wrapped in a sling and gently held up and to the clinician's left. The left blade is always applied first. It is held in the clinician's left hand and is applied to the left side of the woman's pelvis (but to the right side of the fetus). Unlike other forceps applications, the clinician holds the handle in a horizontal position and below the fetus. The right hand is placed in the vagina alongside the fetal head to protect the vaginal sidewalls. Then the forceps blade is inserted between the right hand and the fetal head, following the cephalic curve of the blade around the head. After insertion, the handle may be supported by an assistant or allowed to dangle.

The right blade is then inserted in a similar fashion by grasping the handle with the right hand and sliding the blade into the vagina alongside the head while protecting the sidewall with the left hand. The forceps should then be locked. When the right blade is applied over the left blade, the lock will articulate normally. The handles are typically separated slightly away from the lock and should not be squeezed together. Because the clinician cannot determine how the blade is applied to the fetal head and face, no effort is made to do so.

Delivery of the head may commence when the application is complete. The clinician applies a small amount of traction to the forceps. Because the shanks of the forceps have a large axis-traction curve, no special maneuvers (eg, the Pajot maneuver) are required to ensure that traction is in the correct vector. The primary motion of the forceps is to raise the handles in a large arc, starting approximately horizontal and ending at or past vertical. This arc will flex the head through the pelvis with the same geometry as the MSV maneuver but with greatly increased leverage because of the length of the forceps. None of the flexing maneuvers of the MSV maneuver are required when Piper forceps are used. The fetus may be held in the sling or laid on the shanks of the forceps during the delivery.

The principal difficulty in applying Piper forceps is a result of the condition that indicates their use. That is, failure of the MSV maneuver implies a tight fit of the fetal head in the maternal pelvis. There may be an insufficient amount of room to place a hand alongside the head. In this situation, the blade must be blindly applied with risk of injury to the woman and fetus. After the Piper forceps are in place, delivery can be accomplished in almost every case. Training in Piper forceps is beyond the scope of the ALSO Vaginal Breech Delivery and Malpresentations workshop, but physicians who anticipate participating in emergency or elective vaginal breech deliveries in settings where they may be called on to deliver a trapped head are encouraged to seek additional training. Elective use of Piper forceps during cesarean delivery can provide an opportunity for training.[49]

Complications of Breech Delivery

Nuchal arm occurs when one or both arms are extended upward behind the neck, which may impede delivery of the head. When this occurs, three delivery options exist. If the fetus is small or the pelvis is large, the head and extended arm may be delivered together. Alternatively, the clinician may attempt to flex the arm and sweep it down over the face and chest. As a maneuver of last resort, the clinician may rotate the fetus 90 to 180 degrees in the direction of the hand to sweep the arm out of its nuchal position (clockwise for a left nuchal arm, counterclockwise for a right nuchal arm).

Medical management of cervical entrapment may be attempted using nitroglycerin to cause rapid but transient relaxation of the uterus. Intravenous nitroglycerin may be administered by an anesthetist, or sublingual spray may be used if available.[67] Because of the rare and emergent nature of head entrapment during vaginal breech birth, there are no published studies of medical management.

Resolution without excessive traction may require cutting the cervix, a procedure known as Duhrssen incisions. Ring forceps are placed in pairs, parallel to each other at 2 o'clock, 10 o'clock and, if possible, 6 o'clock, extending 3 cm to 4 cm into the cervix. A radial incision is made between the ring forceps of each pair. Anesthesia and exposure are major technical problems, and hemorrhage is a major potential complication. This procedure is recommended only in the most extreme life-threatening circumstances.

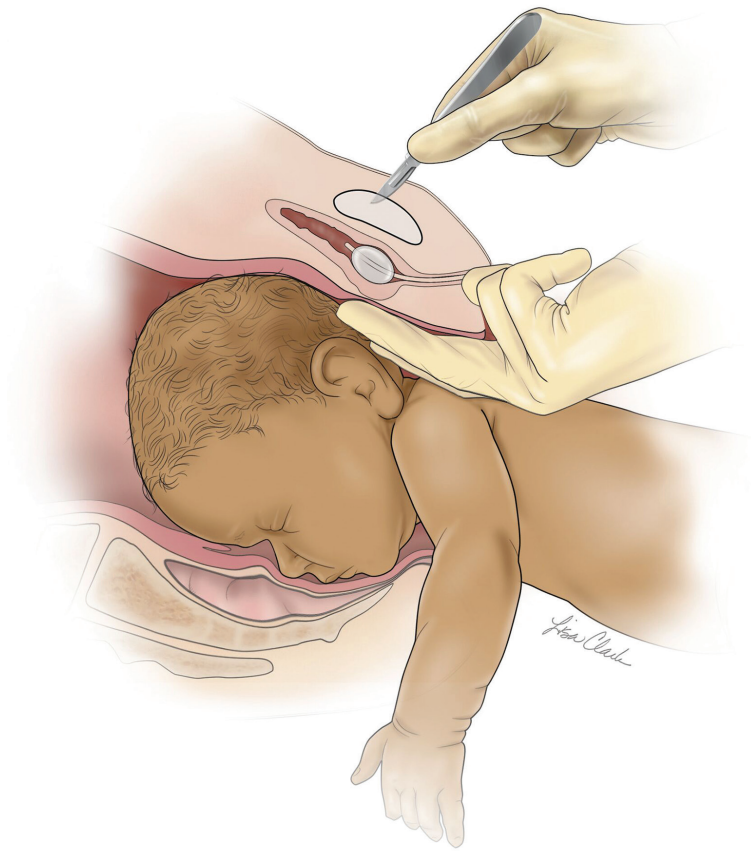
The fetus with hydrocephalus may present as a breech delivery with an entrapped head. The appearance of a meningomyelocele, or spina bifida, may indicate the presence of hydrocephalus, which occurs in approximately one-third of such cases.[68] A prenatal diagnosis of hydrocephalus will require highly individualized management and probable cesarean delivery. An unexpected diagnosis at the time of a breech delivery presents a significant dilemma. If cesarean delivery is available, emergent cesarean delivery for *abdominal rescue* will be required. Decompression of the fetal ventricles, or cephalocentesis, may be detrimental to the fetus, but it is the only way for the delivery to be completed if the fetus is alive and rapid cesarean delivery is not available. Cephalocentesis may be undertaken transvaginally or transabdominally with a long needle.

Symphysiotomy is an emergency maneuver for incising the ligaments of the pelvic symphysis to release a trapped aftercoming head (*Figure 6*). It is rarely used in high-resource settings, but its use in low-resource settings without ready access to emergency cesarean delivery can be lifesaving. Maternal risks include urological and orthopedic injuries.

Transverse Lie, or Shoulder Presentation

In *transverse lie,* the long axis of the fetus is approximately perpendicular or at right angles to that of the woman. In the back-down transverse lie, or *shoulder presentation,* the shoulder is over the pelvic inlet, the head is lying in one of the iliac fossae and the breech in the other. Transverse lie can also occur in the back-up orientation, most commonly in a second twin. On occasion, an unstable or oblique lie will be noted, in which the fetus switches from a breech or vertex to a transverse lie or assumes an intermediate lie.

Figure 6. Symphysiotomy to Free Trapped Aftercoming Head



Transverse lie occurs in approximately 0.3% of singleton deliveries.[69] The common causes of transverse lie are unusual relaxation of the abdominal wall, preterm fetus, placenta previa, abnormal uterus (eg, subseptate uterus, fibroids), contracted pelvis, tumor occluding the birth canal, and polyhydramnios.

Diagnosis

A suspected transverse lie presentation identified by vaginal examination will require confirmation with an ultrasound. On vaginal examination, the fetal head will not be felt and other body parts likely will not be felt in the pelvis. Using Leopold maneuvers, the fetal head may be palpable to the left or right of center near the maternal umbilicus.

Mechanism of Labor and Management of Delivery

Spontaneous delivery of a full-term fetus in transverse lie is impossible. Therefore, cesarean delivery is required in most cases. If transverse lie is identified before the onset of labor or in early labor with intact membranes, an attempt of ECV is reasonable if there are no contraindications to vaginal delivery (eg, placenta previa).

When labor ensues with a back-down transverse lie, the shoulder is forced into the pelvis and an arm may prolapse. With continued labor, a retraction ring may develop. Ultimately, in a neglected labor, the uterus ruptures and the woman and fetus are at risk of mortality. This scenario is rarely seen in modern obstetrics, but it may be encountered in parts of the world where health care access is limited.

Cesarean delivery for a back-down transverse lie may require a low vertical or classical incision for the clinician to successfully deliver one of the fetal poles through the uterine incision. A transverse uterine incision often will be adequate for delivery and has the benefit of allowing a TOL in future pregnancies.[70] If the initial transverse incision does not allow the feet to be reached, a T extension can be performed.

Face Presentation

In a *face presentation,* the head is hyperextended so the occiput is in contact with the fetal back, and the face is the presenting part. The skull that presents to the pelvis is the submentobregmatic diameter, which when the chin (mentum) is anterior, is favorable for most deliveries. Face presentation occurs in 0.2% to 0.3% of singleton deliveries.[69] When the fetus is large or the pelvis is contracted, there is a predisposition for extension of the fetal head. The pendulous abdomen of a grand multipara also predisposes to extension of the fetal head. In exceptional instances, extension also can be caused by enlargement of the neck because of goiter, cystic hygroma, or numerous coils of cord around the neck. Anencephalic fetuses often present with the face because of absent development of the cranium.

Diagnosis

The clinical diagnosis of a face presentation usually is made by vaginal examination. The mouth, nose, and the malar prominences may be palpated. As previously discussed, a face presentation may be confused with a breech presentation, particularly because breech is approximately 20 times more common (*Table 1*).[4,69] The mouth may be mistaken for the anus, and the malar prominences may be mistaken for the ischial tuberosities.

Mechanism of Labor

The key for successful delivery of a face presentation is for the chin to be under the pubic symphysis or for the fetus to be in the mentum anterior position. With further descent of the fetus, the cranial vault can sweep through the posterior pelvis and the head can be delivered by flexion with conversion to an OP delivery.

Although this mechanism does not present the most favorable diameter of the fetal head to the pelvis, if the fetus is not too large and the pelvis is adequate, spontaneous delivery can occur. If the chin rotates or remains posterior, there is no mechanism that allows the fetus to use the space in the posterior pelvis in the hollow of the sacrum, and delivery cannot occur. Forceps or manual rotation of a mentum posterior presentation should not be attempted because of the risk of fetal spinal cord injury.[71]

Management of Delivery

Spontaneous vaginal delivery may occur sometimes with surprising ease. The fetus must rotate to a mentum anterior position. Rotation from mentum posterior or mentum transverse often occurs late in the second stage of labor; therefore,

cesarean delivery should not occur at initial identification of this presentation. Expectant management is recommended even if identified in the second stage of labor if progress is occurring and results of fetal monitoring are not concerning.

A persistent mentum posterior without labor progress in the first or second stages of labor mandates a cesarean delivery. Forceps can be safely and successfully applied to a mentum anterior that is on the perineum. Use of a vacuum extractor is absolutely contraindicated. Likewise, scalp electrode internal monitoring is contraindicated to avoid injury to the face. There is an increased incidence of variable and late decelerations, and oxytocin augmentation should only be used with caution. Parents should be prepared for the infant's face to have significant bruises and edema, but recovery occurs within 24 to 48 hours.

Brow Presentation

In a *brow presentation,* the portion of the fetal head between the orbital ridge and the anterior fontanel presents at the pelvic inlet. The fetal head is in an attitude between full flexion (or occiput) and full extension (or face). The presenting fetal skull is the occipitomental diameter, which is unfavorable for delivery. Delivery of a persistent brow typically cannot occur unless the fetus is small, or the pelvis is large.

Brow presentation occurs in 0.007% of singleton deliveries.[69,72] The causes of this rare presentation are similar to those for face presentation. A brow presentation is typically unstable and often will convert to a face or a vertex presentation.

Diagnosis

Diagnosis of a brow presentation is made by vaginal examination. The frontal sutures, anterior fontanel, orbital ridges, eyes, and root of the nose may be felt. Frequently, the examination is confusing because of edema and unfamiliarity of the presenting features.

Mechanism and Management of Labor

The fetus in persistent brow presentation cannot be delivered vaginally under normal conditions. If the fetus converts to vertex or face presentation, delivery may occur according to the respective mechanisms for these presentations. In the absence of conversion and progress of labor, cesarean delivery is required.

Compound Presentation

In a *compound presentation* an extremity, typically a hand, prolapses alongside the main presenting part, typically the head. Often, no cause is found. This presentation is more common with premature infants and when the fetal presenting part does not completely occlude the pelvic inlet.

Diagnosis

The diagnosis of compound presentation is typically made by vaginal examination. It is critical to distinguish between a hand and a foot prolapsed alongside the head.

Management of Delivery

If labor is progressing normally, no intervention is necessary. Most commonly, the prolapsed limb will deliver spontaneously along with the head, or sometimes the fetus will retract its limb spontaneously. If the prolapsed arm appears to be impeding descent, it should be gently elevated upward, and the head manipulated simultaneously downward. On occasion, cesarean delivery will be necessary. Parents should be told to expect bruising and edema of the prolapsed extremity.

Prolapse of the Umbilical Cord

Prolapse of the umbilical cord is an obstetric emergency. The cord may become compressed or occluded between the presenting part of the fetus and the pelvic brim or sidewall, resulting in asphyxia and mortality. The incidence of cord prolapse is 0.1% to 0.6% in vertex presentations.[73] The incidence of cord prolapse is slightly increased in frank breech presentation, occurring in less than 1% of labors; however, the rate in footling breech presentation is markedly increased at approximately 10%.[57]

Cord prolapse is most common when the fetus does not occlude the pelvic inlet, as in a footling breech presentation. Other factors that may contribute to cord prolapse are prematurity, polyhydramnios, high presenting part, and a long cord. Approximately 50% of umbilical cord prolapses follow obstetric interventions,[74] such as when the membranes are ruptured with the presenting part high out of the pelvis and the gush of fluid may then push the cord down into the vagina. In addition, the cord may have already been coiled beneath the fetal presenting part (occult cord prolapse) such that rupture of the membranes merely

revealed the prolapse but did not cause it. The proportion of cases that are iatrogenic appears to be decreasing, but the use of a balloon catheter for cervical ripening is one modern intervention that can lead to cord prolapse by elevating the presenting part.[75]

Rapid identification and management of cord prolapse may be lifesaving for the fetus. The management steps are:

1. Diagnose the cord prolapse by visual inspection or palpation on immediate vaginal examination. The cord may be extruded from the vagina, coiled in the vagina, or wrapped across the presenting part. The only sign may be a severe variable deceleration or bradycardia after rupture of the membranes
2. Quickly assess the fetal status via fetal heart rate (FHR) monitoring or ultrasound
3. Assess the dilation and status of labor. In the uncommon situation where the fetus can be delivered more quickly and safely by vaginal rather than cesarean delivery, proceed immediately using forceps, vacuum, or in the case of a second twin, total breech extraction, when appropriate
4. If immediate vaginal delivery is not feasible, prepare for cesarean delivery. Elevate the presenting part out of the pelvis to protect the cord from occlusion. This may be performed by placing a hand in the vagina and forcefully but carefully elevating the presenting part. Alternatively, some success has been achieved by filling the bladder rapidly with about 500 cc of saline solution followed by clamping of the catheter. Oxytocin should be discontinued. Tocolysis (eg, terbutaline 0.25 mg subcutaneously) is helpful if the patient is in labor and there are recurrent FHR decelerations or if cesarean delivery will be delayed.[73] Placing the woman in a deep Trendelenburg position also is useful to add gravity to other efforts to elevate the fetus off the cord. The effectiveness of these maneuvers can be measured by monitoring the FHR. Handling of the cord should be minimized to prevent vasospasm of the umbilical arteries
5. Do not attempt to replace the cord in the uterus
6. Perform an emergent cesarean delivery while continuing all efforts to hold the presenting part off the cord. If a delay is encountered, wrap the cord in warm wet packs

Prevention of cord prolapse is difficult but occasionally may be accomplished by identifying risk factors or by identifying a cord presentation by ultrasound. Artificial rupture of the membranes should not be performed when the station is high. If artificial rupture of membranes is essential to manage a difficult obstetric situation, and the head is unengaged and high, the membranes can be needled under double set-up conditions.[75] The same procedure can be used to rupture the membranes when polyhydramnios is present.

Patients in the later stages of pregnancy who are at high risk of cord prolapse (eg, footling breech presentation, polyhydramnios) should be identified. They can be instructed to examine themselves for cord prolapse if their membranes rupture outside of a hospital. If a prolapse is identified, they should assume a deep knee-chest position and maintain the position during transport to the hospital.

Multiple Gestation

Multiple gestation occurred in 3.4% of births in the United States in 2013.[76] The twinning rate rose 76% from 1980 to 2009 with the increase attributed to the use of fertility therapies and increased proportion of women with advanced maternal age.[76] Twin pregnancies are 7 times more likely to result in delivery at less than 32 weeks' estimated gestational age, and multifetal pregnancies have 5 times the risk of stillbirth compared with singleton pregnancies.[77] Congenital anomalies, IUGR, and intrapartum complications also contribute to stillbirth. Dizygotic twins occur in approximately two-thirds of twin gestations, and increase with age, parity, and certain familial and racial circumstances. Monozygotic twins occur in one-third of twin gestations, and there are no known predisposing factors. Morbidity and mortality are higher in monozygotic twins.[78]

Maternal complications are common in multiple gestation. These include gestational hypertension, preeclampsia, gestational diabetes, anemia, hyperemesis, abruption, placenta previa, postpartum hemorrhage, and increased assisted delivery.

Diagnosis

Multiple gestation is now routinely diagnosed by ultrasound during prenatal care in high-resource settings. The intrapartum diagnosis of the second twin occurs uncommonly but is most common when there is a lack of prenatal care. Historical and physical findings suggestive of multiple gesta-

tion and indicating an ultrasound are: uterine size larger than date, hyperemesis gravidarum, early preeclampsia, elevated maternal serum alpha fetoprotein levels, suggestive palpatory or auscultatory findings, polyhydramnios, ovulation induction, and family history of multiple gestations.

Prenatal Management

Several factors are more common in multiple versus singleton gestations:

Prematurity. Prematurity is the greatest threat to multiple gestation fetuses, and prevention of prematurity is the highest priority. Unfortunately, no preventive measures, including bed rest, routine cerclage, and tocolytic drugs, have been shown to effectively prevent preterm labor.[79,80] Although progesterone has been shown to be beneficial in singleton pregnancies with a history of preterm birth (intramuscular weekly progesterone) and singleton pregnancies with a short cervix diagnosed between 16 and 24 weeks' gestation (vaginal progesterone), the role of progesterone in twin pregnancies remains controversial and no benefit has been shown for routine use of vaginal progesterone or intramuscular 17 alpha-hydroxyprogesterone caproate in twin pregnancies with a short cervical length.[81-83] Intramuscular progesterone use in twin pregnancies complicated by a prior preterm delivery may be reasonable based on the singleton date, but there is no evidence to support or refute this practice.[84]

Congenital anomalies and developmental defects. Compared with singleton pregnancies, the rates of congenital anomalies and developmental defects are doubled in twin pregnancies, and higher in monozygotic twin pregnancies.[85] An ultrasound including a detailed anatomic survey is recommended for all women with a multiple gestation at approximately 18 weeks' gestation. A first-trimester ultrasound may be performed to confirm or determine gestational age as part of genetic screening and to determine chorionicity.

Preeclampsia. Compared with singleton pregnancies, preeclampsia occurs twice as often in twin gestations.[86] A daily low-dose aspirin (81 mg/day) taken orally starting after 12 weeks' gestation is recommended for women at risk of preeclampsia, including those with multiple gestations.[87] Iron deficiency is common, and iron supplementation is typically indicated. Women with twin pregnancies are at greater risk of gestational diabetes, but routine screening in early pregnancy is not recommended.[88,89]

Growth restriction, size discordance, and twin-to-twin transfusion syndrome. The possibility of IUGR and discordant growth requires surveillance with ultrasound for interval growth.[90] Size discordance greater than 20% is associated with a sevenfold increase in major neonatal morbidity.[91] Ultrasound examination every 4 weeks starting at approximately 24 weeks' gestation is recommended to assess interval growth and concordance. In pregnancies with monochorionic twins who have an increased risk of twin-to-twin transfusion syndrome, surveillance should start at approximately 16 weeks' gestation with assessment of amniotic fluid volume every 2 weeks. Intervention in the presence of significant discordance before 36 weeks' gestation or twin-to-twin transfusion syndrome is complex, and perinatal consultation is appropriate.

Fetal mortality. Fetal mortality, including stillbirth, is much more common in twin than singleton pregnancies and in monoamniotic/dichorionic than in diamniotic/dichorionic pregnancies. Assessing the FHR at each prenatal visit should be performed via ultrasound (or electronic fetal monitor with two FHR attachments) rather than Doppler ultrasound imaging. When stillbirth of one twin occurs, conservative management of the surviving twin is indicated, at least until 34 weeks' gestation. Surviving twins in monochorionic/diamniotic pregnancies are at risk of neurologic injury when the stillbirth occurs after 14 weeks' gestation, but early delivery is not beneficial because the injury is thought to occur before the time of diagnosis of a single twin demise. Because of the higher risk of intrauterine fetal demise, routine induction of diamniotic/dichorionic twin pregnancies at 38 weeks' gestation and monoamniotic/dichorionic at 36 to 37 weeks' gestation is commonly recommended.[92,93]

Placenta previa. The incidence of placenta previa is increased in multiple gestation and should be detected by the recommended anatomic survey and/or interval growth ultrasounds.

Intrapartum Management

Delivery of twin pregnancies presents a range of challenges. Intrapartum complications include malpresentations, locked twins, cord prolapse, abruption, concerning FHR tracing, dysfunctional

labor, and postpartum hemorrhage. Several of these complications can arise from the way the twins present. Either fetus may be vertex, breech, or in a transverse lie. Theoretically, there are nine combinations of presentations of first and second twins, but for practical purposes there are three (*Table 3*).[94]

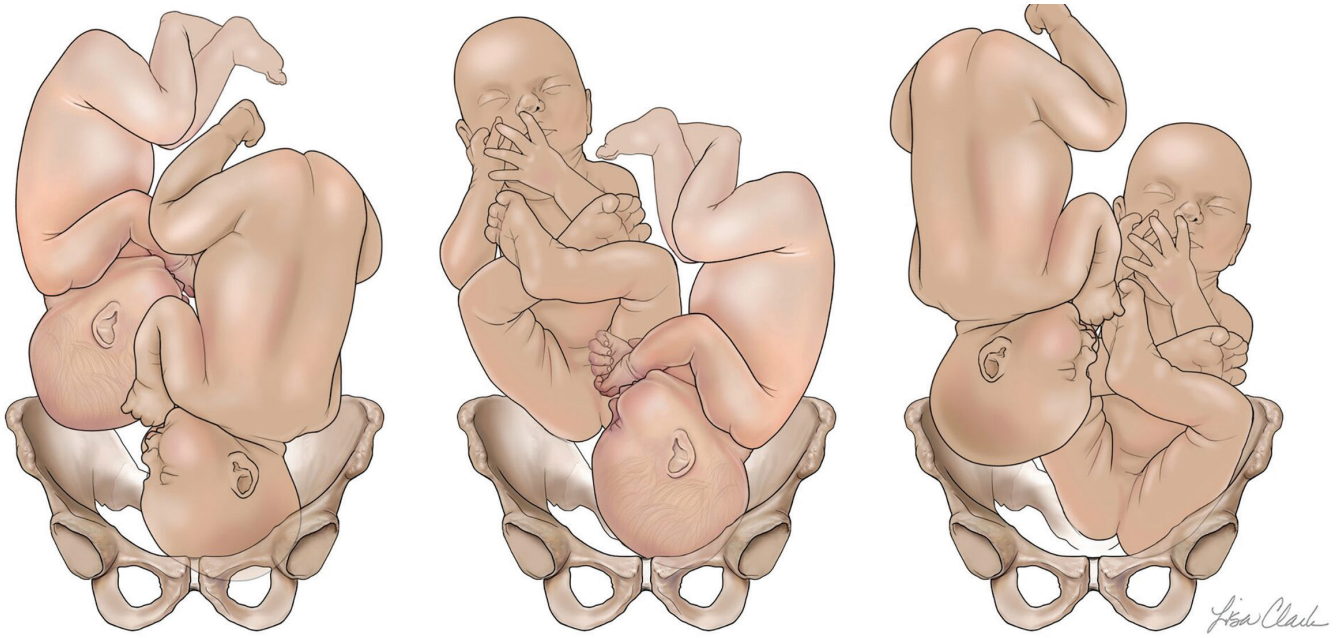
Vertex-vertex presentations are the most common and least complicated. With appropriate monitoring and the ability to respond to an emergency with urgent cesarean or assisted vaginal delivery, labor can be allowed to progress to vaginal delivery of both fetuses.[95] Oxytocin induction or augmentation, epidural analgesia, and other interventions are all acceptable, with caution. The interval between deliveries is not critical if the second fetus is doing well, but oxytocin augmentation is often used when delay is encountered between deliveries. A 2011 Cochrane review showed minimal evidence regarding optimal mode of delivery in twin pregnancies.[96]

Based on a 2013 RCT that showed no benefit from routine cesarean delivery as long as the first twin was in vertex position and at least 32 weeks' gestation, current recommendations are not to perform cesarean delivery for vertex-vertex twins without another indication.[97] A physician experienced in vaginal breech delivery and breech extraction should be available for all twin vaginal deliveries because the second twin can change to a breech or transverse presentation after the delivery of the first twin.

When vaginal delivery is attempted, the time of greatest risk occurs after the delivery of the first twin, when the provider must determine the presentation of the second twin, which may be different from its presentation before the first twin was delivered. A combination of external examination, internal examination, and ultrasound may be used. Assuming the second twin is in breech presentation or a transverse lie, a decision must be made whether to attempt an ECV to vertex, deliver

Table 3. Presentation of Twins

Twin A	Twin B	Percentage of Occurrence
Vertex	Vertex	40
Vertex	Nonvertex	40 (30% breech/10% transverse)
Breech	Vertex/Nonvertex	20



Information from Cruikshank DP. Intrapartum management of twin gestations. Obstet Gynecol. *2007;109(5):1167-1176.*

the second twin as a vaginal breech delivery, or perform a cesarean delivery. The exact obstetric circumstances, the experience of the clinician, condition of the fetus, state of mind of the woman, and the available resources are all factors in the decision. The second twin must be carefully monitored because placental abruption and umbilical cord prolapse are obstetric emergencies that may occur between the delivery of the first and second twin.

If concerning fetal monitoring requires expeditious delivery of the second twin, then a vacuum-assisted vaginal delivery may be performed at a slightly higher station than would typically be considered for a singleton. A mid-pelvic vacuum of the second twin at 0 or +1 station is reasonable if the estimated fetal weight of the second twin is not considerably greater than the first twin.[98] The delivery of twins is best accomplished in the operating room, in case a rapid cesarean delivery is needed. Anesthesia should be on standby.

When the first twin is vertex but the second is nonvertex, the optimal mode of delivery has been controversial. Some clinicians advocate for cesarean delivery, but if a physician experienced in internal podalic version and vaginal breech delivery of the second twin is available, routine cesarean delivery is not necessary.[97] Vaginal breech delivery of the second twin is reasonable for a fetus with an estimated weight greater than 1,500 g (3.3 lb), greater than 32 weeks' gestation and if criteria are met for vaginal delivery of a singleton fetus in breech presentation as discussed previously.[97]

When the second twin is presenting in a transverse or oblique presentation or as a footling breech, then a breech extraction can be performed.[99] The clinician will identify and grasp the feet without rupturing the second gestational sac and bring the feet down to the vagina. Abdominal ultrasound to determine the location of the feet can be helpful. The delivering clinician's second hand or an assistant can apply gentle abdominal pressure to help guide the fetal head upward into the uterine fundus. With the clinician exerting steady downward traction on the feet to maintain the breech as the presenting part, the membranes are then ruptured. After delivery of the umbilicus, the delivery of the arms and head is similar to other vaginal breech deliveries.

Breech extraction of the second twin may also occur as part of an internal podalic version in which the vertex is elevated out of the pelvis before reaching for the feet. Breech extraction and internal podalic version should not be attempted unless the clinician has training and experience. This is perhaps the most difficult and dangerous procedure permissible in modern obstetrics. Breech extraction and podalic version should be performed only if the estimated fetal weight of the second twin is not substantially greater (eg, 20%) than the first.[97,99] The use of a model to simulate internal podalic version or breech extraction to deliver the second twin has been developed and the cited reference includes links to videos demonstrating the simulations.[100]

Undiagnosed twins are rare in areas where ultrasound is frequently used. In the pre-ultrasound era, as many as 50% of twin gestations were unsuspected until delivery. When ultrasound has not been performed, birth attendants should always be alert to this possibility. If a nonvertex second twin unexpectedly presents in a setting without an experienced clinician because of an undiagnosed second twin or no prenatal care, options include ECV (as previously discussed) or cesarean delivery.

Situations mandating cesarean delivery for a twin gestation include cord prolapse, abruption, clinician inability to reach the feet to perform internal podalic version, and breech extraction of a fetus in a transverse lie. A contributing problem occurs when the cervix closes after the first twin is delivered. These situations can arise suddenly, so resources for immediate cesarean delivery should be available. When the first twin is in nonvertex position, cesarean delivery typically is recommended, but high-quality evidence is lacking. Although rare, locking or collision of the twins is a disastrous event that can occur when the first twin is in breech presentation and the second twin is vertex or in a transverse lie. ECV of a breech first twin has been considered to be contraindicated or not technically feasible; however, a 2019 retrospective review of attempted ECVs of breech first twin at one hospital showed success in 10 of the 19 cases with eight subsequent vaginal deliveries. There were no emergent cesarean deliveries or neonatal injuries. Additional studies are needed to evaluate the safety of ECV for a first twin.[101]

Cesarean delivery in women with multiple gestation presents anesthetic and surgical challenges because of the enlarged uterus, the exaggerated physiologic response to pregnancy, and the potential for unusual presentations of the fetuses.

The necessity for a vertical incision in both the skin and uterus is a special consideration when the twins are in unusual or entwined positions. Conjoining of twins is a rare condition and is beyond the scope of this chapter but should be considered if ultrasound shows twins in a face-to-face or back-to-back position.

After delivery, postpartum hemorrhage is relatively common because of the overdistension of the uterus. Clinicians should be fully prepared with IV access, proper oxytocic drugs, and blood products. Neonatal resuscitation is often required because of prematurity or the many potential complications of multiple gestation. Frequently, two infants and the woman need treatment simultaneously. Adequate personnel and equipment must be available.

Global Perspective

The diagnosis, management, and perinatal outcomes of pregnancies complicated by malpresentation in low-resource settings will be strongly influenced by the availability of obstetric ultrasound and urgent cesarean delivery. A greater proportion of fetuses in breech presentation will be delivered vaginally when the diagnosis is delayed because of the lack of access to ultrasound and time to initiate a cesarean delivery can be lengthy. In the TBT, 9.6% of women assigned to the elective cesarean delivery group had vaginal deliveries.[51] The safest route for breech deliveries is not as clear for developing countries, as the TBT showed that the reduction in adverse perinatal outcomes was less pronounced and did not reach statistical significance in countries where the perinatal mortality rate was greater than 20 per 1,000.[51]

A 2015 Cochrane review showed that early ultrasound significantly decreases the number of post-date pregnancies and undiagnosed twins.[102] In settings where routine obstetric ultrasound is uncommon, a higher proportion of twin pregnancies may be diagnosed in the third trimester or even after delivery of the first twin. For these reasons, skills in delivery of fetuses in breech presentation and nonsurgical maneuvers (eg, ECV, internal podalic version, manual rotation from OP position) are essential in low-resource settings.[102] Transverse lie of a dead fetus is a life-threatening complication for women in developing countries and almost unheard of in high-resource settings. Emergent treatment may require destructive delivery, internal podalic version, or laparotomy.

Summary

There are six types of malpositions or malpresentations. Some are common (OP position, breech presentation) and some are rare (transverse lie, brow presentation, face presentation, compound presentation). Diagnosis is made by physical examination and imaging. A high index of suspicion for malpositions and malpresentations is helpful in making the diagnosis. Each variation in position or presentation has its own potential complications. Clinicians should be alert not only to complications resulting from labor and delivery, but also problems that may be causative of the malpresentation.

Vaginal delivery may be considered for four of these presentations: OP, frank breech, mentum anterior position, and compound. With OP position, the clinician has several management choices for delivery. With breech presentation, complex criteria determine if vaginal delivery can occur safely. ECV should be offered for nonvertex presentation at 37 weeks' gestation or greater. A high degree of technical skill and judgment is required to safely deliver fetuses with malpresentations. Multiple gestation presents a wide variety of special challenges to the clinician. If the first twin is in vertex presentation, then vaginal delivery is usually the recommended mode of delivery.

Nursing Considerations: Malpresentations, Malpositions, and Multiple Gestation

- Determine the location of ultrasound equipment every shift and learn how to turn it on
- Identify patients at risk of malpresentations and malpositions
- Apply evidence to advocate for the option of regional anesthesia in selected patients undergoing external cephalic version
- During an emergency breech vaginal delivery, consider positioning the woman at the edge of the bed, establishing intravenous access, and identifying your institution's location of and administration process for nitroglycerin
- During a cord prolapse, consider changing the maternal position and hold the presenting part off of the cord as appropriate. If entering the vagina, do not remove your hand from position until indicated by delivery
- Validate distinct fetal heart rates for multiple gestation

References

1. Cunningham FG, Leveno KJ, Gilstrap LC III, Hauth JC, eds. *Williams Obstetrics*. 22nd ed. New York, NY: McGraw-Hill; 2005.
2. Ali UA, Norwitz ER. Vacuum-assisted vaginal delivery. *Rev Obstet Gynecol*. 2009;2(1):5-17.
3. López-Zeno J. Presentation and Mechanisms of Labor. 2008. Available at https://www.glowm.com/section_view/heading/PresentationandMechanismsofLabor/item/126.
4. Barth WH Jr. Persistent occiput posterior. *Obstet Gynecol*. 2015; 125(3):695-709.
5. Bellussi F, Ghi T, Youssef A, et al. The use of intrapartum ultrasound to diagnose malpositions and cephalic malpresentations. *Am J Obstet Gynecol*. 2017;217(6):633-641.
6. Bellussi F, Ghi T, Youssef A, et al. Intrapartum Ultrasound to Differentiate Flexion and Deflexion in Occipitoposterior Rotation. *Fetal Diagn Ther*. 2017;42(4):249-256.
7. Cheng YW, Shaffer BL, Caughey AB. The association between persistent occiput posterior position and neonatal outcomes. *Obstet Gynecol*. 2006;107(4):837-844.
8. Pearl ML, Roberts JM, Laros RK, Hurd WW. Vaginal delivery from the persistent occiput posterior position. Influence on maternal and neonatal morbidity. *J Reprod Med*. 1993;38(12):955-961.
9. Bueno-Lopez V, Fuentelsaz-Gallego C, Casellas-Caro M, et al. Efficiency of the modified Sims maternal position in the rotation of persistent occiput posterior position during labor: A randomized clinical trial. *Birth*. 2018;45(4):385-392.
10. Le Ray C, Serres P, Schmitz T, et al. Manual rotation in occiput posterior or transverse positions: risk factors and consequences on the cesarean delivery rate. *Obstet Gynecol*. 2007;110(4):873-879.
11. Malvasi A, Tinelli A, Barbera A, et al. Occiput posterior position diagnosis: vaginal examination or intrapartum sonography? A clinical review. *J Matern Fetal Neonatal Med*. 2014;27(5):520-526.
12. Fitzpatrick M, McQuillan K, O'Herlihy C. Influence of persistent occiput posterior position on delivery outcome. *Obstet Gynecol*. 2001;98(6):1027-1031.
13. American College of Obstetricians and Gynecologists' Committee on Practice Bulletins—Obstetrics. Practice Bulletin no. 165: prevention and management of obstetric lacerations at vaginal delivery. *Obstet Gynecol*. 2016;128(1):e1-e15.
14. Bahl R, Van de Venne M, Macleod M, et al. Maternal and neonatal morbidity in relation to the instrument used for mid-cavity rotational operative vaginal delivery: a prospective cohort study. *BJOG*. 2013; 120(12):1526-1532.
15. Burke N, Field K, Mujahid F, Morrison JJ. Use and safety of Kielland's forceps in current obstetric practice. *Obstet Gynecol*. 2012; 120(4):766-770.
16. Gray CJ, Shanahan MM. Breech Presentation. In: *StatPearls* [Internet]. Treasure Island, FL: StatPearls Publishing; 2020.
17. Hemelaar J, Lim LN, Impey LW. The Impact of an ECV Service is Limited by Antenatal Breech Detection: A Retrospective Cohort Study. *Birth*. 2015;42(2):165-172.
18. Andrews S, Leeman L, Yonke N. Finding the breech: Influence of breech presentation on mode of delivery based on timing of diagnosis, attempt at external cephalic version, and provider success with version. *Birth*. 2017;44(3):222-229.
19. Wastlund D, Moraitis AA, Dacey A, et al. Screening for breech presentation using universal late-pregnancy ultrasonography: A prospective cohort study and cost effectiveness analysis. *PLoS Med*. 2019;16(4):e1002778.
20. Hofmeyr GJ, Kulier R. Cephalic version by postural management for breech presentation. *Cochrane Database Syst Rev*. 2012;10: CD000051.
21. Smith C, Crowther C, Wilkinson C, et al. Knee-chest postural management for breech at term: a randomized controlled trial. *Birth*. 1999;26(2):71-75.
22. American College of Obstetricians and Gynecologists' Committee on Practice Bulletins—Obstetrics. Practice Bulletin no. 161: External Cephalic Version. *Obstet Gynecol*. 2016;127(2):e54-e61.
23. Hofmeyr GJ, Kulier R, West HM. External cephalic version for breech presentation at term. *Cochrane Database Syst Rev*. 2015; 4(4):CD000083.
24. Impey LWM, Murphy DJ. External Cephalic Version and Reducing the Incidence of Term Breech Presentation: Green-top Guideline No. 20a. *BJOG*. 2017;124(7):e178-e192.
25. Kok M, Cnossen J, Gravendeel L, et al. Clinical factors to predict the outcome of external cephalic version: a metaanalysis. *Am J Obstet Gynecol*. 2008;199(6):630.e1-e7; discussion e1-e5.
26. Thissen D, Swinkels P, Dullemond RC, van der Steeg JW. Introduction of a dedicated team increases the success rate of external cephalic version: A prospective cohort study. *Eur J Obstet Gynecol Reprod Biol*. 2019;236:193-197.
27. Hutton EK, Hannah ME, Ross SJ, et al; Early ECV2 Trial Collaborative Group. The Early External Cephalic Version (ECV) 2 Trial: an international multicentre randomised controlled trial of timing of ECV for breech pregnancies. *BJOG*. 2011;118(5):564-577.
28. Hutton EK, Hofmeyr GJ, Dowswell T. External cephalic version for breech presentation before term. *Cochrane Database Syst Rev*. 2015;7(7):CD000084.
29. Rosman AN, Guijt A, Vlemmix F, et al. Contraindications for external cephalic version in breech position at term: a systematic review. *Acta Obstet Gynecol Scand*. 2013;92(2):137-142.
30. Sela HY, Fiegenberg T, Ben-Meir A, et al. Safety and efficacy of external cephalic version for women with a previous cesarean delivery. *Eur J Obstet Gynecol Reprod Biol*. 2009;142(2):111-114.
31. ACOG Practice Bulletin no. 205: vaginal birth after cesarean delivery. *Obstet Gynecol*. 2019;133(2):e110-e127.
32. Burgos J, Cobos P, Rodríguez L, et al. Is external cephalic version at term contraindicated in previous caesarean section? A prospective comparative cohort study. *BJOG*. 2014;121(2):230-235, discussion 235.
33. Cluver C, Gyte GM, Sinclair M, et al. Interventions for helping to turn term breech babies to head first presentation when using external cephalic version. *Cochrane Database Syst Rev*. 2015;2(2): CD000184.
34. Coyle ME, Smith CA, Peat B. Cephalic version by moxibustion for breech presentation. *Cochrane Database Syst Rev*. 2012;5(5): CD003928.
35. Goetzinger KR, Harper LM, Tuuli MG, et al. Effect of regional anesthesia on the success rate of external cephalic version: a systematic review and meta-analysis. *Obstet Gynecol*. 2011;118(5):1137-1144.
36. Weiniger CF, Ginosar Y, Elchalal U, et al. Randomized controlled trial of external cephalic version in term multiparae with or without spinal analgesia. *Br J Anaesth*. 2010;104(5):613-618.
37. Weiniger CF, Ginosar Y, Elchalal U, et al. External cephalic version for breech presentation with or without spinal analgesia in nulliparous women at term: a randomized controlled trial. *Obstet Gynecol*. 2007;110(6):1343-1350.

38. Carvalho B, Tan JM, Macario A, et al. Brief report: a cost analysis of neuraxial anesthesia to facilitate external cephalic version for breech fetal presentation. *Anesth Analg*. 2013;117(1):155-159.
39. Weiniger CF, Spencer PS, Weiss Y, et al. Reducing the cesarean delivery rates for breech presentations: administration of spinal anesthesia facilitates manipulation to cephalic presentation, but is it cost saving? *Isr J Health Policy Res*. 2014;3(1):5.
40. Suen SS, Khaw KS, Law LW, et al. The force applied to successfully turn a foetus during reattempts of external cephalic version is substantially reduced when performed under spinal analgesia. *J Matern Fetal Neonatal Med*. 2012;25(6):719-722.
41. Nassar N, Roberts CL, Barratt A, et al. Systematic review of adverse outcomes of external cephalic version and persisting breech presentation at term. *Paediatr Perinat Epidemiol*. 2006;20(2):163-171.
42. Suyama F, Ogawa K, Tazaki Y, et al. The outcomes and risk factors of fetal bradycardia associated with external cephalic version. *J Matern Fetal Neonatal Med*. 2019;32(6):922-926.
43. Fernandez CO, Bloom SL, Smulian JC, et al. A randomized placebo-controlled evaluation of terbutaline for external cephalic version. *Obstet Gynecol*. 1997;90(5):775-779.
44. Coco AS, Silverman SD. External cephalic version. *Am Fam Physician*. 1998;58(3):731-738, 742-744.
45. Collins S, Ellaway P, Harrington D, et al. The complications of external cephalic version: results from 805 consecutive attempts. *BJOG*. 2007;114(5):636-638.
46. ACOG Committee Opinion no. 764: medically indicated late-preterm and early-term deliveries. *Obstet Gynecol*. 2019;133(2):e151-e155.
47. ACOG Committee Opinion no. 765: avoidance of nonmedically indicated early-term deliveries and associated neonatal morbidities. *Obstet Gynecol*. 2019;133(2):e156-e163.
48. Lee HC, El-Sayed YY, Gould JB. Population trends in cesarean delivery for breech presentation in the United States, 1997-2003. *Am J Obstet Gynecol*. 2008;199(1):59.e1-59.e8.
49. Lyons J, Pressey T, Bartholomew S, et al; Canadian Perinatal Surveillance System (Public Health Agency of Canada). Delivery of breech presentation at term gestation in Canada, 2003-2011. *Obstet Gynecol*. 2015;125(5):1153-1161.
50. Danielian PJ, Wang J, Hall MH. Long-term outcome by method of delivery of fetuses in breech presentation at term: population based follow up. *BMJ*. 1996;312(7044):1451-1453.
51. Hannah ME, Hannah WJ, Hewson SA, et al; Term Breech Trial Collaborative Group. Planned caesarean section versus planned vaginal birth for breech presentation at term: a randomised multicentre trial. *Lancet*. 2000;356(9239):1375-1383.
52. Impey LWM, Murphy DJ, Griffiths M. Management of Breech Presentation: Green-top Guideline No. 20b. *BJOG*. 2017;124(7):e151-e177.
53. Greene MF. Vaginal breech delivery is no longer justified. *Obstet Gynecol*. 2002;99(6):1113-1115.
54. Whyte H, Hannah ME, Saigal S, et al; Term Breech Trial Collaborative Group. Outcomes of children at 2 years after planned cesarean birth versus planned vaginal birth for breech presentation at term: the International Randomized Term Breech Trial. *Am J Obstet Gynecol*. 2004;191(3):864-871.
55. Goffinet F, Carayol M, Foidart JM, et al; PREMODA Study Group. Is planned vaginal delivery for breech presentation at term still an option? Results of an observational prospective survey in France and Belgium. *Am J Obstet Gynecol*. 2006;194(4):1002-1011.
56. ACOG Committee Opinion no. 745: mode of term singleton breech delivery. *Obstet Gynecol*. 2018;132(2):e60-e63.
57. Kotaska A, Menticoglou S. No. 384-Management of breech presentation at term. *J Obstet Gynaecol Can*. 2019;41(8):1193-1205.
58. Vlemmix F, Bergenhenegouwen L, Schaaf JM, et al. Term breech deliveries in the Netherlands: did the increased cesarean rate affect neonatal outcome? A population-based cohort study. *Acta Obstet Gynecol Scand*. 2014;93(9):888-896.
59. Joseph KS, Pressey T, Lyons J, et al. Once more unto the breech: planned vaginal delivery compared with planned cesarean delivery. *Obstet Gynecol*. 2015;125(5):1162-1167.
60. Berhan Y, Haileamlak A. The risks of planned vaginal breech delivery versus planned caesarean section for term breech birth: a meta-analysis including observational studies. *BJOG*. 2016;123(1):49-57.
61. van Loon AJ, Mantingh A, Serlier EK, et al. Randomised controlled trial of magnetic-resonance pelvimetry in breech presentation at term. *Lancet*. 1997;350(9094):1799-1804.
62. Cheng YK, Lao T. Fetal and maternal complications in macrosomic pregnancies. *Res Rep Neonatol*. 2014;4:65-70.
63. Zatuchni GI, Andros GJ. Prognostic index for vaginal delivery in breech presentation at term. *Am J Obstet Gynecol*. 1965;93:237-242.
64. Louwen F, Daviss BA, Johnson KC, Reitter A. Does breech delivery in an upright position instead of on the back improve outcomes and avoid cesareans? *Int J Gynaecol Obstet*. 2017;136(2):151-161.
65. Advanced Maternal and Reproductive Education. *Becoming a Breech Expert (BABE) Course Manual*. 6th ed. Sydney, Australia: Advanced Maternal and Reproductive Education; 2017.
66. Wildschut HIJ, van Belzen-Slappendel H, Jans S. The art of vaginal breech birth at term on all fours. *Clin Case Rep*. 2017;5(2):182-186.
67. Dufour P, Vinatier D, Puech F. The use of intravenous nitroglycerin for cervico-uterine relaxation: a review of the literature. *Arch Gynecol Obstet*. 1997;261(1):1-7.
68. Elgamal EA. Natural history of hydrocephalus in children with spinal open neural tube defect. *Surg Neurol Int*. 2012;3:112.
69. Ratcliffe SD, Baxley EG, Cline MK, Sakornbut EL. *Family Medicine Obstetrics*. 3rd ed. Philadelphia, PA: Mosby Elsevier; 2008.
70. Shoham Z, Blickstein I, Zosmer A, et al. Transverse uterine incision for cesarean delivery of the transverse-lying fetus. *Eur J Obstet Gynecol Reprod Biol*. 1989;32(2):67-70.
71. Vialle R, Piétin-Vialle C, Ilharreborde B, et al. Spinal cord injuries at birth: a multicenter review of nine cases. *J Matern Fetal Neonatal Med*. 2007;20(6):435-440.
72. Gardberg M, Leonova Y, Laakkonen E. Malpresentations—impact on mode of delivery. *Acta Obstet Gynecol Scand*. 2011;90(5):540-542.
73. Royal College of Obstetricians and Gynaecologists. Umbilical Cord Prolapse: Green-top Guideline No. 50. 2014. Available at https://www.rcog.org.uk/globalassets/documents/guidelines/gtg-50-umbilicalcordprolapse-2014.pdf.
74. Usta IM, Mercer BM, Sibai BM. Current obstetrical practice and umbilical cord prolapse. *Am J Perinatol*. 1999;16(9):479-484.
75. Holbrook BD, Phelan ST. Umbilical cord prolapse. *Obstet Gynecol Clin North Am*. 2013;40(1):1-14.
76. Martin JA, Hamilton BE, Osterman MJ, et al. Births: final data for 2013. *Natl Vital Stat Rep*. 2015;64(1):1-65.

77. Martin JA, Hamilton BE, Ventura SJ, et al. Births: final data for 2009. *Natl Vital Stat Rep*. 2011;60(1):1-70.

78. Glinianaia SV, Obeysekera MA, Sturgiss S, Bell R. Stillbirth and neonatal mortality in monochorionic and dichorionic twins: a population-based study. *Hum Reprod*. 2011;26(9):2549-2557.

79. Hofmeyr GJ, Hannah M, Lawrie TA. Planned caesarean section for term breech delivery. *Cochrane Database Syst Rev*. 2015;7(7): CD000166.

80. Rafael TJ, Berghella V, Alfirevic Z. Cervical stitch (cerclage) for preventing preterm birth in multiple pregnancy. *Cochrane Database Syst Rev*. 2014;9(9):CD009166.

81. Brizot ML, Hernandez W, Liao AW, et al. Vaginal progesterone for the prevention of preterm birth in twin gestations: a randomized placebo-controlled double-blind study. *Am J Obstet Gynecol*. 2015; 213(1):82.e1-82.e9.

82. Senat MV, Porcher R, Winer N, et al; Groupe de Recherche en Obstétrique et Gynécologie. Prevention of preterm delivery by 17 alpha-hydroxyprogesterone caproate in asymptomatic twin pregnancies with a short cervix: a randomized controlled trial. *Am J Obstet Gynecol*. 2013;208(3):194.e1-194.e8.

83. Brubaker SG, Pessel C, Zork N, et al. Vaginal progesterone in women with twin gestations complicated by short cervix: a retrospective cohort study. *BJOG*. 2015;122(5):712-718.

84. Collins A, Shennan A. A clinical opinion on how to manage the risk of preterm birth in twins based on literature review. *J Matern Fetal Neonatal Med*. 2016;29(7):1125-1130.

85. Glinianaia SV, Rankin J, Wright C. Congenital anomalies in twins: a register-based study. *Hum Reprod*. 2008;23(6):1306-1311.

86. Day MC, Barton JR, O'Brien JM, et al. The effect of fetal number on the development of hypertensive conditions of pregnancy. *Obstet Gynecol*. 2005;106(5 Pt 1):927-931.

87. LeFevre ML; U.S. Preventive Services Task Force. Low-dose aspirin use for the prevention of morbidity and mortality from preeclampsia: U.S. Preventive Services Task Force recommendation statement. *Ann Intern Med*. 2014;161(11):819-826.

88. Committee on Practice Bulletins—Obstetrics. ACOG Practice Bulletin no. 190: Gestational Diabetes Mellitus. *Obstet Gynecol*. 2018; 131(2):e49-e64.

89. Rauh-Hain JA, Rana S, Tamez H, et al. Risk for developing gestational diabetes in women with twin pregnancies. *J Matern Fetal Neonatal Med*. 2009;22(4):293-299.

90. Cleary-Goldman J, D'Alton ME. Growth abnormalities and multiple gestations. *Semin Perinatol*. 2008;32(3):206-212.

91. Yinon Y, Mazkereth R, Rosentzweig N, et al. Growth restriction as a determinant of outcome in preterm discordant twins. *Obstet Gynecol*. 2005;105(1):80-84.

92. Spong CY, Mercer BM, D'alton M, et al. Timing of indicated late-preterm and early-term birth. *Obstet Gynecol*. 2011;118(2 Pt 1):323-333.

93. Committee on Practice Bulletins—Obstetrics; Society for Maternal–Fetal Medicine. Practice Bulletin no. 169: Multifetal Gestations: Twin, Triplet, and Higher-Order Multifetal Pregnancies. *Obstet Gynecol*. 2016;128(4):e131-e146.

94. Cruikshank DP. Intrapartum management of twin gestations. *Obstet Gynecol*. 2007;109(5):1167-1176.

95. Rossi AC, Mullin PM, Chmait RH. Neonatal outcomes of twins according to birth order, presentation and mode of delivery: a systematic review and meta-analysis. *BJOG*. 2011;118(5):523-532.

96. Hofmeyr GJ, Barrett JF, Crowther CA. Planned caesarean section for women with a twin pregnancy. *Cochrane Database Syst Rev*. 2011;(12):CD006553.

97. Barrett JF, Hannah ME, Hutton EK, et al; Twin Birth Study Collaborative Group. A randomized trial of planned cesarean or vaginal delivery for twin pregnancy. *N Engl J Med*. 2013;369(14):1295-1305.

98. Vacca A. *Handbook of Vacuum Delivery in Obstetric Practice*. 3rd ed. Brisbane, Australia: Vacca Research; 2009.

99. Fox NS, Silverstein M, Bender S, et al. Active second-stage management in twin pregnancies undergoing planned vaginal delivery in a U.S. population. *Obstet Gynecol*. 2010;115(2 Pt 1):229-233.

100. Cornette JMJ, Erkamp JS. Internal Podalic Version and Breech Extraction: Enhancing Realistic Sensations in a Simulation Model. *Obstet Gynecol*. 2018;131(2):360-363.

101. Staat BC, Shields A, Eubanks AA, et al. An alternative to cesarean: a description of external cephalic version in noncephalic presenting twin. *J Matern Fetal Neonatal Med*. 2019;1-5.

102. Whitworth M, Bricker L, Mullan C. Ultrasound for fetal assessment in early pregnancy. *Cochrane Database Syst Rev*. 2015;7(7): CD007058.

Shoulder Dystocia

Learning Objectives

1. Identify the risk factors for shoulder dystocia (SD).
2. Describe a systematic team-based approach to managing SD.
3. Explain the appropriate maneuvers to reduce SD using the HELPER[4] mnemonic.

Introduction

Definition

Shoulder dystocia (SD) is the failure to deliver the fetal shoulder(s) after the delivery of the head when attempts at gentle axial traction are unsuccessful. It may be preceded by the classic *turtle sign* (neonatal face and head retracted up against the maternal perineum). Attempts at standardizing the definition of SD have included a head-to-body delivery time interval of 60 seconds or greater, but the use of any ancillary obstetric maneuvers to affect delivery is a more common definition.[1]

Incidence

The overall incidence of SD varies and ranges from 0.1% to 3% of all deliveries.[2] The reported rate is influenced by the populations studied, definition inconsistency, and provider reporting behavior. The incidence of SD increases with every 500 g (approximately 1.1 lb) of birthweight. When birth weight is greater than 4,500 g (approximately 9.9 lb), the incidence of SD increases 10-fold.[2] Despite the additional risks posed by a macrosomic fetus, more than 50% of cases occur with birth weights of less than 4,000 g (approximately 8.8 lb). Although knowledge of risk factors is important, SD is typically unanticipated.[2]

Risk Factors

Antenatal

Many antenatal and intrapartum factors have been associated with an increased incidence of SD (*Table 1*). When a previous delivery has been complicated by SD, there is an incidence of recurrence of at least 10%.[2] The 2017 American College of Obstetricians and Gynecologists (ACOG) Practice Bulletin on SD states that management of subsequent pregnancies after a delivery complicated by SD should involve early conversations with women and their families, but acknowledges that SD most likely will not recur.[3] There are no reliable predictive risk models for recurrent SD. However, individual patient characteristics, such as estimated fetal weight, gestational age, maternal diabetes or glucose intolerance, and the severity of previous neonatal injury, should be considered in delivery planning. Universal elective cesarean delivery due to a history of SD is not recommended.[3]

Macrosomia. The overall incidence of fetal macrosomia is increasing, and is associated with a four- to sixfold greater risk of SD than in normal-weight fetuses.[4,5] There is no consensus on the definition of nondiabetic macrosomia; however, the most common definition of macrosomia is a newborn with birth weight greater than 4,500 g (approximately 9.9 lb) in a woman without diabetes. Antepartum management is affected by the lack of a clear definition of macrosomia and the challenge of accurately predicting birth weight. Birth weight estimation by physical examination and sonographic examination have known inaccuracies.[6-8]

Table 1. Risk Factors for Shoulder Dystocia

Antenatal Risk Factors	Labor-Related Risk Factors
Prior delivery with a shoulder dystocia	Assisted vaginal delivery with vacuum or forceps
Gestational or preexisting diabetes	Labor dystocia
Macrosomia >4,500 g (approximately 9.9 lb)	Prolonged second stage of labor
Male fetal sex	
Maternal obesity (BMI >30 kg/m^2)	
Excessive weight gain during pregnancy	
Postdates pregnancy	
Abnormal pelvic anatomy	
Short stature (<5 ft [1.52 m])	

Information from Baxley EG, Gobbo RW. Shoulder dystocia. Am Fam Physician. *2004;69(7): 1707-1714.*

Diabetes. Diabetes (gestational and preexisting) is considered one of the most important risk factors for SD and has been shown to increase the incidence of SD by up to 6 times the baseline risk.[9,10] Fetuses of women with diabetes often are larger, have larger heads and torsos, and higher percentages of body fat, all potentially contributing to an increased risk of SD and birth trauma.[11] In a large, retrospective cohort study of 36,241 singleton pregnancies stratified by gestational diabetes diagnosis, newborns of women with gestational diabetes and fetal birth weights of 4,000 g (approximately 8.8 lb) or more were shown to have increased rates of SD (10.5% versus 1.6%, P <0.001) and Erb palsy (2.6% versus 0.2%, P <0.001).[12] Management of diabetes decreases the incidence of macrosomia and subsequent SD.[13,14]

Intrapartum

Labor. The identification of the labor risk factors for SD remains challenging, and the prediction of SD during the intrapartum phase remains elusive. Most SDs occur without an identifiable major intrapartum risk factor.[15] Furthermore, a large retrospective analysis did not show any reliable risk factors for brachial plexus palsy among deliveries with or without SD.[16]

Assisted vaginal delivery. Assisted vaginal deliveries with vacuum or forceps, and certainly with the sequential use of both instruments, has a significant risk of subsequent SD and fetal neurologic injury.[17,18] The fetus is normally in a position of flexion and shoulder adduction while in the birth canal. A potential mechanism for SD with assisted vaginal deliveries is when the instrument is placed on the fetal vertex and traction is started. The vertex can be pulled away from the body, which causes the neck to extend, head to deflex, and the shoulder to abduct. This produces a greater bisacromial diameter and increases risk of shoulder entrapment by the maternal symphysis pubis.[19-21] Vacuum and forceps deliveries also increase the risk of a brachial plexus injury (odds ratio [OR] = 2.7; 95% confidence interval [CI] = 2.4-3.1, and OR = 3.4; 95% CI = 2.7-4.3, respectively).[19]

Morbidity and Mortality

Injuries to the woman and newborn can have serious short- and long-term consequences. Prevention strategies and appropriate management and training can reduce these complications.

Soft tissue injuries are the most common maternal complication, with increased rates of third- and fourth-degree perineal tears and subsequent potential for rectovaginal fistula formation. SD was associated with a threefold increase in the risk of third- and fourth-degree perineal lacerations in one retrospective review.[22] The use of internal maneuvers (OR = 2.2; 95% CI = 1.2-4.1), an increased number of maneuvers of four or more (OR = 4.7; 95% CI = 1.8-11.8), Woods screw maneuver (OR = 3.1; 95% CI = 1.6-6.2), reverse Woods screw maneuver (OR = 4.9; 95% CI = 1.6-14.3), and removal of the posterior arm (OR = 2.2; 95% CI = 1.1-4.4) were all associated with a significant increase in the likelihood of third- and fourth-degree perineal lacerations.[22]

Postpartum hemorrhage due to uterine atony or birth canal trauma is also more common. Symphyseal diathesis and uterine rupture occur rarely, but symphyseal separation and transient femoral neuropathy have been associated with the McRoberts maneuver.[23]

Neonatal

Brachial plexus palsies are among the most common and concerning complications of SD. Although most newborns recover within 6 to 12 months, up to 20% will have some degree of permanent injury.[24] Two types of nerve injury are generally described: Erb palsy (more common) involving C5 to C6 nerve roots, which is associated with loss of sensation in the arm and paralysis and atrophy of the deltoid, biceps, and brachialis muscles; and Klumpke palsy involving C8 to T1 nerve roots, which manifests as signs of paralysis of intrinsic hand muscles. Clavicular and humeral fractures are additional potential injuries associated with SD, but simple fractures typically heal without deformity or complication.[24]

There is a common perception in the medicolegal community that the provider has a significant role in causing injury while managing a SD via excessive traction and lateral extension exerted on the fetal neck during delivery. Stretching, tearing, or avulsing cervical nerve roots from the spinal cord are potential mechanisms of injury. Simulation models suggest that rapid jerking or pulling movements are likely to cause significant stretch and injury to the brachial plexus and should be avoided.[25]

Although SD at the time of delivery is commonly attributed to brachial plexus palsies, it is

now accepted that in utero positioning of the fetus (with uterine anomalies [eg, lower uterine segment fibroid tumors, intrauterine septum]) and propulsive labor forces (eg, abnormal labor patterns of dilatation and descent) have been shown to cause brachial plexus palsies.[26] Simulation-based studies evaluating mechanical fetal response during deliveries (with and without SD) have shown the following:

- The posterior brachial plexus experiences the greatest stretch during descent in deliveries without SD
- The anterior brachial plexus experiences similarly quantified stretch during deliveries without SD and deliveries with a unilateral (anterior fetal shoulder impaction against maternal symphysis) and bilateral (anterior fetal shoulder impaction against maternal symphysis and posterior fetal shoulder impaction against maternal sacral promontory) SD in simulation[27]
- Force with provider-applied maneuvers during a SD is reduced with the use of maneuvers described in the HELPER[4] mnemonic compared with continuing the delivery in the lithotomy position
- Delivery of the posterior arm correlates with the most significant reduction in delivery force and nerve stretch, but may be more difficult to perform than other maneuvers.[28]

A review of the medical literature and legal case law involving obstetric brachial plexus injury (OBPI) in the United Kingdom presented a template for reviewing the strength of evidence for clinical negligence in OBPI.[25] This review found evidence-based criteria that predicts which cases of SD are more likely due to propulsive or labor forces and which are more likely due to iatrogenic causes (*Table 2*).[25] However, other studies have disputed the claim that anterior OBPIs are purely iatrogenic.[29]

Birth asphyxia and neonatal encephalopathy due to prolonged delivery time are other potential neonatal injuries associated with SD. The time interval from the delivery of the fetal head to the resolution of the SD and delivery of the fetus that is considered safe is not clear. After the fetal head has delivered during a SD, umbilical cord compression between the fetal body and the maternal pelvis can occur and result in fetal hypoxemia and metabolic acidosis. If compression occurs and there are significant delays in resolving the SD and delivering the fetus, it can result in permanent neurologic damage or death.[29]

One study showed that SD resulted in statistically significant, but clinically insignificant, reductions in mean umbilical artery blood gas parameters when compared with the mean arterial pH in all vaginal deliveries in their institution (7.23 ± 0.082 versus 7.27 ± 0.069, P <0.001).[29] Surprisingly, among the group of 44 patients with SD with recorded intervals, increasing head-to-body delivery interval was not correlated with a lower pH (P = 0.9), higher pCO_2 (P = 0.496), or base deficit (P = 0.618). The time for SD resolution did not correlate with lower 5-minute Apgar scores.[29] Comparing cases of SD stratified by the number of maneuvers (ranging from one to three), no significant differences in umbilical artery pH were shown using pH thresholds of 7.10 and 7.00.[26] As the number of maneuvers required for delivery increased from one to three, the rates of cord pH less than 7.20 were 25.6%, 28.6%, and 25%, respectively. Finally, cord pO_2, pCO_2, and base excess were also comparable among the groups.[26]

A study that specifically evaluated neonatal brain injury as an outcome measure showed that brain injuries were associated with significantly prolonged head-shoulder delivery intervals (10.6 ± 3.0 minutes versus 4.3 ± 0.7 minutes, P = 0.03), and that a head-shoulder delivery interval

Table 2. Mechanisms of Injury in Obstetric Brachial Plexus Injuries

Propulsion Injury	Iatrogenic Injury
Posterior arm injured	Anterior arm injured
No shoulder dystocia	Shoulder dystocia
Up-to-date training	Lack of recent training
Appropriate protocol followed and all maneuvers correctly performed	Incorrect maneuvers/persistence with an ineffective maneuver
No evidence of excess traction	Evidence of excess traction
Correct number of clinicians	Insufficient number of clinicians
Precipitous second stage of labor	Use of fundal pressure
Temporary injury	Permanent injury

Adapted from Draycott T, Sanders C, Crofts J, Lloyd J. A template for reviewing the strength of evidence for obstetric brachial plexus injury in clinical negligence claims. Clin Risk. *2008;14(3):96-100.*

threshold of 7 minutes or more had a sensitivity and specificity of 67% and 74%, respectively, in predicting brain injury.[30] Two 2011 studies examined the risks of severe acidosis (pH <7) and hypoxic ischemic encephalopathy based on head-to-body delivery intervals. For head-to-body delivery intervals less than 5 minutes, the risks were 0.5% and 0.5%; for head-to-body delivery intervals of 5 minutes or greater, the risks were 5.9% and 23.5%. Fifty-seven percent of newborns with depression had head-to-body delivery intervals greater than 4 minutes.[31,32]

During SD, it has empirically been thought that fetal acidemia results from umbilical cord compression. Compression of the fetal neck resulting in cerebral venous obstruction, excessive vagal stimulation, and bradycardia may also contribute to severe clinical deterioration that is out of proportion to the duration of hypoxia.[32]

Given the lack of scientific data concerning the effect of SD on fetal pH levels, there is no clearly established point at which irreversible brain injury can be predicted. However, the important clinical conclusion is that most fetuses with SD can tolerate a delay in delivery, which allows the care team to communicate effectively while executing maneuvers in a calm, organized, and safe manner. It is reasonable to assume that the risk of permanent central neurologic dysfunction may be associated with prolongation of the head-shoulder delivery interval greater than 5 to 7 minutes in a previously uncompromised fetus. After the cord pH level is less than 7, the likelihood of fetal brain asphyxia and neurologic impairment increases.[31] This time interval should be more than adequate to deliver the majority of fetuses with SD using the maneuvers described in this chapter.

Prevention

Risk factor modification before and during pregnancy may be the best way to prevent SD. Although it is commonly accepted that optimizing maternal fitness, weight gain, and control of blood glucose measurements affect the overall health and well-being of the woman and fetus starting labor, there is little direct evidence to support this. The odds of predicting a macrosomic fetus using standard ultrasonography fetal biometry extrapolations in an uncomplicated pregnancy are modest at best. Sonographic estimates of fetal birth weights vary by as much as 22% to 37% between predicted and actual, and are similar to the variance of an experienced provider's estimation (12% and 36%).[33] With these current limitations in prediction accuracy, suspected macrosomia alone is not an indication for induction and rarely an indication for primary cesarean delivery in pregnancies that are not complicated by diabetes.

Although it is commonly thought that induction of labor (IOL) at term versus expectant management for suspected fetal macrosomia in women with and without diabetes may decrease actual birth weight and prevent escalation to the 90th percentile and thereby decrease SD, definitive evidence is still needed, particularly in the population without diabetes. ACOG guidelines recommend consideration of cesarean delivery in women with and without diabetes for fetuses with estimated fetal weights greater than 4,500 g (approximately 9.9 lb) and 5,000 g (approximately 11 lb), respectively.[34] Historically, IOL in women at term has not been shown to decrease the incidence of SD.[35]

Similarly, there is insufficient evidence to establish a threshold of estimated fetal weight that would mandate cesarean delivery.[33] Despite the lack of evidence, most providers in the United States follow the ACOG recommendation that a planned cesarean delivery for SD prevention may be a reasonable management option in pregnancies with suspected fetal macrosomia when the estimated fetal weight is greater than 5,000 g (approximately 11 lb) in women without diabetes or greater than 4,500 g (approximately 9.9 lb) in women with diabetes.[3]

Management

Anticipation

The care team should be prepared for SD at every delivery. Anticipation, preparation, and communication start before an emergency occurs. When antenatal or intrapartum risk factors are present, key personnel should be alerted before delivery. The patient and her family should be educated on the possibility of a potentially difficult delivery and can be shown what they might anticipate. The patient's bladder can be emptied and the room cleared of unnecessary clutter to make room for additional personnel and equipment.

Anxious instincts (and training) may prompt providers to provide immediate axial traction on the fetal head after it delivers in an effort to prevent SD. Gentle axial traction on the fetal

head before the anterior shoulder has delivered may force the anterior shoulder behind the pubic symphysis, thereby causing SD by impeding the natural process of descent, restitution, and delivery.[36] There is no published literature that supports the prophylactic use of any of the maneuvers for managing SD. After the head has delivered, the alternative practice of waiting until the next contraction to allow the woman to push out the shoulders after the fetus has restituted will avoid unnecessary and unintentional axial traction on the fetal head. If the shoulders do not deliver with maternal effort, then SD can be diagnosed.[37,38]

The term *gentle downward traction* has been used in previous ALSO and other obstetric literature. This term should now be avoided. *Downward traction* can imply *downward traction toward the floor*, which could cause lateral anterior brachial plexus injury. As understanding of this potential mechanism of injury evolves, a change to using the term and practice of *axial traction* only is recommended. This delineates a safer application of traction that is directly in line with the long axis of the fetus as it lies in the birth canal axis after restituting. Axial traction is applied in alignment with the fetal cervicothoracic spine. The axial component is typically along a vector estimated to be 25 to 45 degrees below the horizontal plane when the laboring woman is in the lithotomy position.[3]

The amount and degree of gentle axial traction required to deliver the anterior shoulder has been studied in simulation settings and is a frequent question among participants in ALSO workshops. One study using force-sensing devices on 29 vaginal deliveries found that a normal or difficult delivery used 47 to 69 N (approximately 4.59 to 6.12 kg [10.5 to 15.5 lb]), respectively, to deliver the fetal shoulders using gentle downward traction on the head, whereas a SD required a traction force up to 100 N (10.2 kg [22.5 lb]).[39]

Computer and simulation models have suggested that force in excess of 100 N can cause significant stretch and injury to the fetal brachial plexus.[40] Most simulation studies find that participants are more likely to exceed this threshold of force if they have not been involved in recent training, are more experienced, or persist with an initial attempt or technique rather than switching to another maneuver.[41]

When anxiety is mitigated, the care team can calmly respond to emergent SD situations with a systematic and thoughtful approach and avoid using early and excessive force. Workshops and simulations, such as those taught in the ALSO Provider Course, are designed to help.

Developing an Institutional Plan

A critical step in addressing the emergency management of SD is to ensure that all relevant hospital personnel are familiar with their roles and responsibilities.[3] An institutional plan can be designed to define the individual roles, and hospital drills or simulations can be conducted regularly to test and rehearse this plan with labor and delivery staff.

On-Site Assistance

After SD is diagnosed, the presence of additional assistants in the delivery room is critical. Team members should be assigned the duties of recording events, obtaining designated equipment and supplies, and notifying the rest of the team of time intervals. Documentation of which shoulder is lying anteriorly, the maneuvers used, and the duration of each maneuver may be valuable information in prompting the provider to proceed to other maneuvers rather than persisting with an ineffective one. All remaining individuals present at the delivery should have clearly defined roles.

Additional Support Staff

A pre-arranged plan should identify team members who are available to respond to the emergency. The team may consist of a family physician, midwife or obstetrician, a pediatrician or neonatologist, one or two labor and delivery nurses to assist with maneuvers, a nurse capable of caring for the newborn, and an anesthesia clinician. At least one other clinician with maternity or neonatal skills should be called immediately if SD occurs. In a large facility, this may be a neonatologist or neonatal nurse practitioner, whereas a family physician, pediatrician, respiratory therapist, or obstetrician may be called at a smaller facility. In some rural areas, this individual could be an emergency department physician or a physician partner. Anesthesia staff should be called to administer drugs as needed. A unit clerk or hospital operator should be available and prepared to assist in summoning appropriate individuals to the delivery room. This may involve developing a priority list of individuals to contact during an obstetric emer-

gency and may be accomplished in part through an overhead page or other notification methods.

Reduction Maneuvers and the HELPER[4] Mnemonic

Shoulder dystocia becomes obvious after the head emerges and then retracts up against the perineum, commonly referred to as the *turtle sign*. Excessive pulling or traction should not be applied to the fetal head or neck, and fundal pressure must be avoided. These maneuvers are unlikely to free the impaction and may cause fetal and maternal injury while wasting valuable time. If gentle axial traction does not relieve the SD after delivery of the head and restitution, the provider must methodically and quickly move to alternate maneuvers to aid in delivery. The woman and nursing staff should be notified of the diagnosis and other personnel may be summoned. Providers should use the maneuver most likely to result in successful delivery.[42]

Since the creation of the ALSO Program, the HELPER mnemonic has served as a training concept for teaching maternity care providers a process for managing SD. The HELPER[4] mnemonic was introduced in 2020 to describe an approach that includes an R[4] step describing several options to relieve SD based on provider assessment. Although there is some data that show removal of the posterior arm is the most effective interior maneuver to relieve SD,[42] it may not always be possible to safely perform in certain situations. The HELPER[4] mnemonic gives the provider a set of options that can be used to manage SD. Persistence in using only one ineffective or difficult maneuver has been associated with an increased incidence of brachial plexus palsy and maternal injury.[18]

Fundal pressure (placing a hand on the top of the maternal fundus and pushing the fetus and uterus toward the vagina) can be harmful and must be avoided. Fundal pressure unsafely duplicates a directional expulsive force that has already failed to deliver the fetal shoulders and serves only to further impact the anterior shoulder behind the symphysis pubis while risking uterine rupture.[32] Documentation after the delivery should clearly state that no fundal pressure was used.

The maneuvers described by the HELPER[4] mnemonic will allow the provider to perform a safe delivery by accomplishing one or more of these effects:

1. Increase the functional size of the bony pelvis
2. Decrease the bisacromial diameter (width of the presenting shoulders)
3. Change the relationship of the shoulders-bisacromial diameter within the bony pelvis.[3,43]

H *= Call for* ***H****elp Early*

After diagnosing a SD, call for help. Activate the pre-arranged plan for personnel to the labor and delivery department respond with necessary equipment. If such a pre-arranged plan has not yet been developed, the appropriate equipment and personnel should be requested. This includes personnel to assist in neonatal resuscitation and anesthesia staff to ensure that appropriate drugs will be available immediately. As different staff enter the room, each should be given and understand their role. Extraneous people in the delivery room can increase patient and staff confusion and anxiety.

E *=* ***E****valuate and* ***E****xplain*

Previously, the first *E* in the HELPER[4] mnemonic stood for *Evaluate for Episiotomy*. The evidence no longer supports performing a routine episiotomy in the early management of SD. One large systematic review found no evidence supporting the use of episiotomy in the prevention or management of SD.[44] SD is a bony impaction, so simply performing an episiotomy will not cause the shoulder to release.

However, the first *E* in the mnemonic reminds the provider to pause and evaluate the clinical situation. This should include determining which shoulder is anterior, calling it out to the nursing staff in the room, and then assigning and explaining roles to the other individuals in the room. This includes determining who will provide suprapubic pressure, if needed, and in what direction, as well as which staff member will track the time and record the maneuvers used. The provider will also need to explain to the woman and her family that the woman needs to discontinue pushing and listen for instruction if a change in maternal position is needed. The provider attending the delivery should direct the activities of the personnel in the room in the same manner as a cardiopulmonary arrest code. It is important that other personnel listen to directions and act as a team. Furthermore, obtaining a cord gas sample and briefing the neonatal clinician are important for guiding the care of the newborn after delivery.[3,42]

L = Legs (McRoberts Maneuver)

The simplicity of the McRoberts maneuver and its proven effectiveness make it an ideal first step in management. The maneuver requires flexing the maternal hips beyond 90 degrees with abduction and external rotation to a position alongside the maternal abdomen. This simulates the squatting position, which increases the inlet diameter. Nurses and the patient's family members present at the delivery can assist with this maneuver. When SD is anticipated, it is helpful to demonstrate this maneuver to the family members beforehand.

The McRoberts maneuver also straightens the lumbosacral lordosis, which flattens the sacral promontory. This procedure simultaneously flexes the fetal spine, which often pushes the posterior shoulder over the sacral promontory and allows it to fall into the hollow of the sacrum. When this occurs, the maternal symphysis may rotate over the impacted shoulder. Finally, the direction of maternal force in this position is perpendicular to the plane of the inlet. When this maneuver is successful, normal traction should deliver the fetus. Delivery should be attempted in this position for approximately 30 seconds. A retrospective study of 250 women showed that use of the McRoberts maneuver alone relieved 42% of SDs.[45]

P = Suprapubic Pressure (Continuous, Then 'Rocking')

If the McRoberts maneuver alone is not sufficient to relieve the SD, suprapubic pressure can be added to effect delivery. An assistant should attempt external manual suprapubic pressure on the maternal abdomen for no longer than 30 seconds while the delivering provider continues gentle axial traction. The assistant's hand should be placed on the maternal suprapubic area over the fetus's anterior shoulder, applying pressure in a firm, constant manner or in a manner similar to cardiopulmonary resuscitation to cause the shoulder to adduct or collapse anteriorly and pass under the symphysis. The suprapubic pressure should be applied from the side of the woman that will allow the heel of the assistant's hand to move in a downward and lateral motion on the posterior aspect of the fetal shoulder. The delivering provider should direct the assistant regarding the correct direction for applying pressure and advise of its effectiveness. Initially, the pressure can be continuous; however, if delivery is not accomplished, a rocking or cardiopulmonary resuscitation motion is recommended to dislodge the shoulder from behind the pubic symphysis. If this procedure fails after 30 seconds, the next procedure should be immediately attempted.

A retrospective study of 276 deliveries with SDs showed that the use of the McRoberts maneuver and/or suprapubic pressure relieved 58% of SDs.[11] As previously discussed, fundal pressure is never appropriate as it only worsens the impaction and can potentially injure the fetus and/or woman.

E = Consider Episiotomy

If the McRoberts maneuver and suprapubic pressure are not effective in delivering the shoulders, the provider's hand should enter the birth canal posteriorly. The provider's hand should assess the position of the shoulders, the amount of room that is present posteriorly, and which shoulder is anterior and easiest to deliver. The provider's hand may then consider whether an episiotomy is necessary to allow additional room for the internal maneuvers. The use of mediolateral rather than midline episiotomy is recommended to decrease the likelihood of third- and fourth-degree perineal lacerations.[46]

The Four Rs: Removal, Rotatory, Roll, and Repeat

At this point, the provider needs to be aware of the time elapsed and progress to the four Rs of the mnemonic to effect delivery. The four Rs represent: *R*emoval of the posterior arm, use of the *R*otatory internal maneuvers (Rubin II, Woods screw, and reverse Woods screw), *R*oll the patient (Gaskin maneuver), and *R*epeat all maneuvers as necessary.

This R^4 portion of the mnemonic requires the provider to attempt the maneuver they judge to be most effective or that they are most comfortable with using. Internal rotatory maneuvers may prove to be equally safe and effective in some scenarios because removal of the posterior arm may not be possible because of the provider's hand size, maternal anatomy, or provider familiarity with the technique. Rolling the patient is especially useful if she is able to actively move into the all-fours position. ALSO advocates that providers first choose the maneuver they think will most likely result in successful delivery. Having familiarity and regular practice with simulation exercises will allow pro-

viders to be able to use each step in the HELPER[4] mnemonic more effectively.

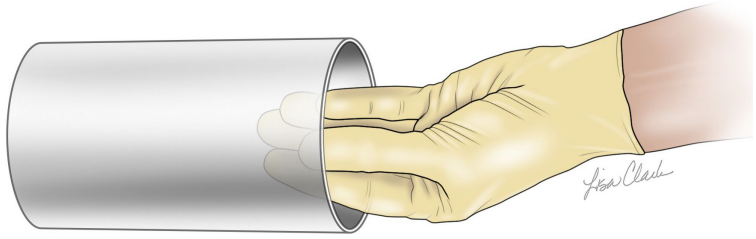
R[1] = Remove the Posterior Arm

Because the risk of neonatal injury increases as the number of additional maneuvers are required, choosing the best single internal maneuver to effect delivery has led to a recommendation that removal of the posterior arm should be the first internal maneuver attempted.[42] Delivery of the posterior arm is now supported as the most effective internal maneuver if the McRoberts maneuver and suprapubic pressure are unsuccessful. In a large retrospective cohort study comparing internal maneuvers, delivery of the posterior arm was shown to be the safest and most effective (84% compared with 24% to 72%).[42]

If the posterior shoulder is not impacted on the maternal sacrum, removal of the posterior arm (a technique described as the Jacquemier or Barnum maneuver) is recommended.[47] As the posterior arm is removed from the birth canal, the bisacromial diameter narrows, resulting in a 20% reduction in shoulder diameter.[42,48] This allows the anterior shoulder to collapse as the fetus descends into the pelvic hollow, which frees the impaction anteriorly.

To perform this maneuver, the provider must insert his or her hand far into the vagina and attempt to locate the posterior arm. If the fetal back is toward the maternal right, the provider inserts their right hand into the vagina at the 6 o'clock position, which should be in front of the fetus's chest. The provider's hand must be lubricated and made small to aid insertion of the whole hand into the posterior vagina. The authors of one study explained this technique by describing how a hand might be inserted into a potato crisp can (*Figure 1*).[40]

Figure 1. Potato Crisp Can Technique



After entering the vagina, confirm the position of the fetus and attempt to locate the front of the chest where the forearm is located. The provider can then apply pressure at the fetus's antecubital fossa to flex the forearm or follow the forearm to grasp at the wrist and deliver by a sweeping motion over the anterior chest wall of the fetus, and then out over the fetal face. Rotation of the fetal trunk to bring the posterior arm anteriorly is sometimes necessary. The fetal upper arm should never be grasped and pulled directly because this may fracture the humerus. If performed correctly, the posterior hand, arm, and shoulder will be delivered. Often, the fetus rotates in a corkscrew manner as the arm is delivered. The anterior shoulder will then rotate backwards under the symphysis and deliver.

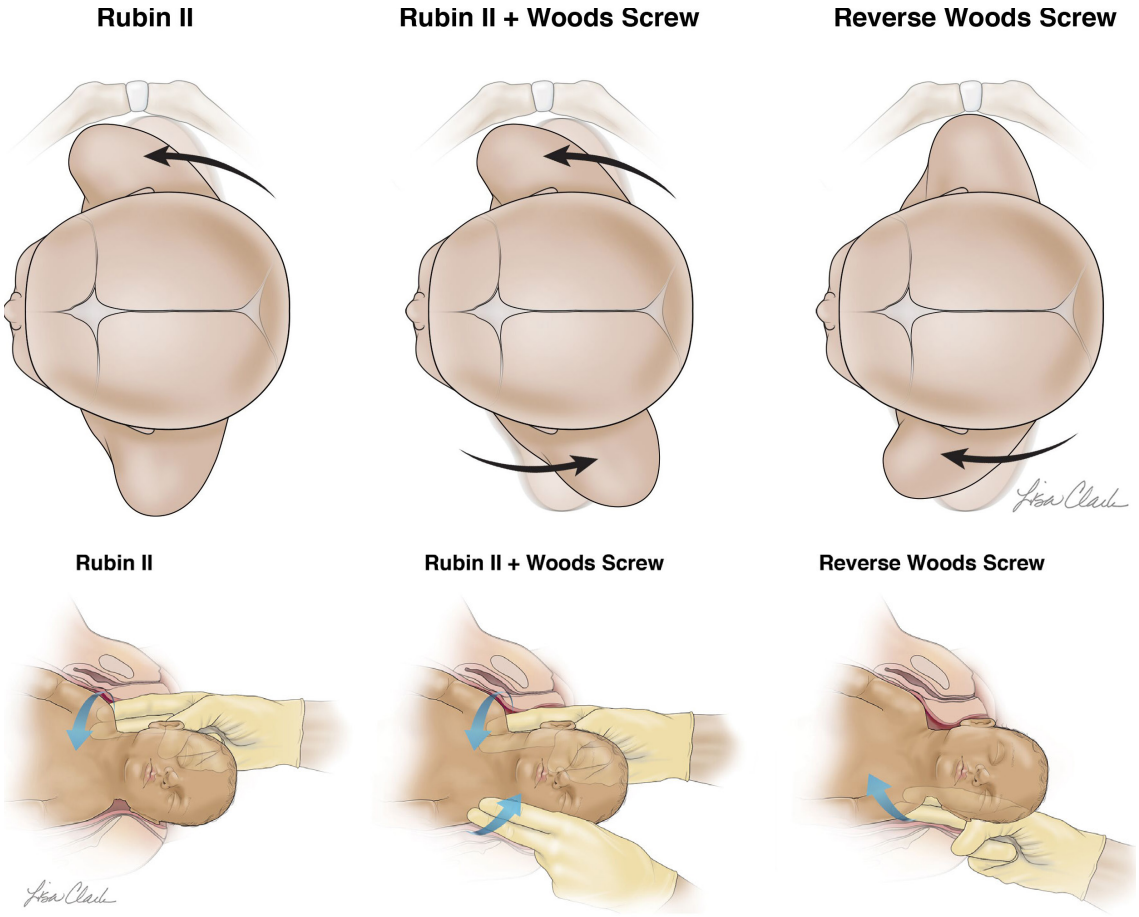
A difficult scenario is sometimes encountered when the arm is located behind the back of the fetus. The provider should suspect this if on entering the vagina the posterior arm cannot be found in front of the fetal trunk. If this occurs, the provider should remove the hand that is in front of the fetal chest and insert the opposite hand behind the fetal back. After the arm is located, it can be displaced under the fetal body and nudged anteriorly to lie in front of the fetal chest. The provider then removes his or her hand from behind the fetal back and reinserts the opposite hand into the birth canal in front of the fetal chest where the arm can now be located. The maneuvers described previously can then be attempted. It is not unusual for a large episiotomy to be cut to accommodate the provider's hands during these maneuvers, and third- and fourth-degree perineal tears are not uncommon.

A 2010 study confirmed that all maneuvers reduce the degree of force and the resultant brachial plexus stretch required to achieve delivery. The greatest effect shown was with delivery of the posterior arm, which yielded a 71% decrease in nerve stretch and an 80% reduction in force.[28] A 2011 study also found that when all maneuvers were evaluated in cases of SD, removal of the posterior arm was associated with the highest success rate of delivering the fetus while not increasing the rate of injury.[42]

R[2] = Rotatory Internal Maneuvers

These maneuvers attempt to manipulate the fetus to rotate the anterior shoulder into an oblique plane under the maternal symphysis. This can be

Figure 2. Rubin Maneuver Converting to a Woods Screw Maneuver



accomplished with the Rubin or Woods screw maneuver.

Many patients may require more than two internal rotary maneuvers.[23] These maneuvers are often the most difficult to understand and can lead to some confusion, but they can be learned effectively with practice. Gaining access for these maneuvers is best achieved using a posterior approach by making use of the space in the sacral hollow. This will allow the provider to perform internal maneuvers using two fingers or potentially the whole hand.

Rubin maneuvers. In 1964, Alan Rubin, described two maneuvers, now typically referred to as the Rubin I and Rubin II maneuvers.[49] The first maneuver (Rubin I) is to rock the fetus's shoulders from side to side once or twice by pushing on the woman's lower abdomen just above her pubic bone. This is the *P* or pressure component of the HELPER[4] mnemonic, which has evolved to applying pressure in a firm, consistent manner or in a manner similar to cardiopulmonary resuscitation. The Rubin II maneuver consists of inserting the first two fingers of one hand vaginally behind the anterior fetal shoulder and pushing the shoulder anteriorly toward the fetal chest. Because there is often insufficient room to insert a hand directly behind the impacted anterior shoulder, it is recommended that the provider insert their hand behind the posterior shoulder where there is more space. After the hand is inside the birth canal, it is slid over the fetal back to the anterior shoulder and pressure is applied (*Figure 2*). This pressure will adduct, or collapse, the fetal shoulder girdle and reduce its diameter. The method recommended by ALSO refers to pressure behind the anterior shoulder as in the Rubin II and can also be termed the anterior Rubin. The McRoberts maneuver can still be applied during this maneuver and may help facilitate its success.

Modified Woods screw maneuver. If the Rubin maneuver is unsuccessful, the Woods

screw maneuver can be attempted. First described in 1943, the Woods screw maneuver calls for the provider to use the opposite hand to approach the posterior shoulder from the front of the fetus and rotate the shoulder toward the symphysis in the same rotatory direction as the provider had been attempting with the Rubin II maneuver (*Figure 2*).[50] ALSO recommends that the provider keep their fingers or hand on the posterior aspect of the impacted anterior shoulder as it has been in position for the Rubin maneuver, and then slide two fingers of the opposite hand in front of the posterior shoulder. The combination of the two maneuvers used concurrently may be more successful than the Woods screw maneuver alone. With this movement, the fetus's shoulders rotate and deliver much like the turning of a screw. The Woods screw maneuver may require an episiotomy to provide room for posterior manipulation, whereas the Rubin II maneuver typically does not.[51]

Reverse Woods screw maneuver. If the Rubin or Woods maneuvers fail, the reverse Woods screw (also called the posterior Rubin maneuver) may be tried. The fingers of the hand that had been on the anterior aspect of the posterior shoulder during the Woods screw maneuver should be removed from the vagina. The fingers of the opposite hand, which have been on the posterior aspect of the anterior shoulder, are slid behind the scapula of the posterior shoulder. After the hand is in place, the provider may attempt to rotate the fetus in the opposite direction as the Woods screw maneuver. This rotates the fetal shoulders out of the impacted position and into an oblique plane from which they can deliver.

There has been significant confusion about these maneuvers, and even leading obstetric texts have described them differently.[52] These maneuvers can be difficult to perform, particularly when the anterior shoulder is partially wedged underneath the symphysis. Sometimes it is necessary to push the posterior or anterior shoulder, back into the pelvis slightly to accomplish the maneuvers.

Whichever internal maneuver is chosen or attempted, it is important to recognize that all of these have been shown in mechanical simulation models to provide a much lower degree of fetal neck rotation, brachial plexus stretch, and amount of force required to accomplish a difficult delivery compared with the McRoberts manuever.[53] This highlights the importance of avoiding excessive traction forces on the fetal neck. Maneuvers that focus on rotating the fetal body core or reducing the bisacromial diameter will cause less stretch on the brachial plexus.

R^3 = Roll the Patient

The Gaskin maneuver (also called the all-fours maneuver) is a safe, rapid, and effective technique for the reduction of SD. Named for Ina May Gaskin, the traditional midwife who first described it, the maneuver requires the patient to roll from the existing supine or dorsal lithotomy position to an all-fours position.[54] The precise mechanism by which this maneuver acts to relieve SD is unknown, but it has been shown that the pelvic diameters increase when laboring women switch from the dorsal recumbent position.[3,55] The maneuver is included here as R^3, although there is less evidence to support its effectiveness when compared with removal of the posterior arm and the rotatory internal maneuvers.[3,42]

X-ray studies indicate that pelvic measurements are least favorable for delivery in the dorsal lithotomy position. By rotating to the all-fours position, the true obstetric conjugate increases by as much as 1 cm and the sagittal measurement of the pelvic outlet increases up to 2 cm.[56] The fetal shoulder often dislodges during the act of turning from the supine to all-fours position, indicating that this movement alone may be sufficient to allow enough pelvic change to dislodge the impaction. In addition, gravitational forces may aid in releasing the impacted fetal shoulder after the change in position.

The Gaskin maneuver may be difficult for a woman who is fatigued or restricted by intravenous lines, fetal monitors, epidural analgesia, or a Foley catheter. The patient will often need assistance to reposition. One facility instructs all patients with epidural analgesia to perform a SD drill and practice moving into the all-fours position.[55] Including this practice as part of prenatal education should be considered. This position may be disorienting to providers who are unfamiliar with it. However, by providing gentle axial traction, the provider can deliver the posterior shoulder first with the aid of gravity in the same direction as if the patient was in the dorsal lithotomy position. The all-fours position is compatible with all intravaginal manipulations for SD, but it

is incompatible with suprapubic pressure. A tip to remember is to *go with gravity first* and provide gentle axial traction to deliver the shoulder closest to the ceiling first. In this case, it is the fetus's posterior shoulder rather than the anterior shoulder. Performing a few routine or simulated deliveries in this position may help the provider be prepared for emergent situations.

R[4] = *R*epeat

The final R in the mnemonic instructs providers to repeat all maneuvers as necessary in whatever order is deemed most likely to effect delivery. This will occur in the lithotomy position or the all-fours position if the patient has been repositioned. Sometimes a change in the position or station of the shoulder occurs and a repeat attempt can be successful.

The order in which these maneuvers are attempted is flexible. However, a logical progression is essential to allow adequate time for each maneuver to potentially accomplish delivery. The suggested length of time for performing each maneuver is meant only as a guideline. Clinical judgment should always guide the progression of the procedures used.

Persistent Shoulder Dystocia After HELPER[4]

Consider Additional Maneuvers

If repeat attempts to resolve SD are unsuccessful, the following techniques have been described as *last resort* or *save* maneuvers.

Posterior axillary sling traction. The technique to deliver the posterior shoulder has been described in two different ways.[57,58] It may prove helpful in cases of SD when the posterior arm is extended or lying under the fetal body. The posterior shoulder is delivered first.

The first variation is for the provider's hand to enter the birth canal along the sacral hollow and deliver the posterior shoulder before delivering the posterior arm. To make access easier to the posterior pelvis, an assistant holds (not pulls) the fetus's flexed head upward toward the anterior shoulder. An episiotomy is helpful if the perineum is too rigid to allow entry. The provider will need to get on one knee below the woman's perineum to use the correct downward vector. The right middle finger is placed into the fetus's posterior axilla from the left side of the pelvis, and the left middle finger is placed into the axilla that lies posteriorly. By using the two middle fingers in the axilla, downward and outward traction is used following the curve of the sacrum. After the shoulder has emerged from the pelvis, the posterior arm can be delivered.[47,59]

Another version of the posterior sling involves the use of a 14 Fr soft plastic suction catheter or firm urinary catheter. This technique was originally described to avoid a symphysiotomy to accomplish the delivery after all other attempted maneuvers failed.[60] More recently, with literature and techniques that focus on delivery of the posterior arm, this technique has been reconsidered.

The provider uses the catheter to create a sling through the axilla of the fetus's posterior shoulder. The provider folds the catheter in half over the operator's right index fingertip and manually inserts the loop around the posterior shoulder and under the axilla. The loop is then retrieved with the left index finger and withdrawn so that the proximal end and tip of the catheter meet to create a sling around the posterior shoulder.[60] Gentle traction is then applied in a posterior downward vector to deliver the posterior shoulder.[61]

If the posterior arm does not deliver, it can often be swept out and delivered because room has been created in the maternal pelvis by delivery of the posterior shoulder. If that maneuver is unsuccessful, the sling can be used with traction to rotate the shoulders.[52] The sling catheter is altered to lateral traction in the direction of the back of the fetus, assisted by pressure behind the anterior shoulder in the opposite direction with the provider's two fingers. As the shoulders rotate, the direction of the sling traction is moved in an arc of 180 degrees to maintain traction at right angles to the axis of the shoulders.[60]

These techniques have their drawbacks. Fracture of the fetus's posterior humerus is frequent.[47] For the woman, tears of the anal sphincter and rectum will typically occur because of the posterior pressure. Posterior axillary sling traction is by no means an original technique and has been described in the past, but it does not seem to have been popularized. However, the success of this technique presupposes that the posterior shoulder is accessible and is not held up by the sacral promontory. If the anterior shoulder is impacted by the symphysis pubis and the posterior shoulder is above the sacral promontory (ie, neither shoul-

der is in the pelvis), only cephalic replacement or symphysiotomy is likely to resolve the problem.

The Zavanelli maneuver. The Zavanelli maneuver requires reversal of the cardinal movements of labor: derestitution (internal rotation), flexion, and subsequent manual replacement of the fetal vertex into the vaginal canal, followed by cesarean delivery.[32,62] It may need to be used in rare cases where both shoulders are impacted in the pelvis.[3] Continuous upward pressure is then maintained on the fetal head until cesarean delivery can be accomplished. Induction of uterine relaxation with intravenous or subcutaneous terbutaline or nitroglycerin administered by the oral, sublingual, or IV route is a valuable adjunct to this procedure, and potentially will prevent uterine rupture. Alternatively, musculoskeletal or uterine relaxation can be induced with halothane or another general anesthetic.[32,62] A surgical team, anesthesia clinician, and physicians capable of performing a cesarean delivery must be present before considering cephalic replacement. The Zavanelli maneuver should not be attempted if a nuchal cord has been previously clamped and cut.[23]

Symphysiotomy. Intentional division of the fibrous cartilage of the symphysis pubis under local anesthesia has been more successfully used in developing countries than in North America and Europe. Reports in the United States are related to its use after a failed Zavanelli maneuver.[63] Because the procedure likely takes 2 minutes or more to accomplish, it should be initiated within 5 to 6 minutes of delivery of the fetal head and should only be used when all other maneuvers have failed and cesarean delivery is not possible.[64] Some women who undergo this procedure might experience chronic symphyseal pain due to separation and could potentially have urethral trauma.

Deliberate clavicular fracture and cleidotomy. As techniques evolve and maneuvers are studied, deliberate clavicular fracture with a live fetus is now considered one of the last maneuvers to use. If all other options have failed, this may be required to avoid fetal death. Direct upward pressure on the midportion of the fetal clavicle or even formal cleidotomy with scissors will result in fracture and reduce the shoulder diameters. This technique should be used with great caution and typically be reserved for extreme cases, given the increased risk of injury to the fetal subclavian vessels and lung tissue.[65]

Documentation

Documentation in the medical record after a completed delivery is an essential risk management tool and several studies point to the lack of standardization and adherence in documentation. The use of comprehensive, standardized procedure notes in cases of SD has been widely advocated.[61,66] Furthermore, use of shared standardized team documentation for discrete elements of the event and delivery should be considered. Using the same discrete elements in the medical record for nurse and provider documentation will strengthen the record of events and avoid contradictions.

It is important to record the elapsed time of the SD, the maneuvers used, and the condition of the woman and newborn after the delivery.[67] Terms such as mild, moderate, or severe SD offer insufficient information about the maternity care provided and are less useful in potential legal proceedings. The documentation should also include the other team members present and umbilical cord venous and arterial cord pH, if obtained. In case subsequent nerve palsy develops, it is important to document which arm was impacted against the pubis and on which arm maneuvers were performed.[49] See the *Appendix* for a sample recording document.[69,70]

A retrospective observational study in the United Kingdom compared documentation of SD under three institutional documentation conditions: nonstandardized, written delivery notes; standardized delivery notes after electronic medical record implementation; and standardized delivery notes in the electronic medical record after SD simulation drills.[71] Standardized electronic notes improved documentation, but the addition of SD drills further increased the rate of SD documentation.[71]

Simulation Training and Institutional Plans

Simulation training and repeated practice of emergency drills has been shown to improve performance in the management of simulated obstetric emergencies including SD.[40] Simulation has evolved to include complex high-fidelity models, but low-fidelity models can still be valuable, especially for team training.

One study in Great Britain showed that a standardized training program with simulated deliveries resulted in an increase in successful delivery

rates and reduction in force amplitudes administered by providers.[40]

High-fidelity training pelvises (ie, models with electronic and computer circuitry) have been shown to improve providers' ability to use minimum traction force and reduce the actual incidence of neonatal injury. However, low-fidelity models (eg, those used by the ALSO Program) can provide the communication and team skills needed to successfully manage SD and limit psychological and medicolegal concerns. Training programs should consider the inclusion of patient-actors with mannequins to increase the fidelity of simulation exercises. Additional studies evaluating the effectiveness of ALSO training and methods for measuring traction forces with low-fidelity models are needed to document course effectiveness in providing providers with improved SD management skills.[28,59,72-74]

Summary

Shoulder dystocia is a relatively uncommon and dangerous event that is difficult to predict. The majority of SDs have no antecedent risk factors.[23] Anticipation and preparation are crucial for successful management.

An institutional plan that assigns duties to each member of the team is highly recommended. The recommended management of SD is based on the HELPER[4] mnemonic, which provides a memory guide and a structured framework for action. The elements of the HELPER[4] mnemonic are all effective, and they should be tried in a logical and calm sequence. Practice on a mannequin is an essential aid to providers who are likely to encounter this obstetric emergency. The time allotted to each maneuver and the exact sequence are best determined by the clinical circumstances and the provider's best judgment while incorporating suggested guidelines.

Nursing Considerations: Shoulder Dystocia

- Prepare for shoulder dystocia in all deliveries. Ensure step stools are present in all delivery rooms
- Identify the turtle sign and facilitate a systematic team approach beginning with clear communication
- When shoulder dystocia is confirmed, document time, initiate HELP from other team members, and assist delivering provider
- Facilitate roles/duties for each team member, including neonatal care and equipment
- Anticipate postpartum hemorrhage and identify the last time the bladder was emptied. Establish intravenous access and identify the location of the hemorrhage cart
- Champion an institutional plan and simulation team training

Appendix: Shoulder Dystocia Documentation

Date _____ Time _____

Person completing form _____

Designation _____

Signature _____

Mother's name _____

Date of birth _____

Hospital number _____

Consultant _____

Called for help at:		**Emergency call via switchboard at:**		
Staff present at delivery of head		**Additional staff attending for delivery of shoulders**		
Name	Role	Name	Role	Time arrived

Procedures used to assist delivery	**By whom**	**Time**	**Order**	**Details**	**Reason if not performed**
❑ McRoberts maneuver					
❑ Suprapubic pressure				From maternal **left / right** (circle as appropriate)	
❑ Episiotomy				Enough access / tear present / already performed (circle as appropriate)	
❑ Delivery of posterior arm				**Right / left** arm (circle as appropriate)	
❑ Internal rotational maneuver					
Description of rotation					
Description of traction	Routine (as in normal vaginal delivery)	Other:		Reason if not routine:	
Other maneuvers used					

Mode of delivery of head	Time of delivery of head _____	**Fetal position during dystocia**	
❑ Spontaneous	Time of delivery of baby _____	Head facing maternal **left**	Head facing maternal **right**
❑ Instrumental — vacuum / forceps	Head-to-body delivery interval _____	**Left** fetal shoulder anterior	**Right** fetal shoulder anterior

Birth weight kg	Apgar	1 min:	5 mins:	10 mins:
Cord gases	Art pH:	Art BE:	Venous pH:	Venous BE:
Explanation to parents ❑ Yes By:				

Neonatal assessment at delivery

Assessment by:

Any sign of arm weakness? ❑ Yes ❑ No Any sign of potential bony fracture? ❑ Yes ❑ No Infant admitted to neonatal intensive care unit? ❑ Yes ❑ No	If yes to any of these questions, for review and follow-up by consultant neonatologist.

Information from Stohl HE, Granat A, Ouzounian JG, Miller DA, Jaque J. Comprehensiveness of Delivery Notes for Shoulder Dystocia. Obstet Gynecol. *2014;123(Suppl 5):25S; Dartmouth-Hitchcock. Guidelines: Shoulder Dystocia Documentation. Available at https://med.dartmouth-hitchcock.org/obstetrics_safety/guidelines_shoulder_dystocia_documentation.html.*

References

1. Beall MH, Spong C, McKay J, Ross MG. Objective definition of shoulder dystocia: a prospective evaluation. *Am J Obstet Gynecol*. 1998;179(4):934-937.
2. Ouzounian JG. Shoulder Dystocia: Incidence and Risk Factors. *Clin Obstet Gynecol*. 2016;59(4):791-794.
3. Committee on Practice Bulletins—Obstetrics. Practice Bulletin No 178: Shoulder Dystocia. *Obstet Gynecol*. 2017;129(5):e123-e133.
4. Ju H, Chadha Y, Donovan T, O'Rourke P. Fetal macrosomia and pregnancy outcomes. *Aust N Z J Obstet Gynaecol*. 2009;49(5):504-509.
5. Bjørstad AR, Irgens-Hansen K, Daltveit AK, Irgens LM. Macrosomia: mode of delivery and pregnancy outcome. *Acta Obstet Gynecol Scand*. 2010;89(5):664-669.
6. Wong SF, Chan FY, Cincotta RB, Oats JJ, McIntyre HD. Sonographic estimation of fetal weight in macrosomic fetuses: diabetic versus non-diabetic pregnancies. *Aust N Z J Obstet Gynaecol*. 2001;41(4):429-432.
7. Henriksen T. The macrosomic fetus: a challenge in current obstetrics. *Acta Obstet Gynecol Scand*. 2008;87(2):134-145.
8. Royal College of Obstetrics and Gynaecologists. Green-top Guideline No 42. Shoulder Dystocia. London, England: Royal College of Obstetricians and Gynaecologists; 2012.
9. Malinowska-Polubiec A, Romejko-Wolniewicz E, Szostak O, et al. Shoulder dystocia in diabetic and non-diabetic pregnancies. *Neuroendocrinol Lett*. 2014;35(8):733-740.
10. Dildy GA, Clark SL. Shoulder dystocia: risk identification. *Clin Obstet Gynecol*. 2000;43(2):265-282.
11. McFarland MB, Langer O, Piper JM, Berkus MD. Perinatal outcome and the type and number of maneuvers in shoulder dystocia. *Int J Gynaecol Obstet*. 1996;55(3):219-224.
12. Esakoff TF, Cheng YW, Sparks TN, Caughey AB. The association between birthweight 4000 g or greater and perinatal outcomes in patients with and without gestational diabetes mellitus. *Am J Obstet Gynecol*. 2009;200(6):672.e1-672.e4.
13. Crowther CA, Hiller JE, Moss JR, McPhee AJ, Jeffries WS, Robinson JS; Australian Carbohydrate Intolerance Study in Pregnant Women (ACHOIS) Trial Group. Effect of treatment of gestational diabetes mellitus on pregnancy outcomes. *N Engl J Med*. 2005; 352(24):2477-2486.
14. Gottlieb AG, Galan HL. Shoulder dystocia: an update. *Obstet Gynecol Clin North Am*. 2007;34(3):501-531, xii.
15. Gemer O, Bergman M, Segal S. Labor abnormalities as a risk factor for shoulder dystocia. *Acta Obstet Gynecol Scand*. 1999;78(8): 735-736.
16. Ouzounian JG, Korst LM, Miller DA, Lee RH. Brachial plexus palsy and shoulder dystocia: obstetric risk factors remain elusive. *Am J Perinatol*. 2013;30(4):303-307.
17. Nesbitt TS, Gilbert WM, Herrchen B. Shoulder dystocia and associated risk factors with macrosomic infants born in California. *Am J Obstet Gynecol*. 1998;179(2):476-480.
18. Brimacombe M, Iffy L, Apuzzio JJ, et al. Shoulder dystocia related fetal neurological injuries: the predisposing roles of forceps and ventouse extractions. *Arch Gynecol Obstet*. 2008;277(5):415-422.
19. Gilbert WM, Nesbitt TS, Danielsen B. Associated factors in 1611 cases of brachial plexus injury. *Obstet Gynecol*. 1999;93(4):536-540.
20. Mollberg M, Hagberg H, Bager B, Lilja H, Ladfors L. Risk factors for obstetric brachial plexus palsy among neonates delivered by vacuum extraction. *Obstet Gynecol*. 2005;106(5 Pt 1):913-918.
21. Øverland EA, Vatten LJ, Eskild A. Pregnancy week at delivery and the risk of shoulder dystocia: a population study of 2,014,956 deliveries. *BJOG*. 2014;121(1):34-41.
22. Gauthaman N, Walters S, Tribe IA, Goldsmith L, Doumouchtsis SK. Shoulder dystocia and associated manoeuvres as risk factors for perineal trauma. *Int Urogynecol J Pelvic Floor Dysfunct*. 2016;27(4): 571-577.
23. Baxley EG, Gobbo RW. Shoulder dystocia. *Am Fam Physician*. 2004;69(7):1707-1714.
24. Chauhan SP, Blackwell SB, Ananth CV. Neonatal brachial plexus palsy: incidence, prevalence, and temporal trends. *Semin Perinatol*. 2014;38(4):210-218.
25. Draycott T, Sanders C, Crofts J, Lloyd J. A template for reviewing the strength of evidence for obstetric brachial plexus injury in clinical negligence claims. *Clin Risk*. 2008;14(3):96-100.
26. Gherman RB, Chauhan S, Ouzounian JG, Lerner H, Gonik B, Goodwin TM. Shoulder dystocia: the unpreventable obstetric emergency with empiric management guidelines. *Am J Obstet Gynecol*. 2006; 195(3):657-672.
27. Allen RH, Cha SL, Kranker LM, Johnson TL, Gurewitsch ED. Comparing mechanical fetal response during descent, crowning, and restitution among deliveries with and without shoulder dystocia. *Am J Obstet Gynecol*. 2007;196(6):539.e1-539.e5.
28. Grimm MJ, Costello RE, Gonik B. Effect of clinician-applied maneuvers on brachial plexus stretch during a shoulder dystocia event: investigation using a computer simulation model. *Am J Obstet Gynecol*. 2010;203(4):339.e1-339.e5.
29. Stallings SP, Edwards RK, Johnson JWC. Correlation of head-to-body delivery intervals in shoulder dystocia and umbilical artery acidosis. *Am J Obstet Gynecol*. 2001;185(2):268-274.
30. Ouzounian JG, Korst LM, Ahn MO, et al. Shoulder dystocia and neonatal brain injury: Significance of the head-shoulder interval. *Am J Obstet Gynecol*. 1998;178:S76.
31. Leung TY, Stuart O, Sahota DS, Suen SS, Lau TK, Lao TT. Head-to-body delivery interval and risk of fetal acidosis and hypoxic ischaemic encephalopathy in shoulder dystocia: a retrospective review. *BJOG*. 2011;118(4):474-479.
32. Lerner H, Durlacher K, Smith S, Hamilton E. Relationship between head-to-body delivery interval in shoulder dystocia and neonatal depression. *Obstet Gynecol*. 2011;118(2 Pt 1):318-322.
33. Chauhan SP, Grobman WA, Gherman RA, et al. Suspicion and treatment of the macrosomic fetus: a review. *Am J Obstet Gynecol*. 2005;193(2):332-346.
34. Bryant DR, Leonardi MR, Landwehr JB, Bottoms SF. Limited usefulness of fetal weight in predicting neonatal brachial plexus injury. *Am J Obstet Gynecol*. 1998;179(3 Pt 1):686-689.
35. Gonen O, Rosen DJ, Dolfin Z, Tepper R, Markov S, Fejgin MD. Induction of labor versus expectant management in macrosomia: a randomized study. *Obstet Gynecol*. 1997;89(6):913-917.
36. Locatelli A, Incerti M, Ghidini A, et al. Head-to-body delivery interval using 'two-step' approach in vaginal deliveries: effect on umbilical artery pH. *J Matern Fetal Neonatal Med*. 2011;24(6):799-803.
37. Menticoglou S. Delivering Shoulders and Dealing With Shoulder Dystocia: Should the Standard of Care Change? *J Obstet Gynaecol Can*. 2016;38(7):655-658.
38. Kotaska A, Campbell K. Two-step delivery may avoid shoulder dystocia: head-to-body delivery interval is less important than we think. *J Obstet Gynaecol Can*. 2014;36(8):716-720.

39. Allen R, Sorab J, Gonik B. Risk factors for shoulder dystocia: an engineering study of clinician-applied forces. *Obstet Gynecol*. 1991; 77(3):352-355.
40. Crofts JF, Fox R, Ellis D, Winter C, Hinshaw K, Draycott TJ. Observations from 450 shoulder dystocia simulations: lessons for skills training. *Obstet Gynecol*. 2008;112(4):906-912.
41. Crofts JF, Ellis D, James M, Hunt LP, Fox R, Draycott TJ. Pattern and degree of forces applied during simulation of shoulder dystocia. *Am J Obstet Gynecol*. 2007;197(2):156.e1-156.e6.
42. Hoffman MK, Bailit JL, Branch DW, et al.; Consortium on Safe Labor. A comparison of obstetric maneuvers for the acute management of shoulder dystocia. *Obstet Gynecol*. 2011;117(6):1272-1278.
43. Gurewitsch ED, Kim EJ, Yang JH, Outland KE, McDonald MK, Allen RH. Comparing McRoberts' and Rubin's maneuvers for initial management of shoulder dystocia: an objective evaluation. *Am J Obstet Gynecol*. 2005;192(1):153-160.
44. Sagi-Dain L, Sagi S. The role of episiotomy in prevention and management of shoulder dystocia: a systematic review. *Obstet Gynecol Surv*. 2015;70(5):354-362.
45. Gherman RB, Goodwin TM, Souter I, Neumann K, Ouzounian JG, Paul RH. The McRoberts' maneuver for the alleviation of shoulder dystocia: how successful is it? *Am J Obstet Gynecol*. 1997;176(3): 656-661.
46. American College of Obstetricians and Gynecologists' Committee on Practice Bulletins—Obstetrics. Practice Bulletin No. 165: Prevention and Management of Obstetric Lacerations at Vaginal Delivery. *Obstet Gynecol*. 2016;128(1):e1-e15.
47. Sentilhes L, Sénat MV, Boulogne AI, et al. Shoulder dystocia: guidelines for clinical practice from the French College of Gynecologists and Obstetricians (CNGOF). *Eur J Obstet Gynecol Reprod Biol*. 2016;203:156-161.
48. Poggi SH, Spong CY, Allen RH. Prioritizing posterior arm delivery during severe shoulder dystocia. *Obstet Gynecol*. 2003;101(5 Pt 2): 1068-1072.
49. Rubin A. Management of Shoulder Dystocia. *JAMA*. 1964;189(11): 835-837.
50. Woods CE. A principle of physics as applicable to shoulder delivery. *Am J Obstet Gynecol*. 1943;45(5):796-804.
51. Ramsey PS, Ramin KD, Field CS. Shoulder dystocia. Rotational maneuvers revisited. *J Reprod Med*. 2000;45(2):85-88.
52. Cluver CA, Hofmeyr GJ. Posterior axilla sling traction for shoulder dystocia: case review and a new method of shoulder rotation with the sling. *Am J Obstet Gynecol*. 2015;212(6):784.e1-784.e7.
53. Poggi SH, Allen RH, Patel CR, Ghidini A, Pezzullo JC, Spong CY. Randomized trial of McRoberts versus lithotomy positioning to decrease the force that is applied to the fetus during delivery. *Am J Obstet Gynecol*. 2004;191(3):874-878.
54. Gaskin IM. *Spiritual Midwifery*. 4th ed. Summertown, TN: Book Publishing Company; 2002.
55. Bruner JP, Drummond SB, Meenan AL, Gaskin IM. All-fours maneuver for reducing shoulder dystocia during labor. *J Reprod Med*. 1998;43(5):439-443.
56. Borell U, Fernstrom I. A pelvimetric method for the assessment of pelvic mouldability. *Acta Radiol*. 1957a;47(5):365-370.
57. Menticoglou SM. A modified technique to deliver the posterior arm in severe shoulder dystocia. *Obstet Gynecol*. 2006;108(3 Pt 2): 755-757.
58. Cluver CA, Hofmeyr GJ. Posterior axilla sling traction: a technique for intractable shoulder dystocia. *Obstet Gynecol*. 2009;113(2 Pt 2): 486-488.
59. Crofts JF, Bartlett C, Ellis D, Hunt LP, Fox R, Draycott TJ. Management of shoulder dystocia: skill retention 6 and 12 months after training. *Obstet Gynecol*. 2007;110(5):1069-1074.
60. Taddei E, Marti C, Capoccia-Brugger R, Brunisholz Y. Posterior axilla sling traction and rotation: A case report of an alternative for intractable shoulder dystocia. *J Obstet Gynaecol*. 2017;37(3): 387-389.
61. Crofts JF, Bartlett C, Ellis D, Fox R, Draycott TJ. Documentation of simulated shoulder dystocia: accurate and complete? *BJOG*. 2008; 115(10):1303-1308.
62. Zelig CM, Gherman RB. Modified Zavanelli maneuver for the alleviation of shoulder dystocia. *Obstet Gynecol*. 2002;100(5 Pt 2): 1112-1114.
63. Goodwin TM, Banks E, Millar LK, Phelan JP. Catastrophic shoulder dystocia and emergency symphysiotomy. *Am J Obstet Gynecol*. 1997;177(2):463-464.
64. Gherman R, Gonik B. Shoulder Dystocia. The Global Library of Women's Medicine. 2008. Available at https://www.glowm.com/section_view/heading/Shoulder%20Dystocia/item/137.
65. Hill MG, Cohen WR. Shoulder dystocia: prediction and management. *Womens Health (Lond)*. 2016;12(2):251-261.
66. Clark SL, Belfort MA, Dildy GA, Meyers JA. Reducing obstetric litigation through alterations in practice patterns. *Obstet Gynecol*. 2008;112(6):1279-1283.
67. Clinical Negligence Scheme for Trusts. Maternity: Clinical Risk Management Standards. London: NHS Litigation Authority; 2006. Available at http://www.nhsla.com/NR/ rdonlyres/002DB3DE-F1A1-4153-AFE1-B59A3D743239/0/ CNSTMaternityStandardsApril-2006final.pdf.
68. Woywodt A, Matteson E. Should eponyms be abandoned? Yes. *BMJ*. 2007;335(7617):424.
69. Stohl HE, Granat A, Ouzounian JG, Miller DA, Jaque J. Comprehensiveness of Delivery Notes for Shoulder Dystocia. *Obstet Gynecol*. 2014;123(Suppl 5):25S.
70. Dartmouth-Hitchcock. Guidelines: Shoulder Dystocia Documentation. Available at https://med.dartmouth-hitchcock.org/obstetrics_safety/guidelines_shoulder_dystocia_documentation.html.
71. Nguyen T, Fox NS, Friedman F Jr, Sandler R, Rebarber A. The sequential effect of computerized delivery charting and simulation training on shoulder dystocia documentation. *J Matern Fetal Neonatal Med*. 2011;24(11):1357-1361.
72. Deering S, Poggi S, Macedonia C, Gherman R, Satin AJ. Improving resident competency in the management of shoulder dystocia with simulation training. *Obstet Gynecol*. 2004;103(6):1224-1228.
73. Johannsson H, Ayida G, Sadler C. Faking it? Simulation in the training of obstetricians and gynaecologists. *Curr Opin Obstet Gynecol*. 2005;17(6):557-561.
74. Black RS, Brocklehurst P. A systematic review of training in acute obstetric emergencies. *BJOG*. 2003;110(9):837-841.

Assisted Vaginal Delivery

Learning Objectives

1. Discuss indications and prerequisites for vacuum and forceps use.
2. Discuss pelvic landmarks and define instrument procedures.
3. Explain the A through J mnemonic as they apply to vacuum extraction and forceps assisted delivery.

Introduction

Assisted vaginal delivery via vacuum device or forceps is an important skill for managing the second stage of labor.[1] Labor is a dynamic event, and may require emergent or elective assisted vaginal delivery for indications including concerning fetal heart tones, prolonged second stage of labor, and maternal exhaustion. Situations that require immediate and competent use of vacuum devices or forceps can arise quickly, even in low-risk labors. Skilled birth attendants in all communities should learn how to provide assisted vaginal delivery.

The rate of assisted vaginal delivery has steadily decreased in the United States, from 9% of live births in 1990 to 3.1% in 2015.[2] Vacuum-assisted vaginal deliveries represented 2.58% of deliveries in 2015[3] and forceps deliveries represented 0.56%.[2,4] The decrease in assisted vaginal deliveries has significantly reduced the number of training opportunities for family physicians and obstetricians. A 2007 survey of chief residents in obstetrics and gynecology residency programs in the United States found that only approximately half of senior trainees thought they were competent in the use of forceps, but more than 90% thought they were competent in vacuum delivery.[5] A 2017 international study found that trainees were equally confident in hands-on assisted vaginal delivery skills despite the low numbers of procedures, but lacked decision making skills in determining when to perform assisted vaginal delivery.[6] A 2014 study of US hospitals teaching obstetrics showed that 3.7% hospitals did not perform vacuum-assisted vaginal deliveries, and 38.3% did not perform forceps deliveries.[7]

As the number of assisted vaginal deliveries has decreased in the United States, the cesarean delivery rate has risen, increasing from 20.7% of deliveries in 1996 and peaking at 32.9% in 2009 before decreasing slightly to 31.9% in 2016.[2] A 2004 study of 124 hospitals showed regional differences in rates of assisted vaginal delivery are significant, ranging from 1% to 23% of deliveries.[8] Performing assisted vaginal deliveries when appropriate may have a significant role in preventing a cesarean delivery. Simulation programs can assist in developing and maintaining the manual skills required.[9]

Prevention

Assisted vaginal delivery is a procedure with an inherent complication rate; therefore, it is preferable to use labor management approaches that minimize the need for an assisted vaginal delivery.[10] Factors that may contribute to the need for assisted vaginal delivery include the use of epidural analgesia and dorsal lithotomy positioning in the second stage of labor. Epidural analgesia increases the rate of assisted vaginal delivery.[11] Early versus late epidural analgesia use did not affect assisted vaginal delivery rates.[12] Upright or lateral positioning during the second stage of labor slightly decreases assisted vaginal delivery rates in women without epidural analgesia but slightly increases second-degree perineal laceration and hemorrhage rates.[13] This is not true of upright or lateral positioning during the second stage of labor with epidural analgesia.[14] The continuous presence of a labor support companion is associated with decreased length of labor and decreased likelihood of assisted vaginal delivery.[15] The use of techniques to increase the effectiveness of maternal efforts during the second stage of labor, such as using a bed sheet held by the laboring woman on one end and the provider on the other, can be helpful, especially in women reporting exhaustion (*Figure 1*).[16]

Oxytocin administration in the second stage of labor in women with epidural analgesia does not affect the rate of assisted vaginal delivery or cesarean delivery, though additional studies are warranted.[17] Placing arbitrary limits on the appropriate duration of the second stage of labor can increase the rate of assisted vaginal delivery. Recent guidelines support allowing more time for progress to occur in the first and second stages of labor.[18] Providers may even choose to exceed these guidelines and continue the second stage of labor

Figure 1. Use of a Bed Sheet to Avoid Assisted Vaginal Delivery

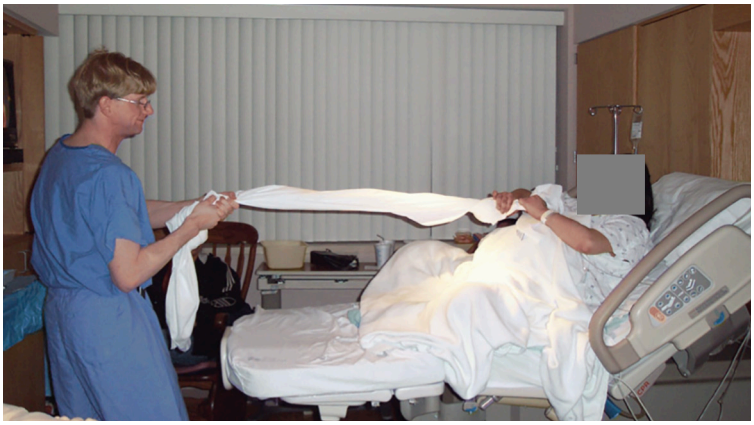


Image from Dresang LT. Using a bed sheet to avoid an assisted delivery. J Am Board Fam Pract. *2004;17(5):394-395.*

if labor is progressing and there is no evidence of fetal compromise.[19]

Predictors of Success and Failure of Assisted Vaginal Delivery

The failure rates of assisted vaginal delivery range from 3.0% to 6.3%.[20] Factors associated with failure in case-control studies include nulliparity, white race, induction of labor, chorioamnionitis, second stage of labor lasting 2 hours or more, fetal occiput posterior (OP) position, low station (versus outlet) at application, larger estimated fetal weight, increasing gestational age, epidural analgesia use, and arrest or maternal exhaustion as indication (compared with fetal indication); however, caution should be used in considering these factors to reliably predict failure.[21,22] Maternal obesity is associated with lower rates of attempted assisted vaginal delivery but a similar rate of success, and is an important consideration when evaluating risks of cesarean delivery in the second stage of labor in this population.[23] Assisted vaginal delivery is also an important consideration in women undergoing labor after cesarean (LAC). In one study, assisted vaginal delivery after LAC was not associated with increased adverse maternal or neonatal outcomes compared with repeat cesarean delivery in the second stage of labor.[24] In another study, assisted vaginal delivery after LAC was associated with a slight increase in maternal morbidity related to perineal lacerations, but a lower rate of neonatal adverse outcomes compared with repeat cesarean delivery.[25]

Instruments

Originally, vacuum devices had a rigid metal cup with a separate suction catheter attached laterally and connected to a foot-operated pedal. Modern vacuum cups can be soft or rigid and are available in many different shapes and sizes. Rigid, plastic posterior cups (eg, Kiwi OmniCup [*Figure 2B*], Mityvac M-cup, Bird or O'Neil cups) have been designed for OP and asynclitic deliveries.[10,26] The flatter cup allows for better placement at the flexion point on the fetal head, which is typically much further back in the maternal pelvis in OP position, with the cup placed underneath the perineal skin. Newer devices allow for an assistant to produce suction by hand using a separate device (*Figure 2A*) or a single handheld device (*Figure 2C* and *2D*). In the United States, the latter are single-use, disposable devices.

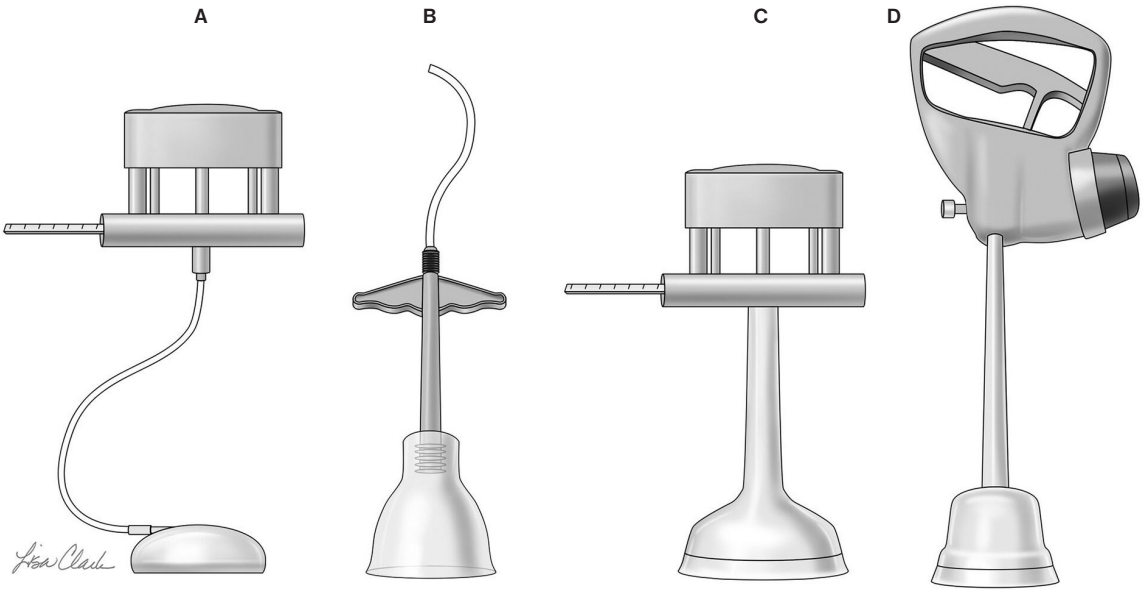
A Cochrane review of 32 studies including 6,597 women undergoing assisted vaginal deliveries showed that forceps achieved a higher rate of vaginal delivery compared with vacuum-assisted delivery.[27] The review also found that a rigid metal cup was more effective for achieving vacuum-assisted delivery than a soft plastic cup, but their use increased the risk of fetal injury.[27] The specific vacuum device used in an ALSO Provider Course may vary based on local practice patterns; however, familiarity with a plastic posterior cup for OP and asynclitic deliveries as well as a soft, bell-shaped cup is encouraged.

There are many types of forceps that are suited for different uses (eg, Piper, Elliot, Kielland). Simpson forceps are adaptable forceps that are available in most labor and delivery departments and are suitable for use with large, molded fetal heads. The ALSO Provider Course uses Simpson forceps to teach outlet delivery. Piper forceps are used in breech deliveries. Kielland forceps are for rotation and should only be used by physicians trained and experienced in the use of rotational forceps. Whereas Simpson forceps have a fenestration, Tucker-McLane forceps do not. Smaller Simpson forceps (ie, *baby Simpsons*) may be used for preterm assisted vaginal delivery.

Indications and Prerequisites for Assisted Vaginal Delivery

The major indications for assisted vaginal delivery are prolonged second stage of labor, maternal indications (eg, maternal conditions such as car-

Figure 2. Types of Vacuum Devices



Examples of different vacuum devices; the cups can vary in shape and size. (A) Some devices use a rigid plastic cup that is disc-shaped and modeled after the original Bird posterior cup. (B) Newer devices allow an assistant to manually generate suction using a separate device. (C and D) Other self-contained, handheld devices allow the user to manually generate suction.

diac disease), and fetal intolerance of the second stage of labor.

Prolonged Second Stage of Labor

A prolonged second stage of labor was redefined in 2012. Previous cutoffs have underestimated the normal duration of the second stage of labor.[28] Updated definitions are described in *Table 1* and allow more time for a woman to deliver than previous guidelines, assuming no evidence of fetal compromise.[19,29,30] See the *Labor Dystocia* chapter for additional information.

There are many causes of a prolonged second stage of labor—some are more safely managed with assisted vaginal delivery than others. Prolonged descent because of soft tissue resistance or the effects of epidural analgesia is less concerning than descent that is slowed because of relative cephalopelvic disproportion (ie, bony pelvis-fetal disproportion), malposition (eg, OP, occiput transverse), or malpresentation (assisted delivery of fetuses in face-mentum anterior presentation is only performed using forceps).[27]

Factors associated with maternal exhaustion include starting to push too early in the labor process and the absence of a labor support companion.[15] Although newer guidelines allow for a longer second stage of labor, the provider may offer assisted vaginal delivery before a prolonged second stage of labor occurs if it is clear that progress is not being made due to exhaustion and other interventions have been already attempted.[19,29,30]

Analgesia may interfere with maternal pushing efforts.[29] Discontinuing epidural analgesia in the second stage of labor to assist with pushing is a

Table 1. Intervals for Defining Prolonged Second Stage (95th Percentile)

Parity	Without Regional Anesthetic	With Regional Anesthetic
Nullipara	3 hours	4 hours
Multipara (parous)	2 hours	3 hours

Information from Cheng YW, Hopkins LM, Caughey AB. How long is too long: Does a prolonged second stage of labor in nulliparous women affect maternal and neonatal outcomes? Am J Obstet Gynecol. *2004;191(3):933-938.*

common practice; however, there is no evidence that this practice decreases the risk of assisted delivery, and the increased degree of pain may be difficult for the woman to tolerate.[31]

Maternal Indications

Intensive Valsalva maneuvers may be contraindicated in patients with cardiorespiratory or intracranial disease, requiring the use of instrumentation in the second stage of labor to achieve delivery.[10] Forceps are commonly chosen in this situation because vacuum delivery requires maternal effort. However, vacuum-assisted vaginal delivery after passive descent with epidural analgesia may also benefit women with concerning cardiopulmonary conditions by reducing the duration of the second stage of labor.[10,32]

Concerning Fetal Status

Concerning fetal statuses include evidence of immediate or impending fetal compromise, such as Category III and some Category II fetal heart rate tracings that do not improve with conservative measures, or evidence of abruption during the second stage of labor.[10] See the *Intrapartum Fetal Surveillance* chapter for guidelines on fetal heart rate tracing interpretation.

Prerequisites for Assisted Vaginal Delivery

Conditions that must exist before an assisted vaginal delivery should be attempted include:[1,10]

1. Vertex presentation of the head with pelvic engagement and position determined
2. Complete cervical dilation
3. Amniotic membrane rupture
4. Lack of suspicion of severe cephalopelvic disproportion
5. Acceptable shoulder dystocia risk
6. Willingness to discontinue the procedure if difficulties occur, including a plan for proceeding to cesarean delivery if needed.

A Cochrane review examining antibiotic prophylaxis found that it had no effect on rates of endometritis or length of hospital stay.[33]

Criteria for Instrument Deliveries

Engagement is defined as the passage of the biparietal diameter of the fetal head through the plane of the pelvic inlet. By definition, clinical evidence of engagement is when the leading edge of the fetal skull is at or below the ischial spines. The distance between the ischial spines and the pelvic inlet is typically thought to be greater than the distance between the leading edge of the fetal skull and the biparietal diameter. However, the fetal skull may be elongated and molded with caput formation after vigorous labor.

Zero station does not prove engagement, especially with a posterior presentation or a large degree of molding.[34] The head is engaged when neither the sinciput nor occiput is palpable above the upper border of the pubis symphysis. Because of the difficulties of clinically estimating engagement and confusion surrounding terminology of stations for mid-instrument application, the American College of Obstetricians and Gynecologists (ACOG) reclassified criteria for instrument deliveries as follows:[1]

1. Outlet forceps or vacuum: The fetal skull has reached the pelvic floor and the fetal head is at or on the perineum. The leading point of the fetal skull is greater than 2 cm from the ischial spines (+3 out of 5 station or +2 out of 3 station or greater). The scalp is visible between contractions without separating the labia. The sagittal suture is in the anterior-posterior diameter or in the right or left occiput anterior (OA) or OP position, but not more than 45 degrees from the midline.

2. Low forceps or vacuum: The leading edge of the fetal skull is 2 cm from the ischial spines (+2 out of 3 station or +2 out of 5 station). The head is not on the pelvic floor.

3. Mid forceps or vacuum: The head is engaged, but the leading edge of the skull is less than 2 cm from the ischial spines (less than +2 out of 3 or less than +2 out of 5 station). This procedure is reserved for experts in the application of forceps and vacuum. Most providers would perform cesarean delivery if delivery needed to be expedited and the fetus was less than 2 cm from the ischial spines. Assisted vaginal delivery at less than 2 cm from the ischial spines has been associated with higher rates of severe neonatal and maternal morbidity/mortality compared with cesarean delivery.[35] In contrast, a retrospective study comparing 391 midpelvic assisted vaginal deliveries with 1,747 low or outlet assisted vaginal deliveries did not show increased rates of severe, short-term maternal and neonatal morbidity. In a twin gestation, delivery of a second twin via forceps or vacuum-assisted delivery at 0 or 1 cm from the ischial spines may be appropriate.[36]

4. Rotation: Vacuum-assisted vaginal delivery would not be considered to aid in fetal head rotation, although autorotation may occur during the vacuum procedure. Attempts to purposefully rotate the head with a vacuum may lead to scalp laceration. See the Delivery of an Occiput Posterior Position Fetus With Forceps section of this chapter for specific details on rotation with Kielland forceps.

Choice of Vacuum Versus Forceps

In the United States, the vacuum extractor is the more commonly preferred instrument when assisted vaginal delivery is indicated. However, provider choice may be dependent on training and experience. The vacuum extractor is easier to apply. Vacuum-assisted vaginal delivery teaches the provider to follow the pelvic curve. Less force is applied to the fetal head, although this may be a liability when a rapid delivery is indicated. Vacuum extractor use causes less maternal trauma than forceps, but it is associated with increased rates of cephalohematoma and retinal hemorrhages.[27] Vacuum extraction is more likely to be unsuccessful than forceps extraction,[27] but it is less likely to lead to third- and fourth-degree perineal lacerations.[27,37,38]

The OP position can present a challenging choice. A vacuum-assisted vaginal delivery of a fetus in OP position is less likely to be successful, but there is an increased incidence of third- and fourth-degree perineal lacerations associated with forceps application.[39]

Recent evidence shows forceps and vacuum devices are equivalent in their ability to expedite delivery of the fetus by experienced providers.[40] The relative safety of vacuum extraction versus forceps for assisted vaginal delivery was assessed in a randomized controlled trial of 313 patients. After 5 years of close follow-up, there were no differences in maternal or fetal outcomes.[41]

Application of Vacuum Instruments

The ABCDEFGHIJ mnemonic may be useful for novice providers and provides a systematic approach for all maternity care providers:[42]

A = **A**sk for help, **A**ddress the patient, and is **A**nesthesia adequate?

Patients may be receiving epidural analgesia, which should be sufficient for pain management. If time allows, a pudendal block can be considered in women not receiving analgesia.[43] All patients should verbally consent to the procedure as time allows. The provider should discuss the potential need for an assisted vaginal delivery with patients as part of routine prenatal care and document the discussion. This will allow for prompt consent by the patient if an expedited delivery is required.

B = **B**ladder empty.

Ensure the bladder is not full because this may lead to shoulder dystocia and bladder injury with an instrument delivery. If needed, the provider should perform a straight catheterization.

C = **C**ervix must be completely dilated.

D = **D**etermine position. Consider shoulder **D**ystocia. Review the HELPER[4] mnemonic.

The fetal head position should be assessed continuously throughout the first stage of labor. It becomes increasingly difficult to determine as fetal caput develops during the second stage of labor. In determining position, the provider should remember that:

1. The anterior fontanel is larger and forms a cross or diamond
2. The posterior fontanel is smaller and forms a Y or triangle shape
3. The provider can feel for an ear and assess which way it bends
4. Molding should be assessed when considering dystocia. It is often an indication of the extent of fetal head compression. Molding can be divided into mild, moderate, and severe categories. If the parietal bones are touching but not overlapped at the sagittal suture line, molding is mild. If the parietal bones are overlapping but can be easily reduced to the normal position by finger pressure, molding is considered to be moderate. Severe molding exists when the overlapping of bones cannot be reduced.[44] Severe molding, as is often found with deflexed and asynclitic heads, often leads to dislodgement of the cup, and may increase risk of intracranial injury.[45]

Bedside or handheld ultrasound may provide a more reliable assessment of fetal position. See the *Malpresentations, Malpositions, and Multiple Gestations* chapter for more information.

When considering shoulder dystocia, assisted vaginal delivery should not be attempted if risk seems too great. For example, caution should be practiced in a patient with diabetes with a large fetus and a prolonged labor because of the risk of shoulder dystocia.[43] If a decision is made to proceed

with assisted vaginal delivery, plans can be made for managing shoulder dystocia if it does occur. The patient can be advised of what to expect. A team member can be designated to apply suprapubic pressure. A step stool can be obtained, if needed.

E = **E**quipment ready (eg, suction, cord clamp, instrument table).

The vacuum device should be prepared and an assistant should be available.

F = Apply the cup over the **F**lexion point, and **F**eel for maternal tissue before and after applying suction.

With the suction off, the center of the cup should be applied 3 cm anterior to the posterior fontanel, centering the sagittal suture under the vacuum. The edge of the cup will be over the posterior fontanel (most cups have a diameter of 5 to 7 cm). This point, located in the midline along the sagittal suture, approximately 3 cm in front of the posterior fontanel and approximately 6 cm from the anterior fontanel, is called the flexion point. The flexion point is important in maximizing traction and minimizing cup detachment. Checking for cup placement using the anterior fontanel as the landmark may be easier because the posterior fontanel will be obscured by the cup. The risk of subgaleal hemorrhage increases if the cup edge is placed on the sagittal suture.[46]

Improper application appears to be common with attempted vacuum-assisted delivery[47] and is thought to be a primary factor in unsuccessful attempts to deliver.[48] A finger sweep should be performed before and after application to ensure no maternal tissue is under the cup to minimize trauma and optimize the vacuum's seal. A Cochrane review showed that rapid application of vacuum pressure is preferable to stepwise application because it results in a faster delivery with no change in maternal or neonatal outcomes.[49]

G = **G**entle traction.

Traction should be applied steadily and at right angles to the plane of the cup. Proper axis traction is the most efficient means of affecting progress with the least amount of force. To avoid birth trauma, rocking movements or torque should never be applied to the vacuum device.[48] As the fetal head flexes, passes beneath the symphysis, and begins to extend, the vacuum handle will rise from an approximately horizontal position to an almost vertical position. The position of the bed and the provider should then be evaluated. If the shaft is bent or a rotary force is applied, the vacuum seal will break, resulting in a pop-off.

Traction should be applied only during contractions and combined with maternal pushing efforts. It is optional to decrease pressure between contractions by triggering the vacuum release valve and then repeating the cycle when the next contraction starts. Some providers advocate maintaining pressure between contractions to prevent loss of station.[50,51]

H = **H**alt traction.

Halt the procedure entirely if 20 minutes has passed, if the cup disengages three times, or if there is no progress in three consecutive pulls.[50] One study recommended allowing no more than 10 minutes of vacuum application because the rate of fetal injury increases significantly between durations of 11 to 20 minutes compared with durations of less than 10 minutes.[52] Nulliparity, midstation vacuum attempts, cup dislodgement, and pulls with greater than three contractions have also been associated with increased neonatal complications.[45]

In many cases, cesarean delivery is a safer option than attempting delivery with a second instrument.[50,53] ACOG guidelines advise against the use of sequential instruments except in an emergency when a cesarean delivery is not immediately possible. Failed assisted vaginal delivery followed by cesarean delivery in the second stage of labor has a greater risk of adverse fetal and maternal outcomes compared with cesarean delivery alone, especially in the setting of nonreassuring fetal heart rate tracings.[54]

I = Evaluate for **I**ncision.

Routine episiotomy with every vaginal delivery is not indicated, and midline episiotomy is associated with increased maternal trauma. Episiotomy may be appropriate if there is a compelling indication.[55] Compared with women who have spontaneous vaginal delivery without episiotomy, the odds of a severe (ie, third- or fourth-degree) perineal laceration are increased in women who undergo vacuum-assisted delivery even without episiotomy (odds ratio [OR] = 3.1; 95% confidence interval [CI] = 1.9-4.3).[55] The use of midline episiotomy with vacuum delivery is associated with increased risk of severe perineal lacerations (OR = 13.7; 95% CI = 10.1-17.3).[55] Retrospective cohort studies have shown mediolateral episiotomy to be associated with a five- to 10-fold decrease in severe perineal laceration rates during assisted vaginal delivery.[20,56]

J = Remove the vacuum cup when the **J**aw is delivered.

The modified Ritgen maneuver (placing external upward pressure with a gloved hand on the fetal chin from the coccygeal region) can be used to extend the head in a controlled manner.

Disadvantages of Vacuum-Assisted Delivery

The disadvantages of using vacuum devices for assisted vaginal delivery include:

1. Delivery is expedited only when there is cooperation from the patient with pushing and/or there is minimal cephalopelvic disproportion
2. Proper traction at a right angle to the plane of the vacuum cup is necessary to avoid losing suction
3. There is an increase in the incidence of cephalohematoma.[7,27]

Contraindications to Vacuum-Assisted Delivery

1. Prematurity: Vacuum-assisted vaginal delivery is generally considered inappropriate before 34 weeks' gestation because of the risk of intraventricular hemorrhage. In certain settings, emergent use of a vacuum extractor to aid delivery before 34 weeks' gestation may be necessary and appropriate if no alternative is available. Rates of intraventricular hemorrhage, extraventricular hemorrhage, and brachial plexus injuries are increased with vacuum-assisted delivery. However, overall rates of injury are low and may be confounded by gestational age.[57]

2. A fetus in nonvertex presentation (eg, breech, face, brow, transverse lie).

3. Incomplete cervical dilatation: Application of a vacuum without complete cervical dilation incurs serious risks of cervical laceration and hemorrhage.

4. Suspected cephalopelvic disproportion: This diagnosis is often made after a trial of vacuum-assisted delivery has failed. The provider should use caution when applying a vacuum device to a fetus with suspected macrosomia especially when presenting at a higher station.

5. The fetal head is not engaged.[10]

Care After Vacuum-Assisted Delivery

The woman and newborn should be examined for evidence of birth trauma. Neonatal risks are discussed in the Neonatal Risks Associated With Assisted Vaginal Delivery section below. Localized caput formation or small cephalohematomas can persist up to a week, but typically resolve within hours after delivery.[4] The newborn should be observed closely for hyperbilirubinemia and subgaleal hematoma, because of the slightly increased incidence of these conditions after vacuum-assisted vaginal delivery. If there is concern that scalp swelling may be due to a subgaleal bleed, then serial head circumference measurements, hematocrit testing, and head imaging are indicated along with potential consultation or transfer to neonatology and neurosurgery specialists.[10,57] An operative note in the patient's medical record is indicated. A sample operative note is included in *Table 2*.[10,43,58]

Forceps Delivery

Simpson forceps consist of two interlocking parts, which are referred to as right and left according to the side of the woman's pelvis in which they lie when applied (*Figure 3*). Each forceps has a handle, a shank, a lock, and a blade. The distal end of the blade is called the toe, and the part nearest the shank is the heel of the blade. The blades are curved on the inner-medial side, producing the cephalic curve conforming to the fetal head. The superior and inferior edges of the blades curve in such a way as to reproduce the pelvic curve, which fits in the hollow of the sacrum and conforms to the maternal pelvis.

The ABCDEFGHIJ mnemonic is again used in the application of forceps:

A = **A**sk for help, **A**ddress the patient, and is **A**nesthesia adequate?

Epidural analgesia or a pudendal block is most effective; local anesthesia can be considered.

B = **B**ladder empty?

Ensure the bladder is not full as this can lead to shoulder dystocia and bladder injury with an instrument delivery. If needed, the provider should perform a straight catheterization.

C = **C**ervix must be completely dilated.

D = **D**etermine position of the fetal head. Think about shoulder **D**ystocia.

E = **E**quipment ready (eg, suction, cord clamp, instrument table).

F = **F**orceps ready.

Many providers will coat the forceps blades with surgical lubricant or soapy water for ease of application. The two forceps blades should be articulated and held up to the vaginal introitus by the

Table 2. Operative Note After Vacuum-Assisted Vaginal Delivery

Indication (eg, prolonged second stage of labor, maternal exhaustion, suspicion for immediate fetal compromise): _____

_____

Diabetes: ☐ Yes ☐ No **If yes, pregestational?:** ☐ Yes ☐ No **Taking medications?:** ☐ Yes ☐ No

Clinical examination: _____

Estimated fetal weight: _____ **Fetal station:** _____ out of _____

Position of head (eg, left occiput anterior, right occiput anterior, occiput posterior): _____

Molding (eg, mild, moderate, significant): _____

Preoperative diagnosis — Term intrauterine pregnancy, prolonged second stage of labor with maternal exhaustion, terminal fetal bradycardia, other (specify): _____

Postoperative diagnosis — Term/preterm intrauterine pregnancy, prolonged second stage of labor with maternal exhaustion, terminal fetal bradycardia, other (specify): _____

Vaginal delivery of a term infant, birth weight _____ g, Apgar scores _____ at 1 minute, _____ at 5 minutes and _____ at 10 minutes

Abandoned/unsuccessful vacuum-assisted vaginal delivery attempt ☐

Procedure — Vacuum-assisted vaginal delivery at _____ station

History — A _____ -year-old G _____ P _____ at _____ weeks admitted for _____ . Prenatal risks – _____ . Verbal and/or written consent obtained before procedure: ☐ Yes ☐ No

First stage of labor — _____ hours, _____ minutes. Spontaneous/augmented/induced labor to complete cervical dilatation. Spontaneous/artificial rupture of membranes performed _____ hours before delivery with _____ fluid. Analgesia with _____ .

Second stage of labor — _____ hours, _____ minutes. Description of second stage of labor. Fetal heart tones were _____ . Vacuum-assisted vaginal delivery was offered to the patient for _____ indication. The risks and benefits of vacuum-assisted delivery, including neonatal and maternal trauma, were discussed and verbal/written consent was obtained.

Procedure — _____ anesthesia was used. The bladder was emptied with a straight catheter. On examination, the cervix was completely dilated and the position of the fetus was _____ , station _____ . The _____ vacuum cup was applied with the center of the cup over the flexion point and the edge of the cup over the posterior fontanel. A finger sweep ensured that no maternal tissue was trapped beneath the cup. Pressure was applied and again, a finger sweep ensured no trapped maternal tissue. During a contraction, moderate traction was used in line with the pelvic axis was applied. With _____ pushes, delivery of a _____ g viable infant occurred with Apgar scores of _____ at 1 minute, _____ at 5 minutes and _____ at 10 minutes.

Number of pop-offs: _____ **Number of pulls:** _____ through _____ contractions

Total time of vacuum device application to fetal head: _____ minutes.

Episiotomy needed: ☐ Yes ☐ No **Clear advancement of vertex with pulls:** ☐ Yes ☐ No

Pediatric service present for delivery: ☐ Yes ☐ No **Complications:** ☐ Yes ☐ No

Third Stage — _____ minutes. 10 units of oxytocin administered intramuscularly after delivery of the anterior shoulder. The third stage of labor was actively managed. Placenta was intact and spontaneous, umbilical cord contained three vessels, blood loss less _____ mL, no maternal tears or trauma noted.

Based on inspection of newborn: _____

Placement of cup appropriate: ☐ Yes ☐ No- describe _____

Fetal injury evident: ☐ No ☐ Yes - describe _____

Newborn resuscitation: _____

Cord gases obtained: ☐ No ☐ Yes – describe results _____

Information from Ali U, Norwitz ER. Vacuum-assisted vaginal delivery. Rev Obstet Gynecol. 2009;2(1):5-17; Hook CD, Damos JR. Vacuum-assisted vaginal delivery. Am Fam Physician. *2008;78(8):953-960; Operative vaginal delivery. ACOG technical bulletin number 196-- August 1994 (replaces no. 152, February 1991).* Int J Gynaecol Obstet. *1994;47(2):179-185.*

provider in exactly the position and attitude they will be in after they are applied. Then the blades are disarticulated, and the left blade is taken in the left hand. The left forceps blade is applied first under usual conditions. It is grasped in the left hand with a pencil grip and is applied to the left side of the patient's pelvis, and also to the left side of the fetus (in OA position). The cephalic curve should be inward toward the vulva, and the shank vertical as the application starts.

The blade is applied to the left side of the fetal head (if OA), typically with two fingers of the right hand inserted deeply into the left posterolateral aspect of the vagina to protect the maternal vaginal tissues and to guide the blade into position. The right thumb on the heel of the blade is used to apply the force of application of the left blade rather than the left hand on the handle or shank of the forceps. The forceps blade should slide almost effortlessly into place as the handle of the forceps moves downward in a large lateral arc.

The right forceps handle is then held in the right hand during insertion, and is applied to the right side of the fetal head (if OA) on the patient's right, with the left hand protecting the maternal right pelvis and guiding the blade into position. The locks should articulate if the blades are correctly applied. Depressing the handles slightly to bring the locks together is sometimes helpful.

The following points can be used to assess the adequacy of the forceps application:

1. The posterior fontanel should be midway between the shanks and 1 cm above the plane of the shanks. This ensures the proper flexion of the head to present the narrowest diameter to the pelvis. If the posterior fontanel is higher than 1 cm above the plane of the shanks, then traction will cause extension of the head, present greater fetal diameters to the pelvis, and make the delivery more difficult.

2. The fenestrations should be barely palpable and admit no more than a fingertip. If more than a fingertip is felt, then the blades are not inserted far enough to be below the fetal malar eminence and can potentially injure the fetal cheeks.

3. The lambdoidal sutures should be above and equidistant from the upper or superior surface of each blade. This ensures the sagittal suture is in the midline in between the blades, where it should be to ensure proper forceps application.

To summarize, ensure the forceps are applied correctly by thinking of the phrase "position for safety" (posterior fontanel, fenestration, sutures: lambdoidal and sagittal).

G = **G**entle traction (Pajot maneuver).

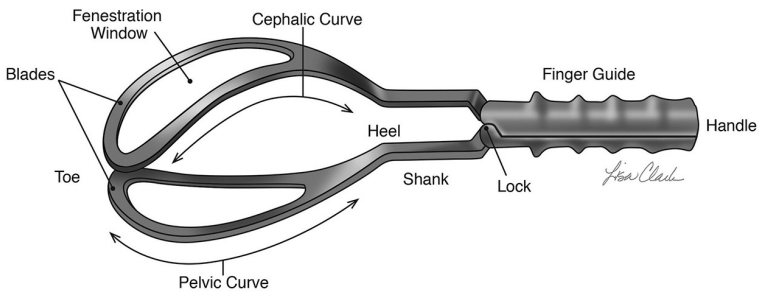
The birth canal curves through the pelvis from the inlet through the outlet, and the curve is often described as an arc or a J-shape when viewed from a sagittal projection. For the provider performing a forceps delivery, the curve starts in a downward direction and then sweeps in a large arc toward the provider, almost completing a 180-degree turn depending on the initial station of the head. The direction of traction on the forceps handles should always be in the same axis as the pelvic curve for any given station of the head. This is the concept of axis traction.

The Pajot maneuver consists of having one of the provider's hands pulling on the forceps handles in the same direction that the handles extend outward and away from the patient. The other hand should be placed on the shanks from above and a downward push exerted. Thus, there are two vectors of force: one approximately horizontal outward and one approximately vertical downward. These vectors summate to a direction of force that is outward and downward. When the fetal skull is more than 2 cm below the ischial spines, this downward and outward force will be in the axis of traction and will bring the head down under the symphysis. Traction in this manner will complete the downward part of the pelvic curve. After the head has come down under the symphysis, the axis of traction begins to stem upward as the head begins to extend under the symphysis.

H = **H**andle elevated to follow the J-shaped pelvic curve.

If the forceps are not removed before the delivery is complete, the shanks will be vertical, or even past vertical as the head extends up and out of the outlet.

Figure 3. Simpson Forceps



I = Evaluate for **I**ncision for episiotomy when the perineum distends.

Episiotomy may be needed if there is inadequate room for the provider to safely guide the forceps into the vagina. However, episiotomy increases the risk of anal sphincter lacerations and is typically not indicated.

J = Remove forceps when the **J**aw is reachable.

The forceps are removed in the reverse order of their application. The right blade is removed first by following the curve of the blade up and over the head anteriorly. The left blade is then removed in a similar fashion. Forceps removal may be accomplished before the head has completely emerged to decrease tension on the perineum.

Delivering a Fetus in Occiput Posterior Position With Forceps

The ALSO Provider Course does not teach forceps rotation. However, forceps delivery of a fetus in OP position is possible. Forceps are applied in the usual manner. The mechanisms of labor are different with this position: extension will not occur, and further flexion of the head is limited by the symphysis pubis. Therefore, horizontal traction is applied to the forceps until the top of the nose appears beneath the symphysis. Slow upward motion then exposes the occiput, followed by downward pressure to deliver the face.[34]

Rotational assisted vaginal delivery is often regarded as a potentially unsafe practice, especially when executed by inexperienced providers. However, studies show great success with these rotational maneuvers and a decreased risk of severe obstetric hemorrhage[59] and perineal laceration.[3,60] The available data regarding the use of Kielland rotational forceps show success in rotating the fetal head to OA position and expediting the vaginal delivery while causing minimal perineal trauma (eg, third- and fourth-degree lacerations) and incurring low rates of postpartum hemorrhage and urinary incontinence.[26,61] Kielland forceps are only used for rotation. When rotation is achieved, they are removed and other forceps (eg, Simpson forceps) are applied to assist delivery. More training for obstetric care providers in this rotational procedure should be considered.

Care After Forceps Delivery

After the fetus is delivered, thorough cervical and vaginal examinations are essential to rule out maternal lacerations. Providers should also be prepared to manage possible postpartum hemorrhage.

The newborn should be examined for evidence of birth trauma (eg, fractured clavicle, brachial plexus injury, cephalohematoma, lacerations-abrasions, facial nerve palsy). Newborns often have visible forceps marks. These typically disappear in 1 to 2 days. These marks should be inspected carefully because they provide evidence of the accuracy of the forceps application.

On the first postpartum day, the delivery can be reviewed with the woman to discuss her perceptions regarding the need for forceps. Any concerns or misconceptions about the delivery should be addressed. A detailed written or dictated operative note similar to the operative note for vacuum-assisted delivery in *Table 2* should be completed.[10,43,58]

Neonatal Risks Associated With Assisted Vaginal Delivery

Assisted vaginal delivery is not a harmless procedure and instruments should be properly applied when indications and prerequisites exist. However, it is important to consider context in neonatal complications of assisted vaginal delivery. Many studies have compared cesarean delivery with assisted vaginal delivery at disparate stations, and the indications for the delivery itself are often contributory and not necessarily overcome by cesarean delivery.[1] One study showed assisted vaginal delivery at low station (fetal skull more than 2 cm below ischial spines) was associated with reduced neonatal risk compared with cesarean delivery, including fewer neonatal intensive care unit admissions, fewer infections, and reduced rates of neonatal respiratory distress.[62] There is no known association between neonatal head injury and duration of vacuum application, number of pulls, or cup dislodgements when standard guidelines are followed.[63]

Assisted vaginal delivery rarely results in intracranial hemorrhage (1 in 650 to 850 deliveries) or neurologic complications (1 in 220 to 385 deliveries).[1] Signs of intracranial hemorrhage include convulsions, lethargy, obtundation, apnea, bulging fontanel, poor feeding, increased irritability, bradycardia, and/or shock.[4]

Vacuum-assisted vaginal delivery is associated with increased risks of cephalohematoma, retinal hemorrhage, and jaundice.[1] Subgaleal hematoma is a rare complication of vacuum delivery that occurs when blood accumulates in the potential space

between the galea aponeurotica and the periosteum of the skull. Signs and symptoms of subgaleal hematomas include diffuse swelling of the head and shock. Subgaleal hematomas can be life threatening and require neonatal intensive care. Cephalohematomas are a less severe collection of blood that accumulates beneath the periosteum of a skull bone (typically a parietal bone) and are characteristically limited to the confines of the cranial bone.[64]

Forceps deliveries carry neonatal risks of facial lacerations, facial nerve palsy, corneal abrasions, external ocular trauma, skull fracture, and intracranial hemorrhage.[1] No differences have been shown between vacuum-assisted and forceps delivery in umbilical cord pH, severe neonatal morbidity, or neonatal death.[27]

Shoulder dystocia rates are increased after assisted vaginal delivery and rates are equivalent between forceps and vacuum device use, according to a meta-analysis of seven studies.[65] Shoulder dystocia in the setting of assisted vaginal delivery is associated with maternal diabetes, chorioamnionitis, arrest as indication, vacuum device use, and estimated fetal weight greater than 4 kg (approximately 8.8 lb), but with only modest predictive ability.[66]

Maternal Risks Associated With Assisted Vaginal Delivery

In the past, maternal risks have been biased by changes in practice over time, disparate levels of provider experience, and lack of statistical power. The comparison group should be cesarean delivery in the second stage of labor as opposed to normal spontaneous vaginal delivery.[1] In large studies, assisted vaginal delivery at low station (+2/3 or greater or +3/5 or greater) is associated with decreased maternal morbidity including reduced rates of blood transfusion and endometritis compared with cesarean delivery in the second stage of labor.[62] These risks must be balanced with the risk of maternal perineal lacerations.

Assisted vaginal delivery is a risk factor for anal sphincter injury; however, it is difficult to separate the procedure from other risk factors associated with its use, including prolonged second stage of labor, fetal size, maternal age and obesity, shoulder dystocia, and episiotomy use.[1] When controlling for these factors, third- and fourth-degree perineal laceration rates were associated with two- and sixfold increases with vacuum device and forceps use, respectively, compared with spontaneous vaginal delivery.[67] However, symptoms of pelvic floor and sexual dysfunction at 1 year postpartum were not different among women undergoing cesarean versus assisted vaginal delivery in the second stage of labor.[68] The station at which assisted vaginal delivery is performed has no known effect on urinary or anal incontinence, sexual dysfunction, or postpartum depression rates.[69,70]

There is preliminary evidence from retrospective cohort studies that mediolateral episiotomy may prevent obstetric anal sphincter injuries (number needed to treat [NNT] = 8 in nulliparous women, 18.3 in primiparous women undergoing vacuum-assisted delivery, 4 in nulliparous women, and 9 in multiparous women undergoing forceps-assisted delivery); however, randomized controlled trials are needed.[56,71] Based on these studies, the Royal College of Obstetricians and Gynaecologists guidelines recommend that mediolateral episiotomy be considered in assisted vaginal deliveries.[72] ACOG guidelines recommend mediolateral episiotomy only when indicated, due to the risks of dyspareunia and long-term perineal pain and uncertain benefits.[1]

Global Perspective

Worldwide, intrapartum complications account for approximately half of all maternal deaths and 2 million stillbirth and neonatal deaths each year.[73] Seventeen of 23 countries in Latin America and the Caribbean, and 40% of sub-Saharan countries neither use nor teach assisted vaginal delivery.[74] Reusable vacuums may be essential for reducing costs, especially in low-resource settings. Forceps, which can be sterilized and reused, may also reduce costs. Assisted vaginal delivery in developing countries may help avoid fistulas resulting from obstructed labor. In addition, using assisted vaginal delivery to avoid potentially unsafe cesarean delivery can decrease the risk of uterine rupture in future pregnancies.[18,75]

In 2012, the World Health Organization began trialing the Odón device, which inserts a polyethylene material around the entire fetal head using an inserter and expedites delivery with provider effort similar to a vacuum-assisted vaginal delivery in terms of force used. To date, the Odón device has only been studied in small trials, but results show the device was easily placed and expedited approximately three-fourths of the deliveries in which it was used. Significant perineal trauma was

rare, although some women experienced cervical laceration. A randomized controlled trial comparing the Odón device with traditional devices used for assisted vaginal delivery is planned.[76,77]

Summary

Although the incidence of assisted vaginal delivery continues to decline, all providers offering labor and delivery care should be familiar with vacuum devices and/or forceps (including indications and complications) and how to properly document any assisted vaginal delivery procedure. Use of assisted vaginal delivery can be essential in emergencies and can help prevent primary cesarean deliveries. Although vacuum devices and forceps are safe to use in most circumstances, there are associated risks of maternal and neonatal complications that must be considered.

Nursing Considerations: Assisted Vaginal Delivery

- Identify the location and types of vacuum devices and forceps used at your institution
- Identify other providers at your institution that may require notification for assisted vaginal deliveries (eg, anesthesia, neonatal intensive care unit)
- Facilitate removal of internal fetal and uterine monitors and initiate external fetal and uterine monitoring
- Communicate the last time the woman's bladder was emptied
- Prepare for the possibility of shoulder dystocia and postpartum hemorrhage: step stools, extra nurses, hemorrhage cart

References

1. Committee on Practice Bulletins—Obstetrics. ACOG Practice Bulletin no. 154: operative vaginal delivery. *Obstet Gynecol*. 2015;126(5): e56-e65.
2. Martin JA, Hamilton BE, Osterman MJ, et al. Births: final data for 2016. *Natl Vital Stat Rep*. 2018;67(1):1-55.
3. Hirsch E, Elue R, Wagner A Jr, et al. Severe perineal laceration during operative vaginal delivery: the impact of occiput posterior position. *J Perinatol*. 2014;34(12):898-900.
4. Doumouchtsis SK, Arulkumaran S. Head trauma after instrumental births. *Clin Perinatol*. 2008;35(1):69-83, viii.
5. Powell J, Gilo N, Foote M, et al. Vacuum and forceps training in residency: experience and self-reported competency. *J Perinatol*. 2007; 27(6):343-346.
6. Crosby DA, Sarangapani A, Simpson A, et al. An international assessment of trainee experience, confidence, and comfort in operative vaginal delivery. *Ir J Med Sci*. 2017;186(3):715-721.
7. Kyser KL, Lu X, Santillan D, et al. Forceps delivery volumes in teaching and nonteaching hospitals: are volumes sufficient for physicians to acquire and maintain competence? *Acad Med*. 2014;89(1):71-76.
8. Clark SL, Belfort MA, Hankins GD, et al. Variation in the rates of operative delivery in the United States. *Am J Obstet Gynecol*. 2007; 196(6):526e1-526e5.
9. Vadnais MA, Dodge LE, Awtrey CS, et al. Assessment of long-term knowledge retention following single-day simulation training for uncommon but critical obstetrical events. *J Matern Fetal Neonatal Med*. 2012;25(9):1640-1645.
10. Ali UA, Norwitz ER. Vacuum-assisted vaginal delivery. *Rev Obstet Gynecol*. 2009;2(1):5-17.
11. Anim-Somuah M, Smyth RM, Jones L. Epidural versus non-epidural or no analgesia in labour. *Cochrane Database Syst Rev*. 2011;(12): CD000331.
12. Sng BL, Leong WL, Zeng Y, et al. Early versus late initiation of epidural analgesia for labour. *Cochrane Database Syst Rev*. 2014;(10): CD007238.
13. Gupta JK, Sood A, Hofmeyr GJ, Vogel JP. Position in the second stage of labour for women without epidural anaesthesia. *Cochrane Database Syst Rev*. 2017;5:CD002006.
14. Kibuka M, Thornton JG. Position in the second stage of labour for women with epidural anaesthesia. *Cochrane Database Syst Rev*. 2017;2:7CD008070.
15. Bohren MA, Hofmeyr GJ, Sakala C, et al. Continuous support for women during childbirth. *Cochrane Database Syst Rev*. 2017;7: CD003766.
16. Dresang LT. Using a bed sheet to avoid an assisted delivery. *J Am Board Fam Pract*. 2004;17(5):394-395.
17. Costley PL, East CE. Oxytocin augmentation of labour in women with epidural analgesia for reducing operative deliveries. *Cochrane Database Syst Rev*. 2013;(7):CD009241.
18. Caughey AB, Cahill AG, Guise JM, Rouse DJ; American College of Obstetricians and Gynecologists; Society for Maternal-Fetal Medicine. Safe prevention of the primary cesarean delivery. *Am J Obstet Gynecol*. 2014;210(3):179-193.
19. Cheng YW, Hopkins LM, Caughey AB. How long is too long: Does a prolonged second stage of labor in nulliparous women affect maternal and neonatal outcomes? *Am J Obstet Gynecol*. 2004;191(3): 933-938.
20. de Vogel J, van der Leeuw-van Beek A, Gietelink D, et al. The effect of a mediolateral episiotomy during operative vaginal delivery on the risk of developing obstetrical anal sphincter injuries. *Am J Obstet Gynecol*. 2012;206(5):404.e1-404.e5.
21. Palatnik A, Grobman WA, Hellendag MG, et al. Predictors of failed operative vaginal delivery in a contemporary obstetric cohort. *Obstet Gynecol*. 2016;127(3):501-506.
22. Verhoeven CJ, Nuij C, Janssen-Rolf CRM, et al. Predictors for failure of vacuum-assisted vaginal delivery: a case-control study. Eur J Obstet Gynecol Reprod Biol. 2016;200:29-34.
23. Ramos SZ, Waring ME, Leung K, et al. Attempted and successful vacuum-assisted vaginal delivery by prepregnancy body mass index. *Obstet Gynecol*. 2017;129(2):311-320.
24. Son M, Roy A, Grobman WA. Attempted operative vaginal delivery vs repeat cesarean in the second stage among women undergoing a trial of labor after cesarean delivery. *Am J Obstet Gynecol*. 2017; 216(4):407.e1-407.e5.
25. Brock CO, Govindappagari S, Gyamfi-Bannerman C. Outcomes of operative vaginal delivery during trial of labor after cesarean delivery. *Am J Perinatol*. 2017;34(8):765-773.
26. Barth WH Jr. Persistent occiput posterior. *Obstet Gynecol*. 2015; 125(3):695-709.
27. O'Mahony F, Hofmeyr GJ, Menon V. Choice of instruments for assisted vaginal delivery. *Cochrane Database Syst Rev*. 2010;(11): CD005455.
28. Spong CY, Berghella V, Wenstrom KD, et al. Preventing the first cesarean delivery: summary of a joint Eunice Kennedy Shriver National Institute of Child Health and Human Development, Society for Maternal-Fetal Medicine, and American College of Obstetricians and Gynecologists Workshop. *Obstet Gynecol*. 2012;120(5):1181-1193.
29. Cheng YW, Shaffer BL, Nicholson JM, Caughey AB. Second stage of labor and epidural use: a larger effect than previously suggested. *Obstet Gynecol*. 2014;123(3):527-535.
30. Myles TD, Santolaya J. Maternal and neonatal outcomes in patients with a prolonged second stage of labor. *Obstet Gynecol*. 2003; 102(1):52-58.
31. Torvaldsen S, Roberts CL, Bell JC, Raynes-Greenow CH. Discontinuation of epidural analgesia late in labour for reducing the adverse delivery outcomes associated with epidural analgesia. *Cochrane Database Syst Rev*. 2004;(4):CD004457.
32. Gei AF, Belfort MA. Forceps-assisted vaginal delivery. *Obstet Gynecol Clin North Am*. 1999;26(2):345-370.
33. Liabsuetrakul T, Choobun T, Peeyananjarassri K, Islam QM. Antibiotic prophylaxis for operative vaginal delivery. *Cochrane Database Syst Rev*. 2017;8:CD004455.
34. Cunningham FG, Leveno KJ, Bloom SL, et al. Forceps and vacuum delivery. In: Cunningham FG, Lenovo KJ, Bloom SL, et al, eds. *Williams Obstetrics*. New York, NY: McGraw-Hill; 2014.
35. Muraca GM, Skoll A, Lisonkova S, et al. Perinatal and maternal morbidity and mortality among term singletons following midcavity operative vaginal delivery versus cesarean delivery. *BJOG*. 2018; 125(6):693-702.
36. Barrett JF. Twin delivery: method, timing, and conduct. *Best Pract Res Clin Obstet Gynaecol*. 2014;28(2):327-338.
37. Caughey AB, Sandberg PL, Zlatnik MG, et al. Forceps compared with vacuum: rates of neonatal and maternal morbidity. *Obstet Gynecol*. 2005;106(5 Pt 1):908-912.
38. Fitzpatrick M, Behan M, O'Connell PR, O'Herlihy C. Randomised clinical trial to assess anal sphincter function following forceps or vacuum assisted vaginal delivery. *BJOG*. 2003;110(4):424-429.

39. Johnson JH, Figueroa R, Garry D, et al. Immediate maternal and neonatal effects of forceps and vacuum-assisted deliveries. *Obstet Gynecol*. 2004;103(3):513-518.
40. Murphy DJ, Koh DK. Cohort study of the decision to delivery interval and neonatal outcome for emergency operative vaginal delivery. *Am J Obstet Gynecol*. 2007;196(2):145.e1-145.e7.
41. Johanson RB, Heycock E, Carter J, et al. Maternal and child health after assisted vaginal delivery: five-year follow up of a randomised controlled study comparing forceps and ventouse. *BJOG*. 2014; 121(Suppl 7):23-28.
42. Bachman JW. Forceps delivery. *J Fam Pract*. 1989;29(4):360.
43. Hook CD, Damos JR. Vacuum-assisted vaginal delivery. *Am Fam Physician*. 2008;78(8):953-960.
44. Vacca A. *Handbook of Vacuum Delivery in Obstetric Practice*. 3rd ed. Brisbane, Queensland, Australia: Vacca Research; 2009.
45. Simonson C, Barlow P, Dehennin N, et al. Neonatal complications of vacuum-assisted delivery. *Obstet Gynecol*. 2007;109(3):626-633.
46. Boo NY, Foong KW, Mahdy ZA, et al. Risk factors associated with subaponeurotic haemorrhage in full-term infants exposed to vacuum extraction. *BJOG*. 2005;112(11):1516-1521.
47. Sau A, Sau M, Ahmed H, Brown R. Vacuum extraction: is there any need to improve the current training in the UK? *Acta Obstet Gynecol Scand*. 2004;83(5):466-470.
48. McQuivey RW. Vacuum-assisted delivery: a review. *J Matern Fetal Neonatal Med*. 2004;16(3):171-180.
49. Suwannachat B, Lumbiganon P, Laopaiboon M. Rapid versus stepwise negative pressure application for vacuum extraction assisted vaginal delivery. *Cochrane Database Syst Rev*. 2012;8(8):CD006636.
50. Yeomans ER. Operative vaginal delivery. *Obstet Gynecol*. 2010; 115(3):645-653.
51. Bofill JA, Rust OA, Schorr SJ, et al. A randomized trial of two vacuum extraction techniques. *Obstet Gynecol*. 1997;89(5 Pt 1):758-762.
52. Murphy DJ, Liebling RE, Patel R, et al. Cohort study of operative delivery in the second stage of labour and standard of obstetric care. *BJOG*. 2003;110(6):610-615.
53. Towner D, Castro MA, Eby-Wilkens E, Gilbert WM. Effect of mode of delivery in nulliparous women on neonatal intracranial injury. *N Engl J Med*. 1999;341(23):1709-1714.
54. Alexander JM, Leveno KJ, Hauth JC, et al; Eunice Kennedy Shriver National Institute of Child Health and Human Development (NICHD) Maternal-Fetal Medicine Units Network (MFMU). Failed operative vaginal delivery. *Obstet Gynecol*. 2009;114(5):1017-1022.
55. Kudish B, Blackwell S, Mcneeley SG, et al. Operative vaginal delivery and midline episiotomy: a bad combination for the perineum. *Am J Obstet Gynecol*. 2006;195(3):749-754.
56. van Bavel J, Hukkelhoven CWPM, de Vries C, et al. The effectiveness of mediolateral episiotomy in preventing obstetric anal sphincter injuries during operative vaginal delivery: a ten-year analysis of a national registry. *Int Urogynecol J Pelvic Floor Dysfunct*. 2018;29(3):407-413.
57. Corcoran S, Daly N, Eogan M, et al. How safe is preterm operative vaginal delivery and which is the instrument of choice? *J Perinat Med*. 2013;41(1):57-60.
58. Operative vaginal delivery. ACOG Technical Bulletin number 196— August 1994 (replaces no. 152, February 1991). *Int J Gynaecol Obstet*. 1994;47(2):179-185.
59. Aiken AR, Alberry MS, Brockelsby JC, Scott JG. Management of fetal malposition in the second stage of labor: a propensity score analysis. *Am J Obstet Gynecol*. 2015;212(3):355e1-355e7.
60. Guerby P, Allouche M, Simon-Toulza C, et al. Management of persistent occiput posterior position: a substantial role of instrumental rotation in the setting of failed manual rotation. *J Matern Fetal Neonatal Med*. 2018;31(1):80-86.
61. Burke N, Field K, Mujahid F, Morrison JJ. Use and safety of Kielland's forceps in current obstetric practice. *Obstet Gynecol*. 2012; 120(4):766-770.
62. Halscott TL, Reddy UM, Landy HJ, et al. Maternal and neonatal outcomes by attempted mode of operative delivery from a low station in the second stage of labor. *Obstet Gynecol*. 2015;126(6):1265-1272.
63. Ghidini A, Stewart D, Pezzullo JC, Locatelli A. Neonatal complications in vacuum-assisted vaginal delivery: are they associated with number of pulls, cup detachments, and duration of vacuum application? *Arch Gynecol Obstet*. 2017;295(1):67-73.
64. Kilani R, Wetmore J. Neonatal subgaleal hematoma: presentation and outcome—radiological findings and factors associated with mortality. *Am J Perinatol*. 2006;23(1):41-48.
65. Dall'Asta A, Ghi T, Pedrazzi G, Frusca T. Does vacuum delivery carry a higher risk of shoulder dystocia? Review and meta-analysis of the literature. *Eur J Obstet Gynecol Reprod Biol*. 2016;204:62-68.
66. Palatnik A, Grobman WA, Hellendag MG, et al. Predictors of shoulder dystocia at the time of operative vaginal delivery. *Am J Obstet Gynecol*. 2016;215:e.1-5.
67. Gurol-Urganci I, Cromwell DA, Edozien LC, et al. Third- and fourth-degree perineal tears among primiparous women in England between 2000 and 2012: time trends and risk factors. *BJOG*. 2013; 120(12):1516-1525.
68. Crane AK, Geller EJ, Bane H, et al. Evaluation of pelvic floor symptoms and sexual function in primiparous women who underwent operative vaginal delivery versus cesarean delivery for second-stage arrest. *Female Pelvic Med Reconstr Surg*. 2013;19(1):13-16.
69. Ducarme G, Hamel JF, Brun S, et al. Sexual function and postpartum depression 6 months after attempted operative vaginal delivery according to fetal head station: A prospective population-based cohort study. *PLoS One*. 2017;12(6):e0178915.
70. Ducarme G, Hamel JF, Brun S, et al. Pelvic floor disorders 6 months after attempted operative vaginal delivery according to the fetal head station: a prospective cohort study. *PLoS One*. 2016;11(12):e0168591.
71. Lund NS, Persson LKG, Jangö H, et al. Episiotomy in vacuum-assisted delivery affects the risk of obstetric anal sphincter injury: a systematic review and meta-analysis. *Eur J Obstet Gynecol Reprod Biol*. 2016;207:193-199.
72. Royal College of Obstetics and Gynaecology. *The Management of Third- and Fourth-Degree Perineal Tears: Greentop Guideline 29*. London: Royal College of Obstetics and Gynaecology; 2015.
73. World Health Organization Odon Device Research Group; Schvartzman JA, Krupitzki H, Betran AP, et al. Feasibility and safety study of a new device (Odón device) for assisted vaginal deliveries: study protocol. *Reprod Health*. 2013;10:33.
74. Ameh CA, Weeks AD. The role of instrumental vaginal delivery in low resource settings. *BJOG*. 2009;116(Suppl 1):22-25.
75. Melah GS, Massa AA, Yahaya UR, et al. Risk factors for obstetric fistulae in north-eastern Nigeria. *J Obstet Gynaecol*. 2007;27(8):819-823.
76. Schvartzman JA, Krupitzki H, Merialdi M, et al; World Health Organization Odon Device Research Group. Odon device for instrumental vaginal deliveries: results of a medical device pilot clinical study. *Reprod Health*. 2018;15(1):45.
77. O'Brien S, Hotton EJ, Lenguerrand E, et al; ASSIST Study Group. The ASSIST Study - The BD Odon Device for assisted vaginal birth: a safety and feasibility study. *Trials*. 2019;20(1):159.

Postpartum Hemorrhage

Learning Objectives

1. Explain the prevention, early recognition and response, and the recommended management of postpartum hemorrhage.
2. Compare quantitative blood loss (QBL) versus estimated blood loss and determine QBL using recommended measurement methods.
3. Describe the components of the "Obstetric Hemorrhage Safety Bundle" created to improve patient safety.

Introduction

Postpartum hemorrhage (PPH) is excessive bleeding after delivery of the fetus, and may occur before or after delivery of the placenta. Providers must learn to recognize excessive bleeding and intervene, preferably before other signs and symptoms of PPH develop (*Table 1*).

Definition, Epidemiology, and Significance

Early PPH is defined as at least 1,000 mL of total blood loss or loss of blood coinciding with signs and symptoms of hypovolemia within 24 hours after delivery of the fetus or intrapartum loss.[1] Primary PPH (occurring within 24 hours of delivery) is more common than secondary PPH (occurring 24 hours to 12 weeks after delivery).[2]

Approximately 5% of obstetric patients will experience PPH.[3-5] PPH is the leading cause of maternal mortality in low-resource countries, and is the cause of 19.7% of maternal mortalities worldwide.[6] Although the proportion of pregnancy-related mortalities due to hemorrhage has been decreasing in the United States, 11.4% of pregnancy-related mortalities from 2006-2010 were due to PPH.[7]

Potential sequelae of PPH include orthostatic hypotension, anemia, and fatigue, which can make breastfeeding and maternal care of the newborn more difficult.[8] PPH may increase the risk of postpartum depression and acute stress reactions.[8,9] Blood transfusion may be necessary, and the associated risks include infection and transfusion reaction.[10] In severe cases, dilutional coagulopathy or disseminated intravascular coagulation (DIC) should be anticipated.

Hemorrhagic shock may lead to Sheehan syndrome (posterior pituitary ischemia with delay or failure of lactation), occult myocardial ischemia, organ failure, or death.[3]

Table 1. Signs and Symptoms of Postpartum Hemorrhage

Signs	Symptoms
Blood loss >1,000 mL	Chest pain
Diaphoresis	Confusion
Hypotension	Dizziness
Hypoxia	Dyspnea
Oliguria	Nausea
Pallor	Palpitations
Syncope	Restlessness
Tachycardia	Weakness

Risk Factors

Risk factors of PPH are listed in *Table 2* and include antepartum and intrapartum conditions. However, 22% of women who develop PPH have no risk factors, so providers must be prepared to respond at every delivery.[4]

Prevention and Early Recognition

In women with mixed degrees of hemorrhage risk, active management reduces mean maternal blood loss at delivery and may reduce the rate of blood loss greater than 500 mL and the need for additional uterotonic agents.[11] The benefits of AMTSL in women at low risk of hemorrhage are less clear.[11]

Active management of the third stage of labor is recommended by the Society of Obstetricians and Gynaecologists of Canada (SOGC),[3] the American College of Obstetricians and Gynecologists (ACOG), the International Federation of Gynecologists and Obstetricians (FIGO), the International Confederation of Midwives (ICM), the Royal College of Obstetricians and Gynae-

Table 2. Postpartum Hemorrhage Risk Factors

Antepartum	Intrapartum	Surgical Interventions
History of PPH (estimated 10% recurrence with subsequent deliveries)	Prolonged labor (first, second, and/or third stage)	Assisted vaginal delivery
Nulliparity	Preeclampsia and related disorders	Cesarean delivery
Grand multiparity (>5 deliveries)	Fetal death	Episiotomy
Coagulopathy (congenital or acquired, including drug use [eg, aspirin, heparin])	Induction or augmentation	
Abnormal placentation	Magnesium sulfate use	
Age >30 years	Chorioamnionitis	
Anemia	Anemia	
Overdistension of the uterus	Dehydration	
Multiple gestation		
Polyhydramnios		
Fetal macrosomia		

PPH = postpartum hemorrhage.

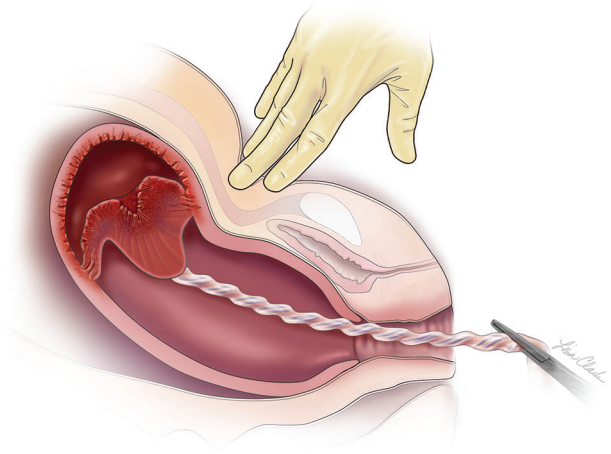
Information from Leduc D, Senikas V, Lalonde AB, et al; Clinical Practice Obstetrics Committee; Society of Obstetricians and Gynaecologists of Canada. Active management of the third stage of labour: prevention and treatment of postpartum hemorrhage. J Obstet Gynaecol Can. *2009;31(10):980-993; Waters JH, Yazer MH. Clinical validation of risk stratification criteria for peripartum hemorrhage.* Obstet Gynecol. *2013;122(1):120-126.*

cologists (RCOG), the Society for Maternal-Fetal Medicine, and the World Health Organization (WHO) for prevention of PPH.[12,13]

Active management of the third stage of labor (AMTSL) includes:[3,11,12,14,15]

1. Administering oxytocin with, or soon after, the delivery of the anterior shoulder. A reduction in the incidence of PPH also occurs if oxytocin is administered after placental delivery[16]

Figure 1. Brandt Maneuver



2. Cutting the cord after a delay of 1 to 3 minutes for infants not requiring resuscitation
3. Controlled cord traction to deliver the placenta. Performing controlled cord traction by grasping the cord with one hand and gently apply traction while simultaneously applying suprapubic (not fundal) pressure with the other hand. This is called the Brandt maneuver (*Figure 1*).
4. Massaging uterus after delivery of the placenta.[19]

Administration of a uterotonic agent is the most important step in reducing PPH.[12,17,18] The benefits of the other steps of AMTSL are less clear.[15,19-21] Earlier definitions of AMTSL did not include transabdominal uterine massage after placental delivery, which is now included in some AMTSL protocols.[19] In addition, initial AMTSL protocols included cutting the umbilical cord immediately after delivery (less than 30 seconds). However, trials have shown that a delay in cord clamping of 1 to 3 minutes has benefits to the newborn without an increase in PPH or neonatal morbidity.[22] These benefits include decreased anemia in preterm and term infants, and decreased intraventricular hemorrhage in very preterm newborns.[23,24]

Oxytocin (10 units intramuscularly [IM] or 5 to 10 units administered as a bolus over 1 to 2 min-

utes intravenously [IV]) is the preferred uterotonic agent for preventing PPH because it is more effective than ergot alkaloids and prostaglandins and has fewer adverse effects.[4] IV oxytocin reduces the risk of severe PPH (number needed to treat [NNT] = 29) and blood transfusion (NNT = 35) compared with IM administration.[25] The IV dosing regimen requires a higher concentration of oxytocin than that used for labor induction or augmentation in many maternity care departments. For example, 10 units of oxytocin in a 50 to 100 mL crystalloid bolus or 30 units of oxytocin in a 500 mL crystalloid administered as a 100 to 150 mL bolus followed by a maintenance infusion would be used to prevent PPH. Using a standard concentration, such as 30 units/500 mL for labor induction, augmentation, AMTSL, and treatment of PPH increases patient safety by reducing drug errors.[25]

Misoprostol has been evaluated for prevention of PPH because of its advantages in low-resource areas. It is inexpensive, heat and light stable, and can be administered without the use of syringes.[26] Misoprostol (oral or sublingual) reduces severe PPH and blood transfusion compared with placebo.[27] However, in a large systematic review, misoprostol was not shown to increase or decrease the risk of maternal mortality compared with placebo or other uterotonic agents.[28] Misoprostol had a greater incidence of adverse effects, including increased shivering and fever, compared with other uterotonic agents.[28] Misoprostol-related fever is typically preceded by shivering; it then follows a typical pattern, starting less than 20 minutes postpartum and peaking at 1 to 2 hours before spontaneously declining over 3 hours. The authors of a meta-analysis recommended that women with this typical manifestation of postpartum fever after peripartum misoprostol administration be monitored initially and treated only if the fever persists beyond 3 hours postpartum.[29]

Misoprostol should be used for prevention of PPH only when oxytocin is not available.[27] The WHO lists misoprostol as an essential drug for preventing PPH, but it is not approved by the US Food and Drug Administration for this indication.[12] A reasonable dose is 600 mcg orally.[28] More research is needed to define the most effective regimens for preventing PPH, especially in low-resource and prehospital settings.

Tranexamic acid has insufficient evidence to support its use for prevention of PPH in routine vaginal deliveries.[30,31]

In addition to oxytocin administered at the time of delivery, specific strategies before, during, and after labor can reduce complications of PPH.

Antenatally, women who are at high risk of invasive placenta (eg, those with prior uterine surgery, prior placenta previa, advanced maternal age, high parity) should undergo ultrasound examination of placental inplantation.[32] Magnetic resonance imaging (MRI) may be useful if the ultrasound is nondiagnostic; however, MRI may be misleading with regard to the presence of uterine invasion.[32,33] Imaging cannot always detect placenta accreta. Women with invasive placenta and others at high risk of PPH (eg, women with anterior placenta previa and a history of cesarean delivery) should deliver at a facility with access to blood products and anesthesia and surgical capabilities. All women should be screened for anemia and treated for reversible causes of anemia (eg, iron deficiency, malaria). Women of African, Southeast Asian, or Mediterranean descent should be offered screening for sickle cell disease or thalassemia by hemoglobin electrophoresis and complete blood count.[34] Providers should identify women who refuse use of blood products (eg, Jehovah's Witnesses) so that the risks and complications of hemorrhage can be discussed and the woman's preferences for prevention and treatment can be fully understood. In some centers, red blood cell salvaging may be available to reduce the need for transfusion.[35]

On hospital admission, women who are at high risk of PPH should have16- to 18-gauge IV lines inserted and undergo baseline laboratory studies, including complete blood count and type and screen or type and cross. The incidence of perineal trauma can be reduced by using perineal warm compresses, using a vacuum device, and restricting episiotomy rather than forceps when assisted vaginal delivery is required, and restricting episiotomy.[36-38] Oxytocin, second-line uterotonic agents (eg, methylergonovine, misoprostol, carboprost), and tranexamic acid should be readily available in the delivery and operating rooms. Because visual estimates of blood loss are often falsely low, quantified blood loss (QBL) should be used to determine blood loss volume.[39] See *Table 3* for suggestions on how to accurately calculate QBL. Vital signs should be assessed, and lochia quantified frequently to detect slow but significant blood loss.

For women at high risk of PPH, cross-matched packed red blood cells (PRBCs) and other blood

products should be readily available in the delivery or operating room. Women with anemia should receive aggressive prevention and treatment of PPH because complications may occur with smaller volumes of blood loss.

Table 3. Tips for Quantification of Blood Loss

Quantification of maternal blood loss is a team effort.

1. Create a list of dry weights for delivery items that may become blood-soaked with directions on how to calculate blood loss.
2. Begin QBL immediately after the infant's birth (prior to delivery of the placenta) and assess and record the amount of fluid collected in a calibrated under-buttocks drape or suction canister. Keep in mind that most of the fluid collected prior to birth of the placenta is amniotic fluid, urine, and feces. If irrigation is used, deduct the amount of irrigation from the total fluid that was collected.
3. Record the total volume of fluid collected in the under-buttocks drape or suction canister.
4. Subtract the pre-placenta fluid volume from the post-placenta fluid volume to more accurately determine the actual blood loss. Keep in mind that most of the fluid collected after the birth of the placenta is blood.
5. Add the fluid volume collected in the drapes and canister to the blood volume measured by weighing blood soaked items to determine the cumulative volume of blood loss or QBL.
6. Weigh all bloodsoaked materials and clots to determine cumulative volume. *1 gram weight = 1 milliliter blood loss volume*
7. The equation used when calculating blood loss of a blood soaked item is WET *Item Gram Weight* – DRY *Item Gram Weight = Milliliters of Blood within the item*

Note. Although a gram is a unit of mass and a milliliter is a unit of volume, the conversion from one to the other is simple.

QBL = quantification of blood loss.

Reprinted from Association of Women's Health, Obstetric and Neonatal Nurses. Quantification of blood loss: AWHONN practice brief number 1. J Obstet Gynecol Neonatal Nurs. *2015;44(1):158-160.*

Table 4. The Four Ts Mnemonic for the Specific Causes of Postpartum Hemorrhage

Four Ts	Specific Cause	Relative Frequency
Tone	Atonic uterus	70%
Trauma	Lacerations, hematomas, inversion, rupture	20%
Tissue	Retained tissue, invasive placenta	10%
Thrombin	Coagulopathies	<1%

Information from Committee on Practice Bulletins-Obstetrics. Practice Bulletin no. 183: Postpartum Hemorrhage. Obstet Gynecol. *2017;130(4):e168-e186; Nyfløt LT, Sandven I, Stray-Pedersen B, et al. Risk factors for severe postpartum hemorrhage: a case-control study.* BMC Pregnancy Childbirth. *2017;17(1):17.*

Diagnosis

Preparation, early recognition, and quick response to excessive blood loss will reduce morbidity associated with primary and secondary PPH.[40] The diagnosis of PPH begins with recognition of excessive bleeding and methodical examination for its cause. The Four Ts mnemonic (Tone, Trauma, Tissue, and Thrombin) can be used to remember specific causes (*Table 4*).

Treatment

Women who are pregnant have increased plasma volume and red cell mass.[2] They are typically healthy and can accommodate mild to moderate blood loss without having signs or symptoms such as orthostasis, hypotension, tachycardia, nausea, dyspnea, oliguria, or chest pain. QBL should be performed in every delivery, and action should be taken before the woman develops symptoms. After excessive blood loss (1,000 mL or greater) is quantified, treatment must be initiated quickly by progressing through the Four Ts mnemonic.[41] Many of the steps in diagnosis and management must be carried out simultaneously (*Figure 2*).

Regardless of the suspected cause of bleeding, additional medical personnel will be needed to assist the delivering provider. Assistants should be directed to start two large-bore IV lines (16- to 18-gauge). When bleeding occurs before placental delivery, attention is directed to its removal and inspection. Manual removal may be required if there is a delay in placental delivery or if it is not intact. Difficulty locating a plane between the placenta and the uterus may signify invasive placenta.[41]

After delivery of the placenta, excessive vaginal bleeding will most often be due to uterine atony (70% of cases). The first maneuver to reduce bleeding is transabdominal (fundal) uterine massage.[1] The lower uterine segment should be supported during vigorous massage to prevent uterine inversion. Oxytocin can be administered next via the (equally effective) IM or IV route.[1,3] If uterine tone does not improve with compression, transabdominal massage (and if necessary, bimanual massage) and oxytocin, additional uterotonic agents can be administered. The antifibrinolytic, tranexamic acid (1 g IV) is recommended if a woman with uncontrolled bleeding is symptomatic, requiring second-line uterotonic agents, or has lost more than 500 mL of blood after vaginal delivery or more than 1,000 mL of blood after cesarean delivery.

Tranexamic acid should be administered as soon as indicated but no later than 3 hours after the onset of bleeding.[42-44] During this time, the genital tract can be explored and lacerations repaired.

If vaginal bleeding persists after uterine atony has been treated and no lacerations or hematomas have been identified, it is useful to explore the uterus (preferably after analgesia administration) to determine if retained placental fragments are responsible for continued bleeding. Uterine exploration also will allow detection of ruptured or partial uterine inversion. Hypotension or shock out of proportion to the amount of blood loss raises the suspicion for concealed hematomas, uterine rupture, or uterine inversion. Anaphylaxis, sepsis, and amniotic fluid or pulmonary embolism also should be considered. Persistent bleeding or lack of clotting may signal coagulopathy, which is sometimes caused by the hemorrhage itself.

Blood loss greater than 1,500 mL requires immediate resuscitation measures using an interdisciplinary team approach, including anesthesia, laboratory, nursing, surgery, and blood bank staff. As part of the initial management of this emergency, providers should perform a primary maternal survey and institute care to support circulation, airway, and breathing (the 'C-A-B'):

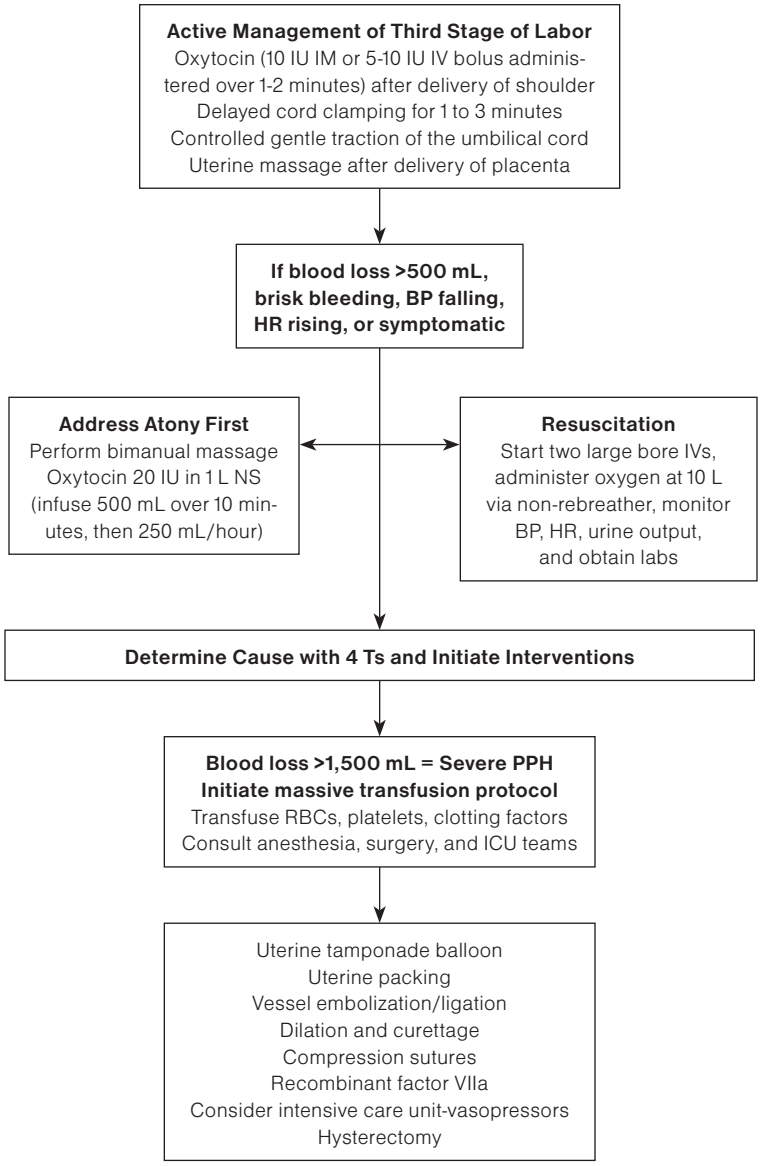
1. Administer IV fluid and possibly blood replacement by starting two large-bore IVs with normal saline or other crystalloid fluids. Elevating the foot of the bed or having an assistant elevate the woman's legs will improve venous return and raise the woman's blood pressure (BP) level
2. Open the airway and administer supplemental oxygen to maintain oxygen saturation of greater than 95%
3. Ventilate the woman with 100% oxygen, if needed.

After the primary maternal survey, obtain stat laboratory tests (type and cross, complete blood count, coagulation studies, and hold a blood tube [red top] for clot evaluation) if not already done when IV access was established. Place a Foley catheter to empty the bladder and monitor urine output. Heart rate and BP should be monitored closely, and times of relevant events (eg, drug and blood product administration, interval QBL measurements) should be documented. Although vital sign changes may be delayed even with significant hemorrhage, the earliest to occur is tachycardia and narrowing pulse pressure. It may be necessary to infuse O-negative blood while waiting for type-specific blood.

Hypothermia worsens coagulopathy and increases peripheral vasoconstriction, which may decrease effectiveness of drugs administered via peripheral IV. Extra, warmed blankets or a fluid warmer can be used to prevent hypothermia.[45]

If available, institute a massive transfusion protocol for any hemorrhage greater than 1,500 mL or

Figure 2. Review of Postpartum Hemorrhage Management



BP = blood pressure; HR = heart rate; ICU = intensive care unit; IM = intramuscular; IU = international units; IV = intravenous; NS = normal saline; PPH = postpartum hemorrhage; RBCs = red blood cells.

Information from various sources.

ongoing symptomatic blood loss.[46] Additional interventions may be needed (see the Massive Transfusion Protocols and Interventions for Intractable Postpartum Hemorrhage sections of this chapter).

In secondary PPH (occurs 24 hours to 12 weeks after delivery), atony is still the most likely cause of bleeding. Bleeding may occur at a slow rate, obscuring the overall volume of blood loss. Endometritis may complicate diagnosis and management. Pelvic ultrasonography or Doppler studies may be used, but nondiagnostic findings are common. Careful curettage may be needed to remove retained tissue.[1]

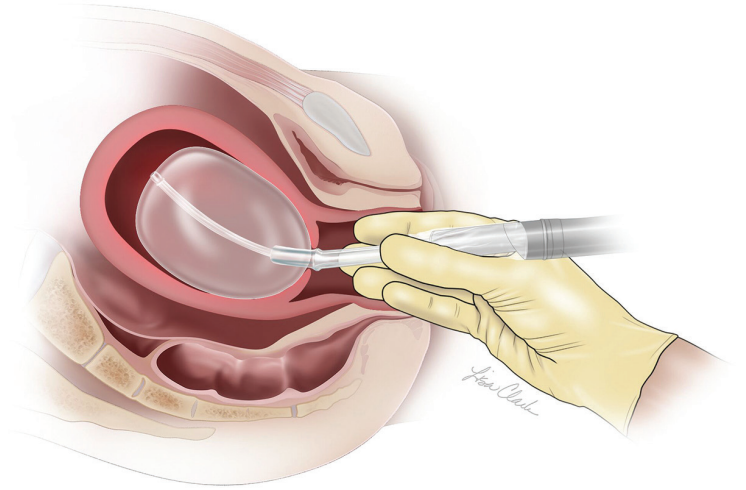
See the *Maternal Resuscitation and Trauma* chapter for recommendations for managing hemorrhage and related emergencies.

Massive Transfusion Protocols

Research involving patients with critical trauma injuries has shown improved survival with use of massive transfusion protocols that recommend infusion of fresh frozen plasma (FFP) and platelets whenever large numbers of PRBCs are needed. The primary goal of a massive transfusion protocol is to expedite release of blood products from the blood bank in ratios that mimic whole blood thereby preventing dilutional coagulopathy. However, site-specific protocols may contain additional steps to bring equipment, nurses, laboratory and blood bank staff, surgeons, and/or other staff with relevant expertise to the bedside. Other protocols may include laboratory parameters for coagulation management.[47]

Typical massive transfusion protocol use ratios of 4 units of FFP and 1 unit of apheresis platelets for every 6 units of PRBCs without waiting for laboratory test results to document coagulopathy.[48] Dilutional coagulopathy may still occur with use of these protocols, so coagulation studies and platelet counts should be obtained frequently and deficiencies corrected with additional FFP, platelets, and/or cryoprecipitate. This can be performed with visoelastic hemostatic assays (VHAs) like thromboelastograph (TEG) or rotational thromboelastometry (ROTEM).[49] Thromboelastometry evaluates clot formation and lysis with a variety of rapid assessment tools to inform providers about specific clotting factor and platelet problems. This allows for targeted use of blood products to reduce cost and transfusion-associated adverse effects.

Figure 3. Uterine Tamponade Balloon Catheter



The uterine tamponade balloon catheter is used for temporary control or reduction of postpartum hemorrhage when conservative management of uterine bleeding is warranted. It is easy to place and rapidly achieves tamponade within the uterine cavity, thereby potentially avoiding a hysterectomy. The tip allows accumulated blood to drain. The balloon of the catheter is inserted into the uterus, making certain that the entire balloon is inserted past the cervical canal and internal ostium. The device is intended for one-time use.

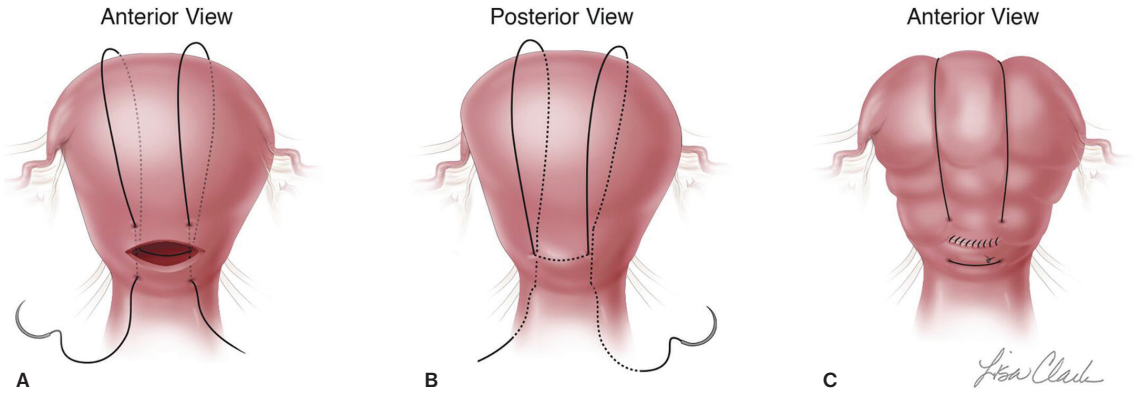
Interventions for Intractable Postpartum Hemorrhage

Intractable hemorrhage may require uterine packing (plain gauze or soaked with vasopressin or carboprost), placement of an intrauterine tamponade device, angiographic embolization, hemostatic drugs (eg, recombinant factor VIIa), or surgery.[50-52] Aortic compression can be performed or antishock garments can be used as temporizing measures.[53,54]

Uterine tamponade devices can be used to treat recalcitrant PPH due to uterine atony, or minimize uterine bleeding while definitive treatment or transport is arranged. A uterine tamponade balloon is placed through the cervix after vaginal delivery or through the uterine incision after cesarean delivery (*Figure 3*). The balloon presses against the hemorrhaging endometrial surface with a force that exceeds the uterine arterial and venous blood pressure. After a balloon is placed, the woman should be frequently assessed for ongoing blood loss from the exit port and increasing fundal height (suggesting accumulating blood).

These devices are contraindicated in women with genital tract infection, cervical cancer, pregnancy, anomalies that distort the uterine cavity (eg, large leiomyoma, congenital anomalies), and those requiring other treatment (eg, arterial embolization, surgical exploration, hysterectomy). Case studies

Figure 4. Anterior Uterine Wall with B-Lynch Suture in Place



A large Mayo needle with No. 2 chromic catgut is used to enter and exit the uterine cavity anteriorly. The suture loops over the fundus and then reenters the uterine cavity posteriorly as shown in Figure 4A and Figure 4B. The suture should be pulled tight at this point. It is looped back over the fundus, and anchored by entering and exiting the anterior lateral lower uterine segment as shown in Figure 4A. The free ends of the suture are tied down securely to compress the uterus as shown in Figure 4C.

of uterine tamponade balloons for PPH have been published and their use is recommended in consensus guidelines.[12,51,55,56] The potential danger of PPH makes the design of a randomized controlled trial to evaluate tamponade balloons unlikely. Manufacturers of uterine tamponade balloons have published instructions for safe use of their devices.

Providers should be familiar with the device that is available to them. Because the balloon is a temporary device and may fail or require a definitive treatment, a surgeon who can perform a hysterectomy should be notified at the time of placement.

Surgical options include B-Lynch uterine suture (*Figure 4*), hemostatic multiple square suturing, surgical ligation of the uterine arteries, or hysterectomy.[1,12] Uterine compression sutures, uterine artery embolization, and ligation may preserve fertility.[57] In women with continued hemorrhaging despite use of bimanual massage, uterotonic agents, and other surgical methods, a plan for rapid hysterectomy must be initiated because continued attempts at uterine conservation may increase the risk of maternal mortality.[1,12]

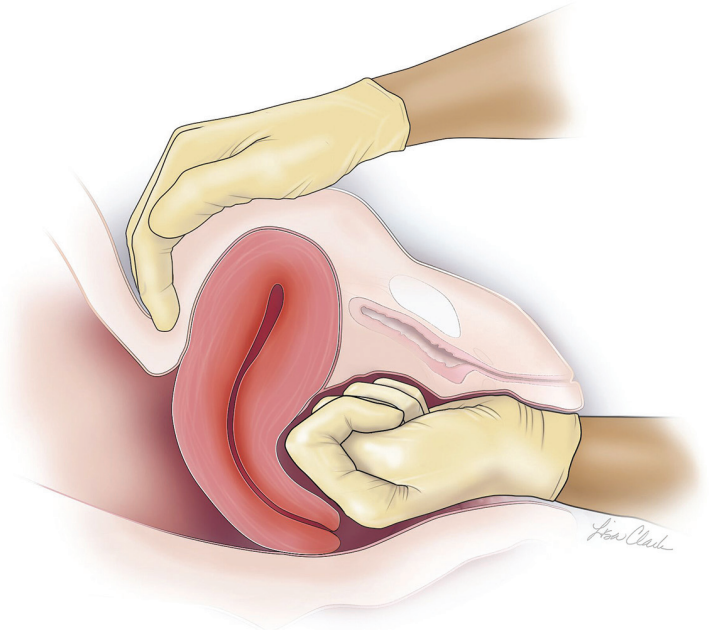
The Four Ts: Cause-Specific Management of Postpartum Hemorrhage

Tone

Uterine atony. Uterine atony is the most common cause of PPH.[58] Because hemostasis after placental separation depends on myometrial contraction, transabdominal massage after delivery of the placenta is a reasonable practice for every delivery and is recommended as a first step if atony occurs.[19] The lower uterine segment should be supported while performing this massage. Placement of a urinary catheter may help maintain uterine tone. Persistent atony (a soft, *boggy* uterus and brisk flow of blood from the vagina) will require bimanual uterine massage while awaiting drugs that promote uterine contraction.

Uterine massage. To perform bimanual massage, the provider places one hand over the lower abdomen to massage the uterine fundus and one hand in the vaginal vault to massage the lower uterine segment. The position of the provider's hands with respect to the uterus depends on the position of the uterus and the woman's body habitus. *Figure 5* shows an anteverted uterus with the

Figure 5. Bimanual Massage for Uterine Atony



provider's hand on the abdomen massaging the posterior aspect of the uterus. Two or more fingers of the vaginal hand are typically used for bimanual massage. Using the entire vaginal hand or fist to compress the uterus may be necessary for severe, persistent atony.

Uterotonic agents. Uterotonic agents include oxytocin, prostaglandins, and ergot alkaloids (*Table 5*). Uterotonic agents stimulate contraction of the myometrium, which constricts spiral arteries and decreases blood flow through the uterus. Oxytocin is an effective first-line treatment for PPH.[1,58] Oxytocin 20 to 40 units can be added to 1 L of normal saline. An initial 500 mL can be infused intravenously over 10 minutes without complications. After this initial infusion, the oxytocin solution can be infused intravenously at 250 mL/hour. If atonic hemorrhage continues, the rate of infusion or oxytocin concentration may be increased (eg, 40 to 80 units of oxytocin in 1 L of normal saline).[56]

If oxytocin alone is insufficient to improve uterine atony and hemorrhage, the choice of second-line agent should be based on the woman's risk factors (eg, the presence of hypertension or asthma) and local maternity care practices. A 2015 study of women experiencing refractory uterine atony after cesarean delivery showed greater hemorrhage-related morbidity with use of carboprost compared with methylergonovine as a second-line uterotonic agent.[59]

Methylergonovine and ergometrine (the latter not available in the United States) are ergot alkaloids that stimulate uterine muscle contraction.[58]

Table 5. Drugs for Prevention and Treatment of Postpartum Hemorrhage

Drug	Dose	Prevention	Treatment	Contraindications/Cautions
First-Line				
Oxytocin	AMTSL Prevention: 10 IU IM or IV infused over 10 minutes PPH Treatment: 20 IU in 1,000 mL NS, infuse 500 mL over 10 minutes then 250 mL/hour Can increase up to 80 IU/L if needed	+	+	Overdose or prolonged use can cause water intoxication Possible hypotension with IV use after cesarean delivery
Second-Line				
Methylergonovine	0.2 mg IM repeat every 2-4 hours	–	+	Avoid in hypertensive disorders of pregnancy including chronic hypertension
Misoprostol[a]	Prevention: 600 mcg PO Treatment: 600 mcg SL (preferred), or 800 PR	+[b]	+	Caution in patients with cardiovascular disease
Tranexamic acid[a]	1 g IV diluted in 100 mL NS administered over 10 minutes (if within first 3 hours of hemorrhage)	–	+	Administer within 3 hours of onset of bleeding. May increase risk of thrombosis. Use with caution in renal impairment and with other clotting factors (eg, prothrombin complex concentrate)
Carboprost	0.25 mg IM (or injected directly into myometrium during surgery) repeated every 15-90 minutes for a total dose of 2 mg	–	+	Relative contraindication in patients with asthma or significant renal, hepatic, or cardiac disease

[a]Misoprostol and tranexamic acid are not approved by the US Food and Drug Administration for use in prevention or treatment of postpartum hemorrhage.

[b]Use only when oxytocin is not available. Use SL route for most rapid onset of action.

AMTSL = active management of third stage of labor; BP = blood pressure; D5W = dextrose 5% in water; IM = intramuscular; IV = intravenous; NS = normal saline; PO = orally; PR = rectally; SL = sublingual.

Information from various sources.

A typical methylergonovine dose is 0.2 mg IM repeated every 2 to 4 hours if needed.[1,56] Because ergot alkaloid agents cause vasoconstriction and raise BP levels, they are contraindicated in women with preeclampsia, gestational hypertension, or chronic hypertension. Other adverse effects include nausea and vomiting. There are many potential serious drug interactions with ergots including vasoconstrictors, protease inhibitors, antifungals, and treatments for hepatitis C.[60]

Carboprost (15 methyl prostaglandin F2-alpha) is a potent uterotonic agent and can be used when adequate tone is not achieved with oxytocin.[27] Carboprost is administered IM in a dose of 0.25 mg, and can be repeated every 15 minutes for a total dose of 2 mg. Carboprost can be injected into the myometrium at the same dose, typically during cesarean delivery or a postpartum surgical procedure to treat severe PPH. Carboprost has been shown to control hemorrhage in up to 88% of patients.[61] In patients who did not benefit, chorioamnionitis or other risk factors for hemorrhage were often present.[61] Hypersensitivity is the only absolute contraindication, but carboprost should typically be avoided in patients with asthma, or significant cardiac, hepatic, or renal disease. Common adverse effects include nausea, vomiting, and diarrhea.[27]

Mechanism of Action	Adverse Effects
Stimulates the upper segment of the myometrium to contract rhythmically, constricting spiral arteries, which decreases blood flow through the uterus	Rare
Vasoconstriction and contracts smooth muscles upper and lower segments of the uterus tetanically	Nausea, vomiting, and increased BP
Generalized smooth muscle contraction	Nausea, vomiting, diarrhea, pyrexia, and shivering
Inhibits breakdown of fibrin and fibrinogen by plasmin	
Improves uterine contractility by increasing the number of oxytocin receptors and causes vasoconstriction	Nausea, vomiting, and diarrhea

The use of misoprostol in addition to oxytocin does not significantly improve treatment of PPH compared with oxytocin alone, especially if prophylactic oxytocin has already been administered as part of AMTSL.[58,62,63] Misoprostol has not been approved by the Food and Drug Administration for the treatment of PPH; however, it is recommended by ACOG.[56] Misoprostol can be administered by sublingual, oral, vaginal, or rectal routes, sometimes in combination.[26,28,64] Oral and sublingual dosing allow more rapid onset of action, but rectal dosing allows for longer duration of action and fewer gastrointestinal adverse effects. Acceptable dosages are 600 mcg sublingually (preferred) or 800 mcg rectally or orally.[12,56] Higher levels and larger doses are associated with more adverse effects including shivering, pyrexia, and diarrhea.[28,58] Even at low doses, misoprostol is associated with more adverse effects than oxytocin.[28,62]

After initial stabilization of a woman with atony, ongoing monitoring is necessary, including checking of vital signs and assessment of any ongoing or recurrent bleeding. Although research is lacking, a common approach to maintain uterine tone is to administer methylergonovine (0.2 mg IM every 4 hours for four doses) or oxytocin (20 to 40 IU in 1 L of normal saline, infusing 500 mL over 10 minutes then 250 mL/hour).[41]

Trauma

Lacerations and hematomas resulting from birth trauma can cause significant blood loss that can be lessened by hemostasis and timely repair. Sutures for hemostasis are placed if direct pressure does not stop the bleeding. Episiotomy increases the risk of anal sphincter lacerations and should be avoided unless urgent delivery is necessary and the perineum is thought to be a limiting factor in achieving delivery.[36] When an episiotomy is needed to facilitate an assisted vaginal delivery

or other indication, a mediolateral episiotomy is recommended instead of the traditional midline episiotomy to decrease the incidence of anal sphincter laceration.[65]

Hematomas can manifest as pain or as a change in vital signs out of proportion to the amount of blood loss observed. Small, nonexpanding vaginal or vulvar hematomas (typically smaller than 4 cm) can be managed conservatively with ice packs, analgesia, and continued observation.[66] Women with large or enlarging hematomas or persistent signs and symptoms of volume loss despite fluid replacement will require intervention (eg, selective arterial embolization, incision and evacuation of the clot).[66,67] If the hematoma is evacuated, the involved area should be irrigated and the bleeding vessels should be ligated. Often, a specific vessel cannot be identified and hemostatic figure-of-eight sutures are placed. Where there is diffuse oozing, a layered closure will help to secure hemostasis and eliminate dead space. Providers may be more successful in achieving hemostasis with a large wound or deep bleeding vessel by using a large needle (eg, CT, CT-1). If oozing occurs from needle entry and exit sites, a needle such as an SH needle may be helpful.

Uterine inversion. Uterine inversion is rare, occurring in approximately 1 in 2,500 deliveries.[68] AMTSL, including the Brandt maneuver, does not appear to increase the incidence of uterine inversion.[68,69] Fundal, adherent, or invasive implantation of the placenta may lead to inversion; the role of fundal pressure and undue cord traction are uncertain.[70] The woman may show signs of shock (pallor, hypotension) without excess blood loss. On inspection, the inverted uterus may be in the vaginal vault or may protrude from the vagina. It appears as a blue-gray mass that may not be readily identifiable as an inverted uterus. The placenta may still be attached, and it may be left in place until after reduction to limit hemorrhage.[68] If oxytocin is being administered, it should be discontinued, and an attempt should be made to replace the uterus quickly.

There are several methods for reduction. The Johnson method involves grasping the protruding fundus with the palm of the hand with fingers directed toward the posterior fornix. The uterus is returned to position by lifting it up through the pelvis and into the abdomen with steady pressure toward the umbilicus.[68] After the uterus is reverted, uterotonic agents should be administered to promote uterine tone and prevent recurrence. If initial attempts to replace the uterus have failed or a cervical contraction ring develops, terbutaline, nitroglycerin, or general anesthesia may allow sufficient uterine relaxation for manipulation.[68]

Uterine rupture. Although rare in an unscarred uterus, clinically significant uterine rupture complicates approximately 0.8% of term labors after cesarean delivery (LACs).[71] The risk is significantly increased in women with a previous classical uterine incision or myomectomy; these women should not undergo a trial of labor and should deliver via elective cesarean at 36 to 37 weeks' gestation.[72]

Risk of uterine rupture is increased to a lesser extent in women with a history of multiple cesarean deliveries, particularly those with no previous vaginal delivery.[73,74] Compared with spontaneous labor, induction in a woman with a uterine scar increases the rate of uterine rupture to 1% to 2%.[71,73-75] The use of prostaglandins for cervical ripening appears to be associated with an increased risk of uterine rupture.[75] Although the evidence with regard to specific prostaglandins is limited, misoprostol (PGE1) is considered to be contraindicated, and the use of the dinoprostone insert (PGE2) remains controversial.[73,75,76] The dinoprostone insert has the advantage of being easily removed if tachysystole or concerning fetal heart rate decelerations occur.

A Foley or double balloon catheter may be considered for cervical ripening if induction is indicated in a woman who would like a LAC.[73]

During labor, the first sign of uterine rupture is typically fetal heart rate changes such as fetal bradycardia.[73,77] Other signs and symptoms include vaginal bleeding, abdominal tenderness, increasing abdominal girth, loss of uterine contractions, elevation of presenting fetal part, maternal tachycardia, or circulatory collapse.[73]

Uterine rupture can harm the fetus and woman. Uterine rupture may require surgical repair of the defect, blood transfusion, or hysterectomy. Small, asymptomatic lower uterine segment defects incidentally noted on postpartum uterine examination can be monitored expectantly.[73] A meta-analysis of studies evaluating morbidity and mortality associated with LAC and elective repeat cesarean delivery (ERCD) in term gestations found that the overall maternal mortality was 9.6/100,000 deliveries (95% CI = 2.1-43.2 per

100,000 deliveries) for ERCD, and 1.9/100,000 deliveries for LAC (95% CI = 0.4-9.5 per 100,000 deliveries). The rates of hysterectomy, hemorrhage, and transfusions did not differ significantly between LAC and ERCD.[75]

Although maternal mortality is reduced by choosing LAC over ERCD, this choice is associated with increased fetal mortality. ERCD is associated with 0.5 perinatal mortalities per 1,000 deliveries compared with 1.3 perinatal mortalities per 1,000 LACs.[75] This LAC perinatal mortality rate is comparable to the perinatal mortality rate of laboring nulliparous women.[71] Hypoxic ischemic encephalopathy also is higher for LAC compared with ERCD, but "it is not possible to know the true relationship due to the low strength of overall evidence."[75]

Tissue

Retained products (placenta, placental fragments, and blood clots) prevent the uterus from contracting enough to achieve optimal tone.

Retained placenta. A small gush of blood with lengthening of the cord and a slight rise of the uterus in the pelvis are the classic signs of placental separation. Firm traction on the umbilical cord with one hand while the other hand applies suprapubic counter-pressure (Brandt maneuver) typically achieves placental delivery. The mean time from delivery until placental expulsion is 8.3 to 9 minutes.[5] A longer interval is associated with an increased risk of PPH, doubling after 10 minutes.[5] Retained placenta, defined as the failure of the placenta to deliver within 30 minutes after birth, occurs in less than 3% of vaginal deliveries.[78] Routine injection of the umbilical vein with saline and oxytocin does not reduce the risk of blood loss or retained placenta.[79]

If the placenta does not deliver after 30 minutes, manual removal of the placenta should be considered.[80] Unless the woman is unstable, establish adequate analgesia before exploring the uterus. Analgesia will make the procedure easier to perform and reduce the woman's emotional and physical distress.

To manually remove the placenta:

1. Discontinue uterine massage and allow the uterus to relax. Subcutaneous or IV terbutaline 0.25 mg, IV nitroglycerin 100 to 200 mcg, or general anesthesia may infrequently be required to relax the uterus. The woman can lose large amounts of blood when drugs for uterine relaxation are administered, so it is imperative to rapidly accomplish the removal and then reverse the relaxation with uterotonic agents[81]
2. Identify the cleavage plane between the placenta and the uterine wall. Advance fingertips in the plane until the entire placenta is free
3. Cup the detached cotyledons in the hand. Deliver the placenta intact if possible
4. After examining the uterine cavity and the placenta to ensure that the entire placenta and membranes have been removed, massage the uterus and administer oxytocin.

If the cleavage plane cannot be identified or parts of the plane cannot be developed completely, prepare for surgical removal of the placenta:

1. Ensure the woman has oxygen, two large-bore IV catheters administering replacement fluids, and adequate anesthesia, and ensure that proper surgical setup is available and appropriately trained providers are present. The reason the plane cannot be developed completely may due to an invasive placenta, so a physician with hysterectomy skills should be called
2. Curette the uterine cavity with a large blunt curette or large suction catheter. Take care to avoid perforating the soft, postpartum uterus
3. Use ring forceps to grasp and remove membranes and placental tissue.

Invasive placenta can be life-threatening.[78] The incidence has increased to at least 0.04% of deliveries and is likely related to the increase in cesarean delivery rates.[32] Other risk factors include prior invasive placenta, placenta previa (especially in combination with prior cesarean deliveries, increasing to 61% with placenta previa and four or more prior cesarean deliveries), advanced maternal age, and high parity.[32,78]

Classification is based on the depth of invasion. Placenta accreta adheres to the myometrium, placenta increta invades the myometrium, and placenta percreta penetrates the myometrium to or beyond the serosa.[32,78] The usual treatment for invasive placenta is hysterectomy. However, conservative management is sometimes successful in select women. Conservative treatment options include partial removal of the placenta, arterial embolization, methotrexate use, and/or watchful waiting.[32,82] Women treated for a retained placenta must be monitored for late sequelae, including infection and late postpartum bleeding.[32,82]

Thrombin

Coagulation disorders, a rare cause of PPH, are unlikely to benefit from uterine massage, uterotonic agents, and repair of lacerations.[1] Coagulation defects may cause and/or result from hemorrhage due to inadvertent dilution from IV fluids or PRBC-only resuscitation. Coagulopathy should be suspected in women who have not benefited from the usual measures to treat PPH, are not forming blood clots, or are bleeding from puncture sites.[41]

Many women taking drugs such as heparin or aspirin or women with chronic coagulopathies (eg, idiopathic thrombocytopenic purpura, thrombotic thrombocytopenic purpura, von Willebrand disease, hemophilia) are identified before delivery, which allows advanced planning to prevent PPH. Coagulopathic bleeding before or during labor can be the result of HELLP syndrome (hemolysis, elevated liver enzymes, and low platelet count) or DIC.[41] Obstetric conditions that can cause DIC include preeclampsia with severe features, amniotic fluid embolism, sepsis, placental abruption (often associated with cocaine use or hypertensive disorders), massive PPH, and prolonged retention of a dead fetus.[83]

Because DIC is characterized by decreasing platelet and fibrinogen levels and increasing fibrinogen degradation products (FDPs), evaluation should include a platelet count, prothrombin time, partial thromboplastin time, fibrinogen level, and fibrin split products (D-dimer). However, rapid laboratory testing may not be available and FDP levels are normally increased at term, so clinical suspicion is critical in the acute setting because there is no standard cut-off for diagnosing DIC using test results in patients with PPH.[84] An empty whole blood tube (red top) can be filled with maternal blood. The blood should clot within 5 to 10 minutes.

Management of coagulopathy consists of treating the underlying disease process, serially evaluating the coagulation status, replacing appropriate blood components (guided by a massive transfusion protocol for severe hemorrhage), and supporting intravascular volume.[84,85]

Tranexamic acid may reduce complications from hemorrhage and could be considered as an adjuvant therapy to uterotonic agents when the woman has had more than 500 mL of blood loss after vaginal delivery, more than 1,000 mL of blood loss after cesarean delivery, or has been hemodynamically unstable and bleeding for less than 3 hours. Tranexamic acid reduces blood loss by decreasing breakdown of fibrin and fibrinogen. An acceptable dosage is IV 1 g.[44] A study of 20,060 women with PPH after vaginal or cesarean delivery found mortality due to bleeding was reduced in women treated with tranexamic acid (relative risk [RR] 0.81; 95% CI = 0.65-1.00; NNT = 250).[42] Study results showed no increases in thrombotic events,[42] though thrombotic events associated with tranexamic acid use in the management of nonobstetric hemorrhage have been a concern.[30]

Poststabilization Care and Debriefing

Postpartum hemorrhage can be frightening for the woman, her family, and her medical caregivers. Nine percent of women screen positive for posttraumatic stress disorder (PTSD) due to traumatic childbirth.[86] Treatment of a woman with PPH does not conclude with control of bleeding and stabilization of her vital signs. Screening for, diagnosing, and treating acute stress disorder (occurring in the first month post-trauma) or PTSD is warranted to prevent long-term emotional sequelae. In addition to support of the health care team, women with acute stress symptoms benefit from cognitive behavioral therapy.[87]

Preventing Postpartum Hemorrhage Complications: A Systems-Based Approach

Complications of PPH are too common, even in high-resource countries and well-staffed delivery suites. Based on analysis of systems errors identified in a 2010 Joint Commission Sentinel Event Alert, the commission recommended that hospitals "identify specific triggers for responding to changes in the mother's vital signs and clinical condition and develop and use protocols and drills for responding to changes, such as hemorrhage and preeclampsia. Hospitals should use drills to train staff on protocols, to refine local protocols, and to identify and fix systems problems that would prevent optimal care."[88] This call to action is supported by studies that show a standardized approach to prevention and treatment of maternal hemorrhage improves patient outcomes.[89-91] See the *Safety in Maternity Care* chapter for more information about team-based care.

The Council on Patient Safety in Women's Health Care subsequently outlined essential

steps that labor and delivery departments should take to decrease the incidence and severity of PPH.[13] The Obstetric Hemorrhage Patient Safety Bundle includes recommendations for improved readiness, recognition, prevention, stage-based response, and reporting (see page 15).[13] Suggested steps include creating a hemorrhage cart and using huddles, rapid response teams, and massive transfusion protocols.

ALSO training can be part of a systems approach to improve patient care. The use of interdisciplinary team training with in situ simulation has been shown to improve perinatal safety.[92,93] Studies of ALSO training in Colombia, Guatemala, Honduras, and Tanzania have shown improved adherence with obstetric best practices, prevention, and management of obstetric complications including PPH.[94,95]

Global Perspective

Although there is risk of PPH at every delivery, severe complications of PPH including maternal mortality are most common in low-resource countries.[12,96] Some risk factors for PPH may be more significant in low-resource countries (eg, prolonged labor and chronic anemia from malnutrition or malaria). Lack of skilled attendants, lack of access to drugs to prevent and treat hemorrhage, and great distances from medical centers capable of providing blood transfusions and surgery further increase the risk of PPH morbidity and mortality.[96,97] Uterine atony accounts for the majority of PPH in all settings. It also is important to consider causes that are more common in low-resource areas such as uterine rupture after prolonged labors and genital tract lacerations in women with female genital mutilation.[96,98]

If used at every delivery, AMTSL would reduce PPH by approximately 30% to 50%.[11,96] Oxytocin is the preferred drug for PPH prevention and treatment; however, it requires controlled temperatures and the use of vials and needles.[99] A single-dose, prefilled syringe has been developed to decrease complexity of use.[100] If a health center cannot use or store oxytocin safely, misoprostol may be the preferred drug for prevention and treatment of PPH.[12,96,99] Misoprostol availability in some countries may be limited because of legal or political concerns related to the potential diversion of misoprostol for pregnancy termination. Heat-stable carbetocin 100 mcg IM is a reasonable option to prevent PPH in settings where cold storage of oxytocin is not possible.[101]

Other prevention strategies include detecting and correcting maternal anemia before delivery and avoiding unnecessary instrumental deliveries and routine episiotomy.[34,36,37,102] Treatment possibilities being evaluated for use in low-resource countries include the use of antishock garments and uterine tamponade with a hydrostatic condom catheter (sterile rubber catheter fitted with a condom, placed into the uterus through the vagina and inflated with 250 to 500 mL of saline).[53-55,96] Proprietary devices are effective for uterine atony but may not be readily available because of financial and logistical concerns.

Additional details regarding PPH in developing countries are available at www.aafp.org/globalalso.

Summary

Postpartum hemorrhage is unpredictable and can occur in women with no risk factors. AMTSL and QBL should be used routinely. AMTSL includes oxytocin after delivery of the fetal anterior shoulder and controlled cord traction with the Brandt maneuver. Uterine massage after delivery of the placenta is a reasonable approach and is included in some AMTSL protocols. Delayed cord clamping (1 to 3 minutes after delivery) may be considered to decrease risk of infant anemia without increasing maternal hemorrhage risk.[22]

Management of PPH requires rapid diagnosis and treatment using a standardized approach. Diagnosis and treatment occur simultaneously using the Four Ts mnemonic. Uterine atony (*T*one) is responsible for the majority of PPH and can be effectively treated with uterine massage and uterotonic agents (oxytocin, misoprostol, methylergonovine, and 15-methyl prostaglandin F2a). Oxytocin remains the first-line medical treatment for PPH due to atony. *T*rauma (eg, perineal lacerations, hematomas) is the second most common cause of PPH and may require intervention. The third most common cause of PPH, *T*issue, requires careful uterine examination to remove clots and retained placenta as well as anticipation of rare cases involving invasive placenta. For women with suspected coagulopathy (eg, DIC), clotting factors need to be replaced and the cause of coagulopathy identified and corrected (*T*hrombin).

Early recognition, systematic evaluation and treatment, and prompt fluid resuscitation mini-

mize the morbidity and mortality associated with PPH, regardless of cause. Massive transfusion protocols and use of the Obstetric Hemorrhage Patient Safety Bundle provide standardized and systems-based means of preparing for and responding to PPH.

Nursing Considerations: Postpartum Hemorrhage

- Identify women at risk of PPH to increase early recognition and prevention
- Empty bladder, establish IV access, obtain laboratory tests
- Determine the location of the PPH cart at the start of every shift and be familiar with the drugs and equipment it contains, including balloon catheter for uterine compression
- Identify your institution's massive transfusion protocol
- Facilitate team efforts for optimizing patient safety
- Champion efforts at your institution to implement the "Obstetric Hemorrhage Safety Bundle" and use of QBL for all deliveries

IV = intravenous; PPH = postpartum hemorrhage; QBL = quantification of blood loss.

COUNCIL ON PATIENT SAFETY
IN WOMEN'S HEALTH CARE
safe health care for every woman

PATIENT SAFETY BUNDLE

Obstetric Hemorrhage

READINESS

Every unit

- Hemorrhage cart with supplies, checklist, and instruction cards for intrauterine balloons and compressions stitches
- Immediate access to hemorrhage medications (kit or equivalent)
- Establish a response team - who to call when help is needed (blood bank, advanced gynecologic surgery, other support and tertiary services)
- Establish massive and emergency release transfusion protocols (type-O negative/uncrossmatched)
- Unit education on protocols, unit-based drills (with post-drill debriefs)

RECOGNITION & PREVENTION

Every patient

- Assessment of hemorrhage risk (prenatal, on admission, and at other appropriate times)
- Measurement of cumulative blood loss (formal, as quantitative as possible)
- Active management of the 3rd stage of labor (department-wide protocol)

RESPONSE

Every hemorrhage

- Unit-standard, stage-based, obstetric hemorrhage emergency management plan with checklists
- Support program for patients, families, and staff for all significant hemorrhages

REPORTING/SYSTEMS LEARNING

Every unit

- Establish a culture of huddles for high risk patients and post-event debriefs to identify successes and opportunities
- Multidisciplinary review of serious hemorrhages for systems issues
- Monitor outcomes and process metrics in perinatal quality improvement (QI) committee

Standardization of health care processes and reduced variation has been shown to improve outcomes and quality of care. The Council on Patient Safety in Women's Health Care disseminates patient safety bundles to help facilitate the standardization process. This bundle reflects emerging clinical, scientific, and patient safety advances as of the date issued and is subject to change. The information should not be construed as dictating an exclusive course of treatment or procedure to be followed. Although the components of a particular bundle may be adapted to local resources, standardization within an institution is strongly encouraged.

The Council on Patient Safety in Women's Health Care is a broad consortium of organizations across the spectrum of women's health for the promotion of safe health care for every woman.

May 2015

Reprinted from Council on Patient Safety in Women's Health Care. AIM-Supported Patient Safety Bundles. Obstetric Hemorrhage. Available at https://safehealthcareforeverywoman.org/.

References

1. Committee on Practice Bulletins-Obstetrics. Practice Bulletin no. 183: postpartum hemorrhage. *Obstet Gynecol*. 2017;130(4):e168-e186.
2. Rajan PV, Wing DA. Postpartum hemorrhage: evidence-based medical interventions for prevention and treatment. *Clin Obstet Gynecol*. 2010;53(1):165-181.
3. Leduc D, Senikas V, Lalonde AB; CLINICAL PRACTICE OBSTETRICS COMMITTEE. Active management of the third stage of labour: prevention and treatment of postpartum hemorrhage. *J Obstet Gynaecol Can*. 2009;31(10):980-993.
4. Magann EF, Evans S, Hutchinson M, et al. Postpartum hemorrhage after vaginal birth: an analysis of risk factors. *South Med J*. 2005; 98(4):419-422.
5. Magann EF, Evans S, Chauhan SP, et al. The length of the third stage of labor and the risk of postpartum hemorrhage. *Obstet Gynecol*. 2005;105(2):290-293.
6. Say L, Chou D, Gemmill A, et al. Global causes of maternal death: a WHO systematic analysis. *Lancet Glob Health*. 2014;2(6):e323-e333.
7. Creanga AA, Berg CJ, Syverson C, et al. Pregnancy-related mortality in the United States, 2006-2010. *Obstet Gynecol*. 2015;125(1):5-12.
8. Thompson JF, Heal LJ, Roberts CL, Ellwood DA. Women's breastfeeding experiences following a significant primary postpartum haemorrhage: A multicentre cohort study. *Int Breastfeed J*. 2010;5:5.
9. Sentilhes L, Gromez A, Clavier E, et al. Long-term psychological impact of severe postpartum hemorrhage. *Acta Obstet Gynecol Scand*. 2011;90(6):615-620.
10. US Department of Health and Human Services, Office of the Assistant Secretary for Health. Report of the US Department of Health and Human Services. The 2009 national blood collection and utilization survey report. Washington, DC; 2011.
11. Begley CM, Gyte GML, Devane D, et al. Active versus expectant management for women in the third stage of labour. *Cochrane Database Syst Rev*. 2019;(2):CD007412.
12. World Health Organization. WHO recommendations for the prevention and treatment of postpartum haemorrhage. WHO Press. Geneva; 2012.
13. Main EK, Goffman D, Scavone BM, et al; National Partnership for Maternal Safety. Council on Patient Safety in Women's Health Care. National Partnership for Maternal Safety: Consensus bundle on obstetric hemorrhage. *Obstet Gynecol*. 2015;126(1):155-162.
14. Mavrides E, Allard S, Chandraharan E, et al; Royal College of Obstetricians and Gynaecologists. Prevention and Management of Postpartum Haemorrhage: Green-top Guideline No. 52. *BJOG*. 2017; 124(5):e106-e149.
15. Hofmeyr GJ, Mshweshwe NT, Gülmezoglu AM. Controlled cord traction for the third stage of labour. *Cochrane Database Syst Rev*. 2015;1(1):CD008020.
16. Soltani H, Hutchon DR, Poulose TA. Timing of prophylactic uterotonics for the third stage of labour after vaginal birth. *Cochrane Database Syst Rev*. 2010;(8):CD006173.
17. Salati JA, Leathersich SJ, Williams MJ, et al. Prophylactic oxytocin for the third stage of labour to prevent postpartum haemorrhage. *Cochrane Database Syst Rev*. 2019;(4):CD001808.
18. Gallos ID, Papadopoulou A, Man R, et al. Uterotonic agents for preventing postpartum haemorrhage: a network meta-analysis. *Cochrane Database Syst Rev*. 2018;(12):CD011689.
19. Hofmeyr GJ, Abdel-Aleem H, Abdel-Aleem MA. Uterine massage for preventing postpartum haemorrhage. *Cochrane Database Syst Rev*. 2013;(7):CD006431.
20. Du Y, Ye M, Zheng F. Active management of the third stage of labor with and without controlled cord traction: a systematic review and meta-analysis of randomized controlled trials. *Acta Obstet Gynecol Scand*. 2014;93(7):626-633.
21. Chen M, Chang Q, Duan T, et al. Uterine massage to reduce blood loss after vaginal delivery: a randomized controlled trial. *Obstet Gynecol*. 2013;122(2 Pt 1):290-295.
22. McDonald SJ, Middleton P, Dowswell T, Morris PS. Effect of timing of umbilical cord clamping of term infants on maternal and neonatal outcomes. *Cochrane Database Syst Rev*. 2013;(7):CD004074.
23. Rabe H, Diaz-Rossello JL, Duley L, Dowswell T. Effect of timing of umbilical cord clamping and other strategies to influence placental transfusion at preterm birth on maternal and infant outcomes. *Cochrane Database Syst Rev*. 2012;(8):CD003248.
24. Committee on Obstetric Practice. Committee Opinion no. 684: delayed umbilical cord clamping after birth. *Obstet Gynecol*. 2017; 129(1):e5-e10.
25. Adnan N, Conlan-Trant R, McCormick C, et al. Intramuscular versus intravenous oxytocin to prevent postpartum haemorrhage at vaginal delivery: randomized controlled trial. *BMJ*. 2018;362:k3546.
26. Bellad MB, Tara D, Ganachari MS, et al. Prevention of postpartum haemorrhage with sublingual misoprostol or oxytocin: a double-blind randomised controlled trial. *BJOG*. 2012;119(8):975-982, discussion 982-986.
27. Tunçalp Ö, Hofmeyr GJ, Gülmezoglu AM. Prostaglandins for preventing postpartum haemorrhage. *Cochrane Database Syst Rev*. 2012;8(8):CD000494.
28. Hofmeyr GJ. Gülmezoglu AM, Novikova N, Lawrie TA. Postpartum misoprostol for preventing maternal mortality and morbidity. *Cochrane Database Syst Rev*. 2013;(7):CD008982.
29. Elati A, Weeks A. Risk of fever after misoprostol for the prevention of postpartum hemorrhage: a meta-analysis. *Obstet Gynecol*. 2012; 120(5):1140-1148.
30. Novikova N, Hofmeyr GJ, Cluver C. Tranexamic acid for preventing postpartum haemorrhage. *Cochrane Database Syst Rev*. 2015;(6): CD007872.
31. Sentilhes L, Winer N, Azria E, et al; Groupe de Recherche en Obstétrique et Gynécologie. Tranexamic acid for the prevention of blood loss after vaginal delivery. *N Engl J Med*. 2018;379(8):731-742.
32. Committee on Obstetric Practice. Committee Opinion no. 529: placenta accreta. *Obstet Gynecol*. 2012;120(1):207-211.
33. Einerson BD, Rodriguez CE, Kennedy AM, et al. Magnetic resonance imaging is often misleading when used as an adjunct to ultrasound in the management of placenta accreta spectrum disorders. *Am J Obstet Gynecol*. 2018;218(6):618.e1-618.e7.
34. ACOG Committee on Obstetrics. ACOG Practice Bulletin no. 78: hemoglobinopathies in pregnancy. *Obstet Gynecol*. 2007;109(1): 229-237.
35. Milne ME, Yazer MH, Waters JH. Red blood cell salvage during obstetric hemorrhage. *Obstet Gynecol*. 2015;125(4):919-923.
36. Jiang H, Qian X, Carroli G, Garner P. Selective versus routine use of episiotomy for vaginal birth. *Cochrane Database Syst Rev*. 2017; 2(2):CD000081.
37. O'Mahony F, Hofmeyr GJ, Menon V. Choice of instruments for assisted vaginal delivery. *Cochrane Database Syst Rev*. 2010;(11): CD005455.
38. Aasheim V, Nilsen ABV, Reinar LM, Lukasse M. Perineal techniques during the second stage of labour for reducing perineal trauma. *Cochrane Database Syst Rev*. 2017;(6):CD006672.

39. Association of Women's Health, Obstetric and Neonatal Nurses. Quantification of blood loss: AWHONN practice brief number 1. *J Obstet Gynecol Neonatal Nurs*. 2015;44(1):158-160.
40. Driessen M, Bouvier-Colle MH, Dupont C, et al. Pithagore6 Group. Postpartum hemorrhage resulting from uterine atony after vaginal delivery: factors associated with severity. *Obstet Gynecol*. 2011; 117(1):21-31.
41. Evensen A, Anderson JM, Fontaine P. Postpartum hemorrhage: prevention and treatment. *Am Fam Physician*. 2017;95(7):442-449.
42. WOMAN Trial Collaborators. Effect of early tranexamic acid administration on mortality, hysterectomy, and other morbidities in women with post-partum haemorrhage (WOMAN): an international, randomised, double-blind, placebo-controlled trial. *Lancet*. 2017; 389(10084):2105-2116.
43. Gayet-Ageron A, Prieto-Merino D, Ker K, et al; Antifibrinolytic Trials Collaboration. Effect of treatment delay on the effectiveness and safety of antifibrinolytics in acute severe haemorrhage: a meta-analysis of individual patient-level data from 40 138 bleeding patients. *Lancet*. 2018;391(10116):125-132.
44. Pacheco LD, Hankins GDV, Saad AF, et al. Tranexamic acid for the management of obstetric hemorrhage. *Obstet Gynecol*. 2017;130(4): 765-769.
45. Bogert JN, Harvin JA, Cotton BA. Damage control resuscitation. *J Intensive Care Med*. 2016;31(3):177-186.
46. Lyndon A, Lagrew D, Shields LE, Main E. Improving health care response to obstetric hemorrhage version 2.0: a California Quality Improvement Toolkit. 2015. Available from https://www.cmqcc.org/resources-tool-kits/toolkits/ob-hemorrhage-toolkit.
47. Pacheco L, Saade G, Costantine M, et al. An update on the use of massive transfusion protocols in obstetrics. *Am J Obstet Gynecol*. 2016;214(3):340-344.
48. Kacmar RM, Mhyre JM, Scavone BM, et al. The use of postpartum hemorrhage protocols in United States academic obstetric anesthesia units. *Anesth Analg*. 2014;119(4):906-910.
49. Solomon C, Collis RE, Collins PW. Haemostatic monitoring during postpartum haemorrhage and implications for management. *Br J Anaesth*. 2012;109(6):851-863.
50. Schmid BC, Rezniczek GA, Rolf N, et al. Uterine packing with chitosan-covered gauze for control of postpartum hemorrhage. *Am J Obstet Gynecol*. 2013;209(3):225.e1-225.e5.
51. Georgiou C. Balloon tamponade in the management of postpartum haemorrhage: a review. *BJOG*. 2009;116(6):748-757.
52. Lavigne-Lissalde G, Aya AG, Mercier FJ, et al. Recombinant human FVIIa for reducing the need for invasive second-line therapies in severe refractory postpartum hemorrhage: a multicenter, randomized, open controlled trial. *J Thromb Haemost*. 2015;13(4):520-529.
53. Soltan MH, Sadek RR. Experience managing postpartum hemorrhage at Minia University Maternity Hospital, Egypt: no mortality using external aortic compression. *J Obstet Gynaecol Res*. 2011; 37(11):1557-1563.
54. El Ayadi AM, Butrick E, Geissler J, Miller S. Combined analysis of the non-pneumatic anti-shock garment on mortality from hypovolemic shock secondary to obstetric hemorrhage. *BMC Pregnancy Childbirth*. 2013;13:208.
55. Tindell K, Garfinkel R, Abu-Haydar E, et al. Uterine balloon tamponade for the treatment of postpartum haemorrhage in resource-poor settings: a systematic review. *BJOG*. 2013;120(1):5-14.
56. American College of Obstetricians and Gynecologists. Postpartum hemorrhage from vaginal delivery. Patient safety checklist no. 10. *Obstet Gynecol*. 2013;121:1151-1152.
57. Doumouchtsis SK, Nikolopoulos K, Talaulikar V, et al. Menstrual and fertility outcomes following the surgical management of postpartum haemorrhage: a systematic review. *BJOG*. 2014;121(4): 382-388.
58. Mousa HA, Blum J, Abou El Senoun G, et al. Treatment for primary postpartum haemorrhage. *Cochrane Database Syst Rev*. 2014;(2): CD003249.
59. Butwick AJ, Carvalho B, Blumenfeld YJ, et al. Second-line uterotonics and the risk of hemorrhage-related morbidity. *Am J Obstet Gynecol*. 2015;212(5):642.e1-642.e7.
60. Lexicomp. Methylergonovine: Drug Information. Waltham, MA: Uptodate; 2012.
61. Oleen MA, Mariano JP. Controlling refractory atonic postpartum hemorrhage with Hemabate sterile solution. *Am J Obstet Gynecol*. 1990;162(1):205-208.
62. Blum J, Winikoff B, Raghavan S, et al. Treatment of post-partum haemorrhage with sublingual misoprostol versus oxytocin in women receiving prophylactic oxytocin: a double-blind, randomised, non-inferiority trial. *Lancet*. 2010;375(9710):217-223.
63. Widmer M, Blum J, Hofmeyr GJ, et al. Misoprostol as an adjunct to standard uterotonics for treatment of post-partum haemorrhage: a multicentre, double-blind randomised trial. *Lancet*. 2010;375(9728): 1808-1813.
64. Winikoff B, Dabash R, Durocher J, et al. Treatment of post-partum haemorrhage with sublingual misoprostol versus oxytocin in women not exposed to oxytocin during labour: a double-blind, randomised, non-inferiority trial. *Lancet*. 2010;375(9710):210-216.
65. Waldman R. ACOG Practice Bulletin no. 198: Prevention and management of obstetric lacerations at vaginal delivery. *Obstet Gynecol*. 2019;133(1):185.
66. Chandraharan E, Arulkumaran S. Surgical aspects of postpartum haemorrhage. *Best Pract Res Clin Obstet Gynaecol*. 2008;22(6): 1089-1102.
67. Distefano M, Casarella L, Amoroso S, et al. Selective arterial embolization as a first-line treatment for postpartum hematomas. *Obstet Gynecol*. 2013;121(2 Pt 2)(Suppl 1):443-447.
68. You WB, Zahn CM. Postpartum hemorrhage: abnormally adherent placenta, uterine inversion, and puerperal hematomas. *Clin Obstet Gynecol*. 2006;49(1):184-197.
69. Gülmezoglu AM, Widmer M, Merialdi M, et al. Active management of the third stage of labour without controlled cord traction: a randomized non-inferiority controlled trial. *Reprod Health*. 2009;6:2.
70. Shah-Hosseini R, Evrard JR. Puerperal uterine inversion. *Obstet Gynecol*. 1989;73(4):567-570.
71. Cunningham GF; National Institutes of Health Consensus Development Conference Panel. National Institutes of Health Consensus Development conference statement: vaginal birth after cesarean: new insights March 8-10, 2010. *Obstet Gynecol*. 2010;115(6): 1279-1295.
72. Society for Maternal-Fetal Medicine. Prior classical cesarean delivery - counseling and management. 2014. Available at https://www.smfm.org/publications/86-prior-classical-cesarean-delivery-counseling-and-management.
73. Committee on Practice Bulletins-Obstetrics. Practice Bulletin no. 184: vaginal birth after cesarean delivery. *Obstet Gynecol*. 2017; 130(5):e217-e233.

74. Landon MB, Hauth JC, Leveno KJ, et al; National Institute of Child Health and Human Development Maternal-Fetal Medicine Units Network. Maternal and perinatal outcomes associated with a trial of labor after prior cesarean delivery. *N Engl J Med*. 2004;351(25): 2581-2589.
75. Guise JM, Eden K, Emeis C, et al. Vaginal birth after cesarean: new insights. *Evid Rep Technol Assess (Full Rep)*. 2010;(191):1-397.
76. Hauk L; American Academy of Family Physicians. Planning for labor and vaginal birth after cesarean delivery: guidelines from the AAFP. *Am Fam Physician*. 2015;91(3):197-198.
77. Guise JM, McDonagh MS, Osterweil P, et al. Systematic review of the incidence and consequences of uterine rupture in women with previous caesarean section. *BMJ*. 2004;329(7456):19-25.
78. Wu S, Kocherginsky M, Hibbard JU. Abnormal placentation: twenty-year analysis. *Am J Obstet Gynecol*. 2005;192(5):1458-1461.
79. Mori R, Nardin JM, Yamamoto N, et al. Umbilical vein injection for the routine management of third stage of labour. *Cochrane Database Syst Rev*. 2012;(3):CD006176.
80. Weeks AD. The retained placenta. *Best Pract Res Clin Obstet Gynaecol*. 2008;22(6):1103-1117.
81. Axemo P, Fu X, Lindberg B, et al. Intravenous nitroglycerin for rapid uterine relaxation. *Acta Obstet Gynecol Scand*. 1998;77(1):50-53.
82. Timmermans S, van Hof AC, Duvekot JJ. Conservative management of abnormally invasive placentation. *Obstet Gynecol Surv*. 2007;62(8):529-539.
83. Levi M. Pathogenesis and management of peripartum coagulopathic calamities (disseminated intravascular coagulation and amniotic fluid embolism). *Thromb Res*. 2013;131(Suppl 1):S32-S34.
84. Collins P, Abdul-Kadir R, Thachil J. Subcommittees on Women' s Health Issues in Thrombosis and Haemostasis and on Disseminated Intravascular Coagulation. Management of coagulopathy associated with postpartum hemorrhage: guidance from the SSC of the ISTH. *J Thromb Haemost*. 2016;14(1):205-210.
85. Gutierrez MC, Goodnough LT, Druzin M, Butwick AJ. Postpartum hemorrhage treated with a massive transfusion protocol at a tertiary obstetric center: a retrospective study. *Int J Obstet Anesth*. 2012; 21(3):230-235.
86. Beck CT, Gable RK, Sakala C, Declercq ER. Posttraumatic stress disorder in new mothers: results from a two-stage U.S. national survey. *Birth*. 2011;38(3):216-227.
87. Roberts NP, Kitchiner NJ, Kenardy J, Bisson JI. Early psychological interventions to treat acute traumatic stress symptoms. *Cochrane Database Syst Rev*. 2010;(3):CD007944.
88. Joint Commission. Preventing maternal death. *Jt Comm Perspect*. 2010;30(3):7-9.
89. Shields LE, Wiesner S, Fulton J, Pelletreau B. Comprehensive maternal hemorrhage protocols reduce the use of blood products and improve patient safety. *Am J Obstet Gynecol*. 2015;212(3):272-280.
90. Rizvi F, Mackey R, Barrett T, et al. Successful reduction of massive postpartum haemorrhage by use of guidelines and staff education. *BJOG*. 2004;111(5):495-498.
91. Main EK, Cape V, Abreo A, et al. Reduction of severe maternal morbidity from hemorrhage using a state perinatal quality collaborative. *Am J Obstet Gynecol*. 2017;216(3):298.e1-298.e11.
92. Riley W, Davis S, Miller K, et al. Didactic and simulation nontechnical skills team training to improve perinatal patient outcomes in a community hospital. *Jt Comm J Qual Patient Saf*. 2011;37(8): 357-364.
93. American College of Obstetricians and Gynecologists Committee on Patient Safety and Quality Improvement. Committee Opinion no. 590: preparing for clinical emergencies in obstetrics and gynecology. *Obstet Gynecol*. 2014;123(3):722-725.
94. Sorensen BL, Rasch V, Massawe S, et al. Advanced life support in obstetrics (ALSO) and post-partum hemorrhage: a prospective intervention study in Tanzania. *Acta Obstet Gynecol Scand*. 2011; 90(6):609-614.
95. Dresang LT, González MM, Beasley J, et al. The impact of Advanced Life Support in Obstetrics (ALSO) training in low-resource countries. *Int J Gynaecol Obstet*. 2015;131(2):209-215.
96. Lalonde A; International Federation of Gynecology and Obstetrics. Prevention and treatment of postpartum hemorrhage in low-resource settings. *Int J Gynaecol Obstet*. 2012;117(2):108-118.
97. Ronsmans C, Graham WJ. Lancet Maternal Survival Series steering group. Maternal mortality: who, when, where, and why. *Lancet*. 2006;368(9542):1189-1200.
98. UNICEF Office of Research - Innocenti. Changing a Harmful Social Convention: Female Genital Mutilation/Cutting. 2005. Available at https://www.unicef-irc.org/publications/396-changing-a-harmful-social-convention-female-genital-mutilation-cutting.html.
99. Hundley VA, Avan BI, Sullivan CJ, Graham WJ. Should oral misoprostol be used to prevent postpartum haemorrhage in home-birth settings in low-resource countries? A systematic review of the evidence. *BJOG*. 2013;120(3):277-285, discussion 86-87.
100. Pantoja T, Abalos E, Chapman E, et al. Oxytocin for preventing postpartum haemorrhage (PPH) in non-facility birth settings. *Cochrane Database Syst Rev*. 2016;4(4):CD011491.
101. Widmer M, Piaggio G, Nguyen TMH, et al. Gülmezoglu AM; WHO CHAMPION Trial Group. Heat-Stable Carbetocin versus Oxytocin to Prevent Hemorrhage after Vaginal Birth. *N Engl J Med*. 2018;379(8): 743-752.
102. Al-Zirqi I, Vangen S, Forsen L, Stray-Pedersen B. Prevalence and risk factors of severe obstetric haemorrhage. *BJOG*. 2008;115(10): 1265-1272.

Maternal Resuscitation and Trauma

Learning Objectives

1. Describe the aspects of maternal physiology that affect maternal resuscitation and response to trauma during pregnancy
2. Describe the evaluation and management of major and minor trauma in pregnancy
3. Describe the technique and timing for resuscitative hysterotomy (perimortem cesarean delivery)

Introduction and Epidemiology

Cardiac arrest is the final pathway in many life-threatening diseases. Although 475,000 people die each year from cardiac arrest in the United States, it is still rare during pregnancy.[1] The rate of cardiac arrest in pregnancy appears to have increased in the United States and is now estimated to occur in 1 in 12,000 women admitted for delivery.[2] Maternal survival ranges from 17% to 59%,[2,3] and fetal survival ranges from 61% to 80%.[4,5] From 1990 to 2013, the maternal mortality ratio for the United States doubled, whereas the ratio in the developing world decreased by 44%.[6] *Figure 1* shows the pregnancy-related mortality rates for 1987-2016. Among surviving neonates, 88% to 100% are neurologically intact.[7]

Approximately 50% of maternal mortality is potentially preventable by an improved health care system.[8] Although this is not a daily occurrence, the prudent provider is skilled in the techniques of cardiopulmonary resuscitation (CPR) including basic life support (BLS) and advanced cardiac life support (ACLS). Providers must be familiar with the underlying causes of arrest, including those unique to pregnancy and those present in the general population. Furthermore, they must understand the aspects of maternal physiology that influence resuscitative efforts and the evaluation and management of trauma in pregnancy (see the Trauma section below). Fetal outcomes are directly related to the well-being of the woman.

Etiology and Differential Diagnosis

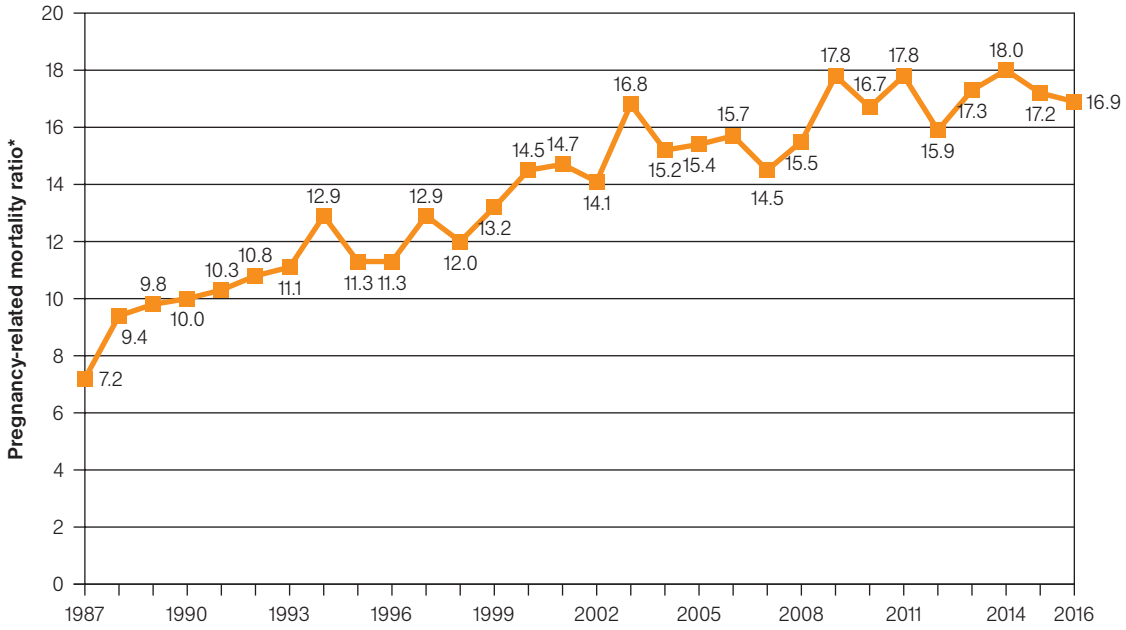
Maternal resuscitation may be required because of pregnancy specific conditions, conditions not specific to pregnancy, or trauma.

Providers should be familiar with conditions specific to pregnancy and childbirth and try to identify these potentially reversible causes of cardiac arrest during resuscitation attempts.[8] Therefore, this section first examines causes of cardiac arrest unique to pregnancy, including amniotic fluid embolism (AFE), magnesium toxicity, preeclampsia/eclampsia, and postpartum hemorrhage (PPH). Then the causes of cardiac arrest that are not limited to pregnancy, including acute coronary syndrome, cerebrovascular accident, aortic dissection, and pulmonary embolism (PE), are reviewed. *Table 1* summarizes the conditions that may be associated with cardiac arrest.[9] Finally, complications caused by trauma are discussed.

The same reversible causes of cardiac arrest that occur in women who are not pregnant can occur during pregnancy.[10] Increasingly, women are presenting for maternity care with serious medical conditions including type 1 diabetes, cystic fibrosis, hemoglobinopathies, steroid dependent asthma, opioid overdose, congenital and acquired heart disease, and transplanted organs. Advanced reproductive technologies make it possible for older women and women with medical conditions typically associated with infertility to become pregnant. These demographic- and condition-specific risk factors increase the likelihood of cardiac arrest.[11-18]

Amniotic fluid embolism. The pathophysiology of AFE is not completely understood. Historically, the condition has been thought to be a hypersensitivity reaction to fetal or amniotic fluid antigen in the maternal circulation; however, recent evidence shows that it may result from the activation of the immune system's complement cascade.[19] During labor or other procedures, amniotic fluid or some other fetal substance enters the maternal circulation and triggers a massive anaphylactic reaction, activation of the complement cascade, or both. The trigger is not certain. Initially, the combination of pulmonary artery vasospasm, pulmo-

Figure 1. Trends in Pregnancy-Related Mortality in the United States: 1987-2016



**Note: Number of pregnancy-related deaths per 100,000 live births per year.*

Reprinted from the Centers for Disease Control and Prevention. Pregnancy Mortality Surveillance System. 2020. Available at https: //www.cdc.gov/reproductivehealth/maternalinfanthealth/pregnancy-mortality-surveillance-system.htm.

Table 1. Nonobstetric and Obstetric Causes of Cardiopulmonary Arrest in Pregnancy

Nonobstetric Causes	Obstetric Causes
Anesthesia complications	Amniotic fluid embolism
Aortic dissection	Eclampsia
Bleeding (nonuterine)	HELLP syndrome
Cardiac disease (acute coronary syndrome, myocardial infarction)	Magnesium toxicity
Cerebrovascular accident	Postpartum hemorrhage
Disseminated intravascular coagulopathy	Preeclampsia
Opioid overdose	Uterine atony
Sepsis	Peripartum cardiomyopathy
Thromboembolism	
Trauma	

Information from Cantwell R, Clutton-Brock T, Cooper G, et al. Saving Mothers' Lives: Reviewing maternal deaths to make motherhood safer: 2006-2008. The Eighth Report of the Confidential Enquiries into Maternal Deaths in the United Kingdom. BJOG. *2011;118(Suppl 1):1-203; Campbell TA, Sanson TG. Cardiac arrest and pregnancy.* J Emerg Trauma Shock. *2009;2(1):34-42; Jeejeebhoy FM, Zelop CM, Lipman S, et al; American Heart Association Emergency Cardiovascular Care Committee, Council on Cardiopulmonary, Critical Care, Perioperative and Resuscitation, Council on Cardiovascular Diseases in the Young, and Council on Clinical Cardiology. Cardiac Arrest in Pregnancy: A Scientific Statement From the American Heart Association.* Circulation. *2015;132(18):1747-1773.*

nary hypertension, and elevated right ventricular pressure cause hypoxia. Hypoxia leads to myocardial and pulmonary injury, left heart failure, and acute respiratory distress syndrome. Subsequent manifestations of this syndrome are massive hemorrhage with uterine atony and disseminated intravascular coagulopathy (DIC). Cardiopulmonary bypass has been shown to be successful in women with life-threatening AFE during labor and delivery.[20] This condition is discussed in more detail in the Amniotic Fluid Embolism section of this chapter.

Preeclampsia/eclampsia. Preeclampsia/eclampsia develops after 20 weeks' gestation and can produce severe hypertension, which can ultimately result in diffuse organ system failure. If untreated, it may result in maternal and fetal morbidity and mortality. The spectrum of hypertensive diseases in pregnancy is addressed in the *Hypertensive Disorders of Pregnancy* chapter.

Excess magnesium sulfate. Iatrogenic overdose is possible in women with preeclampsia/eclampsia who receive magnesium sulfate, particularly if the woman becomes oliguric. Cardiac and pulmonary manifestations include respiratory depression,

prolonged atrioventricular conduction, complete heart block, and cardiac arrest. Magnesium sulfate should be discontinued in this scenario because the risks greatly outweigh the benefits. The treatment of choice for magnesium toxicity is calcium gluconate (1 g administered intravenously [IV] over 3 minutes. Repeat doses may be necessary). Many adult code carts do not contain calcium gluconate. If calcium gluconate is not available, 500 mg calcium chloride administered IV over 5 to 10 minutes is an acceptable and equally effective alternative. Empiric calcium administration may be lifesaving.[21,22]

Postpartum hemorrhage. PPH accounts for a large percentage of maternal death and is discussed in detail in the *Postpartum Hemorrhage* chapter. Remember the Four Ts (Tone, Trauma, Tissue, and Thrombin) and invisible hemorrhage (uterine rupture).

Aortic dissection/pulmonary embolism/stroke. Women who are pregnant are at increased risk of spontaneous aortic dissection, life-threatening PE, and stroke. Use of fibrinolytic drugs has been shown to be an effective treatment for a massive, life-threatening PE or ischemic stroke in women who are pregnant, but are relatively contraindicated.[23]

Nonaccidental trauma and drug overdose. The rate of intimate partner violence (IPV) increases during pregnancy,[24] and homicide and suicide are leading causes of death during pregnancy.[25] Drug errors that may have caused the arrest should be considered and new IV preparations may be reformulated for each drug. Opioid use during pregnancy increased by 300% from 2002 to 2014.[9] The incidence of acute cardiac events among women increased by 50% during this 13-year period.[9]

Acute coronary syndromes. Women who are pregnant may experience acute coronary syndromes, typically in association with other medical conditions. Because fibrinolytic drugs are relatively contraindicated in pregnancy, percutaneous coronary intervention is the reperfusion strategy of choice for ST-elevation myocardial infarction.[23]

Cardiac disease has become the leading cause of maternal deaths overall, exceeding the rates of death from sepsis, hypertension, thrombosis, and amniotic fluid embolism.[26] In the United States, between 1995 and 2006, the rate of hospitalization for postpartum women with chronic heart disease approximately tripled.[27]

Maternal Physiologic Changes in Pregnancy

Cardiovascular

Pregnancy is a high-flow, low-resistance state. The uterine arteries lack autoregulation, so uterine perfusion decreases with any decrease in maternal blood pressure. The uteroplacental vascular bed functions as a maximally dilated, passive, low-resistance system so that uterine blood flow is determined by perfusion pressure.

Management of cardiac arrest or trauma must balance the need for sufficient fluid volume to preserve uteroplacental blood flow with the tendency of the capillaries to leak because of the pregnancy-related reduction of oncotic pressure. As summarized in *Table 2*, these adaptations of pregnancy make the maternal-fetal unit susceptible to deleterious effects of ineffective circulation.[6,28]

Up to 30% of the cardiac output flows to the uterus in women who are pregnant, compared with less than 2% in woman who are not pregnant.[10] In women who are pregnant and in the supine position, the weight of the gravid uterus can compress the aorta and inferior vena cava enough to sequester approximately 27% of the blood volume in the lower extremities by 20 weeks' gestation.[29] A surgeon would have to completely occlude the vessel manually to achieve this same degree of compression of the vena cava. Delivery or left uterine displacement (LUD) relieves aortocaval compression and increases cardiac output in women with hypotension.[30]

The thorax is less compressible by external pressure because of the cephalad displacement of the abdominal contents, hypertrophied breasts, and the presence of the gravid uterus. Women who are pregnant with hemorrhage may lose 1,200 to 1,500 mL of their blood volume before exhibiting signs of hypovolemia.[31] The first indication of significant hemorrhage may be an abnormal fetal heart rate pattern or maternal tachycardia. Fluid resuscitation is especially important in pregnancy.

Estrogen increases excitability in uterine muscle fibers and is thought to have a similar effect on cardiac excitability. Although catecholamine levels do not appear to change during pregnancy, estrogen increases sensitivity to them by increasing the number of myocardial alpha-adrenergic receptors. This effect may increase the propensity for supraventricular arrhythmias.[32]

Table 2. Physiologic Changes of Pregnancy That Affect Resuscitation

Cardiovascular		Effect
Increased	Plasma volume	Dilutional anemia results in decreased oxygen carrying capacity
	Erythrocyte volume	
	Cardiac output up to 50%	Increased CPR circulation demands
	Heart rate by 15 to 20 BPM	Increased CPR circulation demands
	Clotting factors susceptible to thromboembolism	
	Dextrorotation of the heart	Increased ECG left axis deviation
	Estrogen effect on myocardial receptors	Supraventricular arrhythmias
Decreased	Supine blood pressure and venous return with aortocaval compression	Decreased cardiac output by 30%
	Arterial blood pressure by 10 to 15 mm Hg	Susceptible to cardiovascular insult
	Systemic vascular resistance	Sequesters blood during CPR
	Colloid oncotic pressure	Susceptible to third spacing
	Pulmonary capillary wedge pressure	Susceptible to pulmonary edema
Respiratory		**Effect**
Increased	Respiratory rate (progesterone-mediated)	Decreased buffering capacity
	Oxygen consumption by 20%	Rapid decrease of PaO_2 in hypoxia
	Tidal volume (progesterone-mediated)	Decreased buffering capacity
	Minute ventilation	Compensated respiratory alkalosis
	Laryngeal angle	Failed intubation
	Pharyngeal edema	Failed intubation
	Nasal edema	Difficult nasal intubation
Decreased	Functional residual capacity up to 25%	Decreases ventilatory capacity
	Arterial PCO_2	Decreases buffering capacity
	Serum bicarbonate	Compensated respiratory alkalosis
Gastrointestinal		**Effect**
Increased	Intestinal compartmentalization	Susceptible to penetrating injury
Decreased	Peristalsis, gastric motility	Aspiration of gastric contents
	Gastroesophageal sphincter tone	Aspiration of gastric contents
Uteroplacental		**Effect**
Increased	Uteroplacental blood flow by 30% of cardiac output	Sequesters blood in CPR
	Aortocaval compression	Decreases cardiac output by 30%
	Elevation of diaphragm	Aspiration of gastric contents
Decreased	Autoregulation of blood pressure	Uterine perfusion decreases with drop in maternal blood pressure
Renal/Urinary		**Effect**
Increased	Compensated respiratory alkalosis	Decreases buffering capacity and increases acidosis during CPR
	Ureteral dilation, especially right side	Interpretation of x-rays
Decreased	Bladder emptying	Interpretation of x-rays

BPM = beats per minute; CPR = cardiopulmonary resuscitation; ECG = electrocardiography.

Information from Bennett TA, Katz VL, Zelop CM. Cardiac arrest and resuscitation unique to pregnancy. Obstet Gynecol Clin North Am*. 2016;43(4):809-819; Jeejeebhoy FM, Zelop CM, Lipman S, et al; American Heart Association Emergency Cardiovascular Care Committee, Council on Cardiopulmonary, Critical Care, Perioperative and Resuscitation, Council on Cardio-vascular Diseases in the Young, and Council on Clinical Cardiology. Cardiac arrest in pregnancy: a scientific statement from the American Heart Association.* Circulation*. 2015;132(18):1747-1773.*

Pulmonary

Progesterone increases tidal volume and respiratory rate, which increases the amount of expired carbon dioxide and decreases the amount of carbon dioxide dissolved in serum. This chronic hyperventilation results in a compensated respiratory alkalosis with a decreased serum bicarbonate. During pregnancy, maternal apnea is associated with rapid declines in arterial pH and PaO_2. These changes result in decreased buffering capacity compared with the nonpregnant state and make the woman who is pregnant more susceptible to organ damage from hypoventilation and hypoxia due to greater levels of acidosis.

Obesity makes successful CPR more challenging because of difficulty with intubation and the ability to deliver adequate chest compressions.

Chronic hypocapnia ($PaCO_2$ less than 30 mm Hg) is common in late pregnancy. Therefore, a $PaCO_2$ of 35 to 40 mm Hg (within the normal range for adults who are not pregnant) is likely abnormal in pregnancy and may indicate impending respiratory failure.[33-35] Oxygen consumption is increased in pregnancy, so maintenance of arterial oxygenation is especially important during resuscitation.

Women who are pregnant have decreased functional residual capacity and functional residual volume but have increased tidal volume and minute ventilation. It will be necessary to tailor ventilatory support because of these pregnancy-related metabolic changes.

Given these maternal physiologic changes and the cardiovascular constraints from the intra-abdominal presence of the fetus, maternal resuscitative efforts are greatly compromised. Although CPR in adults who are not pregnant may result in achieving up to 30% of the normal cardiac output, CPR during pregnancy may be closer to only 10%.[36]

Fetal oxygen requirements. The fetus of a woman with apnea and no pulse has 2 minutes or less of oxygen reserve because the oxygen tension in the umbilical vein is always less than in the uterine vein. The likelihood of successful resuscitation of the woman and/or fetus decreases after 4 minutes of cardiac arrest. Therefore, the provider has only 4 minutes to effect return of spontaneous circulation (ROSC) before resorting to more drastic interventions, thus the emphasis on the *4-minute rule.*[5,37]

Evacuation of the gravid uterus relieves aortocaval compression and may improve resuscitative efforts by quickly increasing the cardiac output achieved through CPR.[3] The best fetal survival rate occurs when the fetus is delivered no more than 5 minutes after the woman's heart stops beating, and in no cases of resuscitative hysterotomy has a worsening of the maternal condition been shown.[5,37] The American Heart Association (AHA) goal of achieving delivery of the fetus by 5 minutes[28] typically requires the provider to start resuscitative hysterotomy (previously referred to as perimortem cesarean delivery) approximately 4 minutes after maternal cardiac arrest onset.[28]

Resuscitation in Pregnancy

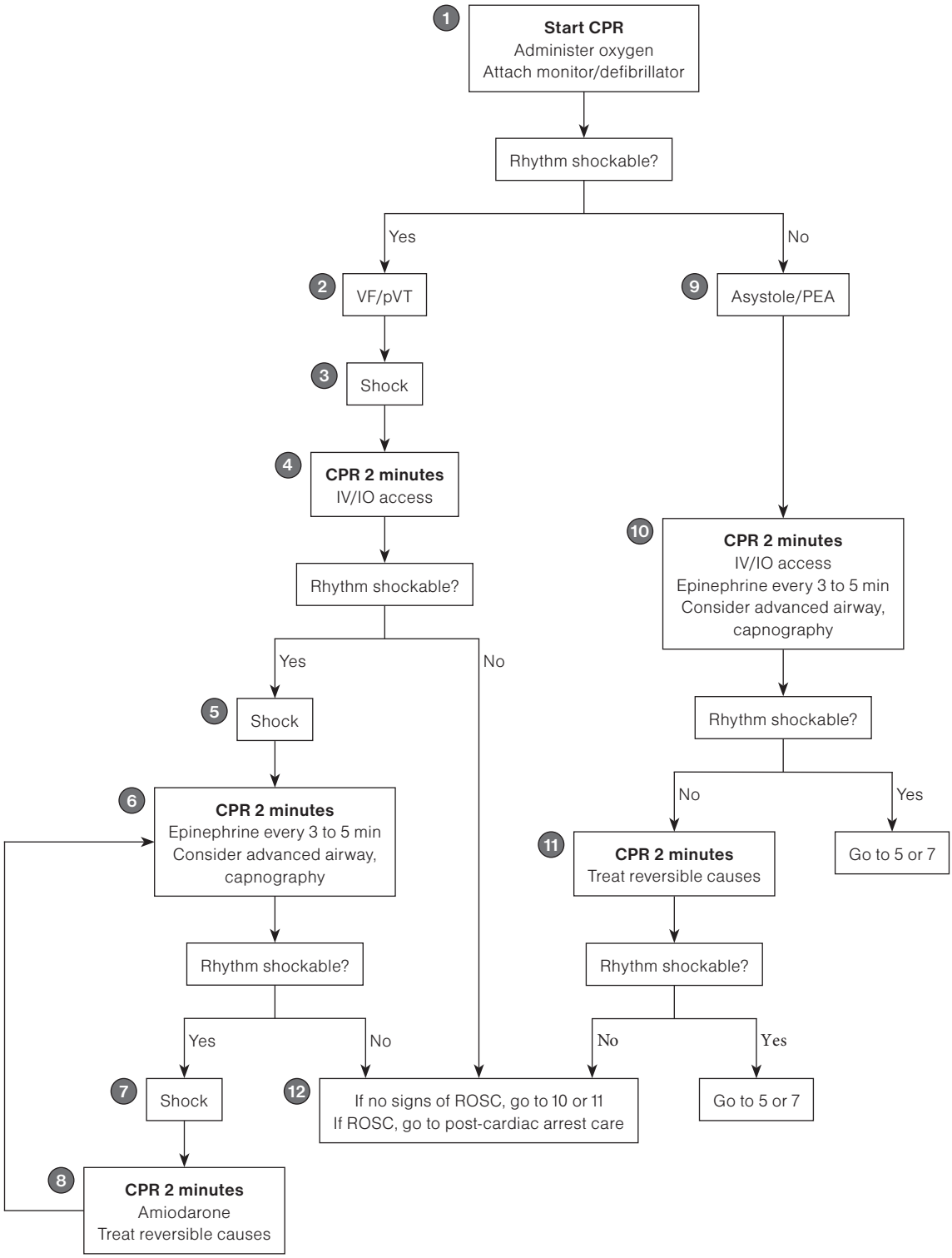
The following discussion includes an overview of the principles of BLS and ACLS but presumes a familiarity with the AHA algorithms and CPR (*Figures 2* and *3*).[28,30] In 2010, the AHA changed the sequence of BLS steps from *airway-breathing-circulation* (A-B-C) to *circulation-airway-breathing* (C-A-B). In pregnancy, the sequence should be: chest compressions/current airway-breathing-uterine displacement (C-A-B-U).[28] ACLS providers in hospitals should tailor the sequence of rescue actions to the most likely cause of arrest. Modifying techniques because of changes in maternal physiology is recommended (*Table 3*).[28,30]

Chest compressions should be performed at a rate of at least 100 per minute at a depth of at least 2 inches while allowing chest recoil between compressions.[28] If available, bag mask ventilation should be performed. Use 100% oxygen and administer 2 breaths per every 30 compressions.[28] Bag mask ventilation with optimal oral and/or nasal airways should be administered by two maternity care team members with 100% oxygen.[38,39] Apply the bag valve mask with a two-hand lifting maneuver and an oral airway or two-nasal airway, and use high-flow oxygenation.[38,39]

Complications of Cardiopulmonary Resuscitation

Women who are pregnant are more susceptible to rib fractures and other iatrogenic injuries (eg, liver lacerations, pneumothorax) as a result of CPR. Therefore, a high index of suspicion is warranted when evaluating for these complications. Thrombocytopenia may predispose women with underlying toxemia to bleeding and hematomas of the liver.

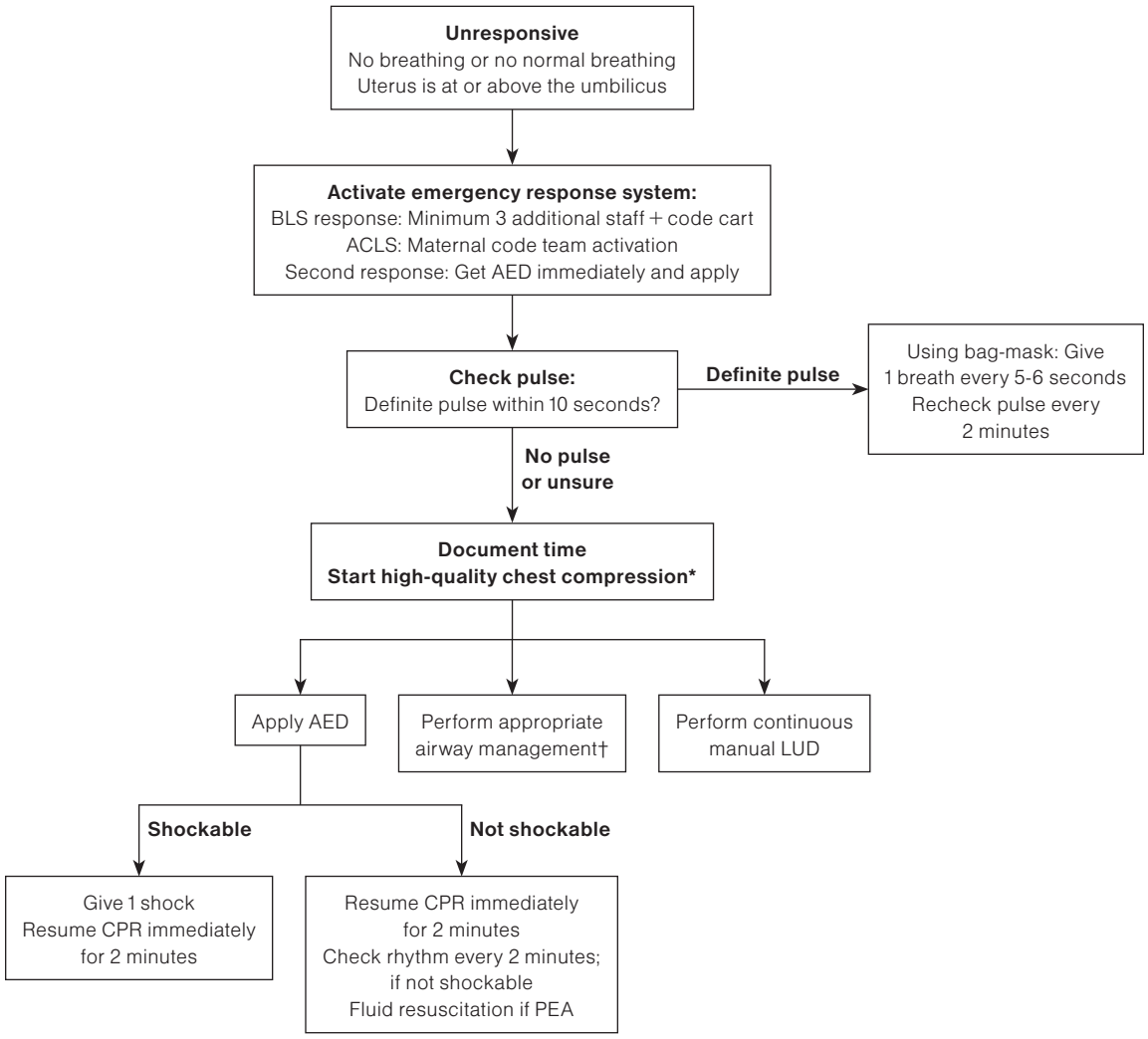
Figure 2. Nonpregnant Adult Arrest Algorithm



CPR = cardiopulmonary resuscitation; IO = intraosseous; IV = intravenous; PEA = pulseless electrical activity; pVT = pulseless ventricular tachycardia; ROSC = return of spontaneous circulation; VF = ventricular fibrillation.

Adapted from American Heart Association. Part 7: Adult Advanced Cardiovascular Life Support. 2015. Available at https://eccguidelines.heart.org/index.php/circulation/cpr-ecc-guidelines-2/part-7-adult-advanced-cardiovascular-life-support/.

Figure 3. Cardiac Arrest in Pregnancy In-Hospital Basic Life Support Algorithm



Chest compressions in pregnancy:

- Use a firm backboard
- Place patient supine
- Place hands in center of chest (as in nonpregnant patient)
- Compress at a rate of at least 100/min
- Compress at a depth of at least 2 inches (5 cm)
- Perishock pause <10 seconds
- Allow complete chest recoil after each compression
- Minimize interruptions
- Perform continuous manual LUD

Appropriate airway management for pregnancy:

- Open airway by using head tilt-chin lift maneuver (if not a trauma victim)
- Administer 100% O_2 at ≥15 L/min
- When available, perform bag-mask ventilation
  - Seal mask, ensure no leak around mask; 2-handed technique preferred
  - Deliver each rescue breath over 1 second
  - Give 2 breaths for every 30 compressions
  - Give a sufficient tidal volume to produce visible chest rise or fog within face mask. If not seen, reopen airway and improve seal. Consider using oral airway.
- Avoid excessive ventilation

ACLS = advanced cardiac life support; AED = automated external defibrillator; BLS = basic life support; CPR = cardiopulmonary resuscitation; LUD = left uterine displacement; PEA= pulseless electric activity.

Reprinted from Jeejeebhoy FM, Zelop CM, Lipman S, et al; American Heart Association Emergency Cardiovascular Care Committee, Council on Cardiopulmonary, Critical Care, Perioperative and Resuscitation, Council on Cardiovascular Diseases in the Young, and Council on Clinical Cardiology. Cardiac arrest in pregnancy: a scientific statement from the American Heart Association. Circulation*. 2015;132(18):1747-1773.*

Table 3. Possible Modifications of Resuscitative Efforts in Pregnancy/Special Circumstances

Action	Rationale
Basic Life Support	
Manual left uterine displacement	Decreases aortocaval compression
Defibrillation: Remove fetal and uterine monitors	May prevent monitors from functioning in the future
Advanced Cardiac Life Support	
Early tracheal intubation, use short laryngoscope handle and smaller endotracheal tube	Difficult ventilation with pharyngeal edema, breast hypertrophy, diaphragmatic elevation
Consider other etiologies (eg, magnesium toxicity, hypoxemia)	Tocolytic therapy Increased oxygen requirement
Consider placement of adhesive defibrillator pads, or left breast displacement to place pad at apex	Dextrorotation of heart, breast hypertrophy
Verify endotracheal tube with CO_2 detector	Esophageal detector more likely to not re-inflate after a compression
Ventilation volumes and rates altered	Tailor ventilatory support to oxygenation and ventilation
Start resuscitative hysterotomy at 4 minutes of cardiac arrest	Decreases aortocaval and venous compression
Remove fetal and uterine monitors	Allows quicker access to abdomen for resuscitative hysterotomy. Recommend removing prior to shocking, and clear patient prior to defibrillation
No Change	
Defibrillation regimen	Early return of effective maternal circulation
Pharmacologic therapy	Early return of effective maternal circulation

Information from Jeejeebhoy FM, Zelop CM, Lipman S, et al; American Heart Association Emergency Cardiovascular Care Committee, Council on Cardiopulmonary, Critical Care, Perioperative and Resuscitation, Council on Cardiovascular Diseases in the Young, and Council on Clinical Cardiology. Cardiac arrest in pregnancy: a scientific statement from the American Heart Association. Circulation*. 2015;132(18):1747-1773.*

Modifications of Cardiopulmonary Resuscitation in Pregnancy

As summarized in *Table 2*, many unique aspects of maternal physiology influence the conduct of resuscitative maneuvers in pregnancy. Left lateral tilt chest compressions result in less effective compressions than in the supine position.[40,41] To prevent aortocaval compression by the uterus after 20 weeks' gestation,[42] the gravid uterus should be manually moved to the left so the weight of the uterus is shifted off the aorta, vena cava, and pelvic great vessels.[43] This maneuver should occur continuously during resuscitation and take precedence over initiating other interventions. In addition, prompt placement of the cardiac backboard during pregnancy is important to ensure high-quality chest compressions can be delivered. This is shown in *Figure 4.*

Manual Left Uterine Displacement

A systematic review showed that chest compressions were less forceful when a patient was in full-body left lateral tilt compared with the supine position.[44] Furthermore, manual LUD has been shown to reduce the incidence of hypotension and reduce ephedrine requirements compared with 15-degree left lateral tilt in women undergoing routine cesarean delivery.[45] Based on these findings and the 2015 AHA recommendations for cardiac arrest in pregnancy, left lateral tilt is no longer a recommended method of uterine displacement during CPR.[28,30] Instead, manual LUD provides an alternative technique for aortocaval decompression that allows the woman to remain supine and receive concurrent, higher quality chest compressions while not delaying the onset of effective chest compressions because of a need to facilitate a

total body tilt. Manual LUD also allows for easier delivery of defibrillation, IV access, and intubation, as well as quicker access to the abdomen should resuscitative hysterotomy be required. Two common methods include the two-handed technique (*Figure 4*) and a one-handed technique.

The most important component of high-quality CPR in the pregnant woman is to perform effective chest compressions. In summary, this is achieved by keeping the woman supine and manually providing LUD (eg, displace the uterus leftward) paired with performing chest compressions at a rate of at least 100 per minute (at a depth of at least 2 inches) with the woman placed on a firm backboard.

Resuscitative Hysterotomy

Despite all the appropriate maneuvers, including continuous LUD, the ability to deliver high-quality compressions is severely limited in pregnancy. CPR in women who are pregnant should never be considered effective circulation. Therefore, resuscitative hysterotomy may be needed to relieve the aortocaval compression caused by the fetus and restore maternal circulation (*Tables 4* and *5*).[46]

Few published cases describe vaginal delivery during a cardiac arrest in pregnancy.[4] Maternity care providers involved in an intrapartum cardiac arrest resuscitation may conduct a vaginal examination, provided that CPR is being adequately performed by the medical team. If the cervix is fully dilated and the fetal head is at an appropriately low station, immediate assisted vaginal delivery can be considered.[4,28]

Historically, resuscitative hysterotomy was performed to facilitate separate burials for the woman and fetus. The procedure has re-emerged as a means to increase the survival of the woman and fetus after maternal cardiac arrest.[5,37,47] The AHA recommends that if ROSC has not occurred after 4 minutes of ACLS, then delivery by resuscitative hysterotomy should be performed within 5 minutes of resuscitative efforts.[28,30]

Resuscitative hysterotomy should be considered when:

- The woman does not benefit within 4 minutes from attempts to restore ROSC
- The woman is at 20 weeks' gestation or greater or has a uterus that is at the level of the umbilicus or higher[28,48]
- Appropriate facilities and skilled personnel are available to perform the procedure and to care

Figure 4. Two-Handed Manual Left Uterine Displacement Technique

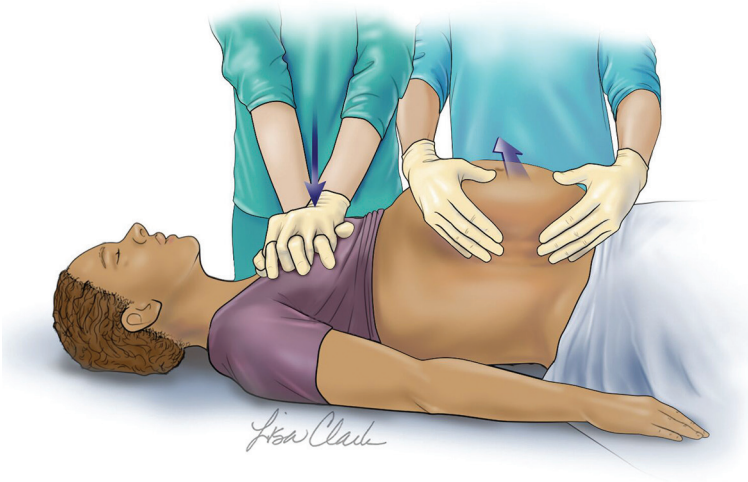


Table 4. Managing Cardiac Arrest in Pregnant Women

Activate cardiac arrest team (code blue)
Lay woman in the supine position on a backboard and manually displace the uterus to the left
Use 100% oxygen when ventilating. Secure advanced airway early in the resuscitation
Remove fetal and/or uterine monitors
Administer typical ACLS drugs and doses
Entire team should prepare for a possible resuscitative hysterotomy
If no ROSC by 4 minutes of resuscitation, prepare for resuscitative hysterotomy
Team should not wait for surgical equipment to begin the procedure; only a scalpel is required
Team should not spend time on lengthy antiseptic procedures. An abbreviated antiseptic pour should be performed, or the step should be eliminated entirely
Perform resuscitative hysterotomy in location of the arrest. No need to delay procedure for transportation
Delivery should occur within 5 minutes of the beginning of cardiac arrest if there is no ROSC with effective CPR
Continuous manual LUD should be performed throughout the cesarean delivery until the fetus is delivered
If maternal viability is not possible (fatal injury or prolonged pulselessness), the team does not have to wait to begin the resuscitative hysterotomy
Assisted vaginal delivery should be considered if the cervix is fully dilated and the fetal head is at an appropriately low station

ACLS = advanced cardiac life support; CPR = cardiopulmonary resuscitation; LUD = left uterine displacement; ROSC = return of spontaneous circulation.

Information from Jeejeebhoy FM, Zelop CM, Lipman S, et al; American Heart Association Emergency Cardiovascular Care Committee, Council on Cardiopulmonary, Critical Care, Perioperative and Resuscitation, Council on Cardiovascular Diseases in the Young, and Council on Clinical Cardiology. Cardiac arrest in pregnancy: a scientific statement from the American Heart Association. Circulation. *2015;132(18):1747-1773.*

for the woman and newborn after the procedure (*Table 6*).

The maternity care team must remain calm and avoid chaos. This may be better achieved through performing in-situ practice drills with all relevant clinical personnel present.[3,49] The best fetal survival rates are achieved when resuscitative hysterotomy is performed within 5 minutes of ineffective maternal circulation.[5,37] In a cohort of 45 surviving newborns delivered within 5 minutes of maternal death, there was a 98% intact neurologic status. This status decreased to 33% for 9 infants surviving a delivery that occurred 16 to 25 minutes after maternal death.[50]

In a 2012 systematic review of 94 published cases of maternal cardiac arrests, 54% of women survived to hospital discharge. Resuscitative hysterotomy was performed in 76 (87%) viable pregnancies.[51] Although approximately two-thirds of the arrests occurred in highly monitored areas of the hospital and 89% of the arrests were witnessed, resuscitative hysterotomy was determined to have benefited the woman in approximately 32% of cases and to have caused no harm in any of the others. In this series, in-hospital arrest and resuscitative hysterotomy within 10 minutes were associated with better maternal outcomes.[51] It may still be worthwhile to pursue delivery after this period. Some fetuses have survived up to 20 to 30 minutes after ineffective circulation.[3,5,37]

There still may be benefit, and resuscitative hysterotomy should be considered even if delivery does not occur within 4 to 5 minutes.[46] Injury-free survival rates as high as 50% have been shown for women and neonates undergoing resuscitative hysterotomy as late as 25 minutes after maternal cardiac arrest.[52] As more rapid resuscitative hysterotomy has been associated with improved survival,[53] the procedure can also be considered as soon as initial resuscitative measures are unsuccessful without waiting for 4 minutes to elapse when maternal ROSC is unlikely.[46,52]

In the prehospital trauma care setting, resuscitative hysterotomy may still be helpful beyond a 5-minute time frame if resuscitative efforts are ongoing.[54] A 2012 systematic review showed that the predictors of maternal survival for women undergoing resuscitative hysterotomy were maternal arrest occurring in-hospital and resuscitative hysterotomy occurring less than 10 minutes after maternal arrest.[51]

With resuscitative hysterotomy, maternal survival rates increase because removal of the fetus results in an improvement in maternal circulation during CPR. A 2005 study showed sudden and often profound improvement in 12 of 18 women, including return of pulse and blood pressure at the time the uterus was evacuated.[37] Uterine evacuation during resuscitative hysterotomy at term can raise cardiac output and facilitate resuscitation by relieving aortocaval compression. When there is an obvious gravid uterus, the resuscitative hysterotomy team should be activated at the onset of maternal cardiac arrest.[28,30]

Resuscitative hysterotomy (see the *Cesarean Delivery* chapter) should not be delayed to listen for fetal heart tones or perform an ultrasonography study to document gestational age or fetal viability. Omitting or delaying a resuscitative hysterotomy may lead to the unnecessary death of both the woman and fetus. In a randomized simulation setting, the total time to incision for resuscitative hysterotomy performed in the labor room was sig-

Table 5. Steps in Performing Resuscitative Hysterotomy

Initiate immediate CPR and ACLS with continuous manual LUD
Initiate resuscitative hysterotomy at 4 minutes of ACLS and no ROSC
Attempt procedure with an obviously gravid uterus at or above the level of the umbilicus or known gestation greater than 20 weeks
Prepare equipment/personnel for resuscitative hysterotomy and neonatal resuscitation. Avoid delays in obtaining fetal heart tones or waiting for an obstetrician or surgeon to perform the resuscitative hysterotomy
The first skilled clinician available should initiate the resuscitative hysterotomy in the location of arrest. Do not transport the woman elsewhere for surgery
Perform resuscitative hysterotomy with a vertical midline skin incision, and a vertical uterine incision. Use a modified sterile technique. Give infant to attendant for drying and warming and/or resuscitation
Pack the uterus with moist lap sponges. Discontinue continuous manual LUD. Continue performing ACLS throughout the procedure
When hemodynamically stable, remove the placenta and close uterus with No. 0 absorbable suture. Close anatomically, depending on available personnel and location. Obtain hemostasis with interrupted No. 0 absorbable suture and transfer to intensive care unit

ACLS = advanced cardiac life support; CPR = cardiopulmonary resuscitation; LUD = left uterine displacement; ROSC = return of spontaneous circulation.

Information from Jeejeebhoy FM, Zelop CM, Lipman S, et al; American Heart Association Emergency Cardiovascular Care Committee, Council on Cardiopulmonary, Critical Care, Perioperative and Resuscitation, Council on Cardiovascular Diseases in the Young, and Council on Clinical Cardiology. Cardiac arrest in pregnancy: a scientific statement from the American Heart Association. Circulation*. 2015;132(18):1747-1773.*

nificantly shorter than that of the same procedure performed after moving to the operating room.[55] Personnel with the appropriate skill and equipment should perform this procedure. An obviously gravid uterus, which correlates to approximately 20 weeks' gestational age or a fundal height at or above the umbilicus, is deemed clinically to be large enough to cause aortocaval compression.[28,30]

An immediate cesarean delivery is the best way to optimize the condition of the woman and fetus, thus the procedure should occur at the location of the arrest. A pregnant woman with in-hospital cardiac arrest should not be transported to the operating room for cesarean delivery.[28]

Consent from family members before performing the procedure is helpful but not necessary. It is the responsibility of the health care provider to perform a resuscitative hysterotomy if the above criteria are met. As long ago as 1931, it was concluded that "a civil action for damages might follow for the negligence or the malpractice of the surgeon or obstetrician in failing to follow the usual and customary practice" regarding resuscitative hysterotomy.[56] Present-day health care providers and facilities should remain aware of the medicolegal consequences of delayed resuscitation.[57] The surgical team should be prepared to care for the newborn. A newborn can lose 30% of available energy reserves in the first 5 minutes in a cold, moist environment, so immediate drying and warming is indicated.

Useful instruments for performing a resuscitative hysterotomy are listed in *Table 7*. If such instruments are not available, a scalpel to perform the delivery and a blanket for the infant are the items that are immediately necessary. Antibiotics and wound management will be necessary if the woman survives.

Some medical conditions indicate a need for simultaneous CPR and resuscitative hysterotomy (eg, mitral and aortic stenosis, cardiomyopathy, pericardial disease, core temperature less than 34°C [93.2°F], pulmonary/cardiac injury or disease, carbon monoxide poisoning).[23] If the woman's hemodynamic status is stabilized, then resuscitative hysterotomy is no longer indicated. Sufficiently restoring maternal function to maintain the pregnancy may be of value for the fetus and the family, even if recovery is incomplete. This is especially important the further from term the woman is. Even patients sustaining irreparable trauma deserve attentive assessment and life support.

Table 6. Resuscitative Hysterotomy Decision Factors to Consider for Maternal Arrest

	Key Factors
Response Teams	Immediate and effective communication that an emergency is occurring
	Aware of the fastest routes to the labor and delivery unit, emergency department, and all intensive care units
	Closed-loop communication during resuscitation
	Assignment of roles, detailed transcriber with times. Code leader should be an individual with knowledge of the treatment of pregnant women who is not task-saturated, can communicate effectively, and periodically reassess management goals and outcomes
	Is resuscitative hysterotomy within the rescuer's skill set?
Maternal Factors	Consider if CPR efforts are effective:
	Assess if the woman has benefited from any arrest interventions
	Consider if there are reversible causes for the arrest
Infant Factors	Estimate gestational age and consider survival rate
	Increased fetal viability is estimated at 23 to 24 weeks' gestation
	Consider the status of the fetus at the time of arrest
	Key Interventions
Response Teams	Ensure appropriate equipment and supplies are available. The most important item is a scalpel; resuscitative hysterotomy must not be delayed while waiting for a cesarean tray
	Notify neonatal support personnel
	Immediately prepare for resuscitative hysterotomy at first recognition of maternal arrest
Maternal Factors	Continuous LUD to relieve aortocaval compression.
	CPR compression depth of at least 2 inches; 100 compressions/minute, changing compressors every 2 minutes (do not discontinue chest compression, except during defibrillation)
	Perform intubation early, administer oxygen at 100%. Consider the increased risk of pregnancy-related complications in airway management
	Defibrillate according to AHA guidelines
	Administer IV drugs above the diaphragm
	Continue CPR throughout, and make an incision by 4 minutes after the start of maternal arrest to deliver the fetus by 5 minutes after the start of cardiac arrest
Infant Factors	Neonatal survival may be greatest (if past viability at 23 to 24 weeks' gestation) when the fetus is delivered within 5 minutes

ACLS = advanced cardiac life support; BLS = basic life support; AHA = American Health Association; CPR = cardiopulmonary resuscitation; IV = intravenous; LUD = left uterine displacement.

Information from various sources.

Table 7. Resuscitative Hysterotomy Kit

Obstetric Supplies
Knife handle, No. 10 scalpel blade
Bandage and dissecting scissors
Sterile laparotomy sponges
Betadine splash
Cord clamps
Gloves

Pediatric Supplies
Neonatal blankets
Self-inflating resuscitation bag (infant, child)
Resuscitation masks (neonate, infant)
DeLee suction trap
Bulb syringe

Alternative Interventions

Direct cardiac massage performed through an extension of a vertical midline abdominal incision may provide enhanced organ perfusion.[6] Systematic reviews of extracorporeal life support showed maternal survival rates of 77.8% to 80% and fetal survival rates of 65.1% to 70%.[58,59]

Trauma in Pregnancy

Trauma complicates 6% to 7% of all pregnancies and is the leading nonobstetric cause of death among women who are pregnant.[10,60] The most common traumatic injuries to women who are pregnant are: motor vehicle collisions (MVCs) (48% to 55%), falls (22% to 25%), assault (17% to 22%), suicide (3.3%), IPV, homicide and gunshot wounds (4%), poisoning, and burns (1%).[61,62] Nine out of 10 traumatic injuries during pregnancy are minor; however, 60 to 70% of fetal losses are because of minor injuries.[61] Trauma can also be divided into minor and major.

Women who are pregnant frequently present to emergency departments, urgent care, or primary care offices with minor trauma (eg, accidents, cuts, sprained ankle without a fall). Women with minor trauma do not report abdominal pain, vaginal bleeding, or loss of fluid, and report feeling adequate fetal movement.[63] However, even minor trauma can be associated with fetal demise.[64] Major trauma may include MVCs or falls in which rapid compression, deceleration, or shearing forces have been applied in some way to the abdomen/uterus. Women with major trauma may report abdominal pain, vaginal bleeding, or loss of fluid, and may report decreased fetal movement. Major trauma is associated with intermediate and long-term adverse pregnancy outcomes including preterm labor, placental abruption, and perinatal morbidity.[64]

This section includes a review of the anatomic and physiologic changes of pregnancy important in evaluating and treating women with trauma. The evaluation and treatment of major trauma is reviewed, and an evaluation protocol for women who are pregnant who experience blunt abdominal trauma and falls is presented. This section concludes with a review of injuries sustained by women who are pregnant in MVCs and physical assaults.

Anatomy and Physiology Relating to Trauma

Many anatomic and physiologic changes of pregnancy relate to the occurrence, diagnosis, and management of trauma.[31,62,65] During the first trimester, the thick-walled uterus is well-protected from trauma by the pelvic girdle. In the second trimester, relatively abundant amniotic fluid volume protects the fetus. Despite this, fetal injury rates increase after 24 weeks' gestation as the uterus rises out of the pelvis.[4] By the third trimester, the now thin-walled and prominent uterus is subject to potential blows, penetration, or rupture. As the pregnancy approaches term, the relative fluid volume is decreased, which reduces the cushioning effect around the fetus. The fetal head at this point in gestation is typically protected within the bony pelvis.

The placenta is an inelastic organ attached to an elastic organ (the uterus). Forces of acceleration or deceleration may deform the uterus and shear the placenta off its implantation site, which creates a placental abruption. The risk of placental abruption is independent of the placenta's location. Uterine rupture can also occur and is associated with fundal trauma by direct high force. Uterine rupture often results in fetal death.[66,67]

Gastrointestinal. Gastric emptying time is prolonged during pregnancy, so the provider should always assume the stomach of a woman who is pregnant is full. Early gastric tube decompression should be considered. The intestines are relocated to the upper part of the abdomen and may be shielded by the uterus. Signs of peritoneal irritation (eg, distention, rebound tenderness,

guarding, rigidity) are frequently detected on examination after trauma but may be less pronounced during pregnancy.

Urinary. Renal blood flow and the glomerular filtration rate are increased in pregnancy. Blood urea nitrogen and serum creatinine levels are decreased. Glycosuria is common because of a lowered threshold of excretion. There may be bilateral or unilateral hydronephrosis with ureteral dilation. These changes may affect interpretation of laboratory and x-ray studies when trauma occurs.

Trauma Assessment in Pregnancy

When major trauma occurs in pregnancy, evaluation and treatment of the woman is the priority. This approach also serves the best interests of the fetus. The diagnosis and treatment of the woman who is pregnant and who has experienced major trauma does not differ significantly from the care of a woman with trauma who is not pregnant, except for the recognition of and adjustment for the anatomic and physiologic changes of pregnancy. The *Advanced Trauma Life Support, Student Course Manual* (10th Edition)[68] outlines a primary trauma survey:

A = Airway maintenance with restriction of cervical spine motion

B = Breathing and ventilation

C = Circulation with hemorrhage control

D = Disability (assessment of neurologic status)

E = Exposure/Environmental control: completely undress the patient but prevent hypothermia.

The primary trauma survey of resuscitation holds true in pregnancy and is more important because of the risks to the fetus from maternal hypotension and hypoxia.[68]

The primary maternal survey with resuscitation (*Table 8*) addresses life support and resuscitation.[31] Fluid resuscitation should be pursued aggressively.[65] A woman who is pregnant can lose a significant amount of blood volume before showing signs and symptoms of hypotension and shock. Crystalloid and early type-specific packed red blood cells are indicated to restore the physiologic hypervolemia of pregnancy. The woman should be returned to the left lateral position after a thorough physical examination.

The primary fetal survey (*Table 8*) is performed after initial assessment and stabilization of the

Table 8. Maternal and Fetal Surveys With Resuscitation

Primary Maternal Survey

Patent airway

Adequate ventilation and oxygenation

- Early intubation
- Maintain appropriate PCO_2

Effective circulatory volume

- Fluid support
- Blood replacement

Decrease uterine compression of inferior vena cava

- Manual left uterine displacement

Baseline laboratory evaluation

- Add fibrinogen

Adjuncts

- Maintain relative hypervolemia
- Pulse oximetry
- Arterial blood gases

Secondary Maternal Survey

X-ray

Focused assessment sonography in trauma

Diagnostic peritoneal lavage

Monitor uterine contractions

Evaluation of perineum

- Assess for vaginal bleeding
- Assess for ruptured membranes
- Assess cervix for dilation and effacement

Central venous pressure

Urine output

Baseline laboratory tests

- Serum bicarbonate
- Rh factor
- Kleihauer-Betke test
- Coagulation factors
- Assess for admission
- Complete blood count
- Type and screen

Primary Fetal Survey

Maternal abdominal examination

- Assess for abruption
- Assess for uterine rupture
- Fundal height

Palpate

- Uterine activity

Fetal heart rate pattern and movement

Adjuncts

- X-ray evaluation for uterine rupture
- Assess for admission

Information from Jeejeebhoy FM, Zelop CM, Lipman S, et al; American Heart Association Emergency Cardiovascular Care Committee, Council on Cardiopulmonary, Critical Care, Perioperative and Resuscitation, Council on Cardiovascular Diseases in the Young, and Council on Clinical Cardiology. Cardiac arrest in pregnancy: a scientific statement from the American Heart Association. Circulation*. 2015;132(18):1747-1773; Nash P, Driscoll P. ABC of major trauma. Trauma in pregnancy.* BMJ*. 1990;301(6758):974-976.*

woman. The major diagnostic details relating to the fetal primary survey are: fetal viability and well-being, likelihood of fetal injury and fetomaternal transfusion, gestational age, placental abruption, preterm labor, rupture of membranes, fetal presentation, and uterine rupture.

The secondary maternal survey (*Table 8*) is the same as for women who are not pregnant. Indications for abdominal computed tomography (CT), focused assessment sonography in trauma, and diagnostic peritoneal lavage (DPL) have the same indications as in women who are not pregnant. If DPL is performed, the catheter should be placed above the umbilicus while using an open technique. Indicated x-ray studies can be performed without concern for radiation injury to the fetus because the immediate diagnostic benefits far outweigh any theoretical risk to the fetus.[69] However, shielding the fetus and minimizing exposure is prudent when possible.

The use of abdominal ultrasonography by a skilled operator should be considered in detecting pregnancy and possible causes of the cardiac arrest, but this should not delay other treatments. Ultrasonography provides fetal assessment (eg, amniotic fluid volume, fetal presentation, estimated gestational age) and is occasionally helpful in cases of anterior abruption. Pelvic examination can be performed after documentation of placental location.

Laboratory testing in women who are pregnant and severely injured should include the same tests performed in women who are not pregnant. Monitoring with a central venous pressure line and measuring urine output can be extremely helpful.

Coagulopathy can accompany abruption, amniotic fluid embolism, and other obstetric events related to trauma. Obtaining fibrinogen, platelet count, and fibrin split products (D-dimer) measurements is recommended. The fibrinogen level may double in late pregnancy. A normal fibrinogen level may indicate early DIC, which can be seen with placental abruption.[31]

Care of Women With Major Trauma

Certain maternal injuries, such as visceral injury or retroperitoneal hemorrhage, require immediate surgery to save the lives of the woman and fetus. Uterine rupture and placental abruption threaten the fetus more directly. Uterine rupture manifests as signs and symptoms of hypovolemia and hemoperitoneum. The fetus will often be acidotic or dead, and may appear on x-ray or ultrasonography in a nontypical position with extended limbs. Placental abruption manifests with classical signs and symptoms: contractions, rigid and tender uterus, expanding uterine height, abnormal fetal heart rate tracing or fetal demise, and coagulopathy; vaginal bleeding may or may not be present. Both conditions require surgery for the woman and fetus.

Appropriate imaging studies should not be avoided because of the presence of a fetus. Typically, concerns about ionizing radiation and fetal harm start between 5 to 10 rads.[65,69] The average CT scan of the abdomen and pelvis is approximately 2 to 2.5 rads.

After completing the primary trauma survey, place two large-bore IV catheters and administer supplemental oxygen to maintain a saturation of 95% or greater. Aggressive transfusion of blood products may provide volume and improve oxygen-carrying capacity.[31] A massive transfusion protocol using a 1:1:1 ratio of one unit of packed red blood cells, one unit of fresh frozen plasma, and one 6 pack of platelets has been shown to decrease mortality.[70] The use of 1 g tranexamic acid intravenously within the first hour of trauma or within 3 hours of PPH has been associated with a decreased mortality rate (see *Postpartum Hemorrhage* chapter).[70,71] The use of pressors should be avoided until adequate volume replacement has been completed. The uterus should be displaced from the vena cava and aorta. After maternal stabilization, the secondary maternal and fetal survey can be completed.[72]

Fetal injuries are highly variable. Skull and brain injuries are common when the maternal pelvis is fractured and the fetal head is engaged. Fetal contrecoup injuries may occur. Penetrating injuries may be present from gunshot or stab wounds. With a penetrating injury, the risk of fetal morbidity and mortality is higher than the maternal risk. Women who are pregnant have a lower risk of injury from penetrating trauma,[61] because the gravid uterus acts as a shield to the maternal organs. Maternal mortality from penetrating wounds is less than 4% in pregnancy;[61] however, 60% to 90% of stab or gunshot wounds to the maternal abdomen result in fetal injury or death.[73] Surgical exploration of the maternal abdomen is almost always necessary in cases of penetrating abdominal injury.

Routine administration of Rh_o(D) immune globulin is indicated in women with significant abdominal trauma who are unsensitized Rh negative. The reported incidence of fetomaternal hemorrhage after trauma is 8% to 30% (with a range of 2.5 to 115 mL of blood).[74] Indications for tetanus prophylaxis do not change in pregnancy and appropriate candidates should be vaccinated.

The Kleihauer-Betke (KB) test should be considered for women with significant blunt uterine trauma to assess the degree of fetomaternal hemorrhage, regardless of their Rh status.[75-77] When pregnant women with trauma have an Injury Severity Score of more than 2 (*Appendix*), a positive KB test is an effective predictor of adverse perinatal outcomes, particularly in women with more severe trauma.[64,78]

The mean estimated blood volume of injected fetal blood is typically less than 15 mL; for more than 90% of women, it is less than 30 mL. Administering 300-mcg of Rh_o(D) immune globulin will treat a 15-mL red blood cell hemorrhage, or a 30 mL total blood volume hemorrhage. Serial testing may be appropriate to assess ongoing hemorrhage.[79,80]

Unfortunately, the KB test is often not readily available for acute clinical management. Fetal heart monitoring, uterine monitoring, or ultrasonography evaluation may be more useful in the acute setting. An alternative approach of calculating the total blood volume of a fetus has been recommended to expedite therapy.[81] First, estimate the fetal weight by ultrasonography, then multiply the estimated fetal weight in kilograms by the total blood volume of a fetus (approximately 100 mL/kg) to determine the maximum total dose of Rh_o(D) immune globulin.[81] A 300-mcg vial will treat 30 mL of total blood loss, so two vials would treat the total fetal blood loss of a 600-g fetus. In the case of a 3.5-kg fetus, 3.5 kg multiplied by 100 mL/kg would produce an estimated total fetal blood volume of 350 mL. This approach will overestimate the maternal exposure to fetal blood because only a limited amount of fetal blood will typically enter maternal circulation, even in severe trauma.[81]

Resuscitative hysterotomy may be required for several reasons: it may be difficult to treat the traumatic maternal condition around the gravid uterus, the obstetric pathology contributes to the woman's worsening condition (as in abruption with coagulopathy), or the fetus is acidotic. Resuscitative hysterotomy may improve the woman's status but may increase her risk of hypovolemia. After 23 to 24 weeks' gestation, resuscitative hysterotomy also may save the fetus.[54]

Care of Women With Minor Trauma

The most common trauma-related occurrence among women who are pregnant is a minor trauma (eg, fall, minor MVC, blunt abdominal trauma), which causes little or no maternal injury. In minor cases, the provider often must judge whether examination or monitoring is necessary. Being cautious and thorough is recommended because seemingly insignificant trauma can result in fetal injury or demise.[64,82] Placental abruption typically becomes apparent shortly after injury. Fetal monitoring of women who experience trauma beyond 20 weeks' gestation should be initiated as soon as the woman is stabilized. Monitoring via cardiotocography should occur for a minimum of 4 to 6 hours.[31,75] In one study, all women shown subsequently to have a placental abruption experienced eight or more contractions per hour in the first 4 hours of monitoring (see the *Late Pregnancy Bleeding* chapter).[83]

Monitoring should be continued for a minimum of 24 hours if there is presence of six uterine contractions per hour,[62] abnormal fetal heart rate patterns, vaginal bleeding, significant uterine tenderness, serious maternal injury, or rupture of membranes occurs during the initial 4- to 6-hour monitoring period. If none of these findings are present, then the woman may be discharged with instructions to return if vaginal bleeding, fluid leakage, decreased fetal movement, or severe abdominal pain occurs.[31,65] A guideline for managing minor trauma is presented in *Table 9*.

The presence of six uterine contractions per hour[62] and fetal red blood cells in the maternal circulation are good indicators of fetal risk from abruption. Ultrasonography has poor sensitivity (24%) for detection of abruptio placenta, but it is highly specific (96%). This results in a positive predictive value of 88% if abruption is seen during ultrasonography and a negative predictive value of 53% if abruption is not seen.[84]

If the woman is hospitalized, then betamethasone therapy should be considered if she is between 24 and 36 6/7 weeks' gestation and delivery appears likely. Tetanus vaccination is safe in pregnancy. Care should be taken to avoid

complications of thromboembolism (eg, consider low-molecular weight heparin or sequential compression devices).[72]

Motor Vehicle Collisions

Motor vehicle collisions are a leading cause of death among women who are pregnant, accounting for approximately 50% of all trauma that occurs during pregnancy.[62]

Women who are pregnant and do not wear a seat belt during a MVC have approximately twice the risk of preterm delivery and four times the risk of fetal death compared with women who wear a seat belt.[85] Seat belt use decreases during pregnancy because some women think that the seat belt will harm the fetus and because the belt can be uncomfortable.[82,85-88] At 6, 7, 8, and 9 months' gestation, 53%, 60%, 66%, and 56% of women who are pregnant, respectively, reported discomfort while wearing a seat belt.[82] The chest and uterine fundus should be 10 inches from the airbag cover.[89] During pregnancy, the distance to the inferior aspect of the steering wheel decreases by 3.07 to 6.52 cm by 6 to 9 months' gestation.[82] Seat belt use should be a major issue of prenatal counseling in every pregnancy.

Proper use of seat belts during a MVC may be the best predictor of maternal and fetal outcomes. Women who properly used a seat belt during a MVC sustained minor injuries compared with serious injury and death in those who did not properly use a seat belt.[85,86] Lack of seat belt use during pregnancy is associated with an increased risk of fetal death.[90] There is an 84% reduction in adverse fetal outcomes in women who wear seat belts.[91] Incorrect seat belt use can contribute to intrauterine injury.[73] The lap belt should be placed as low as possible under the protuberant portion of the abdomen and the shoulder belt positioned off to the side of the uterus between the breasts and over the midportion of the clavicle. Placement of the lap belt over the dome of the uterus significantly increases pressure transmission to the uterus and has been associated with significant uterine and fetal injury. The lap and shoulder restraints should be applied as snugly as comfort will allow. Counseling about seat belt should occur early in pregnancy because women injured in the first 24 weeks have a higher risk of preterm delivery.[92]

Airbag deployment reduces injury to women who are pregnant.[85] Women who are pregnant are not at increased risk of adverse pregnancy outcomes while traveling in a vehicle that is equipped with an airbag during a MVC.[93] The American College of Obstetricians and Gynecologists (ACOG)[94,95] and the National Highway Traffic and Safety Administration[96] recommend that women who are pregnant wear lap and shoulder seat belts and do not turn off airbags.

Direct Assault

Direct assault on the abdomen can occur as a result of IPV. Women who are abused are a frequently undetected high-risk group. ACOG and

Table 9. Management of Minor Trauma in Pregnancy

Interventions	Risk factors	Discharge criteria
Primary maternal and fetal survey	Maternal heart rate >110 BPM	Resolution of contractions
Laboratory tests: blood type, Rh, hematocrit, Kleihauer-Betke test, coagulation studies	Injury Severity Score >9 (*Appendix*)	Normal fetal heart assessment
Consider obstetric ultrasonography	Evidence of placental abruption	Intact membranes
If greater than 20 weeks' gestation, monitor for contractions and fetal heart rate status	Fetal baseline heart rate >160 BPM or <110 BPM	No uterine tenderness
If <6 contractions/hour and no risk factors, monitor for 4 hours, then discharge	Ejection during motor vehicle collision	No vaginal bleeding
If >6 contractions/hour or risk factors, monitor for at least 24 hours	Motorcycle or pedestrian collision	All women who are Rh-negative receive 300 mcg Rh_o(D) immune globulin (more if indicated by Kleihauer-Betke test)

BPM = beats per minute.

Information from American College of Surgeons. Chapter 12: Trauma in Pregnancy and Intimate Partner Violence. In: American College of Surgeons. Advanced Trauma Life Support Student Course Manual*. 10th ed. Chicago, IL: American College of Surgeons; 2018; Mendez-Figueroa H, Dahlke JD, Vrees RA, Rouse DJ. Trauma in pregnancy: an updated systematic review.* Am J Obstet Gynecol. *2013;209(1):1-10.*

the US Preventive Services Task Force recommend universal screening for intimate partner abuse.[97-99] Every provider should be alert to the possibility of IPV when a patient presents with a vague or inconsistent history of trauma. IPV may escalate in pregnancy, and the abdomen is the most frequent target. Prenatal care should include routine screening for IPV, and identified patients should be appropriately counseled and referred. In women who have experienced IPV, an assessment for depression and suicidal ideation should accompany assessment for immediate safety.[100]

Indicators that suggest the presence of IPV include:

- Injuries inconsistent with the stated history
- Diminished self-image, depression, or suicide attempts
- Self-abuse
- Frequent visits to the emergency department or provider's office
- Symptoms suggestive of substance abuse
- Self-blame for injuries
- Partner insists on being present for interview and examination, and attempts to control the discussion

Amniotic Fluid Embolism

Amniotic fluid embolism (AFE) is rare, with an estimated incidence of 1/15,200 deliveries in North America and 1/53,800 deliveries in Europe.[101] In the past, the maternal mortality rate from this complication was initially estimated at 85%, with half of the deaths occurring within the first hour.[102] With the advent of intensive care units, population-based data now show that the case fatality rate and perinatal mortality associated with AFE are 13% to 30% in North America and 9% to 44% in Europe.[101]

Amniotic fluid embolism remains one of the most catastrophic conditions that can occur in women who are pregnant. AFE is catastrophic for the fetus as well, with a neurologically intact survival rate of only 39%.[102] A 2015 Australia/New Zealand population-based descriptive study showed 33 cases of AFE out of an estimated cohort of 613,731 women giving birth; the estimated incidence was 5.4/100,000 women giving birth. Five women died (case fatality rate 15%) and the estimated rate of maternal mortality due to AFE was 0.8/100,000 women giving birth. Two of the 36 infants died.[103]

Predisposing Factors and Pathophysiology

Risk factors associated with an increased risk of AFE include advanced maternal age, placental abnormalities, operative deliveries, eclampsia, polyhydramnios, cervical lacerations, and uterine rupture.[101] At the time of the initial description of AFE in 1941, it was thought of as a mechanical event in which a bolus of amniotic fluid enters the systemic circulation because of a tetanic contraction, moves through the pulmonary circuit, and produces a massive perfusion failure, bronchospasm, and shock.[104] The density of particulate matter in the fluid was thought to be related to the lethality of the event.

The number of cases failing to fit this picture led to a reconsideration of the pathophysiology, which suggests that the syndrome can occur with simple exposure to small amounts of amniotic fluid. A good case can be made to include AFE in a group of anaphylactoid syndromes occurring in late pregnancy or labor. Pathophysiologic studies show that left heart failure and pulmonary vasospasm are the prime etiologic factors in cardiovascular collapse, but the underlying mechanism may be an anaphylactic-like event with an associated 41% incidence of atopy or allergy.[102] Histological findings in one study showed the postpartum presence of inflammatory cells as well as mast cells within the myometrium or postpartum acute myometritis. This concept suggests an inflammatory response and a mast cell mediated-anaphylactoid reaction, independent of classic antigen-antibody-mediated anaphylaxis in AFE.[105] The hemodynamic response in AFE is biphasic, with initial pulmonary hypertension and right ventricular failure followed by left ventricular failure.[101] DIC is the most common complication, possibly because of the large amount of tissue factor in amniotic fluid.[106]

Clinical Picture

The clinical picture develops rapidly. The syndrome begins with respiratory distress (tachypnea and dyspnea) accompanied by restlessness, cyanosis, nausea, vomiting, and seizures. Unexpected cardiovascular collapse occurs, and profound DIC may follow. Finally, the woman becomes comatose and dies. In many cases, these events progress quickly so that only the most rudimentary diagnostic studies and resuscitative efforts can be made.[106]

Diagnosis

Definitive diagnosis of this condition is typically made postmortem. Clinical diagnosis relies primarily on clinical observations. If time permits, helpful laboratory values include blood gases and coagulation factors. The differential diagnosis includes other catastrophic causes of cardiopulmonary compromise, such as massive PE, bilateral pneumothorax, myocardial infarction, or gastric fluid aspiration. Obstetric conditions that mimic AFE include severe abruption, uterine rupture, inverted uterus, and uterine atony. In specific circumstances, consider septic shock and eclampsia.[106]

Management

The management of AFE is primarily supportive. An aggressive medical approach seems justified and certainly can do no harm for the women who survive the initial catastrophic event. AFE outcomes may be improved through establishing a resuscitation process, with checklists and training.[107]

When a woman receiving obstetric care collapses unexpectedly, the BLS algorithm and then the appropriate ACLS algorithm should be attended to, with the airway secured and ventilation ensured, using endotracheal intubation if necessary. Oxygen should be administered at 100%. Two large-bore IV catheters should be placed, and aggressive fluid replacement using crystalloid solution should begin. Pressor agents, such as dopamine, will likely be required.[107]

Blood should be drawn for a complete hemogram, coagulation panel, and chemistry panel, including electrolytes and renal function. Arterial blood gases should be obtained. Urinary output should be monitored via indwelling Foley catheter, and a portable chest x-ray and a 12-lead electrocardiogram should be obtained. A cardiac monitor should be applied and ACLS should be initiated. Hemodynamic monitoring will probably be required via an arterial access line and possibly a Swan-Ganz catheter. Therefore, the woman should be cared for in the intensive care unit.

Coagulation factors should be assessed every 2 hours and aggressive blood component therapy initiated as needed with packed red cells, platelets (if the platelet count is less than 50,000/mcL), fresh frozen plasma, or cryoprecipitate. Viscoelastic hemostatic assays (ie, thromboelastography, thromboelastometry) are increasingly being used for managing DIC in the setting of trauma and may prove to be useful for DIC secondary to AFE.[108,109]

Given the possible anaphylactoid nature of the condition, epinephrine use should be considered.[110] Positive end expiratory pressure is typically required to prevent alveolar collapse and to recruit atelectatic alveoli. Fluids, dopamine, and furosemide should be administered based on hemodynamic parameters.[19]

Summary

With approximately half of maternal deaths being preventable,[8] the next challenge will be to improve maternal mortality one woman at a time. CPR and ACLS modified for maternal physiology, prompt diagnosis, and resuscitative hysterotomy provide important tools to meet that challenge.[28,30]

Regardless of the cause of the maternal collapse, care facilities should perform regular in situ drills to ensure the entire labor and delivery staff is functioning with the same set of assumptions.[30] Documentation should be thorough, but fact-focused and nonspeculative in what can be emotionally and medicolegally charged scenarios.[81]

Nursing Considerations: Maternal Resuscitation and Trauma

- Determine the location of the code cart at the start of every shift
- Use the primary trauma survey in women with trauma who are pregnant and their fetus
- Identify the institutional process for calling a code
- Assign roles/duties, including a recorder, as help arrives
- If a resuscitative hysterotomy is initiated, assist in facilitating the 4-minute rule by avoiding an ultrasound or FHR tracing, and by not transferring the woman to the operating room
- Champion the creation of a resuscitative hysterotomy cart/box and simulation team training

FHR = fetal heart rate.

References

1. American Heart Association. Cardiac arrest statistics. Available at http://cpr.heart.org/AHAECC/CPRAndECC/General/UCM_477263_Cardiac-Arrest-Statistics.jsp.
2. Mhyre JM, Tsen LC, Einav S, et al. Cardiac arrest during hospitalization for delivery in the United States, 1998-2011. *Anesthesiology*. 2014;120(4):810-818.
3. Dijkman A, Huisman CM, Smit M, et al. Cardiac arrest in pregnancy: increasing use of perimortem caesarean section due to emergency skills training? *BJOG*. 2010; 117(3):282-287.
4. Baghirzada L, Balki M. Maternal cardiac arrest in a tertiary care centre during 1989-2011: a case series. *Can J Anaesth*. 2013;60(11):1077-1084.
5. Katz VL, Dotters DJ, Droegemueller W. Perimortem cesarean delivery. *Obstet Gynecol*. 1986;68(4):571-576.
6. Bennett TA, Katz VL, Zelop CM. Cardiac arrest and resuscitation unique to pregnancy. *Obstet Gynecol Clin North Am*. 2016;43(4):809-819.
7. Murugan R, Darby JM. *Rapid Response System: A Practical Guide*. Oxford, UK: Oxford University Press; 2018; 1-344.
8. Saucedo M, Deneux-Tharaux C, Bouvier-Colle MH; French National Experts Committee on Maternal Mortality. Ten years of confidential inquiries into maternal deaths in France, 1998-2007. *Obstet Gynecol*. 2013; 122(4):752-760.
9. Salihu HM, Salemi JL, Aggarwal A, et al. Opioid drug use and acute cardiac events among pregnant women in the United States. *Am J Med*. 2018;131(1):64-71.e1.
10. Atta E, Gardner M. Cardiopulmonary resuscitation in pregnancy. *Obstet Gynecol Clin North Am*. 2007;34(3): 585-597, xiii.
11. Kikuchi J, Deering S. Cardiac arrest in pregnancy. *Semin Perinatol*. 2018;42(1):33-38.
12. Krans E, Cochran G, Bogen DL. Caring for opioid dependent pregnant women: prenatal and postpartum care considerations. *Clin Obstet Gynecol*. 2015;58(2):370-379.
13. Vargas R, Repke JT, Ural SH. Type 1 diabetes mellitus and pregnancy. *Rev Obstet Gynecol*. 2010;3(3):92-100.
14. Geake J, Tay G, Callaway L, Bell SC. Pregnancy and cystic fibrosis: Approach to contemporary management. *Obstet Med*. 2014;7(4):147-155.
15. Murphy VE. Managing asthma in pregnancy. *Breathe (Sheff)*. 2015;11(4):258-267.
16. Dalmo S. ACOG Publishes Guidelines on Hemoglobinopathies in Pregnancy. *Am Fam Physician*. 2007;76(8): 1229-1230.
17. Stout K. Pregnancy in women with congenital heart disease: the importance of evaluation and counselling. *Heart*. 2005;91(6):713-714.
18. Deshpande NA, Coscia LA, Gomez-Lobo V, et al. Pregnancy after solid organ transplantation: a guide for obstetric management. *Rev Obstet Gynecol*. 2013;6(3-4):116-125.
19. Clark SL, Romero R, Dildy GA, et al. Proposed diagnostic criteria for the case definition of amniotic fluid embolism in research studies. *Am J Obstet Gynecol*. 2016; 215(4):408-412.
20. Stanten RD, Iverson LI, Daugharty TM, et al. Amniotic fluid embolism causing catastrophic pulmonary vasoconstriction: diagnosis by transesophageal echocardiogram and treatment by cardiopulmonary bypass. *Obstet Gynecol*. 2003;102(3):496-498.
21. Sibai BM. Hypertension. In: Gabbe SG, Niebyl JR, Simpson JL, eds. *Obstetrics: Normal and Problem Pregnancies*. 4th ed. New York: Churchill Livingstone; 2002: 945-1004.
22. Berg O, Lee RH, Chagolla B. CMQCC Preeclampsia Toolkit: magnesium sulfate. 2014. Available at www.cmqcc.org/resource/magnesium-sulfate-toolkit-pdf.
23. Vanden Hoek TL, Morrison LJ, Shuster M, et al. Part 12: cardiac arrest in special situations: 2010 American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. *Circulation*. 2010;122(18)(Suppl 3):S829-S861.
24. O'Doherty LJ, Taft A, Hegarty K, et al. Screening women for intimate partner violence in healthcare settings: abridged Cochrane systematic review and meta-analysis. *BMJ*. 2014;348:g2913.
25. Metz TD, Rovner P, Hoffman MC, et al. Maternal deaths from suicide and overdose in Colorado, 2004-2012. *Obstet Gynecol*. 2016;128(6):1233-1240.
26. Cantwell R, Clutton-Brock T, Cooper G, et al. Saving mothers' lives: reviewing maternal deaths to make motherhood safer: 2006-2008. The eighth report of the confidential enquiries into maternal deaths in the United Kingdom. *BJOG*. 2011;118(Suppl 1):1-203.
27. Kuklina E, Callaghan W. Chronic heart disease and severe obstetric morbidity among hospitalisations for pregnancy in the USA: 1995-2006. *BJOG*. 2011;118(3): 345-352.
28. Jeejeebhoy FM, Zelop CM, Lipman S, et al; American Heart Association Emergency Cardiovascular Care Committee, Council on Cardiopulmonary, Critical Care, Perioperative and Resuscitation, Council on Cardiovascular Diseases in the Young, and Council on Clinical Cardiology. Cardiac arrest in pregnancy: a scientific statement from the American Heart Association. *Circulation*. 2015;132(18):1747-1773.
29. Rossi A, Cornette J, Johnson MR, et al. Quantitative cardiovascular magnetic resonance in pregnant women: cross-sectional analysis of physiological parameters throughout pregnancy and the impact of the supine position. *J Cardiovasc Magn Reson*. 2011;13:31.
30. Lavonas EJ, Drennan IR, Gabrielli A, et al. Part 10: special circumstances of resuscitation: 2015 American Heart Association guidelines update for cardiopulmonary resuscitation and emergency cardiovascular care. *Circulation*. 2015;132(18)(Suppl 2):S501-S518.
31. Advanced Trauma Life Support Subcommittee, American College of Surgeons Committee on Trauma, International Advanced Trauma Life Support Working Group. Trauma in pregnancy and intimate partner violence. In: *Advanced Trauma Life Support Student Course Manual*. Tenth ed. Chicago, IL: American College of Surgeons; in press.
32. Page RL. Treatment of arrhythmias during pregnancy. *Am Heart J*. 1995;130(4):871-876.

33. Weinberger SE, Weiss ST, Cohen WR, et al. Pregnancy and the lung. *Am Rev Respir Dis*. 1980;121(3):559-581.
34. Hegewald MJ, Crapo RO. Respiratory physiology in pregnancy. *Clin Chest Med*. 2011;32(1):1-13.
35. LoMauro A, Aliverti A. Respiratory physiology in pregnancy: physiology masterclass. *Breathe (Sheff)*. 2015; 11(4):297-301.
36. Gatti F, Spagnoli M, Zerbi SM, et al. Out-of-hospital perimortem cesarean section as resuscitative hysterotomy in maternal posttraumatic cardiac arrest. *Case Rep Emerg Med*. 2014;2014:121562.
37. Katz V, Balderston K, DeFreest M. Perimortem cesarean delivery: were our assumptions correct? *Am J Obstet Gynecol*. 2005;192(6):1916-1921, discussion 1920-1921.
38. Carleton SC, Reardon RF, Brown CA. Bag mask ventilation. In: Brown CA, Sakles JC, Mick NW, eds. *The Walls Manual of Emergency Airway Management*, 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins;2018:90.
39. Mushambi MC, Kinsella SM, Popat M, et al; Obstetric Anaesthetists' Association. Difficult airway society. Obstetric Anaesthetists' Association and Difficult Airway Society guidelines for the management of difficult and failed tracheal intubation in obstetrics. *Anaesthesia*. 2015;70(11):1286-1306.
40. Goodwin AP, Pearce AJ. The human wedge. A manoeuvre to relieve aortocaval compression during resuscitation in late pregnancy. *Anaesthesia*. 1992;47(5):433-434.
41. Rees GA, Willis BA. Resuscitation in late pregnancy. *Anaesthesia*. 1988;43(5):347-349.
42. Ueland K, Novy MJ, Peterson EN, Metcalfe J. Maternal cardiovascular dynamics. IV. The influence of gestational age on the maternal cardiovascular response to posture and exercise. *Am J Obstet Gynecol*. 1969; 104(6):856-864.
43. Cyna AM, Andrew M, Emmett RS, et al. Techniques for preventing hypotension during spinal anaesthesia for caesarean section. *Cochrane Database Syst Rev*. 2006; (4):CD002251.
44. Jeejeebhoy FM, Zelop CM, Windrim R, et al. Management of cardiac arrest in pregnancy: a systematic review. *Resuscitation*. 2011;82(7):801-809.
45. Kundra P, Khanna S, Habeebullah S, Ravishankar M. Manual displacement of the uterus during caesarean section. *Anaesthesia*. 2007;62(5):460-465.
46. ACOG Practice Bulletin no. 212: pregnancy and heart disease. *Obstet Gynecol*. 2019;133(5):e320-e356.
47. Lipman S, Cohen S, Einav S, et al; Society for Obstetric Anesthesia and Perinatology. The Society for Obstetric Anesthesia and Perinatology consensus statement on the management of cardiac arrest in pregnancy. *Anesth Analg*. 2014;118(5):1003-1016.
48. Rose CH, Faksh A, Traynor KD, et al. Challenging the 4- to 5-minute rule: from perimortem cesarean to resuscitative hysterotomy. *Am J Obstet Gynecol*. 2015;213(5): 653-656, 653.e1.
49. Adams J, Cepeda Brito JR, Baker L, et al. Management of maternal cardiac arrest in the third trimester of pregnancy: a simulation-based pilot study. *Crit Care Res Pract*. 2016;2016:5283765.
50. Whitty JE. Maternal cardiac arrest in pregnancy. *Clin Obstet Gynecol*. 2002;45(2):377-392.
51. Einav S, Kaufman N, Sela HY. Maternal cardiac arrest and perimortem caesarean delivery: evidence or expert-based? *Resuscitation*. 2012;83(10):1191-1200.
52. Benson MD, Padovano A, Bourjeily G, Zhou Y. Maternal collapse: challenging the four-minute rule. *EBioMedicine*. 2016;6:253-257.
53. Beckett VA, Knight M, Sharpe P. The CAPS Study: incidence, management and outcomes of cardiac arrest in pregnancy in the UK: a prospective, descriptive study. *BJOG*. 2017;124(9):1374-1381.
54. Battaloglu E, Porter K. Management of pregnancy and obstetric complications in prehospital trauma care: prehospital resuscitative hysterotomy/perimortem caesarean section. *Emerg Med J*. 2017;34(5):326-330.
55. Lipman S, Daniels K, Cohen SE, Carvalho B. Labor room setting compared with the operating room for simulated perimortem cesarean delivery: a randomized controlled trial. *Obstet Gynecol*. 2011;118(5):1090-1094.
56. Weber CE. Postmortem cesarean section: review of the literature and case reports. *Am J Obstet Gynecol*. 1971; 110(2):158-165.
57. Zaami S, Marinelli E, Montanari Vergallo G. Assessing malpractice lawsuits for death or injuries due to amniotic fluid embolism. *Clin Ter*. 2017;168(3):e220-e224.
58. Sharma NS, Wille KM, Bellot SC, Diaz-Guzman E. Modern use of extracorporeal life support in pregnancy and postpartum. *ASAIO J*. 2015;61(1):110-114.
59. Moore SA, Dietl CA, Coleman DM. Extracorporeal life support during pregnancy. *J Thorac Cardiovasc Surg*. 2016;151(4):1154-1160.
60. Mirza FG, Devine PC, Gaddipati S. Trauma in pregnancy: a systematic approach. *Am J Perinatol*. 2010; 27(7):579-586.
61. El Kady D. Perinatal outcomes of traumatic injuries during pregnancy. *Clin Obstet Gynecol*. 2007;50(3): 582-591.
62. Mendez-Figueroa H, Dahlke JD, et al. Trauma in pregnancy: an updated systematic review. *Am J Obstet Gynecol*. 2013;209(1):1-10.
63. Smith R, Crane P; Perinatal Joint Practice Committee. *Post-trauma care in pregnancy*. University of Michigan; 2003.
64. Melamed N, Aviram A, Silver M, et al. Pregnancy course and outcome following blunt trauma. *J Matern Fetal Neonatal Med*. 2012;25(9):1612-1617.
65. Brown S, Mozurkewich E. Trauma during pregnancy. *Obstet Gynecol Clin North Am*. 2013;40(1):47-57.
66. Hill CC, Pickinpaugh J. Trauma and surgical emergencies in the obstetric patient. *Surg Clin North Am*. 2008; 88(2):421-440, viii.
67. Kvarnstrand L, Milsom I, Lekander T, et al. Maternal fatalities, fetal and neonatal deaths related to motor vehicle crashes during pregnancy: a national population-based study. *Acta Obstet Gynecol Scand*. 2008; 87(9):946-952.

68. Advanced Trauma Life Support Subcommittee. American College of Surgeons Committee on Trauma, International Advanced Trauma Life Support Working Group. Initial assessment and management. In: *Advanced Trauma Life Support Student Course Manual*. 10th ed. Chicago, IL: American College of Surgeons; 2018.
69. American College of Obstetricians and Gynecologists' Committee on Obstetric Practice. Committee Opinion no. 656: guidelines for diagnostic imaging during pregnancy and lactation. *Obstet Gynecol*. 2016;127(2): e75-e80.
70. Cameron P, Knapp BJ. Chapter 254: trauma in adults. In: Tintinalli JE, Stapczynski JS, John Ma O, et al. *Tintinalli's Emergency Medicine: A Comprehensive Study Guide*, 8th ed; 2016.
71. World Health Organization. WHO recommendation on tranexamic acid for the treatment of postpartum haemorrhage. 2017. Available at http://www.who.int/reproductivehealth/publications/tranexamic-acid-pph-treatment/en/.
72. Pearce C, Martin SR. Trauma and considerations unique to pregnancy. *Obstet Gynecol Clin North Am*. 2016;43(4):791-808.
73. Chames MC, Pearlman MD. Trauma during pregnancy: outcomes and clinical management. *Clin Obstet Gynecol*. 2008;51(2):398-408.
74. Bhatia K, Cranmer HH. Trauma in Pregnancy. In: Marx J, Hockberger R, Walls R. *Rosen's Emergency Medicine: Concepts and Clinical Practice*, 8th ed. Philadelphia, PA: Elsevier Saunders; 2015.
75. Pearlman MD, Tintinallli JE, Lorenz RP. A prospective controlled study of outcome after trauma during pregnancy. *Am J Obstet Gynecol*. 1990;162(6):1502-1507, discussion 1507-1510.
76. Barraco RD, Chiu WC, Clancy TV, et al; EAST Practice Management Guidelines Work Group. Practice management guidelines for the diagnosis and management of injury in the pregnant patient: the EAST Practice Management Guidelines Work Group. *J Trauma*. 2010;69(1): 211-214.
77. Hyde LK, Cook LJ, Olson LM, et al. Effect of motor vehicle crashes on adverse fetal outcomes. *Obstet Gynecol*. 2003;102(2):279-286.
78. Trivedi N, Ylagan M, Moore TR, et al. Predicting adverse outcomes following trauma in pregnancy. *J Reprod Med*. 2012;57(1-2):3-8.
79. Krywko DM, Shunkwiler SM. Kleihauer Betke Test. StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2020.
80. Huls CK, Detlefts C. Trauma in Pregnancy. *Semin Perinatol*. 2018;42(1):13-20.
81. American College of Obstetricians and Gynecologists. Update Series ACOG. Major and minor trauma in pregnancy. Volume 35; 2009. Available at http://www.acogupdate.com/.
82. Auriault F, Brandt C, Chopin A, et al. Pregnant women in vehicles: Driving habits, position and risk of injury. *Accid Anal Prev*. 2016;89:57-61.
83. Shah AJ, Kilcline BA. Trauma in pregnancy. *Emerg Med Clin North Am*. 2003;21(3):615-629.
84. Glantz C, Purnell L. Clinical utility of sonography in the diagnosis and treatment of placental abruption. *J Ultrasound Med*. 2002;21(8):837-840.
85. Vladutiu CJ, Weiss HB. Motor vehicle safety during pregnancy. *Am J Lifestyle Med*. 2012;6(3):241-249.
86. Chibber R, Al-Harmi J, Fouda M, El-Saleh E. Motor-vehicle injury in pregnancy and subsequent feto-maternal outcomes: of grave concern. *J Matern Fetal Neonatal Med*. 2015;28(4):399-402.
87. Lam WC, To WW, Ma ES. Seatbelt use by pregnant women: a survey of knowledge and practice in Hong Kong. *Hong Kong Med J*. 2016;22(5):420-427.
88. Karbakhsh M, Ershadi Z, Khaji A, Rahimi-Sharbaf F. Seat belt use during pregnancy in Iran: attitudes and practices. *Chin J Traumatol*. 2010;13(5):275-278.
89. Sirin H, Weiss HB, Sauber-Schatz EK, Dunning K. Seat belt use, counseling and motor-vehicle injury during pregnancy: results from a multi-state population-based survey. *Matern Child Health J*. 2007;11(5):505-510.
90. Luley T, Fitzpatrick CB, Grotegut CA, et al. Perinatal implications of motor vehicle accident trauma during pregnancy: identifying populations at risk. *Am J Obstet Gynecol*. 2013;208(6):466.e1-466.e5.
91. Klinich KD, Flannagan CA, Rupp JD, et al. Fetal outcome in motor-vehicle crashes: effects of crash characteristics and maternal restraint. *Am J Obstet Gynecol*. 2008;198(4):450.e1-450.e9.
92. Weiss HB, Sauber-Schatz EK, Cook LJ. The epidemiology of pregnancy-associated emergency department injury visits and their impact on birth outcomes. *Accid Anal Prev*. 2008;40(3):1088-1095.
93. Schiff MA, Mack CD, Kaufman RP, et al. The effect of air bags on pregnancy outcomes in Washington State: 2002-2005. *Obstet Gynecol*. 2010;115(1):85-92.
94. American College of Obstetricians and Gynecologists. Car safety for pregnant women, babies, and children. 2016. Available at https://www.acog.org/Patients/FAQs/Car-Safety-for-Pregnant-Women-Babies-and-Children.
95. ACOG Committee on Obstetric Practice. ACOG Committee Opinion no. 443: air travel during pregnancy. *Obstet Gynecol*. 2009;114(4):954-955.
96. National Highway Traffic Safety Administration. If you're pregnant: seat belt recommendations for drivers and passengers. Available at https://icsw.nhtsa.gov/safercar/parents/SeatBelts/Pregnancy-Seat-Belt-Safety.htm.
97. ACOG Committee Opinion no. 518: Intimate partner violence. *Obstet Gynecol*. 2012;119(2 Pt 1):412-417.
98. Moyer VA; U.S. Preventive Services Task Force. Screening for intimate partner violence and abuse of elderly and vulnerable adults: U.S. preventive services task force recommendation statement. *Ann Intern Med*. 2013; 158(6):478-486.
99. U.S. Preventive Services Task Force. Final recommendation statement: intimate partner violence and abuse of elderly and vulnerable adults. Available at https://www.uspreventiveservicestaskforce.org/Page/Document/RecommendationStatementFinal/intimate-partner-violence-and-abuse-of-elderly-and-vulnerable-adults-screening.

100. Cronholm PF, Fogarty CT, Ambuel B, Harrison SL. Intimate partner violence. *Am Fam Physician*. 2011;83(10): 1165-1172.

101. Conde-Agudelo A, Romero R. Amniotic fluid embolism: an evidence-based review. *Am J Obstet Gynecol*. 2009; 201(5):445.e1-445.e13.

102. Clark SL, Hankins GD, Dudley DA, et al. Amniotic fluid embolism: analysis of the national registry. *Am J Obstet Gynecol*. 1995;172(4 Pt 1):1158-1167, discussion 1167-1169.

103. McDonnell N, Knight M, Peek MJ, et al. the Australasian Maternity Outcomes Surveillance System (AMOSS). Amniotic fluid embolism: an Australian-New Zealand population-based study. *BMC Pregnancy Childbirth*. 2015;15:352.

104. Steiner PE, Lushbaugh CC. Landmark article, Oct. 1941: Maternal pulmonary embolism by amniotic fluid as a cause of obstetric shock and unexpected deaths in obstetrics. By Paul E Steiner and C. C. Lushbaugh. *JAMA*. 1986;255(16):2187-2203.

105. Tamura N, Farhana M, Oda T, et al. Amniotic fluid embolism: pathophysiology from the perspective of pathology. *J Obstet Gynaecol Res*. 2017;43(4):627-632.

106. Clark SL. Amniotic fluid embolism. *Obstet Gynecol*. 2014;123(2 Pt 1):337-348.

107. Hession PM, Millward CJ, Gottesfeld JE, et al. Amniotic fluid embolism: using the medical staff process to facilitate streamlined care. *Perm J*. 2016;20(4):97-101.

108. Hurwich M, Zimmer D, Guerra E, et al. A case of successful thromboelastographic guided Rrsuscitation after postpartum hemorrhage and cardiac arrest. *J Extra Corpor Technol*. 2016;48(4):194-197.

109. Ekelund K, Hanke G, Stensballe J, et al. Hemostatic resuscitation in postpartum hemorrhage - a supplement to surgery. *Acta Obstet Gynecol Scand*. 2015;94(7): 680-692.

110. Rudra A, Chatterjee S, Sengupta S, et al. Amniotic fluid embolism. *Indian J Crit Care Med*. 2009;13(3):129-135.

Appendix A
Injury Severity Score

The Injury Severity Score (ISS) is an anatomical scoring system that provides an overall score for patients with multiple injuries. Each injury is assigned an Abbreviated Injury Scale (AIS) score and is allocated to one of six body regions (head, face, chest, abdomen, extremities, external). Only the highest AIS score in each body region is used. The three most severely injured body regions with the highest scores have their score squared and added together to produce the ISS score.

An example of the ISS calculation is shown below.

Region	Injury Description	AIS	Square Top Three
Head and neck	Cerebral contusion	3	9
Face	No injury	0	
Chest	Flail chest	4	16
Abdomen	Minor contusion of liver	2	
	Complex rupture spleen	5	25
Extremity	Fractured femur	3	
External	No injury	0	
Injury Severity Score:			**50**

Information from Baker SP, O'Neill B, Haddon W Jr, Long WB. The Injury Severity Score: a method for describing patients with multiple injuries and evaluating emergency care. J Trauma. *1974;14:187-196.*

The ISS score takes values from 0 to 75. If an injury is assigned an AIS of 6 (unsurvivable injury), the ISS score is automatically assigned to 75. The ISS score is essentially the only anatomical scoring system in use and correlates linearly with mortality, morbidity, hospital stay, and other measures of severity.

Its weaknesses are that any error in AIS scoring increases the ISS error, many different injury patterns can yield the same ISS score, and injuries to different body regions are not weighted. Additionally, as a full description of patient injuries is not known before full investigation and operation, the ISS (along with other anatomical scoring systems) is not useful as a triage tool.

Cardiac Complications of Pregnancy

Learning Objectives

1. Perform prenatal cardiac screening for history or risk factors associated with heart disease in pregnancy.
2. Provide basic reproductive counseling for patients of childbearing age with a history of congenital heart disease.
3. Explain the evaluation and management of peripartum cardiac complications.

Pregnancy (and the preconception and interconception periods) is a time of opportunity for health care professionals to affect a woman's overall health by providing preconception care and counseling, ameliorating certain cardiovascular risk factors, and detecting or managing underlying disease. Many women with cardiac disease can safely undertake pregnancy, though some conditions are riskier than others.

Background and Epidemiology

Cardiovascular disease (CVD) complicates approximately 0.2% to 4% of pregnancies.[1,2] The severity of CVD in pregnancy has increased and CVD related postpartum hospitalizations have tripled.[3] In the United States, more pregnancies are affected by chronic conditions such as hypertension, diabetes, obesity, and substance use that are associated with ischemic heart disease and peripartum cardiomyopathy. Additionally, more women with congenital heart disease (CHD) survive into reproductive age.[4]

Between 2011 and 2014, over 33% of pregnancy-related deaths in the United States were attributed to CVD, including cardiomyopathy,[5] which surpassed infection, hemorrhage, and thrombosis. Most women who die of CVD in the antepartum period are not identified, but 70% die within the first 6 weeks' postpartum and 29% die between 6 weeks and 1 year postpartum.[4] A review of pregnancy-related CVD deaths in California found that approximately 25% of these deaths were potentially preventable.[4] Provider-related factors include delayed response, ineffective care, misdiagnosis, failure to consult, and lack of continuity of care. Patient-related factors include underlying medical conditions, obesity, substance abuse, and delay in seeking care.[4]

The proportion of maternal deaths attributed to cardiac causes in the United States has risen from 4.2 maternal deaths per 100,000 live births between 2006 and 2010 to 4.8 per 100,000 between 2011 and 2016 (see *Figures 1* and *2*).[6]

Racial Disparities and Cardiovascular Disease

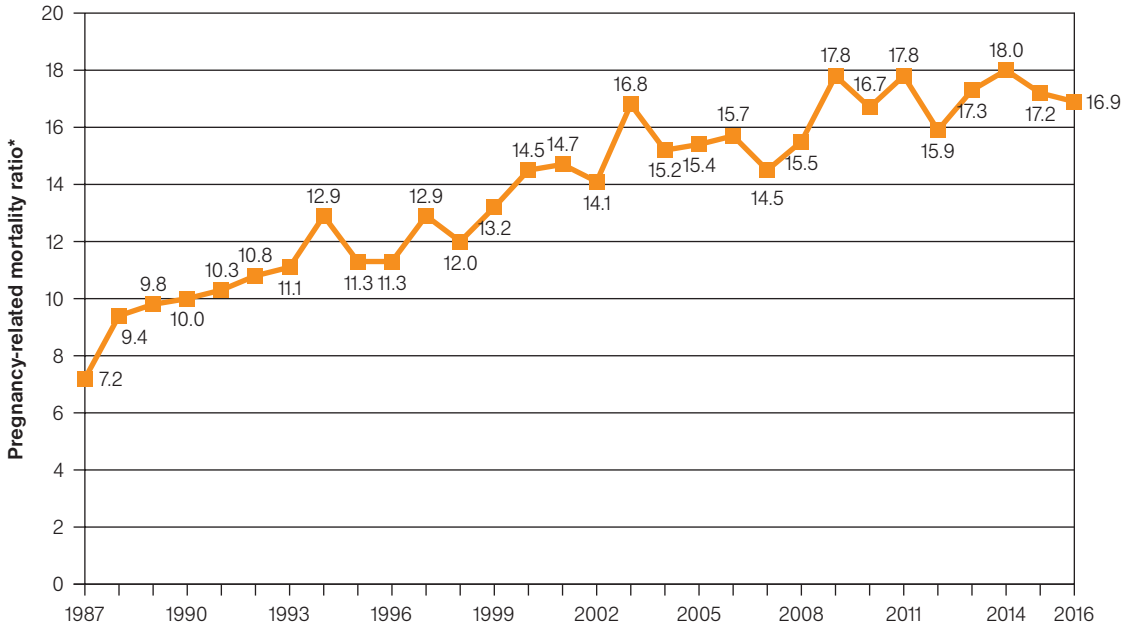
Non-Hispanic black women have more than three times the pregnancy-related mortality risk of non-Hispanic white women,[6] but when CVD deaths are considered, the CVD pregnancy-related mortality rates among black women are eight times higher than in white women.[4] Ethnic differences are also seen in younger women at the time of diagnosis and black women exhibit more severe symptoms, which may be attributable to late initiation of care and higher rates of obesity and hypertensive disorders.

Normal Physiology of Pregnancy

During normal pregnancy, blood volume increases approximately 1,500 mL (1,000 mL plasma volume and 500 mL red blood cell volume).[7] Heart rate and stroke volume increase, leading to an increase in cardiac output from approximately 4 L/minute (prepregnancy) to 6 L/minute at term.[8] As much as 500 mL/minute is supplied to the uterus and placenta by the end of pregnancy.

A decrease in systemic vascular resistance and vascular tone results in alterations to blood pressure. Systolic blood pressure decreases toward the end of the first trimester before returning to baseline in the third trimester. Left ventricular (LV) wall mass increases. After mid-pregnancy, the enlarging uterus compresses the inferior vena cava when a woman lies supine, resulting in a positional decrease in preload and a drop in cardiac output. Because of the necessary physiologic changes of pregnancy, some women with cardiac conditions do not tolerate tachycardia or volume augmentation.

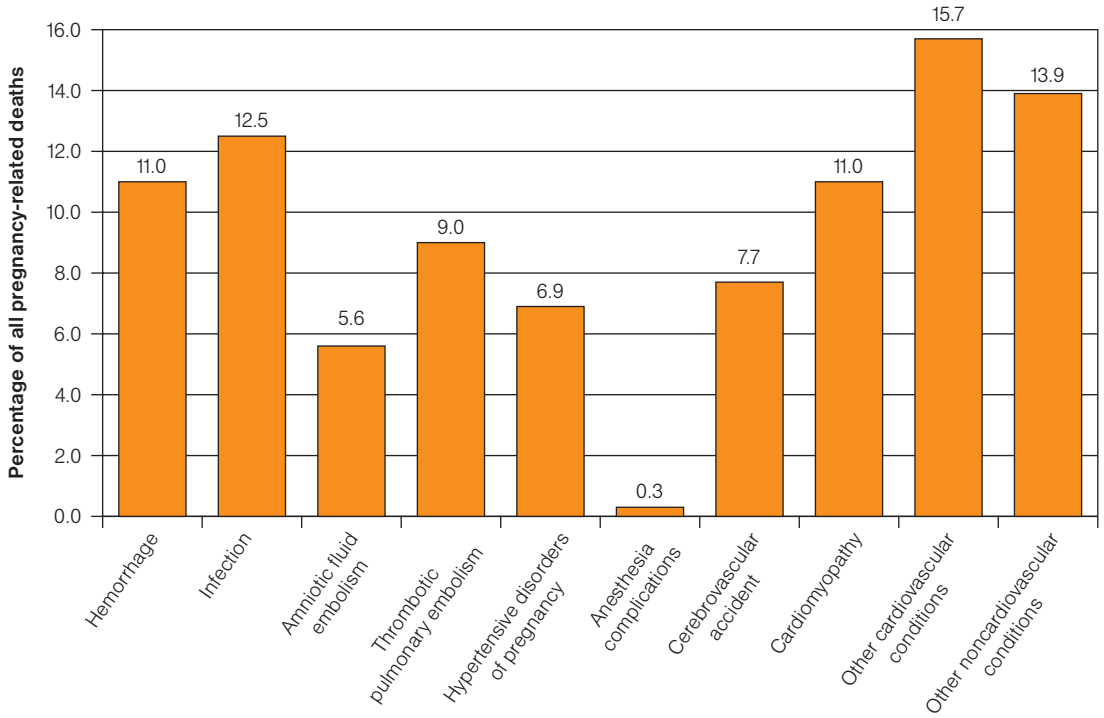
Figure 1. Trends in Pregnancy-Related Mortality in the United States, 1987-2016



**Note: Number of pregnancy-related deaths per 100,000 live births per year.*

Reprinted from Centers for Disease Control and Prevention. Pregnancy Mortality Surveillance System. 2020. Available at https://www.cdc.gov/reproductivehealth/maternalinfanthealth/pmss.html.

Figure 2. Causes of Pregnancy-Related Death in the United States: 2011-2016



Note: Number of pregnancy-related deaths per 100,000 live births per year.

Reprinted from Centers for Disease Control and Prevention. Pregnancy Mortality Surveillance System. 2020. Available at https://www.cdc.gov/reproductivehealth/maternalinfanthealth/pmss.html.

Table 1 summarizes these changes during pregnancy.

During labor, oxygen consumption increases, and systolic and diastolic blood pressure rise during contractions. This response may be blunted by the use of regional analgesia. Uterine contractions also serve to intermittently transfer blood from the uterus back into central circulation. Some women with underlying CVD will experience physiologic decompensation during labor and develop pulmonary edema.

Immediately after delivery, vena caval compression is substantially reduced, and the high rate of uterine blood flow is reduced as the uterus clamps down and the wide-open uteroplacental supply ceases. These factors shift the augmented blood volume of pregnancy away from the uterus and back into central circulation.[9]

Table 1. Cardiovascular Changes in Normal Pregnancy

	Second Trimester	Immediate Postpartum
Heart Rate	+	++
Stroke Volume	+	++
Systemic Vascular Resistance	-	+
Systolic Blood Pressure	-	+

Table 2. Cardiovascular Disease Risk Factors in Pregnant Women

Age >40 years

Black race

Obesity (BMI >35, kg/m^2)

Diabetes

Hypertension

BMI = body mass index.

Information from Hameed AB, Morton CH, Moore A. Improving Health Care Response to Cardiovascular Disease in Pregnancy and Postpartum. 2017. Available at https://www.cdph.ca.gov/Programs/CFH/DMCAH/RPPC/CDPH%20Document%20Library/CMQCC_CVD_Toolkit.pdf.

Preconception Counseling

The American Academy of Family Physicians (AAFP) 2016 position paper advocated for addressing preconception care at all routine primary care visits.[10] Women of reproductive age with obesity, hypertension, diabetes, mental health disorders, or substance abuse disorders (including alcohol and tobacco) should be supported in obtaining treatment and making lifestyle changes. If indicated, drugs with the lowest teratogenic risk profiles should be used. Using shared decision making, women should be encouraged to use highly effective contraception, such as long-acting, reversible methods, until health is optimized for pregnancy.

A multidisciplinary approach to preconception counseling for those with heart disease is ideal to discuss risks and benefits of pregnancy, and optimize prognosis for future pregnancies.[11]

Genetic and Familial Issues

Inheritance risk varies by specific lesion. Generally, a woman with CHD has a 3% to 4% likelihood that her newborn will have CHD, and it need not be the same type. If the woman has a left-sided lesion, the newborn's risk is even higher.[12] Fetal echocardiography should be offered at approximately 20 weeks' gestation. Genetic counseling is appropriate, ideally offered prior to conception.

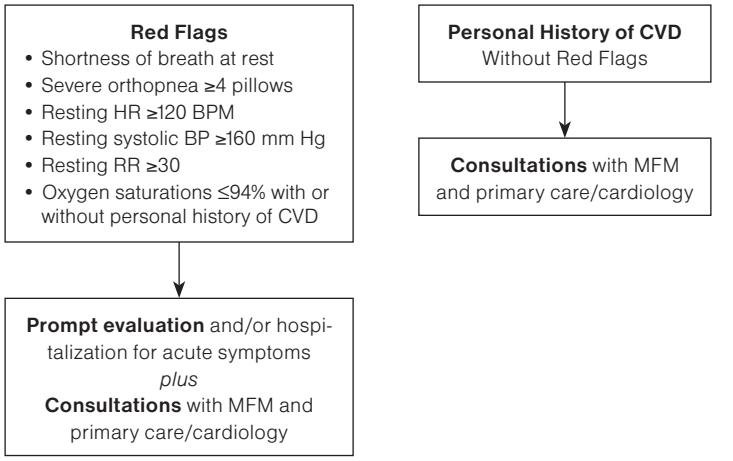
History, Examination, Symptom Review

The initial prenatal visit provides an opportunity to obtain a personal and familial cardiac history, assess the woman's risk of undiagnosed cardiac disease (*Table 2*), and perform a physical examination. Pregnancy and CVD commonly cause fatigue, edema, dyspnea, and reduced exercise tolerance, making it difficult to differentiate normal pregnancy symptoms from life-threatening CVD. Consequently, it is important for health care providers to recognize physical examination findings and red flag signs and symptoms suggestive of CVD (*Figure 3*).[13,14]

Cardiac disease is among the leading causes of intensive care unit admission and maternal death in the developed world. International migrants to developed countries have a higher risk of some cardiac lesions (eg, valvular rheumatic heart disease) than women born into high-income countries, and particular care must be taken.[15]

Terminology appropriate to the patient's level of understanding should be used. If a language

Figure 3. Possible Indicators (Red Flags) of Cardiac Disease in Pregnant/Postpartum Women



BP = blood pressure; BPM = beats per minute; CVD = cardiovascular disease; MFM = maternal fetal medicine.

Reprinted from Hameed AB, Morton CH, Moore A. Improving Health Care Response to Cardiovascular Disease in Pregnancy and Postpartum. 2017. Available at https://www.cdph.ca.gov/Programs/CFH/DMCAH/RPPC/CDPH%20Document%20Library/CMQCC_CVD_Toolkit.pdf.

barrier makes it difficult to obtain a history, expert translation assistance should be obtained.

A cardiovascular risk assessment should be performed in all women to assess CVD risk when prenatal care is initiated.[14] Inquire about symptoms such as chest pain, palpitations, syncope, and dyspnea, and characterize their severity. Modifiable risk factors include traditional risk factors (eg, hypertension, hyperlipidemia, smoking, diabetes) as well as poor diet, sedentary lifestyle, and obesity. Other important, emerging risk factors include polycystic ovarian syndrome, history of pregnancy-related disorders (eg, gestational hypertension, preeclampsia, autoimmune disorders, sleep apnea) and psychosocial factors (eg, depression, anxiety, low socioeconomic status, drug use, racial and ethnic disparities).[16] Drugs should be reviewed at the initial prenatal visit, especially if a preconception evaluation was not performed. The level of physical activity should be assessed. If the woman reports limitations to physical activity, further questioning and investigation is needed.

The physical examination may reveal a rapid or irregular heart rate, but a minor degree of tachycardia is common in normal pregnancy, or rales, which are not a normal finding. Most women who are pregnant also have a soft crescendo-decrescendo systolic murmur over the aortic or pulmonic valve indicative of increased flow. A diastolic or holosystolic murmur or a loud systolic murmur is not normal. Dependent edema is to be expected late in pregnancy but is not a prominent sign in the early stages. Jugular veins may be full after mid-pregnancy, related to the increased circulating volume and the effect of the enlarging uterus on intra-abdominal pressure.[17]

The California Department of Public Health, in conjunction with the California Maternal Quality Care Collaborative, has generated tools that may be useful for identifying possible cardiac disease in women who are pregnant (*Figures 3* and *4*).[13]

Tests

The electrocardiogram (ECG) is a first-line test if ischemia or arrhythmia is suspected.

A 1981 study that evaluated 157 women longitudinally through pregnancy and into the postpartum period, described changes associated with normal pregnancy as follows:

- A gradual increase in heart rate, which by the third trimester was 32% higher than baseline
- A change in QRS axis, which can be leftward or rightward
- No significant changes in electric intervals or wave amplitudes[18]

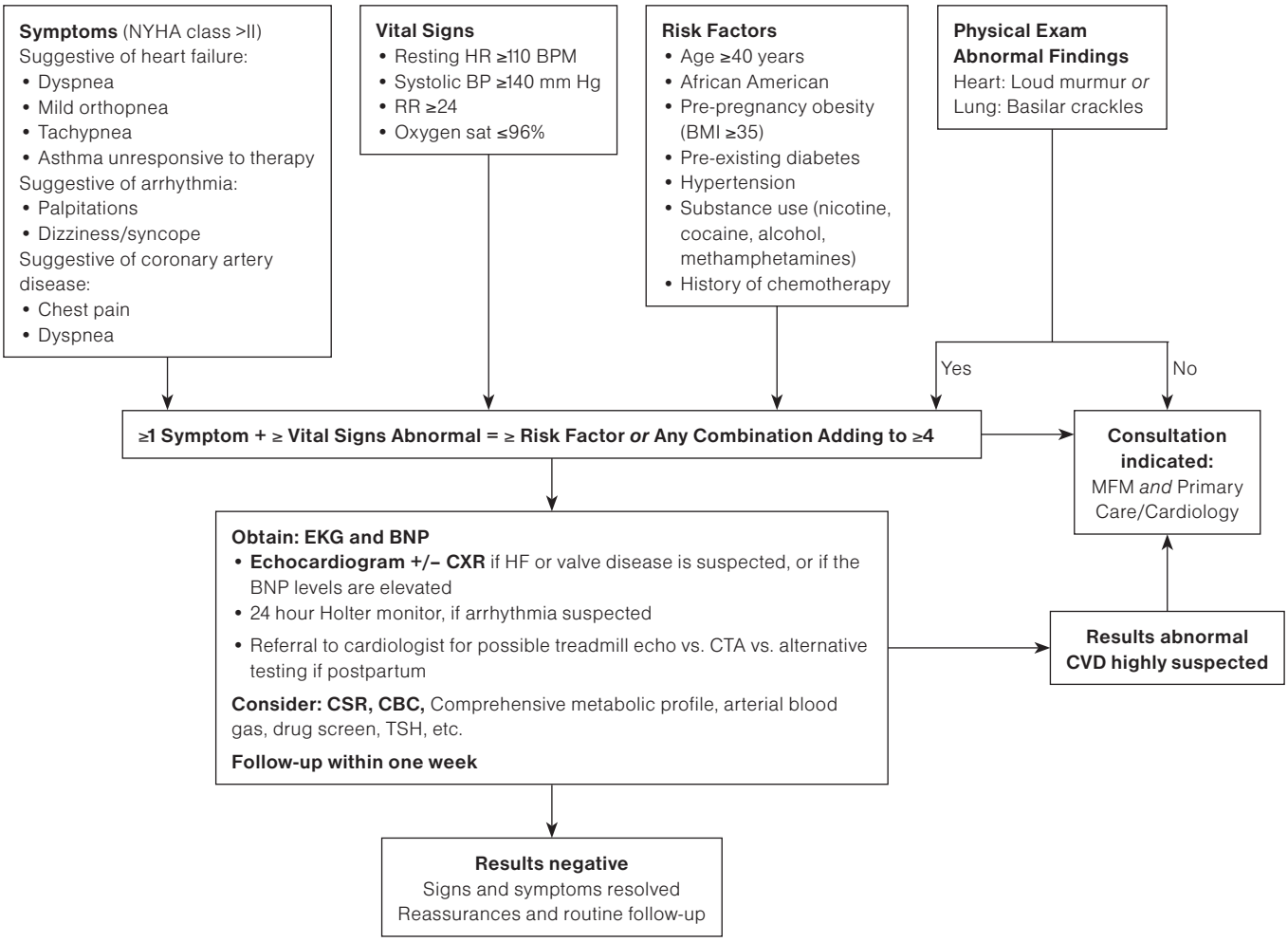
Q-waves are more commonly seen in leads V4-V6 during pregnancy compared with nonpregnant controls, and T-wave abnormalities, such as flattening and inversion, are significantly more common during pregnancy and seen in a majority of women by the third trimester.[19] These changes are unsurprising because the mechanical and electrical axis of the heart shifts during normal pregnancy as the diaphragm rises and the heart is rotated. The LV wall also increases in thickness during normal pregnancy.

Echocardiography is commonly used to diagnose cardiac conditions or monitor disease progression. It can assess structure and function and may be performed at any time during pregnancy transthoracically or, if needed, transesophageally, though the latter typically requires sedation. The echocardiographer must be aware of the normal changes pregnancy brings to the examination: increased LV dimension, volume, wall thickness, and mass; increased right ventricular dimensions and volume; increased left atrial size and volume; increased stroke volume; slightly increased aortic

root diameter. Left and right ventricular ejection fraction are unchanged, as is pulmonary artery pressure.[20] It involves no ionizing radiation and is safe for the woman and fetus. Similarly, pregnancy is not a contraindication for an agitated saline (bubble) study nor a dobutamine stress test.[21,22]

Chest x-ray is used occasionally in cardiac assessment. With a maternal chest x-ray, minimal ionizing radiation is delivered to the fetus (estimated dose of less than 0.0001 mGy per procedure, well below the 50 mGy limit thought to pose any fetal risk).[21] Thoracic CT angiography is estimated to deliver less than 1 mGy to the fetus, coronary CT angiography between 1 to 3 mGy, and standard coronary angiography less than 0.1 mGy.[21] Fluoroscopy (eg, to pass a catheter from the femoral artery to the heart) delivers 0.09 to 0.24 mGy per minute of exposure. Nuclear medicine studies are seldom used in pregnancy but may be considered under rare circumstances; consultation with a nuclear medicine subspecialist and radiation physicist would be recommended.

Figure 4. CMQCC-Suggested Cardiac Evaluation During Pregnancy



California Department of Public Health, 2017: supported by Title V funds. Developed in partnership with California Maternal Quality Care Collaborative Cardiovascular Disease in Pregnancy and Postpartum Taskforce. Visit: www.CMQCC.org for details.

BMI = body mass index; BNP = brain natriuretic peptide; BP = blood pressure; BPM = beats per minute; CBC = complete blood count; CVD = cardiovascular disease; CTA = computed tomography angiography; CXR = chest x-ray; EKG = electrocardiogram; HR = heart rate; MFM = maternal fetal medicine; NYHA = New York Heart Association; RR = respiratory rate; TSH = thyroid-stimulating hormone.

Reprinted from Hameed AB, Morton CH, Moore A. Improving Health Care Response to Cardiovascular Disease in Pregnancy and Postpartum. 2017. Available at https://www.cdph.ca.gov/Programs/CFH/DMCAH/RPPC/CDPH%20Document%20Library/CMQCC_CVD_Toolkit.pdf.

Magnetic resonance imaging (MRI) is considered safe in pregnancy, though there are theoretical concerns about acoustic noise affecting fetal hearing. Cardiac MRI may be used as needed in selected cases. However, most radiology subspecialists are reluctant to use MRI contrast agents in pregnancy. These agents typically localize to and easily cross the placenta, and gadolinium may enter the fetal circulation. Concerns about gadolinium accumulation and long-term fetal exposure to these compounds limit their use in the United States.[21,23] Exercise stress testing may be performed in pregnancy, though experts recommend a submaximal protocol.[22]

The clinical use of cardiac biomarkers during pregnancy is limited by the lack of knowledge regarding their normal ranges during this period. For example, brain natriuretic peptide (BNP) is higher during pregnancy, though levels seem to be below 20 pg/mL in most cases.[24] BNP does rise between late pregnancy and the early puerperium; as many as 6% of healthy women have values over 100 pg/mL in the early postpartum period. Preeclampsia also elevates BNP levels, sometimes to levels greater than 150 pg/mL. Troponins I and T increase somewhat during pregnancy, but typically remain below the upper limit of nonpregnancy normal range. Creatinine kinase MB, while remaining within the nonpregnant normal range during the course of pregnancy, typically increases within the first 24 hours postpartum and may exceed the normal limit.[24]

Presenting Signs and Symptoms and Predictors of Poor Outcomes

Several scoring systems (*Tables 3*, *4*, and *5*) designed to predict maternal cardiac complications among women with known cardiac disease have been published. These conditions include a prior cardiac event (eg, arrhythmia, previous stroke or transient ischemic attack, heart failure), New York Heart Association (NYHA) functional class III or IV (*Table 6*), left heart obstruction, reduced systemic ventricular function (ie, ejection fraction less than 40%), moderate or severe systemic or pulmonary valve regurgitation, cyanotic heart disease, and cardiac drugs prior to pregnancy.[25-28] The modified World Health Organization (WHO) system seems to have the best predictive value for cardiac outcomes, though it performs poorly as a predictor of fetal/neonatal outcomes[29] and is probably most useful in determining which patients should be referred for subspecialty care. However, low scores in the Cardiac Disease in Pregnancy (CARPREG),[25] Zwangerschap bij Aangeboren Hartafwijkingen (ZAHARA),[26] or WHO[28] classification systems cannot be interpreted as obviating cardiac risk. A woman with known significant, cardiac disease should always be referred to a center of expertise for preconception counseling. This allows for a discussion of the challenges, risks to the woman and her potential offspring, and provides an opportunity for reproductive life planning. The effect of cardiac drugs on a developing fetus can be addressed, the role of catheter-based or surgical interventions prior to pregnancy can be considered, and, in concert with the cardiology subspecialist, a plan developed for optimizing the cardiac condition prior to pregnancy. Telemedicine holds promise as a way of overcoming distance and other logistical obstacles to focused preconception counseling.

When preconception counseling suggests that pregnancy should be avoided entirely or deferred until cardiac lesions are corrected, it is important to discuss highly effective contraception. Gener-

Table 3. Cardiac Disease in Pregnancy Classification System

CARPREG Classification System[a]

1 point for each of the following:

- Baseline NYHA classification >II, or cyanosis
- Left heart obstruction (mitral valve area <2 cm^2 or aortic valve area <1.5 cm^2 or peak LV outflow tract gradient >30 mm Hg)
- Reduced systemic ventricle ejection fraction (<40%)

Score 0 → 5% risk of cardiac event

Score 1 → 27% risk of cardiac event

Score >1 → 75% risk of cardiac event

[a]Endpoint: prediction of cardiac event during pregnancy, defined as pulmonary edema, sustained tachyarrhythmia or bradyarrhythmia requiring treatment, stroke, cardiac arrest, or cardiac death.

CARPREG = Cardiac Disease in Pregnancy; LV = left ventricle; NYHA = New York Heart Association.

Information from Siu SC, Sermer M, Colman JM, Alvarez AN, et al; Cardiac Disease in Pregnancy (CARPREG) Investigators. Prospective multicenter study of pregnancy outcomes in women with heart disease. Circulation. *2001;104(5):515-521.*

ally, the alternative to pregnancy is not celibacy. Contraceptive management should also consider the cardiac condition.[28,30] In some cases, it may be desirable to avoid estrogen-dependent hormonal contraception. At least 25% of women with complex CHD have had an unplanned pregnancy,[30] which can be dangerous. Contraception is discussed in more detail later in this chapter (*Table 7*).

Specific Conditions

Congenital

As more children survive CHD, the burden of disease has shifted. The median age of individuals living with CHD increased from 11 years in 1985 to 25 years in 2010. With this shift, more women with CHD are attempting pregnancy,[31] but survival to reproductive age cannot guarantee a normal obstetric outcome. The risk of complications for women and fetuses is increased above that of the general obstetric population. For the fetus, these are typically related to a shortened gestational length and lower birth weight, though the risk of pregnancy loss or perinatal death is increased in some specific conditions.[29,32]

Women with CHD often have comorbidities. Neurologic, developmental, mental health, pulmonary, hepatic, renal, hematologic, and endocrinologic conditions are common.[33] The risk of stroke is significantly higher than in the general population.

Neurodevelopmental delay is common in pediatric CHD, ranging between 20% and 70%, depending on the specific lesion. Though information on the neurodevelopmental concerns of adults with CHD is limited, they are likely to be prevalent and may be related to fetal development (cerebral blood flow is often abnormal in fetuses and neonates when complex CHD is present) or follow cardiopulmonary bypass, or both. Additional time for history-taking and counseling should be anticipated.

Depression and anxiety are common in adults with CHD, and these patients should undergo screening and treatment, if necessary. Pulmonary function may be compromised by abnormal lung or bronchial development related to enlarged or distorted cardiac anatomy, or as a result of thoracotomy. Restrictive lung disease is identified by pulmonary function testing in more than 40% of adults with CHD. Hepatic circulation may be impaired by congestion and portal hypertension, resulting in cellular atrophy or fibrosis, or by ischemia and hypoperfusion, leading to hepatic necrosis. Hepatic fibrosis is present in virtually all patients who have undergone the Fontan procedure. The prevalence of renal impairment is estimated at 50% in adults with CHD, although the etiology is unknown; cyanotic CHD is especially predictive of chronic kidney disease, perhaps related to renal hypoxia and erythrocytosis.

Endocrinologic abnormalities are common in CHD. Hypothyroidism manifests in approximately 10% of patients with CHD, with major associations with Down syndrome, cyanotic CHD, and a history of amiodarone use. Abnormalities of bone, calcium, and vitamin D metabolism affect bone health and increase the risk of fracture in adult CHD. The prevalence of

Table 4. Zwangerschap bij Aangeboren Hartafwijkingen Classification System

ZAHARA Classification System (congenital heart disease only)[a]	Points assigned:
History of arrhythmia	1.5
Cardiac medication prior to pregnancy	1.5
NYHA classification >II prior to pregnancy	0.75
Left heart obstruction (peak gradient >50 mm Hg or aortic valve area <1.0 cm^2)	2.5
Systemic AV valve regurgitation, moderate/severe	0.75
Pulmonic valve regurgitation, moderate/severe	0.75
Mechanical valve prosthesis	4.25
Cyanotic heart disease (corrected or uncorrected)	1

Score	Risk of cardiac event during pregnancy
<0.5	2.9%
0.51 - 1.5	7.5%
1.51 - 2.5	17.5%
2.51 - 3.5	43.1%
>3.51	70%

[a]Endpoint: cardiac complications, defined as clinically significant arrhythmia requiring treatment, clinically significant episode of heart failure requiring treatment, cardiovascular complications (ie, MI, stroke, thromboembolism); endocarditis.

MI = myocardial infarction; NYHA = New York Heart Association; ZAHARA = Zwangerschap bij Aangeboren Hartafwijkingen.

Information from Drenthen W, Boersma E, Balci A, et al. Predictors of pregnancy complications in women with congenital heart disease. Eur Heart J. *2010;31(17):2124-2132.*

impaired glucose tolerance and overt diabetes is increased in this population, with a hazard ratio between 1.35 and 2.85.[34]

Non-cardiac comorbidities among adults with CHD increase the risk of perioperative complications in non-cardiac surgery in general. Specifics are not known for obstetric surgery, but the additional physiologic changes of pregnancy are also likely to increase the possibility of complications. The American Heart Association (AHA) recommends that elective non-cardiac surgery which includes the potential for cesarean delivery inherent in all labors, should "take place in a regional ACHD [adult congenital heart disease] center with specialists familiar with and experienced in the management of those with CHD."[34]

Table 5. Modified World Health Organization Classification of Maternal Cardiovascular Risk

WHO Pregnancy Risk Category I

No detectable increase in maternal mortality; mild or no risk in morbidity

Uncomplicated small mild pulmonary stenosis or patent ductus arteriosus; mitral valve prolapse; successfully repaired ASD, VSD, PDA, or anomalous pulmonary venous drainage; isolated atrial or ventricular ectopic beats

WHO Pregnancy Risk Category II

Small increase in risk of maternal mortality; moderate increase in risk of maternal morbidity

If otherwise well and uncomplicated: unrepaired ASD or VSD; repaired tetralogy of Fallot; arrhythmias

WHO Pregnancy Risk Category II-III

Moderate increase in maternal mortality and morbidity

Mild LV dysfunction; hypertrophic cardiomyopathy; disease of a native or tissue valve which does not fall into category I or IV; Marfan syndrome in the absence of aortic dilation; bicuspid aortic valve with aortic root dilation <4.5 cm

WHO Pregnancy Risk Category III

Significantly increased risk of maternal mortality or severe morbidity

Mechanical valve; systemic right ventricle; Fontan circulation; unrepaired cyanotic congenital heart disease; other complex congenital heart disease; Marfan syndrome with aortic dilation 4-4.5 cm; bicuspid aortic valve with aortic dilation 4.5-5 cm

Expert counseling required. In the event of pregnancy, immediate specialist referral and ongoing subspecialist care (cardiac, obstetric, maternal-fetal medicine) recommended

WHO Pregnancy Risk Category IV

High risk of maternal mortality or severe morbidity

Pulmonary arterial hypertension, any cause; severe LV dysfunction (EF <30%, NYHA class III or IV); previous peripartum cardiomyopathy with any residual LV impairment; severe mitral stenosis; severe aortic stenosis; severe aortic coarctation; Marfan syndrome with aortic dilation >4.5 cm; bicuspid aortic valve with aortic dilation >5 cm

Pregnancy is inadvisable. If pregnancy occurs, termination should be offered. In the event of pregnancy, ongoing subspecialist care and immediate specialist referral is required (see III, above)

ASD = atrial septal defect; EF = ejection fraction; LV = left ventricle; NYHA = New York Heart Association; PDA = patent ductus arteriosus; VSD = ventricular septal defect; WHO = World Health Organization.

Adapted from Canobbio MM, Warnes CA, Aboulhosn J, et al. Management of Pregnancy in Patients With Complex Congenital Heart Disease: A Scientific Statement for Healthcare Professionals From the American Heart Association. Circulation. *2017;135(8):e50-e87.*

Table 6. New York Heart Association Classes of Heart Failure

NYHA Class	Patient Symptoms
I	No limitation of physical activity Ordinary physical activity does not cause undue fatigue, palpitation, dyspnea (shortness of breath)
II	Slight limitation of physical activity Comfortable at rest Ordinary physical activity results in fatigue, palpitation, dyspnea (shortness of breath)
III	Marked limitation of physical activity Comfortable at rest Less than ordinary activity causes fatigue, palpitation, or dyspnea
IV	Unable to carry on any physical activity without discomfort Symptoms of heart failure at rest If any physical activity is undertaken, discomfort increases

NYHA = New York Heart Association.

Information from Simpson LL. Maternal cardiac disease: update for the clinician. Obstet Gynecol. *2012;119(2 Pt 1):345-359.*

Specific Congenital Lesions

When considering cardiac disease in pregnancy, it is important to keep the normal physiologic changes in mind. Lesions, which decompensate with tachycardia, increased cardiac output, or decreased systemic vascular resistance will be poorly tolerated during pregnancy. Women with CHD who already have heart failure or arrhythmias are also at risk of poor outcomes. The AHA distinguishes between *simple* and *complex* CHD in adults (*Table 8*);[22] however, patients with simple CHD may have an increased pregnancy

risk in the presence of cardiac or other comorbidities. The AHA also reiterates that "patients with complex CHD should be managed and delivered at a regional or tertiary center where a multidisciplinary team with knowledge and experience in adult CHD is available."[22]

Brief synopses of simple and complex CHD are given in *Appendices A* and *B*, respectively.

Ischemic Heart Disease

Ischemic heart disease is uncommon during pregnancy, though as maternal age and body mass index increases, the prevalence may be expected to increase. In the large ROPAC registry of 1,321 pregnant women with heart disease, 66% had CHD, whereas 25% had valvular, 7% had cardiomyopathy, and only 2% had ischemic disease.[35] Though the number of women with ischemic disease was small (25 total), their pregnancies tended to end earlier (mean duration = 36 weeks' gestation) and with a higher proportion of cesarean deliveries (60%) than the other subgroups. Maternal mortality, reassuringly, was zero in this group.

A US population-based study of acute myocardial infarction (AMI) during pregnancy showed an incidence of 6 per 100,000 deliveries, with 27% of AMIs occurring in the postpartum period.[36] The risk of AMI increased stepwise with maternal age: women 40 years or older had an event rate of 30 per 100,000 deliveries. Black

Table 7. Current Guidelines for Suggested Contraception in Patients With Cardiovascular Disorders

	Peripartum Cardiomyopathy	Valvular Disease on no Anticoagulation	Valvular Disease on Anticoagulation	Congenital Cardiac Defect
Combined Hormonal Contraceptives: Pill, Patch, Ring **Risks include:** thromboembolism, stroke, myocardial infarction, lipid abnormalities **Risk of unintended pregnancy:** User dependent up to 9/100	Based on individual patient profile in consultation with cardiologist	Based on individual patient profile in consultation with cardiologist	Avoid	Based on individual patient profile in consultation with cardiologist
Progestin only **Risk of unintended pregnancy:** User dependent up to 9/100	Recommended	Recommended	Recommended	Based on individual patient profile in consultation with cardiologist
Progestin Injection **Risks include:** fluid overload **Risk of unintended pregnancy:** 6/100	Recommended	Recommended	Recommended	Based on individual patient profile in consultation with cardiologist
Progestin Implant **Risk of unintended pregnancy:** Less than 1/100	Recommended	Recommended If mechanical valve, antibiotic prophylaxis	Recommended If mechanical valve, antibiotic prophylaxis	Based on individual patient profile in consultation with cardiologist
Copper IUD **Contraindicated in:** Allergy to copper Wilson's disease **Risk of unintended pregnancy:** Less than 1/100	Recommended	Recommended	Recommended if mechanical valve, antibiotic prophylaxis	Based on individual patient profile in consultation with cardiologist
Levonorgestrel IUD **Risk of unintended pregnancy:** Less than 1/100	Recommended	Recommended	Recommended If mechanical valve, antibiotic prophylaxis	Based on individual patient profile in consultation with cardiologist

IUD = intrauterine device.

Reprinted from Hameed AB, Morton CH, Moore A. Improving Health Care Response to Cardiovascular Disease in Pregnancy and Postpartum. 2017. Available at https://www.cdph.ca.gov/Programs/CFH/DMCAH/RPPC/CDPH%20Document%20Library/CMQCC_CVD_Toolkit.pdf.

Table 8. Classification of Congenital Cardiac Lesions

Simple CHD

Mild pulmonary valve stenosis

Small, uncomplicated ASD, VSD, or PDA

Successfully repaired ASD, VSD, PDA, or anomalous pulmonary venous connection

Complex CHD: Moderate Severity

Aorta-to-left-ventricle fistula

Anomalous pulmonary venous return, partial or total

Atrioventricular canal defect, partial or complete

Coarctation of the aorta

Ebstein anomaly

Infundibular right ventricular outflow obstruction, significant

Ostium primum ASD

PDA, not corrected

Pulmonary valve regurgitation, moderate or severe

Pulmonary valve stenosis, moderate or severe

Sinus of Valsalva fistula or aneurysm

Sinus venosus ASD

Subvalvular or supravalvular aortic stenosis, except hypertrophic obstructive cardiomyopathy

Tetralogy of Fallot

VSD with: absent valve, aortic regurgitation, coarctation of the aorta, mitral disease, RV outflow tract obstruction, straddling tricuspid/mitral valve, or subaortic stenosis

Complex CHD: Great Complexity

Conduits, valved or unvalved

Cyanotic CHD, all forms

Double-outlet ventricle

Eisenmenger syndrome

Fontan procedure

Mitral atresia

Single ventricle

Pulmonary atresia, all forms

Transposition of the great arteries

Tricuspid atresia

Truncus arteriosus (including hemitruncus)

Other abnormal atrioventricular or ventriculo-arterial connections (crisscross heart, isomerism, heterotaxy syndromes, ventricular inversion

ASD = atrial septal defect; CHD = congenital heart disease; PDA = patent ductus arteriosus; RV = right ventricle; VSD = ventricular septal defect.

Adapted from Warnes CA, Liberthson R, Daneilson GK, Dore A, et al. Task Force 1: The changing profile of congenital heart disease in adult life. J Am Coll Cardiol. *2001;37(5):1161-1198.*

women were twice as likely as white women to sustain AMI in every age group. Other risk factors were hypertension, history of thrombosis, and to a lesser extent, anemia, diabetes, and smoking. The study showed no statistically significant association with obesity. It is possible that the United States may be an outlier among high-income countries because a population-based study in the United Kingdom from the same period showed an almost 10-fold lower incidence (0.7 MIs per 100,000 pregnancies) and no deaths.[37]

Acute coronary events during pregnancy, while rare, have a mortality risk of 5% to 10%.[2] When an acute coronary event is diagnosed during pregnancy, the patient should be treated in a center able to perform angiography and percutaneous coronary intervention (PCI). No information is available on the safety of drug-eluting stents during pregnancy. The decision between medical management and percutaneous intervention should be made by a cardiology subspecialist. PCI is typically recommended in STEMI and in unstable patients with NSTEMI who are not pregnant, and there is no clear rationale for treating women who are pregnant differently.[2,38] Emergency coronary bypass grafting carried a high risk of fetal loss in older literature, but contemporary data on fetal risk are lacking.

Women who are pregnant seem to be vulnerable to spontaneous coronary artery dissection (SCAD), which manifests in a more severe form than in women who are not pregnant.[39] SCAD typically occurs in the third-trimester, though a few cases have been reported in the second trimester and in the postpartum period. Almost all patients present with chest pain, 75% have ECG changes of ST-segment-elevation myocardial infarction,[40] and LV function is significantly impaired. In the largest case series, 44% of patients had an ejection fraction less than 40% at presentation, and 24% developed cardiogenic shock.[39]

Treatment of SCAD in pregnancy is more complicated than in patients who are not pregnant. In one study, of the 54 patients with SCAD that received conservative medical management (eg, aspirin, antiplatelet or anticoagulation medications, beta-blockers, and/or nitrates), a large number required further intervention. Of the patients receiving PCI (44 patients), only 50% had a completely successful procedure, whereas 25% had extension of the dissection. Ultimately, 54 patients

were treated with CABG. Given the high risk and need for surgical intervention, patients with suspected SCAD should be treated at a tertiary facility that can manage the complexity of this condition.[41]

Valvular

Stenotic lesions are poorly tolerated in pregnancy, while regurgitant lesions typically do not decompensate.[42] Normal pregnancy is characterized by an increased heart rate, increased plasma volume, and increased cardiac output, all of which are problematic for stenotic valves, with intolerance of volume loading and inability to increase cardiac output.

In aortic stenosis, the LV is hypertrophied and poorly compliant. Systolic and/or diastolic dysfunction may be present. Increased volume loading may result in heart failure. Aortic valve gradients typically increase during pregnancy. Women with severe aortic stenosis (valve area less than 1.0 cm peak velocity gradient greater than 4 m/second, mean velocity gradient greater than 40 mm Hg) are at particularly high risk, and those who are symptomatic prior to pregnancy should be counseled against becoming pregnant.[42] Beta blockers and diuretics are indicated if heart failure develops, though care must be taken not to drop preload too aggressively. Percutaneous valvuloplasty may be needed if medical management is unsatisfactory.[42] Valve replacement is better performed outside of pregnancy, as maternal and fetal mortality is high when it is urgently required during pregnancy.

Cardiac function in mitral stenosis is dependent on a slow heart rate and normovolemia. As heart rate increases, time for LV filling (in diastole) is decreased. Left atrial pressure is increased with hypervolemia, leading to pulmonary edema. Severe mitral stenosis (valve area less than 1.5 cm^2) falls into WHO pregnancy risk category IV, and those women should be counseled against pregnancy. Labor and the immediate postpartum period are particularly risky for patients with heart failure. Beta blockers and diuretics should be continued during pregnancy.[42] Anticoagulation is indicated in women with atrial fibrillation during pregnancy, as it is for other individuals. Percutaneous balloon commissurotomy has been performed during pregnancy with satisfactory results, though pre-pregnancy intervention is preferable.[42]

Aortic regurgitation (AR) generally does not pose significant challenges during pregnancy.[42] The increased heart rate and decreased systemic vascular resistance characteristic of pregnancy serve to diminish the amount of regurgitant flow. Acute, de-novo aortic regurgitation, however, predisposes to heart failure, as does chronic severe AR with LV dysfunction. If medical management is required, it typically will be with diuretics or afterload-reducing agents.[42]

Mitral regurgitation is seldom an issue during pregnancy, for similar reasons to AR.[42] Severe symptoms prior to pregnancy, however, should be managed surgically and pregnancy deferred until stability is achieved.

Patients who have undergone previous valve replacement are at risk of other complications. Bioprosthetic valves have a limited lifespan. Up to 35% of porcine valves and 20% of human cadaveric valves fail within 15 years of placement,[43] and pregnancy may accelerate valve deterioration. Alternatively, mechanical heart valves, although less likely to fail, are prone to thrombosis, and women with these valves require lifelong anticoagulation therapy. The choice of anticoagulant drug involves a trade-off between maternal and fetal interests. Warfarin is teratogenic and associated with an increased risk of pregnancy loss because it crosses the placenta, anticoagulates the fetus, and predisposes the fetus to hemorrhagic complications. Heparin, including low-molecular-weight heparins, does not cross the placenta so does not incur the same risks of teratogenesis and fetal/neonatal hemorrhage. However, heparin is much less protective against valve thrombosis, a potentially lethal maternal event. In women with a mechanical heart valve, discussions about anticoagulation should precede conception, and management during pregnancy should be a joint effort between cardiology subspecialists and maternity care providers.

Pulmonary Hypertension

Pulmonary hypertension (PH) of any etiology endangers pregnancy. The WHO, American Heart Association, European Society of Cardiology, and other experts recommend against pregnancy when PH is present[28,44] because maternal mortality is high. There has been some improvement from the late-20th-century data. Between 1978 and 1996, the risk of maternal death was 38% among women with PH and decreased to 25% in the decade between 1997 and 2007.[45] Within the overall

category of PH, there are subsets of primary, or idiopathic, pulmonary arterial hypertension, PH associated with CHD, PH associated with left heart disease, and PH due to other causes. In the largest contemporary case series, 50% of women with PH required hospitalization at least once during pregnancy.[44] There were no maternal deaths in this series among women with mild or moderate PH (right ventricle systolic pressure between 30 to 50 mm Hg or 50 to 70 mm Hg, respectively), but 25% of women with severe PH (right ventricle systolic pressure greater than 70 mm Hg) died, most in the first week after delivery. Heart failure was a common complication, occurring in approximately 28% of the women, arrhythmias occurred in approximately 7%, and thrombotic or thromboembolic events occurred in 3%. Most patients with PH (63%) underwent cesarean delivery. Of the newborns, 19% had low birthweight, and the perinatal mortality rate was 9%.

The reasons pregnancy is risky in women with PH are thought to be related to the interplay between the physiologic demands of pregnancy and the functional limits on the right ventricle. An increase in plasma volume overloads the right ventricle, which cannot appropriately increase its output against resistance. In addition, there may be intracardiac shunting, and right-to-left shunting increases when systemic vascular resistance drops in pregnancy, which does not occur in cases of PH. The resultant cyanosis and hypoxemia, which is not correctable with supplemental oxygen, further increases pulmonary artery pressures. As is typical in many types of heart disease, additional volume shifts during labor, at delivery, and during the postpartum period are poorly tolerated.

Pre-pregnancy counseling plus access to reliable and affordable contraception, backed up by safe and legal abortion, is important for women with PH of any etiology. For those who do become pregnant, surveillance for development of arrhythmias and heart failure is crucial, and management must be multidisciplinary and performed in a facility that can support obstetric and complex cardiac cases. Insofar as possible, measures are taken to reduce pulmonary vascular resistance and to optimize right ventricle function. Activity limitation typically is recommended, and hospitalization for intensified monitoring is common past mid-pregnancy. Serial echocardiography is indicated. Anticoagulation, therapeutic or prophylactic, is appropriate in many patients. Options for medical therapy outside of pregnancy include diuretics, calcium channel blockers, endothelin receptor antagonists, phosphodiesterase-5 inhibitors, and prostacyclin analogues. Extensive human data on all these drugs is not currently available, but many have been used in human pregnancy.[44] For example, the ROPAC investigators had pregnant women treated with phosphodiesterase inhibitors, endothelin receptor antagonists, and prostacyclin analogues.[44] Diuretics and calcium channel blockers are generally understood as safe in human pregnancy. It is crucial that a woman with PH who is considering pregnancy or already pregnant be treated in conjunction with a cardiology subspecialist experienced in managing PH.

Cardiomyopathies

Not all cardiomyopathy seen in pregnancy is peripartum cardiomyopathy. It is important to remember that this is a diagnosis of exclusion.

Cardiomyopathy is a functional disorder of heart muscle, defined by the AHA as, "… a heterogeneous group of diseases of the myocardium associated with mechanical and/or electrical dysfunction that usually (but not invariably) exhibit inappropriate ventricular hypertrophy or dilatation and are due to a variety of causes that frequently are genetic."[46] They may be primary (involving only the heart) or secondary (part of a multisystem constellation of disorders). Primary cardiomyopathies are classified as genetic, acquired, or mixed. Among primary genetic cardiomyopathies are hypertrophic cardiomyopathy (HCM), arrhythmogenic right ventricular cardiomyopathy/dysplasia, LV noncompaction, and several ion channelopathies (long QT syndrome, short QT syndrome, Brugada syndrome, catecholaminergic polymorphic ventricular tachycardia). HCM is the most common cardiomyopathy, affecting 1 in 500 individuals in the United States. It is autosomal dominant, has been reported in association with at least 11 mutations, and may manifest at young ages. Women with HCM are at risk during pregnancy and may produce offspring with the same disease. The group of acquired cardiomyopathies includes myocarditis, which is inflammatory in cause, those triggered by infection, cocaine exposure, or hypersensitivity reactions to many different drugs; stress cardiomyopathy, also known as Tako-Tsubo; peripartum cardiomyopathy, a type

of dilated cardiomyopathy; and a reversible type of dilated cardiomyopathy (DCM) precipitated by prolonged ventricular or supraventricular tachycardia. The third type of primary cardiomyopathy is mixed, primarily nongenetic but with a few familial cases reported. This third group includes DCM and primary restrictive nonhypertrophic cardiomyopathy.

For the provider, it may be more useful to consider these diseases by their function or management. HCM and DCM will be discussed here.

Hypertrophic cardiomyopathy is diagnosed by echocardiographic measurement of LV wall thickness without LV dilation, and is characterized by diastolic dysfunction.[46,47] Some patients have obstruction to the left ventricular outflow tract (LVOT), which, if present, is worsened by tachycardia and by decreased filling volume. Patients may be asymptomatic or they may develop heart failure and/or arrhythmias. Because HCM is an autosomal dominant condition, there is a 50% risk of a woman with HCM birthing an infant with HCM, so genetic counseling is important. Pregnancy does not typically worsen cardiac status unless a woman is already symptomatic or has significant LVOT obstruction. Increased cardiac volumes are better tolerated than dehydration. Tachycardia will be problematic in the presence of LVOT obstruction because it decreases preload and LV filling, and worsens outflow tract obstruction.

Beta blockers are a mainstay of HCM therapy and should not be discontinued during pregnancy. Acute decreases in preload, as may be seen with the sympathectomy that accompanies neuraxial analgesia for labor, must be avoided, but regional analgesia is important in labor because it limits catecholamine response and tachycardia related to pain. Prostaglandins of the E series (ie, dinoprostone, misoprostol) have a vasodilatory effect and should only be used with caution. Pushing in the second stage of labor should be avoided, so vaginal delivery typically is aided with vacuum or forceps.[47] In a review of 408 cases spanning 40 years and a wide range of disease severity, 62% of women with HCM who were pregnant had a vaginal delivery, 29% had a cardiac complication or a worsening of symptoms (dyspnea being the most common outcome), and there was 1 maternal death.[48] The rate of preterm birth was 26%, there was no excess of stillbirth or growth restriction compared with the general population, but there was a 3% risk of what was described as *fetal bradycardia*.[48] In the European ROPAC dataset, a contemporary cohort of 60 women with HCM had a 23% incidence of major cardiac complications (heart failure or arrhythmia), 5% fetal deaths, and no maternal deaths.[49] Fetal bradycardia was not mentioned, 47% of infants were preterm, 32% were small for gestational age, and only 40% were delivered vaginally.

By contrast, DCM is a disorder of LV systolic function whereby contractility is diminished and the LV is dilated. Except for peripartum cardiomyopathy, which is discussed below, DCM is uncommon among women of reproductive age, so data are limited. The rate of cardiac complications in DCM during pregnancy has been reported between 39% and 60%,[50,51] with outcomes being worse among women with higher NYHA classification disease. In the overall cohort, low birthweight occurred in 40% of births and preterm birth in 23%, and for women with NYHA class III/IV disease, the rate of fetal/neonatal complications was 67%. Women with chronic heart failure are generally treated during pregnancy as they would be outside of pregnancy, except that angiotensin-converting enzyme (ACE) inhibitors and angiotensin receptor blockers are avoided because of adverse fetal effects. They may continue beta blockers and diuretics. Afterload reduction also may be needed, in which case hydralazine or nitrates are recommended.[22]

Peripartum cardiomyopathy (PPCM), a type of dilated cardiomyopathy, is defined by the European Society of Cardiology as "an idiopathic cardiomyopathy presenting with heart failure secondary to LV systolic dysfunction toward the end of pregnancy or in the months following delivery, where no other cause of heart failure is found. It is a diagnosis of exclusion. The LV may not be dilated but the ejection fraction is nearly always reduced below 45%."[52] The National Heart, Lung and Blood Institute has defined PPCM as the development of heart failure in the last month of pregnancy or in the first 5 months after delivery, the absence of recognizable heart disease prior to the last month of pregnancy, and LV systolic dysfunction by echocardiographic criteria (LV ejection fraction less than 45% and/or fractional shortening less than 30%.[53] The cause of PPCM remains unknown, but many possible mechanisms have been proposed, such as autoimmunity,

inflammation or myocarditis, selenium deficiency, sodium retention, oxidative stress, imbalance of angiogenic factors, endocrine triggers, and genetic explanations.[52,53] There seems to be some overlap of genetic variants between PPCM and other dilated cardiomyopathies.[54]

PPCM manifests clinically with the signs and symptoms of any other type of heart failure characterized by LV dysfunction[52] (eg, dependent edema, exertional dyspnea, orthopnea, fatigue). In mild cases of PPCM, symptoms are easily confused with discomforts of normal pregnancy or the demands of the postpartum period. Most patients develop PPCM postpartum. Although this remains a relatively rare disease (1:2,000 US births),[55] the index of suspicion should be high because of the potential for morbid or fatal outcomes. In a recent, prospectively collected, North American cohort study of patients with PPCM, 6% had received an LV assist device (LVAD) or heart transplant, or died, within 1 year of diagnosis.[55]

Standard therapy for heart failure is appropriate in PPCM.[52,55] Medical therapies are beta blockers and ACE inhibitors or angiotensin receptor blockers, though the latter two are deferred until after delivery. Hydralazine or nitrates are options for reducing afterload. Anticoagulation is important. Diuretics, inotropes, or mechanical support devices (intra-aortic balloon pump, LVAD) are sometimes required. Because a majority of women with PPCM will recover systolic function within 6 to 12 months, LVAD may be a temporizing intervention rather than a bridge to inevitable transplantation. More than 60% of women with a diagnosis of PPCM recover normal LV function within 6 months after diagnosis,[56] though recovery is much less likely when the initial LVEF is less than 30%. Clinical recovery of ventricular function, however, may not hold up in a subsequent pregnancy. Data, though limited, suggests that approximately 20% of women with LVEF that normalized after a pregnancy with PPCM nevertheless experienced significant LV dysfunction with a subsequent pregnancy.[57]

Preeclampsia and hypertension often are present in patients with PPCM. In a recent meta-analysis of 22 studies and 979 cases, the prevalence of preeclampsia among women with PPCM was 22%, more than 4 times the national prevalence.[58] Of those diagnosed with PPCM, 37% had some type of hypertension during pregnancy (ie, preeclampsia, gestational hypertension, chronic hypertension). However, this is likely an underestimate, as hypertensive disorders often are used as exclusion criteria when diagnosing PPCM.

Pregnancy-related hypertensive disorders (eg, preeclampsia, gestational hypertension, chronic hypertension with superimposed preeclampsia) can lead to fluid overload even without PPCM. A recent study of 30 cases of PPCM and 53 cases of heart failure associated with hypertension showed that heart failure typically presented before delivery and was associated with cardiac hypertrophy with preserved EF and better prognosis compared with PPCM.[59]

Racial differences should be noted in cases of PPCM. Black women tend to be significantly younger than white women (average age 26 years versus 30 years at the time of diagnosis), are more likely to be diagnosed in the postpartum period, and tend toward increased mortality and lower recovery of ejection fraction.[60] Although these diseases of preeclampsia and PPCM are associated and may coexist, they are considered to be separate conditions, and the diagnosis of preeclampsia in the setting of PPCM should not delay treatment.[61]

Arrhythmia

Occasional ectopic beats in pregnancy are common and not a cause for alarm. New-onset sustained arrhythmias are uncommon in a structurally normal heart, though women with a history of arrhythmia or a structural cardiac anomaly often exhibit arrhythmias during pregnancy. Women who are pregnant who are referred for outpatient cardiac evaluation because of reports of palpitations most often have isolated premature atrial contractions (PAC) or premature ventricular contractions (PVC).[62] In a case series of 100,000 pregnant women hospitalized for arrhythmias, 104 patients had sinus arrhythmia, sinus tachycardia or sinus bradycardia, 33 had supraventricular tachycardia (SVT), and 24 had PAC or PVC.[63] All episodes of SVT terminated spontaneously or responded swiftly to medical intervention. Atrioventricular block, atrial fibrillation, and sustained ventricular tachycardia or fibrillation were rare.

The occurrence, recurrence, or development of arrhythmia during pregnancy in a structurally abnormal heart, however, is a more serious issue, whether the underlying disease process is a complex congenital lesion, a valvular lesion, or

impaired myocardial function. Specific lesions predispose to specific arrhythmias (eg, atrial fibrillation/flutter, focal ectopic atrial tachycardia with repaired tetralogy of Fallot, atrial fibrillation with mitral stenosis, symptomatic bradycardia after a Fontan procedure, atrial switch operation).[64] Arrhythmias represent a sequela of cardiac abnormality and a marker for adverse maternal outcomes in women with preexisting heart disease.

Evaluation of suspected arrhythmia in a woman who is pregnant and not known to have heart disease is similar to that of nonpregnant patients: 12-lead ECG, Holter monitor or event recorder, and, if indicated, an echocardiogram. A review of drugs may suggest a precipitating factor. Thyroid status should be assessed. Women with no sustained arrhythmia and with a structurally normal heart typically require only reassurance, not pharmacotherapy. However, sustained or hemodynamically significant arrhythmias, or arrhythmia in structural heart disease, need treatment. A detailed discussion of antiarrhythmic therapy is beyond the scope of this chapter. Also, there are few trials focused specifically on treatment of women who are pregnant. In general, treatment during pregnancy does not differ from treatment outside of pregnancy,[64,65] except for the use of amiodarone. Amiodarone's prolonged half-life in the fetus and newborn indicates a long duration of all potential adverse effects (eg, thyroid dysfunction, prolonged QT). Most antiarrhythmic drugs, except adenosine, cross the placenta in sufficient quantities to make it feasible to treat fetal arrhythmias by administering an antiarrhythmic drug to the woman.

Direct current cardioversion (DCCV) may be used during pregnancy for hemodynamically unstable or drug-refractory arrhythmias. Settings are no different. A case report of a sustained uterine contraction and fetal bradycardia at 28 weeks' gestation after maternal DCCV with 50 J has led to a recommendation that fetal monitoring be used during and after the intervention.[66] It is not clear how common such an outcome would be; the authors of the report cite several instances of uneventful DCCV administration during pregnancy.[66]

Pacemakers may be inserted during pregnancy applying the usual rules regarding fluoroscopic radiation. Limited data on the outcome of pregnancies in which the woman had an implanted cardioverter-defibrillator are reassuring.[67]

Women with arrhythmias (aside from occasional benign ectopic beats) should undergo cardiac monitoring during labor.[22] Drugs with arrhythmogenic potential, such as terbutaline, should be avoided if possible.

Preeclampsia and Heart Failure

The etiology of preeclampsia is unclear, but it is thought that placental development has a key role. Deficient spiral artery remodeling in these placentas early in pregnancy leads to intermittent perfusion and oxidative stress, causing the release of antiangiogenic factors resulting in endothelial dysfunction.[68]

Patients with preeclampsia are at high risk of cardiovascular complications. Circulating antiangiogenic factors and endothelial dysfunction may predispose to heart failure transiently as well as predispose to more severe heart failure such as PPCM.[69]

There is evidence that preeclampsia leads to permanent remodeling of vasculature, leading to renal and metabolic changes and predisposing to permanent CVD.[70]

General Principles of Labor Management in Heart Disease

For labor management, a multidisciplinary approach, considering the woman's individual specific risk factors, is recommended (*Table 9*).[11] The majority of patients can be offered a trial of labor.[71]

The risk of stroke is much higher in women with CHD than in the general population. In women with cyanotic lesions, the potential for paradoxical embolism of air or venous clot must always be considered. Therefore, when intravenous access is required, it is preferable to add a filter to the line.

An assisted vaginal delivery is offered to limit pushing when concern exists about increased work or increased intra-abdominal/intrathoracic pressure during the second stage of labor. A shortened second stage often is thought to be beneficial in women with cardiac disease.[72] Also, early epidural analgesia administration often is recommended to decrease sympathetic stimulation and oxygen demands. In patients with fixed cardiac output, careful management of fluid balance is crucial.

Specific instances where cesarean delivery is recommended include dilated aortic root (more than 4 cm) or aortic aneurysm, acute severe congestive heart failure, a history of recent myocardial infarction, severe symptomatic aortic stenosis, warfarin

administration within 2 weeks of delivery, or need for emergency valve replacement immediately after delivery. [11] However, most women with CVD appear to incur no benefit from planned cesarean delivery simply for cardiac indications.[73]

Overall, induction methods, including mechanical and medical, have an acceptable safety profile.[74] Cardiovascular and hemodynamic monitoring are considered on a case-by-case basis. Fluid management may be challenging when CVD is present. Hypervolemia or hypovolemia may be dangerous. Careful measurement of intake and output is crucial. In rare cases, invasive monitoring may still be used to guide fluid management. Bedside echocardiography has an increasingly important role. Cardiac rhythm monitoring typically will be indicated. Supplemental oxygen often is administered, though it is unlikely to improve cyanosis due to intracardiac shunting.

Endocarditis Prophylaxis

Although the simple presence of maternal CHD is not an indication for prophylaxis,[75] the AHA and American College of Cardiology recom-

Table 9. Multidisciplinary Approach For Labor Management

When planning delivery in a woman with known heart disease, adopting a systematic approach is helpful. The following questions should be addressed, ideally with the involvement of a cardiologist, maternity care provider, and anesthesiologist:

1. Where should she deliver: at what institution? What services might she require? Women at risk of aortic rupture, for example, should be delivered at an institution with ready access to cardiovascular surgery. If the infant also has a significant cardiac lesion - and some are inherited - what specific neonatal services must be available on-site?
2. Where should she deliver? Most deliveries can be managed in a labor and delivery unit, but some women may need to deliver in a cardiac care or intensive care unit. Some women may need to deliver in a cardiac-equipped operating room
3. Can she undergo labor, or should she deliver by cesarean delivery? Cardiac indications for cesarean delivery (eg, dilated aortic root, fresh myocardial infarction) should be considered. Usual obstetric indications apply
4. If she is to undergo labor, can labor be managed expectantly, awaiting spontaneous labor? Should labor be induced? In addition to standard obstetric or fetal reasons for labor induction, many women with cardiac pathology have labor induced so all needed services and personnel can be present
5. What is the plan for pain management during labor? Most women with significant cardiac disease benefit from epidural analgesia, which decreases catecholamine release and blunts some of the hemodynamic changes caused by labor. Epidural analgesia should be sited early in the labor process
6. Does she need additional monitoring? Women with cardiac disease are at particular risk of arrhythmia and heart failure. Many should undergo cardiac rhythm monitoring. Some should have monitoring of filling pressures in the left or right heart, though invasive intravascular monitoring is not commonly used. Noninvasive or minimally invasive monitoring (eg, bedside echocardiography, pulse contour wave analysis) may be substituted. In select cases of cyanotic CHD, it may be necessary to monitor oxygen saturation to determine shunt fraction
7. How should fluids be managed? Is the cardiac lesion one that benefits from lower or higher effective circulating volume?
8. What drugs should be avoided or used with caution? The adverse effects of any drug being considered in women with heart disease must be understood and prepared for
9. What is the appropriate nurse-to-patient ratio during labor? Nurses should not be expected to care for other patients at the same time, except in cases where heart disease concerns are minimal. In some cases, additional nursing expertise must be sought. It is not unusual for a woman with cardiac disease who is in labor to require a labor and delivery nurse and a cardiac nurse
10. If the woman is laboring and a vaginal delivery is planned, how should the second stage of labor be managed? Common recommendations for labor management in women with cardiac conditions include limiting maternal expulsive efforts and using forceps or vacuum-assisted delivery to shorten the second stage of labor
11. How is the third stage to be managed? Hemorrhage is destabilizing for most women, but uniquely so for women with a fixed cardiac output (ie, aortic stenosis)
12. If cesarean delivery is preferred, should it be performed in the labor and delivery unit where other cesarean deliveries typically are performed, or is the main operating room required? In some cases, for example, if cesarean delivery and valve replacement are to take place under the same general anesthetic, the procedure must be performed in the cardiac operating room
13. What is the postpartum plan? The immediate puerperium is the period of highest risk in many women with cardiac conditions (eg, mitral stenosis), because of the increase in effective circulating volume. Where should the patient be managed for the first 24-48 hours postpartum? what should be the nurse-to-patient ratio? Are there specific therapies that must be implemented?
14. Does she plan to breastfeed? Most women with cardiac disease can breastfeed; many drugs are compatible with breastfeeding, and those which are not known to be safe can often be substituted
15. What is the plan for contraception?
16. Finally, what is the plan for follow-up after she is discharged from the hospital? Women with cardiac disease cannot wait till 6 weeks postpartum to be seen. They may need to be seen sooner by a cardiologist or cardiothoracic surgeon, or they may need to review symptoms with a maternity care provider in an outpatient setting or at home

mend endocarditis prophylaxis in patients with a certain subset of cardiac lesions, when associated with infection. These patients include those with prosthetic valves, a history of endocarditis, CHD associated with unrepaired cyanotic defect including palliative shunts and conduits, defects repaired with prosthetic material or device within the past 6 months, and incompletely repaired defects using prosthetic material.[75]

Endocarditis prophylaxis regimens are administered 30 to 60 minutes before delivery and options include 2 g of ampicillin IV or 1 to 1.5 g of cefazolin sodium IV. In the patient with a penicillin or ampicillin allergy, 1 g of ceftriaxone IV or 600 mg clindamycin IV can be administered, and 1 g of vancomycin IV should be added if enterococcus infection is a concern.[76]

Anticoagulation

Cardiac conditions that may require anticoagulation in pregnancy and the postpartum period include recent thromboembolic events, presence of mechanical heart valves, atrial fibrillation, peripartum cardiomyopathy, and PH.[72]

To minimize the risk of epidural hematoma, epidural or spinal anesthesia (ie, neuraxial anesthesia) is delayed 12 hours after the last dose of prophylactic enoxaparin and 24 hours after the last dose of therapeutic enoxaparin.[77,78] Before placing neuraxial anesthesia, the 2018 Society for Obstetric Anesthesia and Perinatology (SOAP) consensus statement recommends waiting at least 4 to 6 hours after administration of low-dose unfractionated heparin (UFH) (up to 5,000 units 3 times/day), at least 12 hours after administration of intermediate-dose UFH (7,500 or 10,000 units 2 times/day), and 24 hours of more after administration of high-dose UFH (an individual dose greater than 10,000 units or a total daily dose greater than 20,000 units). If the aPTT level is normal or anti-factor Xa is undetectable, the SOAP guideline states such patients are at low risk of neuraxial analgesia complications even when these wait times are not met after UFH.[77,79,80] Low-dose aspirin (eg, 81 mg/day) does not pose substantial risk and does not need to be stopped before labor begins.[81]

Postpartum Management of Cardiovascular Disease

Many changes in hemodynamic status occur in the immediate postpartum period. Cardiac output peaks at delivery, remains elevated for approximately 1 hour, and declines to pre-pregnancy values by approximately 2 weeks' postpartum.[82] Cardiac output and stroke volume do not always return to baseline and, women with hypertensive disorders, cardiac lesions, cardiomyopathy, or PH have a higher than average risk of death postpartum. Close monitoring by maternal-fetal medicine and cardiology subspecialists is imperative for high-risk patients.

Postpartum hemorrhage presents challenges for women with CVD. Those whose cardiac output is highly preload dependent can decompensate rapidly even with modest degrees of blood loss. Tachycardia is dangerous for women with stenotic valvular lesions. Women taking beta blockers typically do not have compensatory tachycardia in response to hypovolemia, so the diagnosis of hemorrhage may be delayed. Intracardiac shunts behave unpredictably with systemic vasoconstriction, a compensatory response to volume loss. Finally, drugs used to treat uterine atony may have cardiac or adverse effects.

Women who present postpartum with new-onset cough, fatigue, or dyspnea, should be treated as having possible CVD resulting in heart failure. Further cardiac evaluation in the postpartum period may be warranted.

Medications in Pregnant and Breastfeeding Women With Cardiovascular Disease

Decision making about prescription drug use in pregnant and breastfeeding women must consider maternal benefits, potential fetal risks, timing of fetal exposure, transplacental passage (or partition into breastmilk), alternative drugs, and the risk of discontinuing drugs. The old Food and Drug Administration pregnancy categories (ie, A, B, C, D, X) were not useful because they focused on teratogenesis and were, therefore, irrelevant after the first trimester. It has been replaced by a more detailed classification, the Physician Labeling Rule available at https://www.fda.gov/Drugs/DevelopmentApprovalProcess/DevelopmentResources/Labeling/ucm093307.htm.

Health care providers are advised to consult specific pregnancy registries whenever possible (https://www.fda.gov/ScienceResearch/SpecialTopics/WomensHealthResearch/ucm134848.htm). Women should be encouraged to enroll in

pregnancy registries to help expand the databases (*Table 10*).

Contraception in Women With Cardiovascular Disease

Women with CVD, like all women, should be educated about contraceptive options. Nonhormonal methods are preferred in most women with CVD because they have a lower risk of cardiovascular events compared with hormonal methods. Hormonal methods should be used with caution.[83]

Table 10. Resources for Medications in Pregnancy and Lactation

The Organization of Teratology Information Specialists (OTIS; https://mothertobaby.org) provides information in English and Spanish for women and health care providers

LactMed is a medications database, hosted by the National Library of Medicine, with information on lactation safety (https://toxnet.nlm.nih.gov/newtoxnet/lactmed.htm)

Motherisk (http://www.motherisk.org/; toll-free 1-877-439-2744) is a free, online and telephone teratogen information service provided by the Hospital for Sick Children (Toronto)

Nursing Considerations: Cardiac Complications of Pregnancy

- Know risk factors and understand racial disparities
- Early recognition is key
- Think filter on intravenous lines, strict intake, and output throughout hospitalization
- Rapid decompensation of cardiac status may occur; frequent assessment is essential, especially in the early postpartum period
- Advocate for a postpartum contraceptive plan before discharge for all women but specifically for woman with cardiac disease that may place future pregnancies at risk

References

1. ACOG Practice Bulletin no. 212: pregnancy and heart disease. *Obstet Gynecol*. 2019;133(5):e320-e356.
2. Regitz-Zagrosek V, Blomstrom Lundqvist C, Borghi C, et al.; European Society of Gynecology (ESG). Association for European Paediatric Cardiology (AEPC); German Society for Gender Medicine (DGesGM); ESC Committee for Practice Guidelines. ESC Guidelines on the management of cardiovascular diseases during pregnancy: the Task Force on the Management of Cardiovascular Diseases during Pregnancy of the European Society of Cardiology (ESC). *Eur Heart J*. 2011;32(24):3147-3197.
3. Kuklina E, Callaghan W. Chronic heart disease and severe obstetric morbidity among hospitalisations for pregnancy in the USA: 1995-2006. *BJOG*. 2011;118(3): 345-352.
4. Hameed AB, Lawton ES, McCain CL, et al. Pregnancy-related cardiovascular deaths in California: beyond peripartum cardiomyopathy. *Am J Obstet Gynecol*. 2015; 213(3):379.e1-379.e10.
5. Centers for Disease Control and Prevention. Pregnancy Mortality Surveillance System. 2018. Available at https://www.cdc.gov/reproductivehealth/maternalinfanthealth/pmss.html.
6. Creanga AA, Syverson C, Seed K, Callaghan WM. Pregnancy-related mortality in the United States, 2011-2013. *Obstet Gynecol*. 2017;130(2):366-373.
7. Pritchard JA, Adams RH. Erythrocyte production and destruction during pregnancy. *Am J Obstet Gynecol*. 1960;79(4):750-757.
8. Clark SL, Cotton DB, Lee W, et al. Central hemodynamic assessment of normal term pregnancy. *Am J Obstet Gynecol*. 1989;161(6 Pt 1):1439-1442.
9. Elkayam U, Goland S, Pieper PG, Silverside CK. High-risk cardiac disease in pregnancy: part I. *J Am Coll Cardiol*. 2016;68(4):396-410.
10. American Academy of Family Physicians. Preconception care (position paper). 2015. Available at http://www.aafp.org/about/policies/all/preconception-care.html.
11. Simpson LL. Maternal cardiac disease: update for the clinician. *Obstet Gynecol*. 2012;119(2 Pt 1):345-359.
12. Warnes CA. Pregnancy and delivery in women with congenital heart disease. *Circ J*. 2015;79(7):1416-1421.
13. Hameed AB, Morton CH, Moore A. Improving health care response to cardiovascular disease in pregnancy and postpartum. 2017. Available at https://www.cdph.ca.gov/Programs/CFH/DMCAH/RPPC/CDPH%20Document%20Library/CMQCC_CVD_Toolkit.pdf.
14. Greenland P, Alpert JS, Beller GA, et al.; American College of Cardiology Foundation. American Heart Association. 2010 ACCF/AHA guideline for assessment of cardiovascular risk in asymptomatic adults: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. *J Am Coll Cardiol*. 2010;56(25):e50-e103.
15. van Hagen IM, Thorne SA, Taha N, et al.; ROPAC Investigators and EORP Team. Pregnancy outcomes in women with rheumatic mitral valve disease: results from the Registry of Pregnancy and Cardiac Disease. *Circulation*. 2018;137(8):806-816.
16. Mehta PK, Wei J, Wenger NK. Ischemic heart disease in women: a focus on risk factors. *Trends Cardiovasc Med*. 2015;25(2):140-151.
17. Nihoyannopoulos P. Cardiovascular examination in pregnancy and the approach to diagnosis of cardiac disorder. In: Oakley C, Warnes CA, eds. *Heart Disease in Pregnancy*, 2nd edition. Malden MA: Blackwell Publishing; 2007.
18. Carruth JE, Mivis SB, Brogan DR, Wenger NK. The electrocardiogram in normal pregnancy. *Am Heart J*. 1981; 102(6 Pt 1):1075-1078.
19. M S, S C, Brid SV. Electrocardiographic QRS axis, Q wave, and T-wave changes in 2nd and 3rd trimester of normal pregnancy. *J Clin Diagn Res*. 2014;8(9): BC17-BC21.
20. Liu S, Elkayam U, Naqvi TZ. Echocardiography in Pregnancy: Part 1. *Curr Cardiol Rep*. 2016;18(9):92.
21. Colletti PM, Lee KH, Elkayam U. Cardiovascular imaging of the pregnant patient. *AJR Am J Roentgenol*. 2013; 200(3):515-521.
22. Canobbio MM, Warnes CA, Aboulhosn J, et al.; American Heart Association Council on Cardiovascular and Stroke Nursing. Council on Clinical Cardiology; Council on Cardiovascular Disease in the Young; Council on Functional Genomics and Translational Biology; and Council on Quality of Care and Outcomes Research. Management of pregnancy in patients with complex congenital heart disease: a scientific statement for healthcare professionals from the American Heart Association. *Circulation*. 2017;135(8):e50-e87.
23. Committee on Obstetric Practice. Committee Opinion no. 723: guidelines for diagnostic imaging during pregnancy and lactation. *Obstet Gynecol*. 2017;130(4): e210-e216. https://www.acog.org/-/media/Committee-Opinions/Committee-on-Obstetric-Practice/co723.pdf?dmc=1&ts=20180421T2050575232.
24. Lau ES, Sarma A. The role of cardiac biomarkers in pregnancy. *Curr Treat Options Cardiovasc Med*. 2017; 19(7):49.
25. Siu SC, Sermer M, Colman JM, et al.; Cardiac Disease in Pregnancy (CARPREG) Investigators. Prospective multicenter study of pregnancy outcomes in women with heart disease. *Circulation*. 2001;104(5):515-521.
26. Drenthen W, Boersma E, Balci A, et al. ZAHARA Investigators. Predictors of pregnancy complications in women with congenital heart disease. *Eur Heart J*. 2010; 31(17):2124-2132.
27. Pieper PG. Pre-pregnancy risk assessment and counselling of the cardiac patient. *Neth Heart J*. 2011;19(11): 477-481.
28. Thorne S, MacGregor A, Nelson-Piercy C. Risks of contraception and pregnancy in heart disease. *Heart*. 2006; 92(10):1520-1525.
29. van Hagen IM, Roos-Hesselink JW, Donvito V, et al. Incidence and predictors of obstetric and fetal complications in women with structural heart disease. *Heart*. 2017;103(20):1610-1618.
30. Miner PD, Canobbio MM, Pearson DD, et al. Contraceptive practices of women with complex congenital heart disease. *Am J Cardiol*. 2017;119(6):911-915.

31. Ntiloudi D, Giannakoulas G, Parcharidou D, et al. Adult congenital heart disease: a paradigm of epidemiological change. *Int J Cardiol*. 2016;218:269-274.
32. Pillutla P, Nguyen T, Markovic D, et al. Cardiovascular and neonatal outcomes in pregnant women with high-risk congential heart disease. *Am J Cardiol*. 2016;117(10): 1672-1677.
33. Gaeta SA, Ward C, Krasuski RA. Extra-cardiac manifestations of adult congenital heart disease. *Trends Cardiovasc Med*. 2016;26(7):627-636.
34. Lui GK, Saidi A, Bhatt AB, et al.; American Heart Association Adult Congenital Heart Disease Committee of the Council on Clinical Cardiology and Council on Cardiovascular Disease in the Young. Council on Cardiovascular Radiology and Intervention; and Council on Quality of Care and Outcomes Research. Diagnosis and Management of Noncardiac Complications in Adults With Congenital Heart Disease: a scientific statement from the American Heart Association. *Circulation*. 2017;136(20):e348-e392.
35. Roos-Hesselink JW, Ruys TPE, Stein JI, et al. ROPAC Investigators. Outcome of pregnancy in patients with structural or ischaemic heart disease: results of a registry of the European Society of Cardiology. *Eur Heart J*. 2013;34(9):657-665.
36. James AH, Jamison MG, Biswas MS, et al. Acute myocardial infarction in pregnancy: a United States population-based study. *Circulation*. 2006;113(12):1564-1571.
37. Bush N, Nelson-Piercy C, Spark P, et al. UKOSS. Myocardial infarction in pregnancy and postpartum in the UK. *Eur J Prev Cardiol*. 2013;20(1):12-20.
38. Elkayam U, Jalnapurkar S, Barakkat MN, et al. Pregnancy-associated acute myocardial infarction: a review of contemporary experience in 150 cases between 2006 and 2011. *Circulation*. 2014;129(16):1695-1702.
39. Naderi S. Spontaneous coronary artery dissection and pregnancy. *Curr Treat Options Cardiovasc Med*. 2017; 19(9):69.
40. Havakuk O, Goland S, Mehra A, Elkayam U. Pregnancy and the risk of spontaneous coronary artery dissection: an analysis of 120 contemporary cases. *Circ Cardiovasc Interv*. 2017;10(3):e004941.
41. Hayes SN, Kim ESH, Saw J; American Heart Association Council on Peripheral Vascular Disease. Council on Clinical Cardiology; Council on Cardiovascular and Stroke Nursing; Council on Genomic and Precision Medicine; Stroke Council. Spontaneous coronary artery dissection: current state of the science: a scientific statement from the American Heart Association. *Circulation*. 2018;137(19):e523-e557.
42. Safi LM, Tsiaras SV. Update on valvular heart disease in pregnancy. *Curr Treat Options Cardiovasc Med*. 2017; 19(9):70.
43. Pessel C, Bonanno C. Valve disease in pregnancy. *Semin Perinatol*. 2014;38(5):273-284.
44. Sliwa K, van Hagen IM, Budts W, et al.; ROPAC investigators. Pulmonary hypertension and pregnancy outcomes: data from the Registry Of Pregnancy and Cardiac Disease (ROPAC) of the European Society of Cardiology. *Eur J Heart Fail*. 2016;18(9):1119-1128.
45. Bédard E, Dimopoulos K, Gatzoulis MA. Has there been any progress made on pregnancy outcomes among women with pulmonary arterial hypertension? *Eur Heart J*. 2009;30(3):256-265.
46. Maron BJ, Towbin JA, Thiene G, et al.; American Heart Association. Council on Clinical Cardiology, Heart Failure and Transplantation Committee; Quality of Care and Outcomes Research and Functional Genomics and Translational Biology Interdisciplinary Working Groups; Council on Epidemiology and Prevention. Contemporary definitions and classification of the cardiomyopathies: an American Heart Association Scientific Statement from the Council on Clinical Cardiology, Heart Failure and Transplantation Committee; Quality of Care and Outcomes Research and Functional Genomics and Translational Biology Interdisciplinary Working Groups; and Council on Epidemiology and Prevention. *Circulation*. 2006;113(14):1807-1816.
47. Lewey J, Haythe J. Cardiomyopathy in pregnancy. *Semin Perinatol*. 2014;38(5):309-317.
48. Schinkel AF. Pregnancy in women with hypertrophic cardiomyopathy. *Cardiol Rev*. 2014;22(5):217-222.
49. Goland S, van Hagen IM, Elbaz-Greener G, et al. Pregnancy in women with hypertrophic cardiomyopathy: data from the European Society of Cardiology initiated Registry of Pregnancy and Cardiac disease (ROPAC). *Eur Heart J*. 2017;38(35):2683-2690.
50. Grewal J, Siu SC, Ross HJ, et al. Pregnancy outcomes in women with dilated cardiomyopathy. *J Am Coll Cardiol*. 2009;55(1):45-52.
51. Billebeau G, Etienne M, Cheikh-Khelifa R, et al. Pregnancy in women with a cardiomyopathy: Outcomes and predictors from a retrospective cohort. *Arch Cardiovasc Dis*. 2018;111(3):199-209.
52. Sliwa K, Hilfiker-Kleiner D, Petrie MC, et al.; Heart Failure Association of the European Society of Cardiology Working Group on Peripartum Cardiomyopathy. Current state of knowledge on aetiology, diagnosis, management, and therapy of peripartum cardiomyopathy: a position statement from the Heart Failure Association of the European Society of Cardiology Working Group on peripartum cardiomyopathy. *Eur J Heart Fail*. 2010;12(8):767-778.
53. Pearson GD, Veille JC, Rahimtoola S, et al. Peripartum cardiomyopathy: National Heart, Lung, and Blood Institute and Office of Rare Diseases (National Institutes of Health) workshop recommendations and review. *JAMA*. 2000;283(9):1183-1188.
54. Ware JS, Li J, Mazaika E, et al.; IMAC-2 and IPAC Investigators. Shared genetic predisposition in peripartum and dilated cardiomyopathies. *N Engl J Med*. 2016; 374(3):233-241.
55. McNamara DM, Elkayam U, Alharethi R, et al. IPAC Investigators. Clinical outcomes for peripartum cardiomyopathy in North America: results of the IPAC Study (Investigations of Pregnancy-Associated Cardiomyopathy). *J Am Coll Cardiol*. 2015;66(8):905-914.
56. Goland S, Bitar F, Modi K, et al. Evaluation of the clinical relevance of baseline left ventricular ejection fraction as a predictor of recovery or persistence of severe dysfunction in women in the United States with peripartum cardiomyopathy. *J Card Fail*. 2011;17(5):426-430.

57. Elkayam U. Risk of subsequent pregnancy in women with a history of peripartum cardiomyopathy. *J Am Coll Cardiol*. 2014;64(15):1629-1636.
58. Bello N, Rendon ISH, Arany Z. The relationship between pre-eclampsia and peripartum cardiomyopathy: a systematic review and meta-analysis. *J Am Coll Cardiol*. 2013;62(18):1715-1723.
59. Ntusi NBA, Badri M, Gumedze F, et al. Pregnancy-associated heart failure: a comparison of clinical presentation and outcome between hypertensive heart failure of pregnancy and idiopathic peripartum cardiomyopathy. *PLoS One*. 2015;10(8):e0133466.
60. Goland S, Modi K, Hatamizadeh P, Elkayam U. Differences in clinical profile of African-American women with peripartum cardiomyopathy in the United States. *J Card Fail*. 2013;19(4):214-218.
61. Arany Z, Elkayam U. Peripartum cardiomyopathy. *Circulation*. 2016;133(14):1397-1409.
62. Shotan A, Ostrzega E, Mehra A, et al. Incidence of arrhythmias in normal pregnancy and relation to palpitations, dizziness, and syncope. *Am J Cardiol*. 1997;79(8):1061-1064.
63. Li J-M, Nguyen C, Joglar JA, et al. Frequency and outcome of arrhythmias complicating admission during pregnancy: experience from a high-volume and ethnically-diverse obstetric service. *Clin Cardiol*. 2008;31(11):538-541.
64. Knotts RJ, Garan H. Cardiac arrhythmias in pregnancy. *Semin Perinatol*. 2014;38(5):285-288.
65. Westermann M, Hameed AB. Arrhythmia. In: Plante LA, ed. *Expecting trouble: Early warnings and rapid responses in maternal medical care*. Boca Raton FL: CRC Press; 2018.
66. Barnes EJ, Eben F, Patterson D. Direct current cardioversion during pregnancy should be performed with facilities available for fetal monitoring and emergency caesarean section. *BJOG*. 2002;109(12):1406-1407.
67. Boulé S, Ovart L, Marquié C, et al. Pregnancy in women with an implantable cardioverter-defibrillator: is it safe? Europace. 2014;16(11):1587-1594.
68. Possomato-Vieira JS, Khalil RA. Mechanisms of endothelial dysfunction in hypertensive pregnancy and preeclampsia. *Adv Pharmacol*. 2016;77:361-431.
69. Garovic VD, August P. Preeclampsia and the future risk of hypertension: the pregnant evidence. *Curr Hypertens Rep*. 2013;15(2):114-121.
70. Chambers JC, Fusi L, Malik IS, et al. Association of maternal endothelial dysfunction with preeclampsia. *JAMA*. 2001;285(12):1607-1612.
71. Oakley C, Child A, Jung B, et al.; Task Force on the Management of Cardiovascular Diseases During Pregnancy of the European Society of Cardiology. Expert consensus document on management of cardiovascular diseases during pregnancy. *Eur Heart J*. 2003;24(8):761-781.
72. Levin H, LaSala A. Intrapartum obstetric management. *Semin Perinatol*. 2014;38(5):245-251.
73. Ruys TPE, Roos-Hesselink JW, Pijuan-Domènech A, et al. ROPAC investigators. Is a planned caesarean section in women with cardiac disease beneficial? *Heart*. 2015;101(7):530-536.
74. Kuczkowski KM. Labor analgesia for the parturient with cardiac disease: what does an obstetrician need to know? *Acta Obstet Gynecol Scand*. 2004;83(3):223-233.
75. Baddour LM, Wilson WR, Bayer AS, et al.; American Heart Association Committee on Rheumatic Fever, Endocarditis, and Kawasaki Disease of the Council on Cardiovascular Disease in the Young, Council on Clinical Cardiology, Council on Cardiovascular Surgery and Anesthesia, and Stroke Council. Infective endocarditis in adults: diagnosis, antimicrobial therapy, and management of complications: a scientific statement for healthcare professionals from the American Heart Association. *Circulation*. 2015;132(15):1435-1486.
76. Committee on Practice Bulletins-Obstetrics. ACOG Practice Bulletin no. 199: use of prophylactic antibiotics in labor and delivery. *Obstet Gynecol*. 2018;132(3):e103-e119.
77. Horlocker TT, Wedel DJ, Rowlingson JC, et al. Regional anesthesia in the patient receiving antithrombotic or thrombolytic therapy: American Society of Regional Anesthesia and Pain Medicine Evidence-Based Guidelines (Third Edition). *Reg Anesth Pain Med*. 2010;35(1):64-101.
78. Committee on Practice Bulletins—Obstetrics. Practice Bulletin no. 177: Obstetric analgesia and anesthesia. *Obstet Gynecol*. 2017;129(4):e73-e89.
79. James A; Committee on Practice Bulletins—Obstetrics. Practice bulletin no. 123: thromboembolism in pregnancy. *Obstet Gynecol*. 2011;118(3):718-729.
80. Leffert L, Butwick A, Carvalho B, et al. members of the SOAP VTE Taskforce. The Society for Obstetric Anesthesia and Perinatology Consensus Statement on the Anesthetic Management of Pregnant and Postpartum Women Receiving Thromboprophylaxis or Higher Dose Anticoagulants. *Anesth Analg*. 2018;126(3):928-944.
81. Askie LM, Duley L, Henderson-Smart DJ, Stewart LA; PARIS Collaborative Group. Antiplatelet agents for prevention of pre-eclampsia: a meta-analysis of individual patient data. *Lancet*. 2007;369(9575):1791-1798.
82. Duvekot JJ, Peeters LL. Maternal cardiovascular hemodynamic adaptation to pregnancy. *Obstet Gynecol Surv*. 1994;49(12)(Suppl):S1-S14.
83. Curtis KM, Tepper NK, Jatlaoui TC, et al. U.S. Medical Eligibility Criteria for Contraceptive Use, 2016. *MMWR Recomm Rep*. 2016;65(3):1-103.

Appendix A.
Congenital Heart Disease - Primer

Shunts

The World Health Organization (WHO) classifies most left-to-right shunts as pregnancy category risk I (eg, repaired atrial septal defect [ASD], ventricular septal defect [VSD], patent ductus arteriosus [PDA]) or II (unoperated ASD or VSD; repaired tetralogy of Fallot).

Left-to-right shunts may occur at the level of the ventricles, atria, or great arteries. A small VSD, PDA, or ASD, typically are well tolerated even when unrepaired.[1,2] However, the risks of low birthweight and preeclampsia are increased in these cases. Over time, a large VSD or PDA will result in pulmonary hypertension (PH), which increases the risk of mortality during pregnancy. As PH worsens, the shunt may reverse to a bidirectional or right-to-left flow, a combination known as Eisenmenger syndrome, which is associated with the highest risks of maternal morbidity and mortality (30% to 50% risk of maternal death). Pregnancy is considered to be contraindicated in women with Eisenmenger syndrome.[3]

In addition to the maternal risk, the rate of newborns with low birthweight and fetal mortality is markedly increased. Women with cyanosis from simple shunt or complex congenital heart disease (CHD) are expected to have a high rate of maternal and fetal adverse outcomes: heart failure, preterm delivery, growth restriction, and mortality. These women are best advised to avoid pregnancy altogether. In these cases, ongoing pregnancy cannot be safely managed at a location without complete cardiac, anesthetic, obstetric, and neonatal support, and are best served at a facility with expertise in the multidisciplinary management of adult CHD.

Tetralogy of Fallot

Tetralogy of Fallot (TOF) is marked by four abnormalities: a large VSD, an aorta that overrides the ventricular septum rather than the left ventricle (LV), obstruction of the right ventricular (RV) outflow tract, and RV hypertrophy. Additional anomalies may be present. Patients may have been treated in earlier years with a palliative procedure such as the Blalock shunt followed by a later repair, or in more recent years, with a complete one-stage repair in infancy, in which the VSD is repaired and the RV outflow tract expanded. Delayed complications such as right heart failure or ventricular arrhythmia may develop. In pregnancy, 6% to 7% of women with repaired TOF will have arrhythmia and 2% to 3% will develop heart failure.[4] However, women with repaired TOF tend to tolerate pregnancy well in the absence of ventricular dysfunction, heart failure, or arrhythmia. Repaired TOF is classified as WHO pregnancy category risk II: small increase in maternal mortality, moderate increase in morbidity.[4]

Aortopathies: Coarctation, Dilation of the Aortic Root, Dissection

Pregnancy increases aortic dilation in normal pregnancy by increasing circulating volume, heart rate, and cardiac output.[5] Histologic changes in connective tissue also occur. This combination of factors may account for the greatly increased risk of aortic dissection during pregnancy. Women with existing connective-tissue disease (eg, Marfan syndrome, Ehlers-Danlos syndrome) are at particularly high risk.

To minimize the risk of aortic dissection, women with an aortopathy or existing aortic dilation need serial aortic-diameter assessment and excellent blood pressure control. Depending on the underlying condition, aortic diameters greater than 4 or 5 cm should be managed prior to pregnancy if at all possible.[5,6] Depending on gestational age and maternal condition, prelabor cesarean delivery may be performed immediately prior to aortic repair. Aortic dissection more commonly manifests in the third trimester (50%) and puerperium (20%),[6] and most are type A, which constitutes a surgical emergency. Maternal mortality due to aortic dissection was reported to be as high as 30% in an older series, and 20% to 28%

more recently, whereas fetal mortality rates have decreased from 50% in the older series to 35% to 48%.[7,8,9] Women with aortic dilation who are pregnant are inherently at risk of aortic dissection and rupture and must be cared for in a facility where cardiothoracic surgery and obstetric and neonatal services are available.

Women with coarctation of the aorta will typically have been diagnosed and undergone repair prior to pregnancy.[10] Pregnancy after repair of coarctation typically is well tolerated, though the risk of hypertensive complications is increased to 20% to 30%.[10] Repaired coarctation is classified as WHO category II-III: moderate increase in risk of maternal mortality and morbidity.

Ebstein Anomaly

In Ebstein anomaly, the leaflets of the tricuspid valve are set low, with the result that the right atrium is enlarged and the RV is small and its function somewhat impaired. Tricuspid regurgitation is present. There may be obstruction to RV inflow or outflow tracts or compromised LV function. Abnormal cardiac conduction is present in 25% of cases and an intra-atrial shunt in 50%. Mild cases may be asymptomatic, but symptoms more commonly appear in adolescence or early adulthood, manifesting as exercise intolerance and/or arrhythmia. Cyanosis or right heart failure may develop. Sudden cardiac death is a reported manifestation.[11] Medical, percutaneous, or surgical treatment can be offered. In one study, 13% of women with Ebstein anomaly who were pregnant developed a cardiac complication, most of which were arrhythmias.[12]

References

1. Bhatt AB, De Faria Yeh D. Pregnancy and adult congenital heart disease. *Cardiol Clin*. 2015; 33:611-623.
2. Deen JF, Jones TK. Shunt lesions. *Cardiol Clin*. 2015;33: 513-520.
3. Canobbio MM, Warnes CA, Aboulhosn J, et al; American Heart Association Council on Cardiovascular and Stroke Nursing. Council on Clinical Cardiology; Council on Cardiovascular Disease in the Young; Council on Functional Genomics and Translational Biology; and Council on Quality of Care and Outcomes Research. Management of pregnancy in patients ith complex congenital heart disease: a scientific statement for healthcare professionals from the American Heart Association. *Circulation*. 2017;135(8):e50-e87.
4. Downing TE, Kim YY. Tetralogy of Fallot: general principles of management. *Cardiol Clin*. 2015;33:531-541.
5. Van Hagen IM, Roos-Hesselink JW. Aorta pathology and pregnancy. *Best Pract Res Clin Obstet Gynecol*. 2014; 28: 537-50.
6. Wanga S, Silversides C, Dore A, de Waard V, Mulder B. Pregnancy and thoracic aortic disease: managing the risks. *Can J Cardiol*. 2016;32:78-85.
7. Immer FF, Bansi AG, Immer-Bansi AS, et al. Aortic dissection in pregnancy: analysis of risk factors and outcome. *Ann Thorac Surg*. 2003;76(1):309-314.
8. Zhu JM, Ma WG, Peters S, et al. Aortic dissection in pregnancy: management strategy and outcomes. *Ann Thorac Surg*. 2017;103(4):1199-1206.
9. Rajagopalan S, Nwazota N, Chandrasekhar S. Outcomes in pregnant women with acute aortic dissections: a review of the literature from 2003 to 2013. *Int J Obstet Anesth*. 2014;23(4):348-356.
10. Vriend JWJ, Drenthen W, Pieper PG, et al, on behalf of the ZAHARA investigators. Outcome of pregnancy in patients after repair of aortic coarctation. *Eur Heart J*. 2005;26:2173-2138.
11. Silversides CK, Kiess M, Beauchesne L, et al. Canadian Cardiovascular Society 2009 Consensus Conference on the management of adults with congenital heart disease: outflow tract obstruction, coarctation of the aorta, tetralogy of Fallot, Ebstein anomaly and Marfan's syndrome. *Can J Cardiol*. 2010;26:e80-e97.
12. Drenthen W, Boersma E, Balci A, et al. Predictors of pregnancy complications in women with congenital heart disease. *Eur Heart J*. 2010;31(17):2124-2132.

Appendix B. Complex Congenital Heart Disease

Transposition of the Great Arteries

The two forms of transposition of the great arteries (TGA) are D-transposition (D-TGA) and L-transposition (L-TGA), also referred to as congenitally corrected TGA (CC-TGA).[1]

In D-TGA, the aorta emerges from the right ventricle and the pulmonary artery from the left, so oxygenated blood is not pumped to the systemic circulation. This condition is incompatible with extrauterine life unless a shunt (atrial septal defect [ASD], ventricular septal defect [VSD], or patent ductus arteriosus) is present; one may be created in the newborn as a first step in palliation if it is not already present.

In L-TGA, the great arteries and the ventricles are transposed: the right ventricle (RV) receives oxygenated blood from the left atrium and conveys it to the systemic circulation via the aorta, whereas the left ventricular inflow is received from the right atrium and sent to the pulmonary artery. The earlier atrial switch operation for D-TGA (the Mustard/Senning procedure) has mostly been supplanted by the arterial switch, which realigns the arteries to the correct ventricles, effectively restoring normal cardiovascular anatomy.

Over time, the RV tends to fail in patients with uncorrected L-TGA and those with D-TGA who have undergone an atrial switch procedure, because the RV is not well suited to serve as the systemic ventricle. They also are at significant risk of arrhythmia. Pregnancy is poorly tolerated in women with a systemic RV, and pregnancy may accelerate the expected progressive dysfunction of these condition.[2] As with other complex congenital heart disease, multidisciplinary management is required in the event of pregnancy. Frequent cardiac assessments are needed, with particular attention given to arrhythmia or heart failure. Labor may be poorly tolerated, depending on functional status. Cardiac rhythm monitoring is indicated in labor, along with regional analgesia. Shortening the second stage of labor with the use of vacuum extraction or forceps typically is recommended.

In the Zwangerschap bij Aangeboren Hartafwijkingen (ZAHARA) study, pregnancy outcomes among 52 women with surgically corrected TGA, most of whom had undergone a Mustard procedure, were as follows: 33% developed cardiac complications during pregnancy, 31% had a preterm delivery, and 19% delivered a newborn who was small for gestational age (SGA). Perinatal mortality was approximately 10%.[3] The 19 women with CC-TGA had significantly better outcomes: 16% developed cardiac complications, 5% had a preterm delivery, 16% delivered an SGA newborn, and there were no perinatal deaths.

At this time, there is little published data on the outcome of pregnancy in women who have undergone the arterial switch procedure,[2] but long-term cardiac outcomes outside of pregnancy seem to be better, with less heart failure, less arrhythmia, and longer survival.[1]

Single-Ventricle Disorders

Single-ventricle physiology does not solely imply a true single ventricle, though that can occur, but includes conditions involving a dominant ventricle supplying the systemic circulation, a second hypoplastic ventricle or remnant, and a shunt (ASD or VSD). These conditions are palliated in childhood, but a second functional ventricle is not created. Instead, the pulmonary artery is connected to the right atrium, or the superior vena cava is connected to the pulmonary artery, directly or via an extracardiac conduit, in a Fontan repair.[4] The single ventricle continues to pump to the systemic and pulmonary circulation systems, but after the shunt is closed, systemic and pulmonary circulations are separated and cyanosis is corrected. In the longer term, cardiac and noncardiac complications develop (eg, venous congestion, ventricular failure, arrhythmias, protein-losing enteropathy, thromboembolism).[2,5] There is little or no capacity to increase cardiac output. Women most likely to tolerate pregnancy with single-ventricle physiology are those with good ventricular function and

who have not had arrhythmias or thromboembolic events. The rate of preterm delivery is nonetheless increased in these women, though it is not feasible to distinguish spontaneous preterm delivery from medically indicated preterm deliveries.[6] Vaginal delivery is a reasonable option, depending on maternal condition and obstetric indications; in the largest series, 75% of women with single-ventricle physiology delivered vaginally.[6]

Experts recommend that women with single-ventricle physiology who are pregnant be treated directly by an adult congenital heart disease (CHD) multidisciplinary team.[2] The potential for decompensation must be considered. Ventricular function should be assessed via echocardiography each trimester and when symptoms develop. Oxygen saturation should be monitored. Worsening oxygen saturation will not correct with supplemental oxygen, because it is an indication of intracardiac venous admixture rather than arterial hypoxemia.

Thromboprophylaxis is appropriate in many cases. Labor and delivery should be managed at an institution with adult CHD and high-level obstetric services. Cardiac rhythm must be monitored. Fluid status can be tenuous, because fluid loading will precipitate heart failure and hypovolemia limits blood flow to the lungs, and the physiologic changes of labor and the immediate postpartum may not be tolerated. As in most cases of complex CHD, labor is best managed under regional analgesia, and the second stage of labor may be shortened electively via vacuum or forceps assisted delivery.

Truncus Arteriosus

Truncus arteriosus is a rare condition in which a single great artery supplies blood to the systemic and pulmonary circulations, and which is accompanied by a VSD. This anomaly typically is corrected in early childhood, in a procedure which separates the pulmonary arteries from the trunk, connects them to a surgically constructed conduit and then to the right ventricle, and closes the VSD. Only a few cases of a woman with a repaired truncus arteriosus undertaking pregnancy have been reported.[7] Delayed complications (not specific to pregnancy) have included truncal valve or conduit failure, ventricular dysfunction, arrhythmia, ischemia, and pulmonary hypertension. Spontaneous vaginal delivery, assisted vaginal delivery, and cesarean delivery have all been used in these women. Truncus arteriosus, like other conotruncal anomalies, is known to have an association with 22q11.2 deletion syndrome, so genetic counseling is indicated.[2]

Women with cyanosis from simple shunt or complex CHD are expected to have a high rate of maternal and fetal adverse outcomes including heart failure, preterm delivery, growth restriction, and mortality. These women are best advised to avoid pregnancy altogether. In the event of a pregnancy, they cannot be safely managed at a location without complete cardiac, anesthetic, obstetric, and neonatal support, and are best served at a facility with expertise in the multidisciplinary management of adult CHD.

References

1. Haeffele C, Lui K. Dextro-transposition of the great arteries: long-term sequelae of atrial and arterial switch. *Cardiol Clin*. 2015; 543-558.
2. Canobbio MM, Warnes CA, Aboulhosn J, et al. Management of pregnancy in patients with complex congenital heart disease: a scientific statement for healthcare professionals from the American Heart Association. *Circulation*. 2017;135(8):e50-e87.
3. Drenthen W, Boersma E, Balci A, et al. Predictors of pregnancy complications in women with congenital heart disease. *Eur Heart J*. 2010;31(17):2124-2132.
4. d'Udekem Y, Iyengar A, Cochrane AD, et al. The Fontan procedure: contemporary techniques have improved long-term outcomes. *Circulation*. 2017; 116:I-157-I-164.
5. Naguib MA, Dob DP, Gatzoulis MA. A functional understanding of moderate to complex congenital heart disease and the impact of pregnancy. Part II: Tetralogy of Fallot, Eisenmenger's syndrome and the Fontan operation. *Int J Obstet Anesthesia*. 2010;19:306-312.
6. Collins RT, Chang D, Sandlin A. National in-hospital outcomes of pregnancy in women with single ventricle congenital heart disease. *Am J Cardiol*. 2017;119:1106-1110.
7. Steckham KE, Bhagra CJ, Siu SC, Silversides CK. Pregnancy in women with repaired truncus arteriosus: a case series. *Can J Cardiol*. 2017;33:1737.e1-1737.e3.

Venous Thromboembolism in Pregnancy

Learning Objectives

1. Apply a standardized risk assessment tool to identify appropriate patients for thromboprophylaxis.
2. Diagnose and treat pregnancy-related venous thromboembolism.
3. Assess for complications of pharmacologic thromboprophylaxis.

Venous Thromboembolism During Pregnancy

Venous thromboembolism (VTE) during pregnancy refers to deep venous thrombosis (DVT) and pulmonary embolism (PE). DVT is diagnosed when a blood clot forms in the deep venous system of the lower extremities. PE is diagnosed when a portion of the blood clot breaks loose and becomes lodged in the pulmonary arteries.[1]

Incidence and Clinical Significance

Venous thromboembolism complicates 0.5 to 2 per 1,000 pregnancies and is a leading cause of maternal mortality in developed countries; VTE accounts for 9.3% of maternal deaths in the United States.[1] The incidence of VTE is higher postpartum than antenatally with the peak incidence in the first week after delivery.[2] DVT is three times more common in pregnancy than PE.[3] The importance of timely diagnosis is underscored by the fact that up to 25% of women with untreated DVT who are pregnant develop PE, and undiagnosed PE has a mortality rate of 30%.[4] Morbidity also is common; After DVT, 29% to 79% of women experience postthrombotic syndrome, characterized by chronic leg pain and swelling, varicose veins, skin discoloration, and ulceration.[5]

Pathophysiology and Risk Factors

Venous thromboembolism develops as a result of multiple interacting risk factors.[6] Classic predisposing factors of hypercoagulation and venous stasis are present in every pregnancy and postpartum period.[7] Hypercoagulability of pregnancy results from an increased concentration of fibrinogen, factor VII, factor VIII, factor X, von Willebrand factor, plasminogen activator inhibitor-1 and plasminogen activator inhibitor-2, combined with decreased anticoagulant free protein S.[1] There is no change in procoagulants factor II, factor V, and factor IX nor in anticoagulants protein C and antithrombin.[1]

Stasis results from increased venous distension and obstruction of the inferior vena cava by the gravid uterus. Reduction in venous flow is evident by 13 weeks' gestation, reaches a nadir at 36 weeks' gestation, and returns to nonpregnant levels approximately 6 weeks postpartum.[8] Vascular damage may occur during cesarean or vaginal delivery, but VTE risk is higher after cesarean delivery (OR 3.7; 95% CI = 3.0-4.6).[9] Additional risk factors for VTE in pregnancy are listed in *Table 1*. The most important risk factor for VTE is a history of VTE; 15% to 25% of VTEs in pregnancy are recurrent events.[1,10] Overall, the risk of VTE is 3 to 4 times higher for a woman who is pregnant than a woman of the same age who is not pregnant.[1]

Thrombophilic Disorders

Inherited or acquired thrombophilic disorders are among the important risk factors for VTE. Approximately 50% of women with VTE in pregnancy have underlying thrombophilic disorders compared with only 10% of the general Western population.[8]

The inherited thrombophilias include: factor V Leiden mutation, prothrombin G20210A mutation, methylenetetrahydrofolate reductase mutation, antithrombin deficiency, and protein C and protein S deficiency.[7] Factor V Leiden and prothrombin G20210A mutations are the most common.[7] Women with protein C and protein S deficiencies have an eightfold increased risk of pregnancy-related VTE compared with women without such deficiencies.[11]

Universal screening for thrombophilia is not recommended; however, testing is recommended in women with a history of VTE that is not attributable to a nonrecurrent risk factor (eg, trauma, surgery, travel, immobilization) and in women who have a first-degree family member with a high-risk thrombophilia (eg, antithrombin deficiency, double heterozygous for prothrombin G20210A mutation and factor V Leiden, fac-

Table 1. Venous Thromboembolism Risk Factors

Age >35 years
Personal or family history of VTE
Cesarean delivery, especially if emergent
Dehydration
Hyperemesis
Hypertensive disorders of pregnancy
Infection/sepsis
Major medical conditions (inflammatory bowel disease, nephrotic syndrome, sickle cell disease, or myeloproliferative disorders)
Mechanical heart valve
Multiparity (>4 deliveries)
Multiple gestation
Obesity
PPH
Prolonged bed rest or immobility
Severe varicose veins
Smoking
Thrombophilic disorders

PPH = postpartum hemorrhage; VTE = venous thromboembolism.

Information from Chan WS, Rey E, Kent NE, et al; VTE in Pregnancy Guideline Working Group; Society of Obstetricians and Gynecologists of Canada. Venous thromboembolism and antithrombotic therapy in pregnancy. J Obstet Gynaecol Can. *2014;36(6):527-553; James A; Committee on Practice Bulletins—Obstetrics. Practice bulletin no. 123: thromboembolism in pregnancy.* Obstet Gynecol. *2011;118(3):718-729; Royal College of Obstetricians and Gynaecologists. Greentop Guideline 37a. Reducing the risk of thrombosis and embolism during pregnancy and the puerperium. 2015.*

tor V Leiden homozygous, prothrombin G20210A mutation homozygous) or VTE before age 50 years in the absence of other risk factors.[7] Thrombophilia screening is no longer recommended for women with pregnancy complications such as IUGR and preeclampsia.[12] American College of Obstetricians and Gynecologists (ACOG) guidelines recommend against routinely screening all women for the MTHFR mutation or homocysteine levels because of a lack of evidence that these factors affect maternal or fetal outcomes.[7] The Royal College of Obstetricians and Gynecologists (RCOG) guidelines recommend thrombophilia testing in women younger than 50 years who have a history of an unprovoked or estrogen-related (pregnancy or contraception) event in a first-degree relative.[13] RCOG also recommends testing for antithrombin deficiency in women with family history of antithrombin deficiency or VTE with no previously identified thrombophilia.[13] RCOG guidelines recommend that women with unprovoked VTE be tested for antiphospholipid antibodies.[13]

Accurate interpretation of screening tests requires knowledge of the effects of pregnancy and other disorders. Normal pregnancy decreases protein S levels.[14] Antithrombin and protein C levels remain normal throughout pregnancy, but protein C resistance increases during the second and third trimesters.[14] Massive thrombus decreases antithrombin levels. Nephrotic syndrome and preeclampsia are associated with decreased antithrombin levels, and liver disease is associated with decreased protein C and S levels.[15]

Antiphospholipid antibodies are the most common and clinically important acquired thrombophilic defects. Antiphospholipid syndrome in pregnancy is diagnosed when at least one clinical and one laboratory criteria are present.[16]

Clinical criteria include:

- Arterial, venous, or small vessel thrombosis of any tissue or organ
- Unexplained fetal death beyond 10 weeks' gestation
- Birth before 34 weeks' gestation of a seemingly healthy fetus due to preeclampsia/eclampsia or placental insufficiency
- Three or more unexplained, consecutive spontaneous miscarriages before 10 weeks' gestation.

Laboratory criteria include:

- Lupus anticoagulant
- Anticardiolipin antibody
- Anti-B2-glycoprotein I on at least two occasions 12 or more weeks apart.[16]

Lupus anticoagulant is more specific but less sensitive than the other two laboratory criteria.[16]

Deep Venous Thrombosis

Clinical Signs and Symptoms

Unlike DVT in women who are not pregnant, more than 80% of DVT in women who are pregnant occur in the left leg,[17] perhaps because of the gravid uterus compressing the left iliac vein. A 2010 systematic review of six studies (not all randomized controlled trials [RCTs]) involving 124 women who were pregnant showed that 88% of DVTs occurred on the left side and 71% were restricted to proximal veins without involving calf veins.[18]

Deep venous thrombosis may have a subtle clinical presentation and may be difficult to distinguish from gestational edema. Typical symptoms are unilateral leg pain and swelling. A difference in lower leg circumference of 2 centimeters or more is associated with a higher risk of DVT (OR 13.62; 95% CI = 4.56-40.67).[19]

The LEFt mnemonic can help identify women who are pregnant who are at higher risk of DVT. *L* stands for Left leg symptoms; *E* stands for Edema (2 cm or more leg circumference discrepancy), and *Ft* stands for First trimester symptoms. The LEFt mnemonic has a better negative than positive predictive value. A study of the mnemonic's effectiveness showed that DVT in pregnancy was diagnosed in 13 of 111 women with at least one LEFt criterion (11.7%; 95% CI = 8.3-20.9) compared with 0 of 46 women with no LEFt criteria (0%; 95% CI = 0.0-7.9).[20] Although only 29.4% of DVTs in another study occurred in the first trimester, a multivariate analysis showed that women presenting with suspected DVT in the first trimester was a significant predictor of DVT.[19] Hyperemesis gravidarum with accompanying dehydration and immobility, ovarian hyperstimulation syndrome, and in vitro fertilization are additional VTE risk factors specific to the first trimester.[13]

Less than 10% of women with signs and symptoms of DVT have the diagnosis confirmed by objective testing.[19] Definitive diagnosis is essential because of the need for acute treatment, evaluation for underlying thrombophilia, and prophylaxis in future pregnancies.

Diagnostic Testing

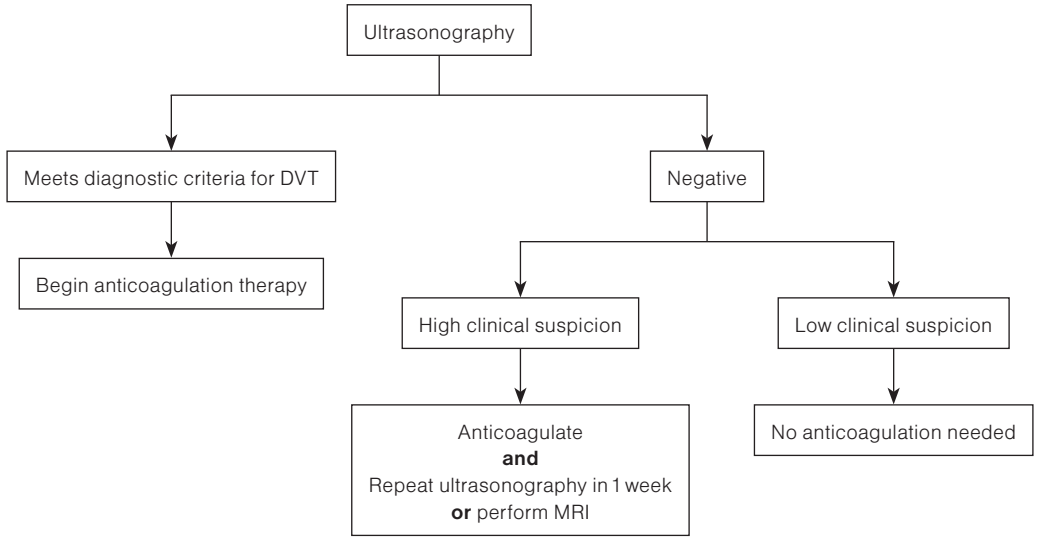
When DVT is strongly clinically suspected, anticoagulation should be administered as soon as possible until results of confirmatory tests are available.[1,12,15] The first-line diagnostic test for DVT is Doppler ultrasonography (*Figure 1*).[1,12,15] A Doppler study indicating DVT in the affected leg is sufficient to recommend a full course of therapeutic anticoagulation.[1,12,15] Negative Doppler results with low clinical suspicion do not require anticoagulation. If iliac vein thrombosis is suspected and Doppler results are negative, magnetic resonance imaging (MRI) or empiric anticoagulation is indicated.[1,21] If empiric anticoagulation is chosen despite negative initial Doppler results, venous Doppler should be repeated at 3 and 7 days.[1]

Because of its high false positive rate in pregnancy, D-dimer is not recommended in the evaluation for acute VTE in pregnancy.[1,15] However, a low D-dimer level does make VTE unlikely.[1,15,22]

Treatment

Low-molecular-weight heparin (LMWH) is the treatment of choice for VTE in pregnancy.[1,12]

Figure 1. Deep Venous Thrombosis Diagnosis With Ultrasonography



DVT = deep venous thrombosis; MRI = magnetic resonance imaging.

Information from various sources.

LMWH is discussed in more detail in the Anticoagulation in Pregnancy section.

Pulmonary Embolism

Clinical Signs and Symptoms

In contrast with DVT, which is equally common during pregnancy and postpartum, at least two-thirds of pregnancy-related PEs occur postpartum.[2] Dyspnea and tachypnea are the most common presenting symptoms of PE. The clinical picture can vary from mild dyspnea and tachypnea accompanied by chest pain to dramatic cardiopulmonary collapse.

Clinical pre-test probability assessments, such as the Wells score, have not been validated for use during pregnancy.[23]

Diagnostic Testing

An approach to the diagnosis of suspected PE using noninvasive testing is outlined in *Figure 2*.[1,15]

When a woman presents with possible PE, stabilization should be the priority. See the *Maternal Resuscitation and Trauma* chapter for more details on stabilization. Consideration should be given to anticoagulation until a more definitive diagnosis is made.[15]

Some experts recommend obtaining a venous Doppler ultrasound before considering a ventilation/perfusion (V/Q) scan or computed tomography pulmonary angiogram (CTPA) to avoid the radiation of these tests.[15] If DVT is diagnosed, anticoagulation is recommended regardless of whether PE is present.[15]

A chest x-ray may help in deciding whether to obtain a V/Q scan or CTPA. A cohort study showed V/Q scan to be preferable to CTPA for diagnosis of PE in women who have negative chest x-ray results; CTPA was more likely to be diagnostic in women with abnormal chest x-ray results.[24] V/Q scans are less likely to be nondiagnostic in women who are pregnant because they are typically young with fewer comorbidities.[25] The choice between V/Q scan and CTPA may be affected by availability.[1] A 2017 Cochrane review found that low-level evidence supports a V/Q scan or a CTPA for diagnosing PE in pregnancy.[26]

Fetal radiation exposure with CTPA is less than 10% of that with a V/Q scan, but the absolute fetal risk of both is low. The fetal radiation dose for CTPA is equivalent to a less than 1 in 1 million risk of cancer at age 15 years compared with a 1 in 280,000 risk with V/Q scan.[27]

Maternal breast radiation exposure is a concern with CTPA.[1] CTPA and V/Q scans involve 20 to 44 and 0.20 to 0.28 mGy of maternal breast absorbed radiation, respectively.[28] The effect of this radiation on future breast cancer risk is controversial.[29,30] For comparison, the American College of Radiology recommends not exceeding 3 mGy for a screening mammogram.[31] When possible, the woman should be involved in choosing between a CTPA and V/Q scan if both tests are available.[15]

The sensitivity of CTPA has increased with technological advances. First-generation single-detector row computed tomography (CT) scanners have had a positive predictive value of only 85%[32] and are only 30% sensitive for subsegmental defects, which account for 20% of symptomatic PE.[32] Newer multidetector row CT scanners allow improved visualization of the segmental and subsegmental pulmonary arteries; they have a positive predictive value of 96% when clinical suspicion is high and a negative predictive value of 99%, comparable with pulmonary angiography, which is now rarely used.[33,34] Multidetector row CT scanners allow quicker scanning of the lung, avoiding respiratory movement and artifacts. The 16-slice CT scanner can image the entire chest with submillimeter resolution in less than 10 seconds.[35] CTPA can identify an alternative diagnosis in approximately two-thirds of cases in which PE is not present; however, it may detect suspicious-appearing, yet benign abnormalities that prompt further evaluation, including biopsy.[36]

Magnetic resonance imaging for diagnosis of PE is an attractive option because it does not expose the fetus to ionizing radiation, and it is as sensitive and specific as CTPA in diagnosing PE.[4,37] Disadvantages of MRI include expense, accessibility, and the fact that it is relatively unstudied in pregnancy.[4,36]

Arterial blood gas determination and electrocardiogram may help determine the clinical likelihood of PE or may suggest other conditions.[15]

When there is high suspicion of PE and V/Q scan or CTPA results are negative or low-probability/equivocal, consideration should be given to continuing anticoagulation and repeating imaging.[15]

Treatment

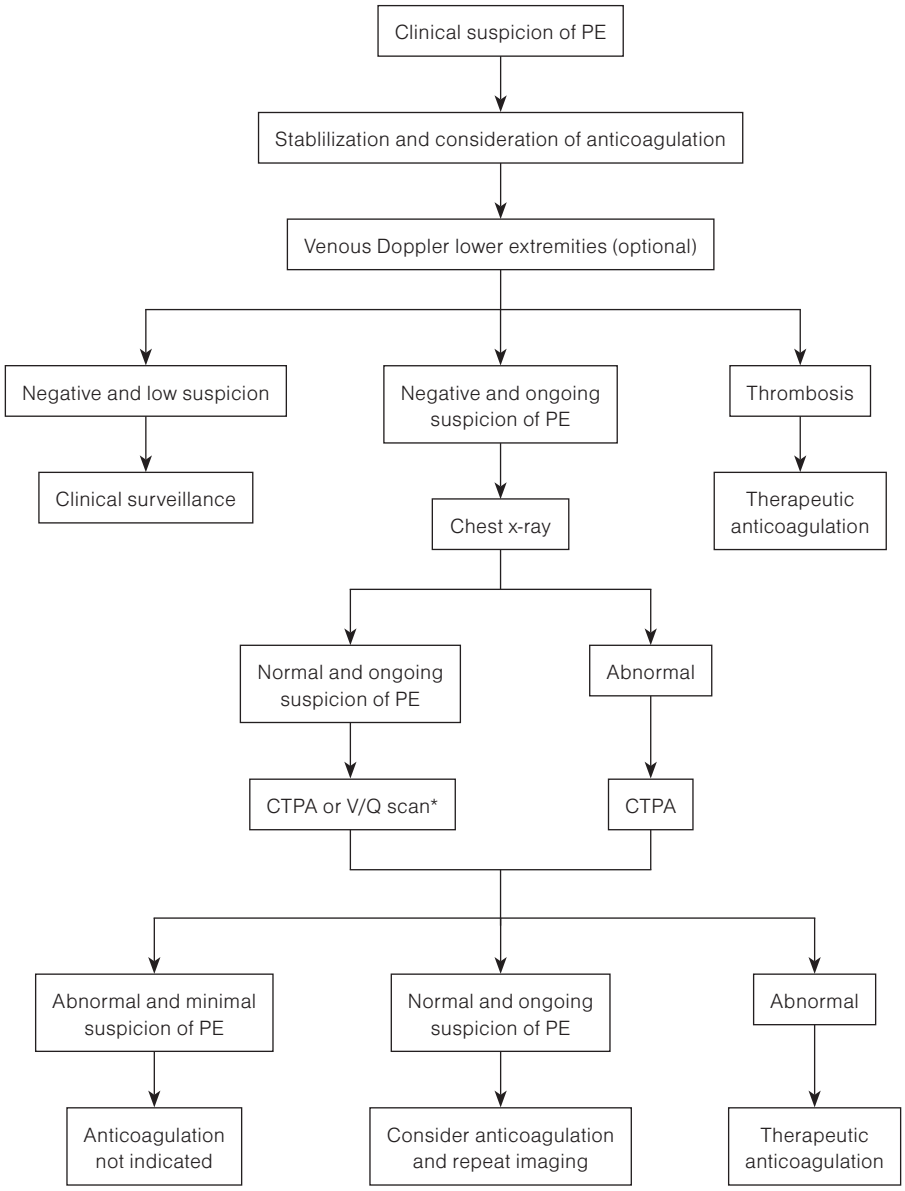
As discussed previously, treatment should begin with stabilization. Anticoagulation may be started empirically while awaiting diagnostic tests. Most

maternal mortality occurs during the first 30 minutes after the event, so prompt action is essential.[38]

Low-molecular-weight heparin is the treatment of choice for PE and DVT.[1,12] If anticoagulation is contraindicated or repeat PE occurs despite adequate anticoagulation, it may be necessary to insert a filter in the inferior vena cava.[15] Anticoagulation is continued after the filter is placed unless contraindicated. More details are discussed in the Anticoagulation in Pregnancy section.

In the case of life-threatening massive PE, intervention with thrombolytic therapy, percuta-

Figure 2. Deep Venous Thrombosis Diagnosis With Ultrasonography



**Consideration of factors including availability of CT versus V/Q, higher radiation to breast tissue with spiral CT, higher sensitivity and specificity with spiral CT, and low but higher fetal radiation with V/Q scan*

CT = computed tomography; CTPA = computed tomography pulmonary angiogram; PE = pulmonary embolism; V/Q = ventilation-perfusion.

Information from James A; Committee on Practice Bulletins—Obstetrics. Practice bulletin no. 123: thromboembolism in pregnancy. Obstet Gynecol. *2011;118(3):718-729; Walker MC, Garner PR, Keely EJ, et al. Changes in activated protein C resistance during normal pregnancy.* Am J Obstet Gynecol. *1997;177(1):162-169.*

neous catheter thrombus fragmentation or surgical embolectomy may be indicated, often depending on local expertise.[15,39] A 2015 Cochrane review of 17 RCTs involving 2,167 participants who were not pregnant showed that thrombolytic therapy compared with heparin for treatment of PE resulted in a lower risk of death (OR 0.57; 95% CI = 0.37-0.87) and recurrence of PE (OR 0.51; 95% CI = 0.29-0.89) but a higher risk of major and minor hemorrhagic events (OR 2.90; 95% CI = 1.95-4.31).[40] Well-designed studies involving women who are pregnant are needed.

Anticoagulation in Pregnancy

When clinical findings and diagnostic testing results indicate DVT or PE, or when the clinical suspicion remains high despite initial negative testing, therapeutic anticoagulation is indicated. Anticoagulation options include LMWHs (eg, dalteparin, enoxaparin, tinzaparin), unfractionated heparin (UFH), and, in the postpartum period, warfarin.

The 2012 American College of Chest Physicians (ACCP) guidelines state, "For pregnant patients, we recommend LMWH for the prevention and treatment of VTE, instead of UFH."[12] The strongest evidence for LMWH comes from studies in patients who are not pregnant. A 2017 Cochrane review showed that, in patients who were not pregnant, the incidence of recurrent VTE events was lower in patients treated with LMWH than patients treated with UFH (OR 0.69; 95% CI = 0.49-0.98; 18 RCTs with 6,238 participants).[41] This persisted to 3 months and the end of follow-up. Major hemorrhage was less common in the LMWH group (OR 0.69; 95% CI = 0.50-0.95; 25 studies with 8,780 participants). There was no difference in mortality rates.[41] Cochrane reviews comparing LMWH with UFH in pregnancy and the early postpartum period yielded poor quality studies and insufficient evidence to recommend one form of heparin over the other.[42,43]

Heparin is considered safe for use during pregnancy and breastfeeding because it does not cross the placenta and is not secreted in breast milk.[44] Although bone loss is possible with UFH and LMWH in pregnancy, the risk is lower with LMWH.[12,45] LMWHs are at least as effective as UFH and are less likely to cause allergy or result in infection from contaminated multidose vials.[1] There is no evidence favoring one LMWH over another.[46,47] LMWH is compatible with breastfeeding.[48-50]

Warfarin is contraindicated during pregnancy, though there are exceptionally rare considerations for its use in a small specific subset of women who are pregnant who require anticoagulation due to the presence of mechanical heart valves. These women should be fully and carefully counselled regarding this unique use of warfarin and its risks.[12] Warfarin crosses the placenta and increases the risk of miscarriage and stillbirth, embryopathy (nasal hypoplasia and/or stippled epiphyses) in the first trimester, central nervous system abnormalities when used in any trimester, and maternal and fetal hemorrhage when used near time of delivery.[12] Despite these risks, RCOG and the American College of Cardiology recommend low-dose warfarin therapy (5 mg/day or less) throughout pregnancy or UFH in the first trimester followed by warfarin therapy in the second and third trimesters to treat women at high-risk who have mechanical heart valves.[13,51] Warfarin is safe for breastfeeding.[15,52,53]

There is limited evidence on the effectiveness of direct thrombin inhibitor (eg, argatroban, bivalirudin, dabigatran, lepirudin) and factor Xa inhibitor (eg, apixaban, rivaroxaban, fondaparinux) use during pregnancy and breastfeeding. A 2015 Cochrane review of 11 RCTs involving 27,945 patients who were not pregnant showed that direct thrombin and factor Xa inhibitors were as effective as LMWH in preventing DVT and PE and had fewer bleeding complications.[54] A 2016 Cochrane review of five RCTs involving 7,897 patients who were not pregnant showed that direct thrombin and factor Xa inhibitors were as equally effective as LMWH in preventing recurrent PE and equivalent in major bleeding complications.[55]

The 2012 ACCP guidelines recommend against using direct thrombin and factor Xa inhibitors in pregnancy.[12] Society of Obstetricians and Gynaecologists of Canada (SOGC) guidelines also recommend against direct thrombin and factor Xa inhibitors in pregnancy.[56] Exceptions include women with a severe heparin allergy or heparin-induced thrombocytopenia.[1,12] A prospective cohort study of 12 pregnancies in 10 women who received fondaparinux thromboprophylaxis because of allergy to LMWH showed no maternal or fetal complications.[57] The baseline laboratory evaluation that should be considered before initiating anticoagulation are listed in *Table 2*.

Therapeutic anticoagulation is recommended for VTE in pregnancy; it should continue for at least 3 months from diagnosis.[12,15,56] After 3 months of therapeutic dosing, anticoagulation can be reduced to intermediate or prophylactic dosing through at least 6 weeks postpartum.[15] Acceptable therapeutic doses for LMWH are listed in *Table 3*. A 2013 Cochrane review of five trials involving 1,508 participants showed that once-daily LMWH was as effective as twice-daily LMWH in patients who are not pregnant.[58] No RCTs have evaluated once- versus twice-daily dosing in pregnancy. Some lower quality studies support once-daily dosing in pregnancy, whereas others do not.[1] In a prospective observational study of 126 women with antenatal VTE who were pregnant, 66% of those who received once-daily LMWH showed no recurrent VTE in either group.[59] A retrospective study of once-daily tinzaparin in 37 women who were pregnant showed two thrombotic events.[60] Dosages of LMWH should be adjusted in the setting of renal insufficiency, particularly in women who have preeclampsia with severe features.[47]

Hospitalization may be indicated for initial anticoagulation for VTE in pregnancy, especially if a woman is unstable, has a large thrombus, or has comorbidities.[1] Initial intravenous (IV) UFH may be preferred when imminent delivery, surgery, or thrombolysis are anticipated.[1] Although pregnancy is a relative contraindication for thrombolysis, there are clinical scenarios when thrombolysis in pregnancy is warranted.[22]

The optimal protocol for monitoring treatment with LMWHs has not been established. It is not necessary to monitor the aPTT as with UFH. Whether to monitor anti-Xa levels is controversial, and the target range is not well established.[61] Anti-Xa levels are typically not monitored, except in women who weigh less than 110 lb or more than 198 lb, have renal insufficiency, or have high-significant risk factors (eg, recurrent VTE).[15] In these women, the target anti-Xa level is 0.6 to 1 units/mL with twice-daily therapeutic LMWH; the target should be slightly higher in women receiving once-daily dosing.[1] Platelet counts are monitored initially after injection in women taking UFH; if thrombocytopenia occurs, it is typically between 7 to 14 days after therapy is initiated.[62] Recommendations vary regarding the need to reevaluate platelet counts after the initiation of LMWH; the SOGC recommends checking platelet count 1 week after initiating LMWH, whereas RCOG guidelines state this practice is not routinely necessary.[15,56]

Intravenous and/or subcutaneous (SQ) forms of UFH may be used instead of LMWH for the initial treatment of DVT or PE in pregnancy. UFH may be chosen over LMWH in some settings because of cost or availability. Recommended dosages and monitoring for LMWH and IV and SQ UFH are listed in *Table 3*.

Postpartum, women should be given a choice of LMWH versus warfarin anticoagulation.[15] By causing an initial decrease in protein C and protein S levels, warfarin can cause a hypercoagulable state in the first days of therapy.[63] With warfarin initiation, LMWH or UFH should be continued until the international normalized ratio (INR) is greater than 2 for at least 24 hours.[15] Typically, this level of anticoagulation is achieved within 5 days.[64] LMWH and warfarin therapy can be started concomitantly in an outpatient setting in select women who are postpartum and medically stable and have a supportive home environment and access to daily monitoring until the INR is therapeutic.[65] Women only requiring 6 weeks of anticoagulation may opt to continue taking LMWH rather than transitioning to warfarin.[1]

Table 2. Baseline Laboratory Tests for Initiating Anticoagulation

Thrombophilia profile: Controversial, best when not pregnant
Creatinine (LMWHs require dose adjustment with abnormal renal function)
Liver function tests (warfarin is contraindicated with significantly abnormal liver function)
CBC with platelet count
PT/INR
aPTT
Anti-Xa level (not recommended routinely; indicated with women <50 kg or >90 kg, recurrent VTE, or renal insufficiency)

aPTT = activated partial thromboplastin time; CBC = complete blood count; INR = international normalized ratio; PT = prothrombin time; LMWH = low-molecular-weight heparin; VTE = venous thromboembolism.

Information from Greer IA, Thomas A. Greentop Guideline 37b. Thromboembolic disease in pregnancy and the puerperium. Guidelines and Audit Committee of the Royal College of Obstetricians and Gynaecologists. 2015.

Delivery and Postpartum Management After Anticoagulation

Delivery. Recommendations vary regarding when to discontinue LMWH before delivery. The 2012 ACCP guidelines recommend that women receiving a therapeutic dose of LMWH should discontinue anticoagulation at least 24 hours before labor induction or scheduled cesarean delivery, and women receiving a once-daily prophylactic dose of LMWH should take half of their dose on the morning of the day before delivery.[12] RCOG guidelines recommend discontinuing LMWH at least 24 hours prior to scheduled induction or cesarean delivery.[13] Women with proximal DVT or PE within 2 weeks of delivery can be transitioned to IV UFH, which should be discontinued

Table 3. Therapeutic and Prophylactic Dosing and Monitoring of Low-Molecular-Weight, Intravenous, and Subcutaneous Unfractionated Heparin

Therapeutic Dosing of Low-Molecular-Weight Heparin

	Enoxaparin	Dalteparin	Tinzaparin
Adjusted (therapeutic) dose	1 mg/kg SQ every 12 hours	100 units/kg SQ every 12 hours or 200 units/kg SQ every 24 hours	175 units/kg SQ every 24 hours

Target anti-Xa level 0.6-1.0 units/mL 4 hours after last injection for twice daily regimen; slightly higher doses for once-daily regimen

Unfractionated Heparin

IV regimen (preferred in massive, life-threatening PE)

- IV bolus of 80 units/kg, followed by a continuous infusion of 18 units/kg/hour
- Monitor aPTT 4 to 6 hours after the loading dose and 6 hours after any dose change. Then check the aPTT at least daily and adjust dosage to achieve aPTT in the therapeutic range of 1.5 to 2.5 times the mean laboratory control value

SQ regimen

- 10,000 units or more (150-200 units/kg, lower dosing for <50 kg) SQ every 12 hours, with dosing adjusted as follows
- Monitor aPTT and adjust SQ dose to achieve aPTT in the therapeutic range of 1.5 to 2.5 times the mean laboratory control value at 6 hours after injection

Prophylactic Dosing of Low-Molecular-Weight Heparin

	Enoxaparin	Dalteparin	Tinzaparin
Prophylactic dose	40 mg/day SQ	5,000 units/day SQ	4,500 units/day SQ
Obesity	60 mg/day SQ	7,500 units/day SQ	75 units/kg/day SQ

Unfractionated Heparin

First trimester	5,000 to 7,500 units SQ every 12 hours
Second trimester	7,500 to 10,000 units SQ every 12 hours
Third trimester	10,000 units SQ every 12 hours, unless aPTT is elevated

Intermediate Dosing

Enoxaparin	Dalteparin
40 mg SQ every 12 hours	5,000 units SQ every 12 hours

aPTT = activated partial thromboplastin time; IV = intravenous; PE = pulmonary embolism; SQ = subcutaneous.

Information from American College of Obstetricians and Gynecologists. ACOG Practice Bulletin no. 196: thromboembolism in pregnancy. Obstet Gynecol. *2018;132(1):e1-e17; Chan WS, Rey E, Kent NE, et al; VTE in Pregnancy Guideline Working Group; Society of Obstetricians and Gynecologists of Canada. Venous thromboembolism and antithrombotic therapy in pregnancy.* J Obstet Gynaecol Can. *2014;36(6):527-553; Greer IA, Thomas A. Greentop Guideline 37b. Thromboembolic disease in pregnancy and the puerperium. Guidelines and Audit Committee of the Royal College of Obstetricians and Gynaecologists. 2015.*

4 to 6 hours before expected time of delivery or administration of epidural analgesia.[12] Women with spontaneous labor may be instructed to discontinue heparin at the onset of regular uterine contractions.[13,56]

Neuraxial anesthesia (epidural/spinal). Recommendations vary regarding how long after the last dose of LMWH it is safe to administer epidural or spinal analgesia. The 2018 Society for Obstetric Anesthesia and Perinatology (SOAP) consensus statement recommends waiting at least 4 to 6 hours after administration of low-dose UFH (up to 5,000 units, three times/day), at least 12 hours after intermediate-dose UFH (7,500 or 10,000 units, twice/day), and 24 hours or more after high-dose UFH (individual doses greater than 10,000 units or greater than 20,000 units/day) before administering neuraxial anesthesia.[66] According to SOAP, a woman is at low risk of adverse effects from neuraxial analgesia if her aPTT level is normal or anti-Xa is undetectable, even when these time limits are not met after UFH administration.[66] The American Society of Regional Anesthesia and Pain Medicine recommends delaying regional analgesia at least 12 hours after the last prophylactic dose of LMWH or 24 hours after the last therapeutic dose of LMWH is administered.[67] Similarly, RCOG and SOGC guidelines recommend that epidural or spinal analgesia should not be administered for 24 hours after the last therapeutic dose of LMWH, and 10 to 12 hours after prophylactic dosing.[15,56]

The Society of Obstetricians and Gynaecologists of Canada recommends that epidural analgesia not be discontinued until 10 to 12 hours after the last prophylactic LMWH dose, and at least 24 hours after the last therapeutic LMWH dose.[56] SOGC guidelines also recommend restarting prophylactic LMWH dosing 6 to 8 hours after initiation of neuraxial analgesia, waiting more than 24 hours if bleeding occurred during the neuraxial block, and waiting more than 4 hours after removal of the neuraxial catheter. When restarting therapeutic LMWH dosing, it is recommended to wait more than 24 hours after initiation of neuraxial analgesia and more than 4 hours after removal of the neuraxial catheter.[56]

Prophylaxis

Prophylaxis against VTE in pregnancy may be required antenatally for women with a history of DVT or PE and for those with a history of thrombophilia. Although better studies are needed, LMWH appears to be the safest and most effective form of thromboprophylaxis in pregnancy.[44,46,68] Prophylactic doses of LMWH are listed in *Table 3*. SQ UFH may be used as a lower cost alternative to LMWH; SQ UFH doses are also listed in *Table 3*. Some experts recommend adjusting prophylactic LMWH dosing in women with obesity, but there are no evidence-based guidelines for this practice.[7] SOGC recommendations for dosing in women with obesity are included in *Table 3*.[56]

Antenatal low-dose aspirin (75 to 100 mg) is recommended in combination with LMWH or UFH for women with antiphospholipid antibody syndrome and a history of three or more pregnancy losses.[12] Adding aspirin also is recommended in women with prosthetic heart valves who are at high risk of thromboembolism.[12]

A U.S. Preventive Services Task Force analysis of 19 RCTs (12 good quality) and two good quality observational studies showed that low-dose aspirin appears to be safe in pregnancy.[69] The most common discontinuation date was on delivery; however, six trials discontinued aspirin use before delivery, as early as 35 weeks' gestation, or when preeclampsia developed. Eleven RCTs (23,332 women) showed aspirin does not cause placental abruption (RR 1.17; 95% CI = 0.93-1.48).[69] Fourteen RCTs (14 studies; n = 22,848) showed no effect on perinatal mortality (RR 0.92; 95% CI = 0.76-1.11).[69] Nine trials (22,760 women) showed no increase in postpartum hemorrhage.[69]

Clinical indications for anticoagulant prophylaxis and recommendations for when to initiate and discontinue therapy are summarized in *Tables 4* (ACCP), *5* (ACOG), *6* (RCOG), and *7* (SOGC). ACCP recommendations have been criticized for having a narrower list of scenarios for which antenatal and postnatal prophylaxis is recommended compared with other organizations. [70] The authors of the ACCP guidelines defend their recommendations and acknowledge that evidence is lacking in many areas, allowing for variations in recommendations and clinical judgment.[71]

A 2017 metaanalysis and systematic review of observational studies found that risks for VTE in pregnancy were less than 3% in women with heterozygous factor V Leiden, heterozygous prothrombin G20210A mutations or compound heterozygous factor V Leiden and prothrombin

Table 4. Clinical Indications for Anticoagulant Prophylaxis per American College of Chest Physicians Guidelines

Indication 1: Personal history of DVT or PE, no known thrombophilia

1A. DVT or PE with thrombogenic event (eg, hip fracture, prolonged surgery)

Antenatal: No prophylaxis

Postpartum: 6 weeks LMWH or warfarin

1B. DVT or PE with no thrombogenic event, pregnancy-related or estrogen-related VTE, history of multiple VTE but not on chronic anticoagulation

Antenatal: Anticoagulation with prophylactic or intermediate dose (0.75 mg/kg SQ twice/day, or fixed dose enoxaparin 40 mg SQ every 12 hours) of LMWH

Postpartum: 6 weeks of LMWH or warfarin

Indication 2: Personal history of DVT or PE, known thrombophilia

2A. Single prior VTE with history of homozygous factor V Leiden or prothrombin 20210A mutation

Antenatal: Anticoagulation with prophylactic LMWH

Postpartum: 6 weeks of LMWH or warfarin

2B. Single prior VTE with thrombophilia other than homozygous factor V Leiden or prothrombin 20210A

Antenatal: Anticoagulation with prophylactic LMWH

Postpartum: 6 weeks of LMWH or warfarin

Indication 3: Women undergoing chronic anticoagulation before pregnancy

Antenatal: Anticoagulation with adjusted dose LMWH or 75% therapeutic dose of LMWH

Postpartum: Resumption of chronic anticoagulation

Indication 4: Women with no history of VTE but known thrombophilia

4A. Homozygous for factor V Leiden or the prothrombin G20210A mutation and positive family history of VTE

Antenatal: Prophylactic- or intermediate-dose of LMWH

Postpartum: 6 weeks anticoagulation with prophylactic- or intermediate-dose LMWH or warfarin

4B. Homozygous for factor V Leiden or the prothrombin G20210A mutation and no family history of VTE

Antenatal: No prophylaxis

Postpartum: 6 weeks LMWH or warfarin

4C. Thrombophilia other than homozygous for factor V Leiden or the prothrombin G20210A mutation and family history of VTE

Antenatal: No prophylaxis

Postpartum: 6 weeks LMWH or warfarin

4D. Thrombophilia other than homozygous for factor V Leiden or the prothrombin G20210A mutation and no family history of VTE

Antenatal: No prophylaxis

Postpartum: No prophylaxis

4E. Antiphospholipid antibody syndrome by laboratory and clinical criteria

Antenatal: Prophylactic LWMH and low-dose aspirin (75 to 100 mg/day)

Postpartum: 6 weeks of LMWH or warfarin

LMWH = low-molecular-weight heparin; PE = pulmonary embolism; VTE = venous thromboembolism.

Information from Bates S, Greer I, Middeldorp S, et al. VTE, thrombophilia, antithrombotic therapy, and pregnancy: Antithrombotic Therapy and Prevention of Thrombosis, 9th ed: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines. Chest. *2012;141:e691s-736s.*

G20210 mutations.[72] In contrast with ACCP guidelines, the authors recommend against LMWH prophylaxis with these thrombophilias even when there is a positive family history of VTE.[72]

Women with mechanical heart valves should be transferred to a high-risk specialist or comanaged with close consultation. The manufacturer of enoxaparin issued a warning against its use for the treatment of women with mechanical heart valves who are pregnant because of an undisclosed number of postmarketing reports of thrombosed valves in patients receiving enoxaparin.[73] Low-dose warfarin therapy may be considered in close consultation with a cardiology subspecialist.

Postcesarean delivery venous thromboembolism prophylaxis. Recommendations and guidelines from ACCP, ACOG, SOGC, and RCOG regarding postcesarean delivery VTE prophylaxis vary; however, current evidence seems to support universal mechanical prophylaxis with sequential compression devices (SCDs).[1,12,56] Evidence supporting pharmacotherapy prophylaxis with LMWH is lacking.

The 2012 ACCP guidelines state that VTE prophylaxis after cesarean delivery should be based on risk factors and only early ambulation is indicated in women without risk factors for VTE prophylaxis other than cesarean delivery. The ACOG guidelines recommend use of pneumatic compression devices during cesarean delivery in all women not already receiving pharmacotherapy prophylaxis.[1,12] The SOGC guidelines recommend postcesarean delivery SCDs when pharmacotherapy prophylaxis is indicated but not possible.[56] The SOGC guidelines also recommend SCDs and pharmacotherapy prophylaxis after cesarean delivery for women who are at high risk.[56]

Evidence supports postcesarean delivery mechanical prophylaxis using a pneumatic compression device until the woman becomes ambulatory. A large hospital system study showed that a 2007 protocol requiring universal use of pneumatic compression devices for all women undergoing cesarean delivery resulted in a significant decrease in maternal deaths due to postcesarean delivery PE (from 7 of 458,097 cesarean deliveries in 2000-2006 to 1 of 465,880 in 2007-2012; $P = 0.038$).[74]

Pharmacotherapy prophylaxis for VTE after cesarean delivery in women with various risk factors is recommended by ACCP, ACOG, RCOG, and SOGC (*Table 8*). A study of 293 women

who underwent cesarean delivery showed that 34.8% met ACCP criteria for pharmacotherapy prophylaxis, 1% met ACOG criteria, and 85% met RCOG criteria.[75] A 2010 Cochrane review concluded that there is not enough evidence to recommend for or against the routine use of LMWH prophylaxis after cesarean delivery.[42]

The expansion of postcesarean delivery pharmacotherapy prophylaxis remains controversial. The authors of a 2016 commentary argued that

Table 5. Clinical Indications for Anticoagulant Prophylaxis per the American College of Obstetricians and Gynecologists 2018 Practice Bulletin

Indication 1: No history VTE, no thrombophilia

Antenatal: Surveillance (VTE risk assessment before or early pregnancy and repeated if new risk factors arise such as immobilization/hospitalization)

Postpartum: Surveillance; no prophylaxis unless multiple risk factors (including first-degree relative with history of thrombotic episode, or other major risk factor such as obesity, prolonged immobility, or cesarean delivery)

Indication 2: VTE diagnosed during pregnancy

Antenatal: Adjusted dose LMWH or UFH

Postpartum: Adjusted dose LMWH or UFH for at least 6 weeks; longer therapy and oral anticoagulant may be indicated

Indication 3: Personal history of single DVT or PE, no known thrombophilia

3A. Single DVT or PE *with thrombogenic event* (eg, surgery, trauma, immobility) not pregnancy- or estrogen-related

Antenatal: Surveillance

Postpartum: Surveillance; no prophylaxis unless additional risk factors (including first-degree relative with history of thrombotic episode, or other major risk factor such as obesity, prolonged immobility, or cesarean delivery)

3B. Single DVT or PE with *no thrombogenic event* (idiopathic), pregnancy- or estrogen-related

Antenatal: Prophylactic, intermediate, or adjusted dose of LMWH or UFH

Postpartum: Prophylactic, intermediate, or adjusted dose of LMWH or UFH for 6 weeks

Indication 4: Personal history of single DVT or PE, known thrombophilia – not on chronic anticoagulation

4A. Single DVT or PE *with low-risk thrombophilia* (factor V Leiden heterozygous; prothrombin G20210A heterozygous; protein C or protein S deficiency, antiphospholipid antibody)

Antenatal: Prophylactic or intermediate dose of LMWH or UFH

Postpartum: Prophylactic or intermediate dose of LMWH or UFH

4B. Single DVT or PE or affected first-degree relative AND patient *with high-risk thrombophilia* (antithrombin deficiency; double heterozygous for prothrombin G20210A mutation and factor V Leiden; factor V Leiden homozygous or prothrombin G20210A mutation homozygous)

Antenatal: Prophylactic, intermediate, or adjusted dose of LMWH or UFH

Postpartum: Prophylactic, intermediate, or adjusted dose of LMWH or UFH (same therapy as antepartum) for 6 weeks

Indication 5: Personal history of multiple DVT or PE with or without thrombophilia

5A. Not on chronic anticoagulation

Antenatal: Intermediate or adjusted dose of LMWH or UFH

Postpartum: Intermediate or adjusted dose of LMWH or UFH (same therapy as antepartum) for 6 weeks

5B. On chronic anticoagulation before pregnancy

Antenatal: Adjusted dose of LMWH or UFH

Postpartum: Resume chronic anticoagulation; oral anticoagulant may be considered

Indication 6: No history of VTE and known thrombophilia

6A. No history of DVT or PE *with low-risk thrombophilia* (factor V Leiden heterozygous; prothrombin G20210A heterozygous; protein C or protein S deficiency, antiphospholipid antibody) *without family history* (first-degree relative) with VTE

Antenatal: Surveillance

Postpartum: Surveillance; prophylaxis if additional risk factors including obesity, prolonged immobility, cesarean delivery

6B. No history of DVT or PE *with low-risk thrombophilia* (factor V Leiden heterozygous; prothrombin G20210A heterozygous; protein C or protein S deficiency, antiphospholipid antibody) *with family history* (first-degree relative) with VTE

Antenatal: Surveillance with no prophylaxis or prophylactic dose of LMWH or UFH

Postpartum: Prophylactic or intermediate dose of LMWH or UFH

6C. No history of DVT or PE *with high-risk thrombophilia* (antithrombin deficiency; double heterozygous for prothrombin G20210A mutation and factor V Leiden; factor V Leiden homozygous or prothrombin G20210A mutation homozygous)

Antenatal: Prophylactic or intermediate dose of LMWH or UFH

Postpartum: Prophylactic or intermediate dose of LMWH or UFH

DVT = deep vein thrombosis; LMWH = low-molecular-weight heparin; PE = pulmonary embolism; UFH = unfractionated heparin; VTE = venous thromboembolism.

Information from American College of Obstetricians and Gynecologists. ACOG Practice Bulletin no. 196: thromboembolism in pregnancy. Obstet Gynecol. *2018;132(1):e1-e17.*

postcesarean delivery pharmacotherapy prophylaxis should not be recommended without further evidence for improved outcomes compared with universal use of mechanical VTE prophylaxis.[76] If only 1 in 465,880 women died from postcesarean delivery VTE with mechanical prophylaxis,[74] the number needed to treat with pharmacotherapy prophylaxis to prevent 1 maternal death would be approximately 1 million women. Authors of a 2018 editorial argue that guidelines recommending postcesarean pharmacotherapy prophylaxis should be reconsidered because the cost-benefit ratios use faulty prediction models that report asymptomatic DVTs and underestimate the

Table 6. Clinical Indications for Anticoagulant Prophylaxis per Royal College of Obstetricians and Gynaecologists Guidelines

Indication 1: Single previous VTE related to major surgery and no other risk factors

Antenatal: Prophylactic dose of LMWH starting at 28 weeks' gestation

Postpartum: 6 weeks of LMWH or warfarin (or other length per postnatal risk assessment)

Indication 2: Single previous VTE (except those with VTE related to major surgery and no other risk factors)

Antenatal: Prophylactic dose of LMWH

Postpartum: 6 weeks of LMWH or warfarin (or other length per postnatal risk assessment)

Indication 3: Single previous VTE associated with thrombophilia

3A. Single previous VTE associated with antithrombin deficiency or antiphospholipid syndrome

Antenatal: Higher-dose LMWH (50%, 75%, or full treatment dose)

Postpartum: 6 weeks of LMWH or warfarin (or until returned to long-term oral anticoagulant)

3B. Single previous VTE associated with thrombophilias other than antithrombin deficiency or antiphospholipid syndrome

Antenatal: Prophylactic dose of LMWH

Postpartum: 6 weeks of LMWH or warfarin (or other length per postnatal risk assessment)

Indication 4: Recurrent VTE

Antenatal: Higher-dose LMWH (50%, 75%, or full treatment dose)

Postpartum: 6 weeks of LMWH or warfarin (or until returned to long-term oral anticoagulant)

Indication 5: Thrombophilia with no history VTE

5A. Antithrombin, protein C or S deficiency or those with more than one thrombophilic defect (including homozygous factor V Leiden, homozygous prothrombin gene mutation, and compound heterozygotes)

Antenatal: LMWH considered

Postpartum: 6 weeks of LMWH or warfarin

5B. Heterozygosity for factor V Leiden or prothrombin gene mutation or antiphospholipid antibodies and three risk factors

Antenatal: Prophylactic dose of LMWH

Postpartum: 6 weeks of LMWH or warfarin

5C. Heterozygosity for factor V Leiden or prothrombin gene mutation or antiphospholipid antibodies and two risk factors

Antenatal: Prophylactic dose of LMWH from 28 weeks' gestation

Postpartum: 6 weeks of LMWH or warfarin

5D. Heterozygosity for factor V Leiden or prothrombin gene mutation or antiphospholipid antibodies and three risk factors

Antenatal: None

Postpartum: At least 10 days of LMWH or warfarin

5E. Four or more current risk factors (other than previous VTE or thrombophilia)

Antenatal: Prophylactic dose of LMWH

Postpartum: 6 weeks of LMWH or warfarin (or other length per postnatal risk assessment)

5F. Three current risk factors (other than previous VTE or thrombophilia)

Antenatal: Prophylactic dose of LMWH from 28 weeks' gestation

Postpartum: 6 weeks of LMWH or warfarin (or other length per postnatal risk assessment)

5G. Two current risk factors (other than previous VTE or thrombophilia)

Antenatal: None

Postpartum: At least 10 days of LMWH or warfarin

Indication 6: First trimester risk factors

6A. Hospital admission for hyperemesis

Antenatal: Prophylactic dose of LMWH during admission

Postpartum: None

6B. Ovarian hyperstimulation syndrome

Antenatal: Prophylactic dose of LMWH during first trimester

Postpartum: None

6C. In vitro fertilization and three other risk factors

Antenatal: Prophylactic dose of LMWH from first trimester

Postpartum: 6 weeks of LMWH or warfarin

LMWH = low-molecular-weight heparin; VTE = venous thromboembolism.

Information from Royal College of Obstetricians and Gynaecologists. Greentop Guideline 37a. Reducing the risk of thrombosis and embolism during pregnancy and the puerperium. 2015.

potential risks of LMWH use.[77] Numbers needed to treat and to harm are not available.[77]

There is also lack of data and consistent recommendations regarding which women should receive pharmacotherapy prophylaxis after a vaginal delivery. The decision to administer pharmacotherapy prophylaxis for VTE after a vaginal delivery in women with risk factors (*Table 1*) can be made on an individual basis.[13,56] The RCOG guidelines recommend heparin prophylaxis after vaginal delivery in women with a body mass index greater than 40 kg/m^2.[13]

Evidence is also lacking regarding the timing of postdelivery initiation of pharmacotherapy prophylaxis for VTE. Based on ACOG and RCOG recommendations, it is reasonable to start or resume heparin prophylaxis 4 to 6 hours after a vaginal delivery, 6 to 12 hours after a cesarean delivery, and 4 hours after discontinuing epidural analgesia.[1,13] There is less than a 1% risk of wound hematoma with LMWH prophylaxis.[48]

Systems

Given the complexity of management decisions and variety of consensus guideline recommendations regarding postpartum VTE prophylaxis, facilities may want to develop consistent local guidelines and standards of care to ensure consistent practice and reduce the morbidity and mortality from VTE in pregnancy. One hospital implemented a protocol based on RCOG guidelines for the prevention of postpartum VTE and found 89.5% adherence to the protocol resulted in an increase in postpartum heparin administration from 0.28% to 33.46%.[78] A larger study (ideally a RCT) would be needed to determine whether an increase in heparin prophylaxis affected VTE rates or wound complications.

The Council on Patient Safety in Women's Health Care and the Alliance for Innovation on Maternal Health (AIM) have developed the Maternal Venous Thromboembolism Prevention patient safety bundle that facilities may use as a tool to manage VTE in a standardized and evidence-based manner (http://safehealthcareforeverywoman.org/patient-safety-bundles/maternal-venous-thromboembolism/).[79] The bundle has four domains (readiness, recognition, response, and reporting/systems learning), which can be used to guide the development of local standards of practice for managing VTE.

In 2018, the California Maternal Quality Care Collaborative (CMQCC) published the Improving Health Care Response to Maternal Venous Thromboembolism patient safety bundle, which focuses on identifying women who are pregnant

Table 7. Clinical Indications for Anticoagulant Prophylaxis per Society of Obstetricians and Gynecologists of Canada Guidelines

Indication 1: Personal history of unprovoked VTE
Antenatal: Prophylactic dose of LMWH or UFH
Postpartum: 6 weeks of LMWH or warfarin

Indication 2: Personal history of VTE related to pregnancy or contraception
Antenatal: Prophylactic dose of LMWH or UFH
Postpartum: 6 weeks of LMWH or warfarin

Indication 3: Personal history of a previous provoked VTE and any low-risk thrombophilia
Antenatal: Prophylactic dose of LMWH or UFH
Postpartum: 6 weeks of LMWH or warfarin

Indication 4: Asymptomatic homozygous factor V Leiden, homozygous prothrombin gene mutation 20210A, combined thrombophilia, or antithrombin deficiency
Antenatal: Prophylactic dose of LMWH or UFH
Postpartum: 6 weeks of LMWH or warfarin

Indication 5: Nonobstetric surgery during pregnancy
Antenatal: Procedure- and patient-dependent
Postpartum: Procedure- and patient-dependent

Indication 6: Strict antepartum bedrest for ≥7 days in a woman with a BMI >25 kg/m^2 at first antenatal visit
Antenatal: Prophylactic dose of LMWH or UFH
Postpartum: 6 weeks of LMWH or warfarin

Indication 7: Multiple pregnancy-related risk factors where risk of VTE is thought to be >1%, especially for women admitted for bedrest
Antenatal: Prophylactic dose of LMWH or UFH
Postpartum: At least 1 to 2 weeks postpartum

Indication 8: Assisted reproductive technology
Antenatal: Prophylactic dose of LMWH or UFH
Postpartum: 6 weeks of LMWH or warfarin

Risk factors: Obesity (BMI >30 kg/m^2); age >35 years; parity ≥3; tobacco use; gross varicose veins; current preeclampsia; immobility (eg, paraplegia, pelvic girdle pain with reduced mobility); family history of unprovoked or estrogen-provoked VTE in first-degree relative; low-risk thrombophilia; multiple pregnancy; in vitro fertilization/assisted reproductive technology.

BMI = body mass index; LMWH = low-molecular-weight heparin; UHF = unfractionated heparin; VTE = venous thromboembolism.

Information from Chan WS, Rey E, Kent NE, et al; VTE in Pregnancy Guideline Working Group. Society of Obstetricians and Gynecologists of Canada. Venous thromboembolism and antithrombotic therapy in pregnancy. J Obstet Gynaecol Can. *2014;36(6):527-553.*

Table 8. Comparison of Recommendations for Postcesarean Delivery Thromboembolism Pharmacotherapy Prophylaxis

Organization	Indication for pharmacotherapy prophylaxis	
ACCP	**Criteria: one major or two or more minor risk factors**	
	Major risk factors (one needed for prophylaxis)	Minor risk factors (two needed for prophylaxis)
	Immobility	BMI >30 kg/m^2
	PPH 1,000 mL with surgery	Multiple pregnancy
	Previous VTE	Emergency cesarean delivery
	Preeclampsia with fetal growth restriction	Tobacco use >10 cigarettes/day
	Thrombophilia	Fetal growth restriction
	Antithrombin deficiency	Thrombophilia
	Factor V Leiden (homozygous or heterozygous)	Protein C deficiency
	Prothrombin G20210A (homozygous or heterozygous)	Protein S deficiency
	Medical conditions	Preeclampsia
	SLE	
	Heart disease	
	Sickle cell disease	
	Blood transfusion	
	Postpartum infection	
ACOG	**Criteria:**	
	Each institution should adopt a risk assessment protocol and implement it in a systematic way	
RCOG	**Criteria:**	
	High risk (at least 6 weeks postnatal prophylactic LMWH)	Two or more of following: (consider longer prophylaxis if >3 risk factors)
	Any previous VTE	Age >35 years
	Anyone requiring antenatal LMWH	Current preeclampsia
	High-risk thrombophilia	Current systemic infection
	Low-risk thrombophilia + family history	Elective cesarean delivery
	Intermediate risk (at least 10 days postnatal prophylaxis LMWH)	Family history of VTE
	Any surgical procedure in the postpartum period except immediate repair of the perineum	Gross varicose veins
	BMI ≥40 kg/m^2	Immobility (eg, paraplegia, pelvic girdle pain with reduced mobility, long distance travel)
	Cesarean delivery in labor	Low-risk thrombophilia
	Medical comorbidities (eg, cancer, heart failure, active SLE, IBD or inflammatory polyarthropathy, nephrotic syndrome, type 1 diabetes with nephropathy, sickle cell disease, current intravenous drug user)	Mid-cavity rotational or assisted delivery
		Multiple pregnancy
		Obesity (BMI ≥30 kg/m^2)
	Readmission or prolonged hospitalization (≥3 days) in the postpartum period	Parity ≥3
		PPH >1 L or blood transfusion
		Preterm delivery in this pregnancy (<37 weeks' gestation)
		Prolonged labor (>24 hours)
		Tobacco use
		Stillbirth in this pregnancy

continues

who may benefit from pharmacotherapy prophylaxis for VTE.[80] This bundle classifies women as low, medium, or high risk of VTE and gives recommendations based on ACOG and ACCP guidelines.[80]

Summary

This chapter aims to improve learner understanding of the risk factors, diagnosis, and management of VTE. Pregnancy is a relatively prothrombotic state, but routine screening for thrombophilia is

Table 8. Comparison of Recommendations for Postcesarean Delivery Thromboembolism Pharmacotherapy Prophylaxis *(continued)*

Organization	Indication for pharmacotherapy prophylaxis
SOGC	**Criteria:** At least one risk factor History of any prior VTE Any high-risk thrombophilia: antiphospholipid syndrome, antithrombin deficiency, homozygous factor V Leiden or prothrombin gene mutation 20210A, combined thrombophilia Strict bed rest prior to delivery for 7 days or more Peripartum or postpartum blood loss of >1 L or blood product replacement, and concurrent postpartum surgery Peripartum/postpartum infection At least two risk factors BMI ≥30 kg/m^2 at first antepartum visit Tobacco use (>10 cigarettes/day antepartum) Preeclampsia Intrauterine growth restriction Placenta previa Emergency cesarean delivery Peripartum or postpartum blood loss of >1 L or blood product replacement Any low-risk thrombophilia (protein C or protein S deficiency, heterozygous factor V Leiden, or prothrombin gene mutation 20210A) Maternal cardiac disease, SLE, sickle cell disease, IBD, varicose veins, gestational diabetes Preterm delivery Stillbirth At least three risk factors Age >35 years Parity ≥2 Any assisted reproductive technology Multiple pregnancy Placental abruption Prelabor rupture of membranes Elective cesarean delivery Maternal cancer

ACCP = American College of Chest Physicians; ACOG = American College of Obstetricians and Gynecologists; BMI = body mass index; IBD = inflammatory bowel disease; IV = intravenous; LMWH = low-molecular-weight heparin; PPH = postpartum hemorrhage; RCOG = Royal College of Obstetricians and Gynaecologists; SLE = systemic lupus erythematosus; VTE = venous thromboembolism.

Information from Palmerola KL, D'Alton ME, Brock CO, Friedman AM. A comparison of recommendations for pharmacologic thromboembolism prophylaxis after caesarean delivery from three major guidelines. BJOG. *2016;123(13):2157-2162; Chan WS, Rey E, Kent NE, et al; VTE in Pregnancy Guideline Working Group; Society of Obstetricians and Gynecologists of Canada. Venous thromboembolism and antithrombotic therapy in pregnancy.* J Obstet Gynaecol Can. *2014;36(6):527-553.*

not recommended. Providers must maintain a high level of suspicion in women presenting with symptoms suggestive of VTE in any trimester. Doppler ultrasound is the initial diagnostic test of choice for DVT or PE in women who are stable, and treatment for DVT or suspected PE should be initiated with positive ultrasound findings. High clinical suspicion in the absence of positive diagnostic studies should not delay treatment, and follow-up testing can be pursued even after therapy is initiated. Priority should be placed on stabilizing women who are unstable, with close consultation as indicated. LMWH is the agent of choice for treatment and prophylaxis.

American College of Chest Physicians, ACOG, SOGC, and RCOG guidelines provide recommendations for treating and preventing VTE; local practice should be well-established based on the best available evidence. Regional anesthesia is not contraindicated in women receiving prophylactic or therapeutic anticoagulation; however, guidelines should be followed regarding safe timing. Recommendations about postcesarean delivery VTE prophylaxis vary. The key to diagnosing these conditions is clinical vigilance coupled with appropriate laboratory or imaging studies, while balancing maternal and fetal well-being in diagnostic and treatment decisions.

Nursing Considerations: Venous Thromboembolism in Pregnancy

- Identify women with risk factors for VTE, including pregnancy/postpartum, mode of delivery, and history
- Be familiar with signs and symptoms of and diagnostic tests for DVT and PE
- Advocate for early ambulation postdelivery
- Champion efforts to implement the Maternal Venous Thromboembolism Prevention Patient Safety Bundle

DVT = deep vein thrombosis; PE = pulmonary embolism; VTE = venous thromboembolism.

COUNCIL ON PATIENT SAFETY IN WOMEN'S HEALTH CARE

safe health care for every woman

PATIENT SAFETY BUNDLE

Maternal Venous Thromboembolism Prevention

READINESS

Every Unit

- Use a standardized thromboembolism risk assessment tool for VTE during:
  - Outpatient prenatal care
  - Antepartum hospitalization
  - Hospitalization after cesarean or vaginal deliveries
  - Postpartum period (up to 6 weeks after delivery)

RECOGNITION & PREVENTION

Every Patient

- Apply standardized tool to all patients to assess VTE risk at time points designated under "Readiness"
- Apply standardized tool to identify appropriate patients for thromboprophylaxis
- Provide patient education
- Provide all healthcare providers education regarding risk assessment tools and recommended thromboprophylaxis

RESPONSE

Every Unit

- Use standardized recommendations for mechanical thromboprophylaxis
- Use standardized recommendations for dosing of prophylactic and therapeutic pharmacologic anticoagulation
- Use standardized recommendations for appropriate timing of pharmacologic prophylaxis with neuraxial anesthesia

REPORTING/SYSTEMS LEARNING

Every Unit

- Review all thromboembolism events for systems issues and compliance with protocols
- Monitor process metrics and outcomes in a standardized fashion
- Assess for complications of pharmacologic thromboprophylaxis

Standardization of health care processes and reduced variation has been shown to improve outcomes and quality of care. The Council on Patient Safety in Women's Health Care disseminates patient safety bundles to help facilitate the standardization process. This bundle reflects emerging clinical, scientific, and patient safety advances as of the date issued and is subject to change. The information should not be construed as dictating an exclusive course of treatment or procedure to be followed. Although the components of a particular bundle may be adapted to local resources, standardization within an institution is strongly encouraged.

The Council on Patient Safety in Women's Health Care is a broad consortium of organizations across the spectrum of women's health for the promotion of safe health care for every woman.

October 2015

Reprinted from Council on Patient Safety in Women's Health Care. AIM-Supported Patient Safety Bundles. Maternal Venous Thromboembolism Prevention. Available at https://safehealthcareforeverywoman.org.

References

1. ACOG Practice Bulletin no. 196: thromboembolism in pregnancy. *Obstet Gynecol*. 2018;132(1):e1-e17.
2. Heit JA, Kobbervig CE, James AH, et al. Trends in the incidence of venous thromboembolism during pregnancy or postpartum: a 30-year population-based study. *Ann Intern Med*. 2005;143(10):697-706.
3. Kourlaba G, Relakis J, Kontodimas S, et al. A systematic review and meta-analysis of the epidemiology and burden of venous thromboembolism among pregnant women. *Int J Gynaecol Obstet*. 2016;132(1):4-10.
4. Doyle NM, Ramirez MM, Mastrobattista JM, et al. Diagnosis of pulmonary embolism: a cost-effectiveness analysis. *Am J Obstet Gynecol*. 2004;191(3):1019-1023.
5. Kreidy R. Pathophysiology of post-thrombotic syndrome: the effect of recurrent venous thrombosis and inherited thrombophilia. *ISRN Vascular Med*. 2011;513503.
6. Gerhardt A, Scharf RE, Zotz RB. Effect of hemostatic risk factors on the individual probability of thrombosis during pregnancy and the puerperium. *Thromb Haemost*. 2003;90(1):77-85.
7. ACOG Practice Bulletin no. 197: inherited thrombophilias in pregnancy. *Obstet Gynecol*. 2018;132(1):e18-e34.
8. Zotz RB, Gerhardt A, Scharf RE. Prediction, prevention, and treatment of venous thromboembolic disease in pregnancy. *Semin Thromb Hemost*. 2003;29(2):143-154.
9. Blondon M, Casini A, Hoppe KK, et al. Risks of venous thromboembolism after cesarean sections: a meta-analysis. *Chest*. 2016;150(3):572-596.
10. Pabinger I, Grafenhofer H, Kyrle PA, et al. Temporary increase in the risk for recurrence during pregnancy in women with a history of venous thromboembolism. *Blood*. 2002;100(3):1060-1062.
11. Friederich PW, Sanson BJ, Simioni P, et al. Frequency of pregnancy-related venous thromboembolism in anticoagulant factor-deficient women: implications for prophylaxis. *Ann Intern Med*. 1996;125(12):955-960.
12. Bates SM, Greer IA, Middeldorp S, et al. VTE, thrombophilia, antithrombotic therapy, and pregnancy: antithrombotic therapy and prevention of thrombosis, 9th ed: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines. *Chest.* 2012;141(2 Suppl):e691S-e736S.
13. Royal College of Obstetricians and Gynaecologists. Green-top Guideline no. 37a. Reducing the risk of venous thromboembolism during pregnancy and the puerperium. London, England: Royal College of Obstetricians and Gynaecologists; 2015.
14. Walker MC, Garner PR, Keely EJ, et al. Changes in activated protein C resistance during normal pregnancy. *Am J Obstet Gynecol*. 1997;177(1):162-169.
15. Royal College of Obstetricians and Gynaecologists. Green-top Guideline no. 37b. Thromboembolic disease in pregnancy and the puerperium: acute management. London, England: Royal College of Obstetricians and Gynaecologists; 2015.
16. Committee on Practice Bulletins—Obstetrics, American College of Obstetricians and Gynecologists. Practice Bulletin no. 132: antiphospholipid syndrome. *Obstet Gynecol*. 2012;120(6):1514-1521.
17. Bates S, Jaeschke R, Stevens S, et al. Diagnosis of DVT: antithrombotic therapy and prevention of Thrombosis, 9th ed: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines. *Chest.* 2012;141(2 Suppl):e351S-e418S.
18. Chan WS, Spencer FA, Ginsberg JS. Anatomic distribution of deep vein thrombosis in pregnancy. *CMAJ*. 2010; 182(7):657-660.
19. Chan WS, Lee A, Spencer FA, et al. Predicting deep venous thrombosis in pregnancy: out in "LEFt" field? *Ann Intern Med*. 2009;151(2):85-92.
20. Righini M, Jobic C, Boehlen F, et al; EDVIGE study group. Predicting deep venous thrombosis in pregnancy: external validation of the LEFT clinical prediction rule. *Haematologica*. 2013;98(4):545-548.
21. Nijkeuter M, Ginsberg JS, Huisman MV. Diagnosis of deep vein thrombosis and pulmonary embolism in pregnancy: a systematic review. *J Thromb Haemost*. 2006; 4(3):496-500.
22. Kearon C, Akl EA, Comerota AJ, et al. Antithrombotic therapy for VTE disease: antithrombotic therapy and prevention of thrombosis, 9th ed: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines. *Chest.* 2012;141(2 Suppl):e419S-e496S.
23. O'Connor C, Moriarty J, Walsh J, et al. The application of a clinical risk stratification score may reduce unnecessary investigations for pulmonary embolism in pregnancy. *J Matern Fetal Neonatal Med*. 2011;24(12): 1461-1464.
24. Cahill AG, Stout MJ, Macones GA, Bhalla S. Diagnosing pulmonary embolism in pregnancy using computed-tomographic angiography or ventilation-perfusion. *Obstet Gynecol*. 2009;114(1):124-129.
25. Lapner ST, Kearon C. Diagnosis and management of pulmonary embolism. *BMJ*. 2013;346:f757.
26. van Mens TE, Scheres LJ, de Jong PG, et al. Imaging for the exclusion of pulmonary embolism in pregnancy. *Cochrane Database Syst Rev*. 2017;1:CD011053.
27. Cook JV, Kyriou J. Radiation from CT and perfusion scanning in pregnancy. *BMJ*. 2005;331(7512):350.
28. Chan WS. Diagnosis of venous thromboembolism in pregnancy. *Thromb Res*. 2018;163:221-228.
29. Remy-Jardin M, Remy J. Spiral CT angiography of the pulmonary circulation. *Radiology*. 1999;212(3):615-636.
30. Allen C, Demetriades T. Radiation risk overestimated. *Radiology*. 2006;240(2):613-614, discussion 614.
31. Parker MS, Hui FK, Camacho MA, et al. Female breast radiation exposure during CT pulmonary angiography. *AJR Am J Roentgenol*. 2005;185(5):1228-1233.
32. Kearon C. Diagnosis of pulmonary embolism. *CMAJ*. 2003;168(2):183-194.
33. Perrier A, Roy PM, Sanchez O, et al. Multidetector-row computed tomography in suspected pulmonary embolism. *N Engl J Med*. 2005;352(17):1760-1768.
34. Quiroz R, Kucher N, Zou KH, et al. Clinical validity of a negative computed tomography scan in patients with suspected pulmonary embolism: a systematic review. *JAMA*. 2005;293(16):2012-2017.

35. Schoepf UJ, Goldhaber SZ, Costello P. Spiral computed tomography for acute pulmonary embolism. *Circulation*. 2004;109(18):2160-2167.
36. Ramzi DW, Leeper KV. DVT and pulmonary embolism: part I. Diagnosis. *Am Fam Physician*. 2004;69(12): 2829-2836.
37. Kline JA, Johns KL, Colucciello SA, Israel EG. New diagnostic tests for pulmonary embolism. *Ann Emerg Med*. 2000;35(2):168-180.
38. Committee on Practice Bulletins—Gynecology, American College of Obstetricians and Gynecologists. ACOG Practice Bulletin no. 84: prevention of deep vein thrombosis and pulmonary embolism. *Obstet Gynecol*. 2007; 110(2 Pt 1):429-440.
39. Pillny M, Sandmann W, Luther B, et al. Deep venous thrombosis during pregnancy and after delivery: indications for and results of thrombectomy. *J Vasc Surg*. 2003;37(3):528-532.
40. Hao Q, Dong BR, Yue J, et al. Thrombolytic therapy for pulmonary embolism. *Cochrane Database Syst Rev*. 2018;12:CD004437.
41. Robertson L, Jones LE. Fixed dose subcutaneous low molecular weight heparins versus adjusted dose unfractionated heparin for the initial treatment of venous thromboembolism. *Cochrane Database Syst Rev*. 2017; 2:CD001100.
42. Bain E, Wilson A, Tooher R, et al. Prophylaxis for venous thromboembolic disease in pregnancy and the early postnatal period. *Cochrane Database Syst Rev*. 2014;(2):CD001689.
43. Che Yaakob CA, Dzarr AA, Ismail AA, et al. Anticoagulant therapy for deep vein thrombosis (DVT) in pregnancy. *Cochrane Database Syst Rev*. 2010;(6): CD007801.
44. Laurent P, Dussarat GV, Bonal J, et al. Low molecular weight heparins: a guide to their optimum use in pregnancy. *Drugs*. 2002;62(3):463-477.
45. Garcia DA, Baglin TP, Weitz JI, Samama MM. Parenteral anticoagulants: antithrombotic therapy and prevention of thrombosis, 9th ed: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines. *Chest.* 2012;141(2 Suppl):e24S-e43S.
46. McColl MD, Greer IA. Low-molecular-weight heparin for the prevention and treatment of venous thromboembolism in pregnancy. *Curr Opin Pulm Med*. 2004;10(5): 371-375.
47. Gouin-Thibault I, Pautas E, Siguret V. Safety profile of different low-molecular weight heparins used at therapeutic dose. *Drug Saf*. 2005;28(4):333-349.
48. Greer IA, Nelson-Piercy C. Low-molecular-weight heparins for thromboprophylaxis and treatment of venous thromboembolism in pregnancy: a systematic review of safety and efficacy. *Blood*. 2005;106(2):401-407.
49. Richter C, Huch A, Huch R. Transfer of low molecular weight heparin during breast feeding. *Thromb Haemost*. 1997;78(suppl):734.
50. Richter C, Sitzmann J, Lang P, et al. Excretion of low molecular weight heparin in human milk. *Br J Clin Pharmacol*. 2001;52(6):708-710.
51. Steinberg ZL, Dominguez-Islas CP, Otto CM, et al. Maternal and Fetal Outcomes of Anticoagulation in Pregnant Women With Mechanical Heart Valves. *J Am Coll Cardiol.* 2017;69(22):2681-2691.
52. Orme ML, Lewis PJ, de Swiet M, et al. May mothers given warfarin breast-feed their infants? *BMJ*. 1977; 1(6076):1564-1565.
53. McKenna R, Cole ER, Vasan U. Is warfarin sodium contraindicated in the lactating mother? *J Pediatr*. 1983; 103(2):325-327.
54. Robertson L, Kesteven P, McCaslin JE. Oral direct thrombin inhibitors or oral factor Xa inhibitors for the treatment of deep vein thrombosis. *Cochrane Database Syst Rev*. 2015;(6):CD010956.
55. Robertson L, Kesteven P, McCaslin JE. Oral direct thrombin inhibitors or oral factor Xa inhibitors for the treatment of pulmonary embolism. *Cochrane Database Syst Rev*. 2015;(12):CD010957.
56. Chan WS, Rey E, Kent NE, et al; VTE in Pregnancy Guideline Working Group. Society of Obstetricians and Gynecologists of Canada. Venous thromboembolism and antithrombotic therapy in pregnancy. *J Obstet Gynaecol Can*. 2014;36(6):527-553.
57. Knol HM, Schultinge L, Erwich JJ, Meijer K. Fondaparinux as an alternative anticoagulant therapy during pregnancy. *J Thromb Haemost*. 2010;8(8): 1876-1879.
58. Bhutia S, Wong PF. Once versus twice daily low molecular weight heparin for the initial treatment of venous thromboembolism. *Cochrane Database Syst Rev*. 2013; (7):CD003074.
59. Voke J, Keidan J, Pavord S, et al; British Society for Haematology Obstetric Haematology Group. The management of antenatal venous thromboembolism in the UK and Ireland: a prospective multicentre observational survey. *Br J Haematol*. 2007;139(4):545-558.
60. Ní Ainle F, Wong A, Appleby N, et al. Efficacy and safety of once daily low molecular weight heparin (tinzaparin sodium) in high risk pregnancy. *Blood Coagul Fibrinolysis.* 2008;19(7):689-692.
61. Shiach CR. Monitoring of low molecular weight heparin in pregnancy. *Hematology.* 2003;8(1):47-52.
62. Linkins LA, Dans AL, Moores LK. Treatment and prevention of heparin-induced thrombocytopenia: antithrombotic therapy and prevention of thrombosis, 9th ed: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines. *Chest.* 2012;141(2 Suppl):e495S-e530S.
63. Wigle P, Hein B, Bernheisel C. Anticoagulation: updated guidelines for outpatient management. *Am Fam Physician*. 2019;100(7):426-434.
64. Hull RD, Raskob GE, Rosenbloom D, et al. Heparin for 5 days as compared with 10 days in the initial treatment of proximal venous thrombosis. *N Engl J Med*. 1990; 322(18):1260-1264.
65. Ageno W, Steidl L, Ultori C, et al. The initial phase of oral anticoagulation with warfarin in outpatients with deep venous thrombosis. *Blood Coagul Fibrinolysis*. 2003;14(1):11-14.

66. Leffert L, Butwick A, Carvalho B, et al. members of the SOAP VTE Taskforce. The Society for Obstetric Anesthesia and Perinatology Consensus Statement on the Anesthetic Management of Pregnant and Postpartum Women Receiving Thromboprophylaxis or Higher Dose Anticoagulants. *Anesth Analg*. 2018;126(3):928-944.

67. Horlocker TT, Vandermeuelen E, Kopp SL, et al. Regional anesthesia in the patient receiving antithrombotic or thrombolytic therapy: American Society of Regional Anesthesia and Pain Medicine Evidence-Based Guidelines (Fourth Edition). *Reg Anesth Pain Med*. 2018;43(3):263-309. Erratum in *Reg Anesth Pain Med*. 2018;43(5):566.

68. Pettilä V, Leinonen P, Markkola A, et al. Postpartum bone mineral density in women treated for thromboprophylaxis with unfractionated heparin or LMW heparin. *Thromb Haemost*. 2002;87(2):182-186.

69. U.S. Preventive Services Task Force. Final Recommendation Statement: low-dose aspirin use for the prevention of morbidity and mortality from preeclampsia: preventive medication. 2014. Available at https://www.uspreventiveservicestaskforce.org/uspstf/document/RecommendationStatementFinal/low-dose-aspirin-use-for-the-prevention-of-morbidity-and-mortality-from-preeclampsia-preventive-medication.

70. De Stefano V, Grandone E, Martinelli I. Recommendations for prophylaxis of pregnancy-related venous thromboembolism in carriers of inherited thrombophilia. Comment on the 2012 ACCP guidelines. *J Thromb Haemost*. 2013;11(9):1779-1781; Epub ahead of print.

71. Bates SM, Greer IA, Middeldorp S, et al. Recommendations for prophylaxis of pregnancy-related venous thromboembolism in carriers of inherited thrombophilia. Comment on the 2012 ACCP guidelines: a rebuttal. *J Thromb Haemost*. 2013;11(9):1782-1784; Epub ahead of print.

72. Croles FN, Nasserinejad K, Duvekot JJ, et al. Pregnancy, thrombophilia, and the risk of a first venous thrombosis: systematic review and bayesian meta-analysis. *BMJ*. 2017;359:j4452.

73. Seshadri N, Goldhaber SZ, Elkayam U, et al. The clinical challenge of bridging anticoagulation with low-molecular-weight heparin in patients with mechanical prosthetic heart valves: an evidence-based comparative review focusing on anticoagulation options in pregnant and nonpregnant patients. *Am Heart J*. 2005;150(1):27-34.

74. Clark SL, Christmas JT, Frye DR, et al. Maternal mortality in the United States: predictability and the impact of protocols on fatal postcesarean pulmonary embolism and hypertension-related intracranial hemorrhage. *Am J Obstet Gynecol*. 2014;211(1):32.e1-32.e9.

75. Palmerola KL, D'Alton ME, Brock CO, Friedman AM. A comparison of recommendations for pharmacologic thromboembolism prophylaxis after caesarean delivery from three major guidelines. *BJOG*. 2016;123(13):2157-2162.

76. Sibai BM, Rouse DJ. Pharmacologic thromboprophylaxis in obstetrics: broader use demands better data. *Obstet Gynecol*. 2016;128(4):681-684.

77. Kotaska A. Postpartum venous thromboembolism prophylaxis may cause more harm than benefit: a critical analysis of international guidelines through an evidence-based lens. *BJOG*. 2018;125(9):1109-1116.

78. Robison E, Heyborne K, Allshouse AA, et al. Implementation of a risk-based heparin protocol for postpartum venous thromboembolism prevention. *Obstet Gynecol*. 2017;130(2):262-269.

79. D'Alton ME. Friedman AM, Smiley RM, et al. National Partnership for Maternal Safety: consensus bundle on venous thromboembolism. *Obstet Gynecol*. 2016;128(4):688-698.

80. Hameed A, Friedman A, Peterson N, et al. The Maternal Venous Thromboembolism Task Force, California Maternal Quality Care Collaborative, Maternal, Child and Adolescent Health Division, Center for Family Health, California Department of Public Health. Improving health care response to maternal venous thromboembolism. (California Maternal Quality Care Collaborative Toolkit to Transform Maternity Care). 2018. Available at https://www.cdph.ca.gov/Programs/CFH/DMCAH/RPPC/CDPH%20Document%20Library/CMQCC_VTE_Toolkit.pdf.

Maternal Sepsis

Learning Objectives

1. Describe prevention strategies for maternal sepsis.
2. Explain the warning signs of sepsis during the perinatal period, and the initial guideline-based diagnosis and management strategies.
3. Identify the evidence-based management of maternal septic shock.

Introduction

Infection, or sepsis, was the third leading cause of maternal mortality (12.8%) in the United States from 2011-2014.[1] A 2017 World Health Organization (WHO) Statement on Maternal Sepsis acknowledges that, in the past 15 years, global health efforts to reduce maternal mortality have focused primarily on the top two causes of maternal mortality: postpartum hemorrhage and hypertensive disorders of pregnancy. The statement calls for improved prevention and treatment of maternal sepsis, which is also the third leading cause of direct maternal mortality internationally.[2] The onset of sepsis in pregnancy can be insidious before rapid deterioration with the onset of septic shock, multiple organ dysfunction, or death.[3] Outcomes for pregnant women with sepsis or septic shock in pregnancy can be improved by early detection and identification of the source of infection, and administration of appropriate therapy.[4]

The WHO in 2011 proposed *near miss* criteria, which include several criteria for organ dysfunction and the diagnosis of sepsis.[5] A high suspicion for and early detection of severe infection are critical in managing sepsis, and mandated guidelines may improve sepsis care.[6] This chapter reviews the most recent evidence and recommends a practical approach to managing maternal sepsis.

Definitions

The 2016 Sepsis-3 guidelines define sepsis as a "life-threatening organ dysfunction caused by a dysregulated host response to infection. Septic shock is a subset of sepsis with circulatory and cellular/metabolic dysfunction associated with a higher risk of mortality."[7]

This classification replaced 2001 concepts of systemic inflammatory response syndrome (SIRS) and severe sepsis (*Table 1*).[8] These criteria were developed for a nonpregnant population, to predict mortality and morbidity and determine if referral to a higher-level facility and intensive care unit (ICU) admission are needed. In 2017, the WHO defined maternal sepsis as "a life-threatening condition defined as organ dysfunction resulting from infection during pregnancy, childbirth, postabortion, or postpartum period."[2]

Most of the evidence regarding maternal sepsis is based on small retrospective studies. Accurate identification of pregnant women at risk of deterioration is difficult, because of physiologic changes and the relatively low incidence of sepsis and septic shock in pregnancy. A meta-analysis and systematic review of 87 studies involving 8,834 women found that SIRS criteria often overlap with normal physiologic changes of pregnancy leading some experts to recommend alternative criteria for the diagnosis of maternal sepsis.[9] An alternative new definition is two standard deviations above the mean for temperature, respiratory rate, and heart rate (38.1° C [100.6° F], 25 breaths per minute, and 107 beats per minute (BPM), respectively) might help in clinical decisions. These findings are consistent with the definition of puerperal fever as greater than 38° C (100.4° F), suggesting that fever greater than 38° C (100.4° F) persisting more than 1 hour warrants evaluation and appropriate intervention. Fever is present in 95% to 100% of cases of intraamniotic infection (chorioamnionitis) and other uterine infections.[9]

Disease Burden

Sepsis causes approximately 11% of maternal deaths.[10] Of an estimated 44,999,260 US hospitalizations for delivery between 1998 and 2008, sepsis was diagnosed in 1:3,333 deliveries (95% confidence interval [CI] = 1:3,151-1:3,540), severe sepsis in 1:10,823 deliveries (95% CI = 1:10,000-1:11,792), and sepsis-related death in 1:105,263 deliveries (95% CI = 1:83,333-1:131,579). During this period, the incidence of sepsis was stable (P = 0.95), but the risk of severe sepsis and

Table 1. Comparison of Sepsis-2 and Sepsis-3 Definitions

Criteria definition	Sepsis-2 (1991 and 2001 consensus terminology)	Sepsis-3 (2016 Definition)
SIRS	2 or more of the following: Temperature >38°C or <36°C Heart rate >90/minutes Respiratory rate >20/minutes or $PaCO_2$ <32 mm Hg (4.3 kPa) White blood cell count >12,000/mm^3 or <4,000/mm^3 or >10% immature bands	Category not used
Sepsis	Defined as 2 or more SIRS criteria in a suspected infection	Present. Suspected or documented infection and an acute increase of ≥2 SOFA points
Severe sepsis and sepsis induced hypotension	Present. Sepsis related organ dysfunction or hypoperfusion	Category not used
Septic shock	Present. Sepsis with persisting hypotension requiring vasopressors to maintain MAP 65 mm Hg and having a serum lactate level >36 mg/dL (4 mmol/L) despite adequate volume resuscitation	Present. Sepsis with persisting hypotension requiring vasopressors to maintain MAP 65 mm Hg and having a serum lactate level >18 mg/dL (2 mmol/L) despite adequatevolume resuscitation

MAP = mean arterial pressure; SIRS = systemic inflammatory response syndrome; SOFA = Sequential Organ Failure Assessment.

Information from Seymour CW, Liu VX, Iwashyna TJ, et al. Assessment of clinical criteria for sepsis: for the Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3). JAMA. *2016;315(8):762-774; Poutsiaka D, Porto M, Perry W, et al. Prospective observational study comparing Sepsis-2 and Sepsis-3 definitions in predicting mortality in critically ill patients.* Open Forum Infect Dis. *2019;6(7):ofz271.*

sepsis-related death increased (P <0.001 and P = 0.02, respectively).[11] Sepsis has been reported as a cause of 6% to 22% of maternal ICU admissions during pregnancy or the postpartum period. It is also associated with preterm delivery.[3,12]

Prevention

Sepsis prevention activities can occur during and after pregnancy including during labor and delivery.

During Pregnancy

Causes of sepsis during pregnancy include pneumonia, influenza, and urinary tract infections (UTIs). Strategies to prevent pyelonephritis in pregnancy may help prevent some cases of maternal sepsis.[13] No Cochrane review or randomized controlled trial (RCT) directly studies this topic. Up to 40% of untreated asymptomatic bacteriuria in pregnancy leads to pyelonephritis and pyelonephritis can lead to maternal sepsis.[13] Asymptomatic bacteria is associated with other adverse pregnancy outcomes including preterm labor and intrauterine growth restriction (IUGR). A urine culture is recommended at the first prenatal visit and penicillins, cephalosporins, and macrolides are first-line treatment. Poorly designed studies have raised concerns about teratogenicity of nitrofurantoin in first trimester and it may be best to choose another antibiotic for first trimester UTI when possible.[14]

During Delivery

Prevention of sepsis in all settings involves good infection control, including hand hygiene. Hand washing by providers before examining women in labor decreased the maternal mortality rate from 18% to less than 3% in one hospital and from 10% to less than 1% in two other hospitals.[15] When hands are not visibly unclean, alcohol-based hand rubs take less time, kill more bacteria, and are less irritating to the hand than soap and water.[15] A 2014 Cochrane review found that existing evidence is insufficient to determine whether use of vaginal chlorhexidine-based products in labor prevents maternal or neonatal infections.[16]

Careful attention to infection prevention can prevent most postpartum sepsis, especially during cesarean delivery. The use of prophylactic antibiotics in women undergoing cesarean delivery reduced the incidence of wound infection (RR 0.40; 95% CI = 0.35-0.46), endometritis (RR 0.38; 95% CI = 0.34-0.42), and serious infectious maternal complication (RR 0.31; 95% CI = 0.20-0.49).[17] A

Cochrane review found that antibiotic prophylaxis for cesarean delivery administered before skin incision rather than after cord clamping decreased the incidence of postpartum endometritis (RR 0.54; 95% CI = 0.36-0.79) and total infectious morbidities (RR 0.57; 95% CI = 0.45-0.72).[18]

The 2018 American College of Obstetricians and Gynecologists (ACOG) guidelines recommend that antibiotic prophylaxis be administered within 60 minutes before the start of the cesarean delivery, or as soon as possible with emergent delivery, with a single intravenous (IV) dose of a first-generation cephalosporin or a combination of clindamycin with gentamicin in women with penicillin allergy.[19] However, a 2016 Cochrane review found insufficient evidence to assess the benefits and harms of different routes of prophylactic antibiotics administered for preventing infectious morbidity in women undergoing cesarean delivery.[20] The addition of azithromycin to standard antibiotic prophylaxis in women undergoing nonelective cesarean delivery who have been in labor or have rupture of membranes (ROM) decreased the composite primary outcome of endometritis, wound infection, or other infections in a 6-week postpartum period.[19,21]

After Delivery

Routine antibiotic prophylaxis after uncomplicated childbirth is controversial, particularly in settings where women are at increased risk of puerperal infectious morbidities. Low-level evidence suggests that routine antibiotic administration after uncomplicated birth reduces the risk of endometritis but does not reduce the incidence of UTI, wound infection, or maternal hospital stay. Cost, severe infectious morbidity, and patient satisfaction were not addressed in these studies.[22] A Cochrane review did not find evidence that antibiotics prevent endometritis after manual removal of the placenta; thus, they are not recommended.[23] This also applies to episiotomy repair after normal vaginal birth.[24] A Cochrane review that only included one RCT of 147 women found a decreased wound complication rate at 2 weeks' postpartum but no difference before hospital discharge or at 6 weeks' postpartum.[25] A postcesarean 48-hour course of oral cephalexin and metronidazole in addition to the standard preoperative cephalosporin prophylaxis for women with obesity showed benefit in one single-center trial. Additional research is needed.[26]

Infections During Pregnancy and Postpartum: Sites That Commonly Lead to Sepsis and Etiologic Organisms

Infectious complications in pregnancy include those related to pregnancy, those unrelated to pregnancy, and those due to nosocomial infections (*Table 2*).

Causes of maternal infection leading to sepsis vary with the state of pregnancy. Sepsis in early pregnancy can be related to spontaneous or elective abortion. In the second and third trimesters, prelabor rupture of membranes (PROM) is associated with an increased risk of intraamniotic infection, or chorioamnionitis. Perineal infec-

Table 2. Common Infections During Pregnancy, Etiologies of Maternal Sepsis

Infection	Common Etiology
Bacterial pneumonia	Pneumococcus Streptococcus A and B *Haemophilus influenzae* *Mycoplasma* *Staphylococcus aureus* *Legionella pneumophila* *Klebsiella pneumoniae* *Pseudomonas aureginosa*
Pyelonephritis	*Escherichia coli* *Klebsiella pneumoniae* *Proteus spp* *Enterobacter*
Chorioamnionitis	*Streptococcus agalactiae* (*Streptococcus B*) *E coli*
Endometritis	*Staphylococcus aureus* Clostridium species
Tissue necrosis	Group A *Streptococcus* (*Streptococcus pyogenes)*
Nonpregnancy-related infections	Malaria Listeriosis Viral hepatitis (E) Varicella Pneumonia Influenza HIV infection Toxoplasmosis Cytomegalovirus

Information from Morgan J, Roberts S. Maternal sepsis. Obstet Gynecol Clin North Am. *2013;40(1):69-87.*

tions, endometritis, wound infections, and mastitis should be considered in the postpartum period.

Infections not related to pregnancy may have an increased incidence in pregnancy. This is true of pyelonephritis and pneumonia, the most common types of infection that lead to sepsis in pregnancy.[11] Every woman with sepsis should undergo urine analysis and culture and chest x-ray.[4] The majority of women of reproductive age are healthy without chronic comorbidities. However, women with a predisposing factor such as HIV, comorbid disease, or immunosuppressed status are predisposed to infection.

Hospital-acquired infections can result from prolonged stays, mechanical ventilation, the insertion of venous and arterial lines and decreased mobility.[27] Urinary catheters raise the risk of UTI and sepsis and should only be used when needed.[28]

Postpartum Endometritis

Postpartum endometritis includes infections of the endometrium, the myometrium, and the parametrium.[29] The severity is consistent with the depth in the uterine wall; however, all uterine infections should be considered serious infections. Most of these infections are secondary to *Enterobacteriaceae* (50%), gram-negative cocci (45%), and/or anaerobes (23%).[30] Infections caused by *Streptococcus pyogenes* (group A *Streptococcus*) or *Streptococcus agalactiae* (group B *Streptococcus* [GBS]) are associated with significant morbidity and mortality.[31] Women who develop postpartum endometritis can also develop an infection of the surgical wound.

Epidemiology

The most important risk factor for postpartum maternal infection is cesarean delivery.[17] Women undergoing primary cesarean delivery with a trial of labor have a 21.2-fold greater risk of endometritis (95% CI = 15.4-29.1) compared with spontaneous vaginal delivery. Women undergoing primary cesarean delivery without a trial of labor have a 10.3 times greater likelihood of endometritis (95% CI = 5.9-17.9) compared with spontaneous vaginal delivery.[32] After ROM occurs, vaginal bacteria can ascend to the uterus. Prolonged ROM doubles the risk of endometritis.[32] In some women, ROM may not be obvious, and it may appear that infection preceded rupture when a subtle rupture occurred first. Risk factors associated with the development of postpartum endometritis are:

- Prolonged labor with ROM
- Lack of prenatal care
- Cesarean delivery after prolonged labor with ROM
- Cesarean delivery in a woman with a body mass index of 25 kg/m^2 or greater
- Multiple vaginal examinations after ROM. However, after adjusting for spontaneous labor, the Bishop score, and ROM on admission, the number of digital examinations has not resulted in a significant increase in infection.

Clinical Presentation and Diagnosis

Postpartum endometritis can occur immediately after or several days after delivery. The timing for development depends on:

- When the infectious process started
- The duration of delivery in the presence of ROM
- The status of the endogenous microflora at the time of infection.

Colonization with bacteria such as *S agalactiae, S pyogenes*, and *Escherichia coli*, as well as other gram-negative facultative anaerobic bacteria, creates a significant risk of developing postpartum endometritis for women in labor, especially those requiring cesarean delivery.[33] The clinical signs of endometritis include:

- Fever (38° C [100.4° F] or greater)[9]
- Uterine tenderness
- Purulent vaginal discharge
- Findings associated with advanced endometritis (ie, pelvic abscess, peritonitis, blood clots, sepsis, death)[34]

A common mnemonic, the Rule of W has been updated to describe the most common causes of postoperative complications: waves (ie, electrocardiogram waves), wind (pneumonia), water (urinary tract), wound (superficial surgical site infection and deep superficial surgical site infection), and walking (venous thromboembolism). In the postcesarean delivery setting, consider postoperative/postpartum fever or womb (for endometritis) as the first most common cause.[35]

- Women with suspected puerperal sepsis should undergo evaluation
- A complete review of current drugs, especially those with serotonergic activity (ie, tramadol, metoclopramide, ondansetron)

- A complete physical examination. Determine if there are physical findings of infection (ie, pneumonia, pyelonephritis, endometritis, infection of the surgical wound)
- Complete blood count
- Pelvic examination and cultures. The importance of endometrial cultures is limited, because the majority of women benefit from empirical antibiotic treatment. Further, it is difficult to obtain an endometrial culture without causing contamination. However, it has been proposed that identification of group A *Streptococcus* infection in endometrial and cervical culture requires further examination, including notification of pediatricians and subsequent isolation[36]
- Electrolytes, urea nitrogen, creatinine, and glucose
- Urine analysis and culture
- Arterial blood gases
- Chest x-ray (if there are respiratory symptoms)
- Other imaging studies if indicated.

In women diagnosed with sepsis due to endometritis, obtain blood cultures before starting empiric antibiotic therapy to optimize the chances of pathogen recovery.[37] However, antibiotic administration should not be delayed to obtain blood cultures.[37,38] If the woman has already received antibiotics, it is critical to use a culture media system that effectively neutralizes antibiotics, increasing the chance of pathogen recovery. Two sets of blood cultures should ideally be obtained:[39] 1) an aerobic bottle, allowing preferential growth of aerobic and facultative anaerobic microorganisms, and 2) an anaerobic bottle, allowing preferential growth of strict anaerobic bacteria. These should be evaluated every 12 hours to assess the growth of microorganisms. Blood cultures have not been shown to be of benefit for endometritis without sepsis.[37]

Antibiotic Management

In general, antibiotic selection should be based on local infectious epidemiology and bacterial resistance profile. This is typically a polymicrobial infection that includes facultative and obligate anaerobes. Appropriate antibiotic options include:

- Piperacillin/tazobactam 4.5 g IV every 6 hours. This antibiotic provides excellent coverage for gram-positive and gram-negative facultative anaerobes, as well as gram-positive and gram-negative anaerobes
- Ampicillin/sulbactam 3 g IV every 6 hours, accompanied by gentamicin, 5 mg/kg of body weight every 24 hours
- Clindamycin 900 mg IV every 8 hours, which is active against 80% of GBS, *Staphylococcus aureus* including methicillin-resistant *S aureus* (MRSA), and obligate anaerobes, accompanied by gentamicin 5 mg/kg of body weight every 24 hours, which provides excellent coverage against gram-negative facultative anaerobes and provides activity against MRSA
- Metronidazole, 500 mg every 8 hours, provides good activity against gram-negative facultative anaerobes, accompanied by gentamicin, 5 mg/kg body weight every 24 hours.[40,41]

The combination of clindamycin and gentamicin is usually appropriate for the treatment of endometritis.[34] There is no evidence that any regimen is associated with fewer adverse effects. Following clinical improvement of uncomplicated endometritis that has been managed with IV therapy, additional oral therapy has not been proven to be beneficial.[34] In addition, antibiotic therapy administered early in the infection usually produces a positive response within 48 hours of onset. Possible reasons for failure of antibiotic therapy include:

- Antibiotic resistance
- Pelvic abscess that requires surgical drainage
- Inappropriate antibiotic dosage
- Late initiation of antibiotic therapy
- Misdiagnosis
- Deep venous thrombosis
- Septic pelvic venous thrombosis
- Thrombosis of the myometrial microvasculature
- Myometrial necrosis
- Drug-induced fever.

If culture results identify the causative bacteria the antibiotic management can be narrowed. Persistent fever may suggest complications: an enlarged and painful uterus suggests myometrial microabscesses, and subcutaneous gas or gas in the uterine walls on x-ray suggests gas gangrene. In these situations, surgical intervention including hysterectomy may be necessary.

Risk factors for septic pelvic thrombophlebitis (SPT) include chorioamnionitis, hypertensive disorders of pregnancy, and cesarean delivery.[42] A high level of suspicion for SPT is warranted in women with persistent, unexplained postpartum fever despite antibiotic therapy for presumed

endometritis. The diagnosis usually can be confirmed by magnetic resonance venography with contrast or pelvic computed tomography scan. The latter has a sensitivity of 78% to 100% and specificity of 63% to 99% for SPT.[43] Unfractionated heparin or low-molecular-weight heparin (ie, enoxaparin) has been proposed in addition to antibiotics to prevent further thrombosis and reduce the spread of septic emboli.[44] The use of heparin for SPT is controversial.[45]

Septic Abortion

The WHO defines unsafe abortion as a procedure to interrupt an unwanted pregnancy that is performed by individuals without the necessary skills or in an environment that does not meet minimum medical standards, or both.[46] It is estimated that unsafe abortion accounts for approximately 13% of maternal deaths worldwide secondary to septic shock with multiple organ failure with or without hemorrhage.[47] Where abortion is illegal, maternal mortality is often high. For example, in Uruguay, where most abortions have been illegal since 1939, unsafe abortion accounted for 28% of maternal deaths from 1995-1999. From 1996-2000, unsafe abortion accounted for 47% of maternal deaths at one large referral hospital.[48]

Sepsis following abortion is usually caused by ascending infection producing endometritis or parametritis, and may occur after a spontaneous abortion, surgical abortion, or unsafe abortion. Although the incidence has decreased with the legalization of abortion in many countries, septic abortion accounts for half of maternal deaths in other countries.[49] Women at highest risk are those with advanced gestation, retention of products of conception, and trauma. Most septic shock and related mortalities are due to a delay in medical care.

Clinical Criteria

Signs and symptoms of sepsis include fever, abdominal pain, vaginal bleeding, purulent discharge, and tenderness of the uterus and adnexa. Symptoms of peritonitis may indicate uterine perforation.[3]

Laboratory Criteria

Diagnosis of septic abortion is clinical, and cultures are usually not indicated. The infection is polymicrobial including vaginal, enteric, and sometimes sexually transmitted pathogens. *Clostridium perfringens* infection can produce gas gangrene, and concomitantly *Clostridium tetani* infection should be considered. *Clostridium sordellii* can be detected by polymerase chain reaction assay.[50]

Antibiotic Management

Management of septic abortion requires early therapy with broad-spectrum antibiotics (eg, ampicillin, gentamycin, clindamycin) and evacuation of the uterus. Ultrasound can be used to document retained products of conception. More aggressive surgical intervention may be required if the woman does not benefit from initial therapy. Urologic or bowel injury must be identified and addressed if present.

Women with established infection, as indicated by fever (ie, temperature of 38° C [100.4° F] or greater), pelvic peritonitis, or tachycardia should be hospitalized for parenteral antibiotic treatment and prompt uterine evacuation. Bacteremia is more common in septic abortion than in other pelvic infections. This can result in septic shock and adult respiratory distress syndrome (ARDS). Management of severe sepsis requires infection eradication and supportive care of the cardiovascular system and other organs. The WHO recommends broad-spectrum antibiotics followed by oral doxycycline 200 mg/day for 10 to 14 days.[51] *C sordellii* has been confirmed in a few cases of maternal sepsis and death following medical abortion; it is uncertain whether doxycycline prophylaxis is effective in preventing *C sordellii* sepsis.[50]

In countries with legalized abortion, a decrease is seen in the maternal mortality rate and ICU admissions because of a decrease in septic abortion. Manual vacuum aspiration and misoprostol are other important therapies to prevent septic abortion.[52]

Intraamniotic Infection

Intraamniotic infection, or chorioamnionitis, involves the amniotic fluid, the membranes, and the placenta and usually occurs in the setting of ROM and/or labor. Risk factors include low parity, multiple digital examinations, use of internal uterine and fetal monitors, and meconium-stained amniotic fluid.[53]

Clinical Criteria

Usually a clinical diagnosis is made based on the presence of maternal fever and fetal tachycar-

dia and there is no need for uterine cultures or bloodwork.

Historically, chorioamnionitis has been defined as maternal fever of 38° C (100.4° F) accompanied by any of these criteria: maternal leukocytosis (15,000 cell/mm^3 or greater), maternal tachycardia (100 BPM or greater), fetal tachycardia (160 BPM or greater), uterine tenderness, and foul-smelling amniotic fluid.

If the temperature is 38° C (100.4° F) or greater but less than 39° C (102.2° F), it should be remeasured in 30 minutes for confirmation. A repeat temperature of 38°C (100.4° F) or greater constitutes a documented fever. The clinical criteria for chorioamnionitis (Triple I) includes the presence of one or more of the following:

- Fetal tachycardia (greater than 160 BPM for 10 minutes or longer)
- Maternal white blood cell count greater than 15,000 mm^3 in the absence of corticosteroid use
- Purulent fluid from the cervical os (cloudy or yellowish thick discharge confirmed visually).

Isolated maternal fever is a temperature of 38° C (100.4° F) and less than 39° C (102.2° F) without other clinical risk factors (ie, fetal tachycardia, leukocytosis, purulent fluid) regardless of whether the temperature is sustained.[54] Intrauterine infections can be found histologically in 20% of term pregnancies and 50% of preterm deliveries. However, clinical infection occurs in 1% to 2% of term pregnancies and 5% to 10% of preterm deliveries.[54]

The infection is typically ascending, followed by PROM with the entry of organisms from the vaginal or intestinal flora. GBS and organisms from sexually transmitted infections may be involved. Rarely, chorioamnionitis is due to hematogenous spread (eg, *L monocytogenes*). The infection is typically polymicrobial.[53]

The early administration of antibiotics for chorioamnionitis can prevent maternal sepsis and neonatal bacteremia. Bacteremia occurs in approximately 10% of cases, especially in group B streptococcal and *E coli* infections.[33]

Isolated maternal fever is a temperature of 38° C (100.4° F) or greater and less than 39° C (102.2° F) with no other risk factors and with or without persistent temperature elevation.[53]

Antibiotic Management

Management of ROM before labor is dependent primarily on gestational age (see the *Preterm Labor and Prelabor Rupture of Membranes* chapter). GBS prophylaxis should be administered if indicated based on Centers for Disease Control and Prevention (CDC) guidelines.[55]

Intraamniotic infection can cause significant maternal morbidity, including dysfunctional labor, postpartum uterine atony with hemorrhage, endometritis, peritonitis, sepsis, ARDS, and death.[56] Expeditious delivery is warranted; however, intraamniotic infection is not an indication for cesarean delivery, which should be reserved for the usual obstetric indications.[57] Antibiotics for intraamniotic infection decrease rates of sepsis and other adverse birth outcomes.[53] Common regimens for treatment of intraamniotic infection are ampicillin accompanied by gentamicin or a third-generation cephalosporin.[54] A short course of antibiotics ending 24 hours after delivery may be adequate because delivery provides control of the infection source.[58] In cases of persistent fever or sepsis after delivery, antibiotics should be continued with the diagnosis changing to endometritis or sepsis. Continued fever despite antibiotic treatment can indicate complications including necrotizing myometritis and pelvic abscess.

Pyelonephritis

Acute pyelonephritis occurs in 0.5% of pregnancies based on a large retrospective cohort.[59] It is a common cause of maternal sepsis,[59] and the most frequent microorganism is *E coli*. All women who are pregnant should be screened for asymptomatic bacteriuria because antimicrobial treatment of asymptomatic bacteriuria during pregnancy decreases the risk of pyelonephritis (average RR 0.23; 95% CI = 0.13-0.41; 11 studies, 1,932 women; very low-quality evidence), low birthweight neonates (average RR 0.64; 95% CI = 0.45-0.93; six studies, 1,437 neonates; low-quality evidence) and preterm birth (RR 0.27; 95% CI = 0.11-0.62; two studies, 242 women; low-quality evidence).[60] There is insufficient evidence for the use of prophylactic antibiotics to prevent recurrent infections after pyelonephritis or recurrent UTI.[61]

Factors that predispose to pyelonephritis in pregnancy include progesterone-mediated ureteral dilatation secondary to smooth muscle relaxation and compression of the ureters by the gravid uterus. In addition to *E coli*, other bacteria involved include *Klebsiella spp, Enterobacter spp,*

Pseudomonas aeruginosa, Serratia spp, and *Citrobacter spp*, and anaerobes such as *Clostridium, Bacteroides spp*, and *Actinomyces spp*. [59]

Clinical Criteria

Symptoms of pyelonephritis include fever, chills, and systemic symptoms, which require hospitalization because of maternal and fetal risks. Other signs and symptoms include flank pain, especially at the costovertebral angle, and nausea and vomiting. Continuous monitoring of the woman and fetus while administering antibiotics are the cornerstones of treatment.[62] Monitor for preterm labor, which can be caused by pyelonephritis. Compared with women without pyelonephritis who are pregnant, women with pyelonephritis who are pregnant are 18 times as likely to have pneumonia, 11 times as likely to have pulmonary edema, and 11 times as likely to have ARDS.[63]

Laboratory Criteria

Urine culture is the diagnostic test of choice; however, this should not delay early treatment. A urine analysis suggestive of infection is sufficient for initiating treatment. Urine culture is especially useful in women with complicated, recurrent UTI or pyelonephritis. It is also advisable to obtain a urine culture in women with a high pretest probability of UTI, but with a negative result on urine dipstick or microscopy.

There is some debate about the definition of a positive culture. The traditional definition is 100,000 colony-forming units (CFU)/mL with a clean catch urine, or 10^3 CFU/mL or greater in patients with an indwelling urethral catheter;[64] this provides high specificity and low sensitivity. The degree of bacteriuria, the sampling method, and the patient's symptoms must be considered when interpreting the results of a urine culture.

Imaging is not required for the diagnosis of pyelonephritis but should be obtained if there is concern of urolithiasis or renal abscess.

Antibiotic Management

Acute UTI should be managed with empirical antibiotic therapy. Management may vary based on infection site (upper versus lower UTI) and gestational age. There are insufficient data to determine the best drug regimen for pyelonephritis during pregnancy. Options include ampicillin accompanied by gentamicin, or a single agent such as cefazolin. These regimens usually are effective within the first 72 hours.[65,66]

Cystitis, or asymptomatic bacteriuria, can be treated with cephalexin, ampicillin, or nitrofurantoin.[14] However, a 2009 population-based case control study found that nitrofurantoin administered during the first trimester was associated with an increased risk of major birth defects including anophthalmia (or microphthalmos), hypoplastic left heart syndrome, atrial septal defects, and cleft lip with cleft palate. This study also implicated trimethoprim/sulfamethoxazole. In late pregnancy, nitrofurantoin and trimethoprim/sulfamethoxazole may cause related hyperbilirubinemia in the newborn. Quinolones and tetracyclines are contraindicated in pregnancy because of tertogenicity.[67]

When assessing for pyelonephritis, consider urolithiasis. This combination can have devastating adverse effects for the woman. Urolithiasis in pregnancy can be managed conservatively; however, invasive treatment may be necessary in cases of persistent pain, fever not responding to antibiotics, worsening renal function or signs of sepsis. Urinary deviation can be performed with a double J stent or placement of percutaneous nephrostomy under ultrasound guidance without risk of radiation.[68] Timely decompression of the kidney will preserve kidney function and help control sepsis. Pyonephrosis is the combination of infection and obstruction (pus under pressure) with a collection of purulent material trapped in the renal collecting system by a stone (calculus), mass or other obstruction. In these situations, in addition to antimicrobial therapy and supportive treatment for sepsis, consultation with interventional or urologic radiology for emergent percutaneous nephrostomy tube or the placement of a ureteral stent may be indicated.[69]

Perineal abscess can manifest as fever and flank pain especially if persistent, or it can be diagnosed incidentally. Accumulation of purulent material around the kidneys occurs frequently secondary to urosepsis of the upper tract, classically in association with urolithiasis. For perirenal abscess or intrarenal abscess greater than 3 cm, percutaneous drainage is widely recommended following medical stabilization and antibiotic therapy. However, many smaller abscesses also require drainage.[70]

Pneumonia

Pneumonia affects 0.5 to 1.5 pregnant women per 1,000, and it is the most common non-

obstetric cause of maternal mortality in the United States.[71] Morbidity and mortality from pneumonia are higher in women who are pregnant because of decreased residual lung volume, increased oxygen requirements, and decreased esophageal sphincter tone.[33]

Women with pneumonia who are pregnant have an increased risk of preterm delivery, cesarean delivery, preeclampsia/eclampsia, and infants with low birth weight and lower APGAR scores.[71]

Clinical Criteria

Pneumonia may manifest as cough, fever, and shortness of breath. In the presence of significant respiratory symptoms, document oxygen saturation. When indicated, obtain a chest x-ray. The woman's abdomen can be shielded to minimize radiation exposure. When considering a diagnosis of pneumonia, include pulmonary embolism in the differential diagnosis.

Laboratory Criteria

Over 90% of community acquired pneumonia in pregnancy is bacterial.[71] The most common organisms are *Streptococcus pneumonia*, *Haemophilus influenzae*, *C pneumoniae*, *Mycoplasma pneumonia*, and *Legionella pneumophila*.[71]

Viral pneumonia is more common and can be more severe in pregnancy. Influenza can be more severe in pregnancy with morbidity and mortality from primary influenza virus infection and potential for secondary pneumonia from bacterial superinfection. Pneumonia develops in 10% to 20% of women with varicella in pregnancy, which often requires ICU care.[72]

Antibiotic Management

Community acquired pneumonia usually responds well to macrolides. For severe pneumonia requiring hospitalization and leading to sepsis, a second- or third-generation cephalosporin should be added.[33]

Influenza can be prevented by vaccination, which ACOG and the CDC recommend administering during any trimester.[73] Both organizations also recommend postexposure prophylaxis with oseltamivir (Tamiflu) 75 mg/day for 10 days.[73] Women with influenza who are pregnant should receive oseltamivir 75 mg twice/day for 5 days in addition to supportive care. If oseltamivir is not available, zanamivir can be used instead.[73]

A Practical Approach to Managing Maternal Sepsis

Clinical care bundles have been proposed to simplify sepsis management and standardize specific actions within limits of 0, 1, 3, 6, and 12 hours *(Figure 1)*.[74] This approach allows the development of a shared mental model.[75] Implemented as a group, these actions may have synergistic beneficial effects on outcomes.[74]

Sepsis Hour 0 Bundle

The Hour 0 Bundle includes two fundamental actions: early recognition and risk stratification. Consider the diagnosis of infection using the clinical criteria for the most prevalent infectious pathologies in pregnancy (*Table 2*). Obtain an adequate history, including an evaluation of signs and symptoms to clarify the source of infection and appropriate treatment.

For risk stratification, consider that maternal physiology includes substantial changes in hemodynamics, respiratory function, and renal function. These changes influence blood loss during delivery; development of infections such as chorioamnionitis, endometritis, pneumonia, and pyelonephritis; need for IV fluids, drug dosages, mode of delivery, and anesthesia. Maternal physiology also affects vital signs and laboratory parameters, making the diagnosis of sepsis and septic shock more difficult in women who are pregnant.[76]

Risk stratification criteria are also incorporated into the sepsis definition. The two most commonly used are SIRS criteria and Quick Sequential

Figure 1. Shared Mental Model for Sepsis Approach (Mnemonic 0,1,3,6,12)

Organ Failure Assessment (qSOFA) and Sequential Organ Failure Assessment (SOFA) scores.

SIRS criteria are defined as the presence of two or more of the following:[74]

- Temperature greater than 38° C (100.4° F) or less than 36° C (96.8° F)
- Heart rate greater than 90 BPM
- Respiratory rate greater than 20 breaths per minute or $PaCO_2$ less than 32 mm Hg
- White blood cell count greater than 12,000/mm^3 or less than 4,000/mm^3 or greater than 10% immature bands.

In the Sepsis-3 consensus, the use of the SIRS criteria was considered nonspecific and its use is no longer recommended.[7] In place of the SIRS criteria, a new clinical approach has been proposed for initial screening of patients with infection, known as qSOFA score, including the following:

- Respiratory rate 22 breaths per minute or greater
- Altered mental status
- Systolic blood pressure 100 mm Hg or less.

If two or more of these criteria are present, an evaluation of organ dysfunction is crucial to diagnose sepsis. For this, the full SOFA score is currently the next step in sepsis diagnosis.[76,77] Values of 2 or more in these results define sepsis (*Table 3)*. Although only 24% of patients with infection who are not pregnant have a qSOFA of 2 or 3, they account for 70% of deaths because of sepsis.[7]

Despite the changes made in the new consensus proposal, a recent meta-analysis recommended SIRS and qSOFA as acceptable alternatives for patients with sepsis. Moreover, The SIRS was significantly superior in terms of sensitivity to the qSOFA for sepsis diagnosis (RR 1.32; 95% CI = 0.40-2.24), and qSOFA was slightly better in predicting hospital mortality, thus the association of both criteria could provide a better model to initiate or escalate therapy in patients with sepsis.[78]

Sepsis-induced organ dysfunction may be subtle; therefore, it should be considered in any patient presenting with infection. In this setting, it is also mandatory to exclude septic shock (ie, the presence of hypotension plus vasopressor use and/or a lactate level greater than 18 mg/dL [2 mmol/L] after adequate fluid management) in any patient with suspected or confirmed infection. Furthermore, even in the absence of hypotension, elevated lactate levels should be considered evidence of

Table 3. The Sequential Organ Failure Assessment (SOFA) Score

	1	2	3	4
Respiration				
PaO_2/FiO_2 ratio	<400	<300	<200 (with respiratory support)	<100 (with respiratory support)
Coagulation				
Platelets × 103/mm^3	<150	<100	<50	<20
Liver				
Bilirubin, mg/dL	1.2-1.9 (20-32)	2.0-5.9 (33-101)	6.0-11.9 (102-204)	>12 (>204)
Cardiovascular				
Hypotension	MAP <70 mm Hg	Dopamine <5 or dobutamine (any dose)	Dopamine >5 or epinephrine <0.1 or norepinephrine <0.1	Dopamine >15 or epinephrine >0.1 or norepinephrine >0.1
Central nervous system				
Glasgow Coma Score	13-14	10-12	6-9	<6
Renal				
Creatinine, mg/dL or urine output	1.2-1.9 (110-170)	2.0-3.4 (171-299)	3.5-4.9 (300-440) or <500 mL/day	>5.0 (>440) or <200 mL/day

MAP = mean arterial pressure.

Adapted from Vincent JL, Moreno R, Takala J, et al. The SOFA (Sepsis-related Organ Failure Assessment) score to describe organ dysfunction/ failure. On behalf of the Working Group on Sepsis-Related Problems of the European Society of Intensive Care Medicine. Intensive Care Med. *1996;22(7):707-710.*

septic shock.[79] However, serum lactate measurements are commonly but not universally available, especially in low-resource settings.

Early Warning Systems and Applicability in Maternal Sepsis

Modified Early Obstetric Warning System

The Modified Early Obstetric Warning System (MEOWS) is a bedside assessment tool designed to identify women at higher risk of severe complications, in which maternal vital signs (temperature, blood pressure, heart rate, oxygen saturation), level of consciousness, and pain are recorded every 12 hours. It was developed as a response to the urgent need for an early warning system adjusted for the physiological adaptations of pregnancy (see *Table 4*).[53]

The MEOWS was recommended as a bedside screening tool by the 2003–2005 triennial Confidential Enquiry into Maternal and Child Health (CEMACH) report for early recognition of severe obstetric complications and periodic record of physiological parameters.[80,81]

Table 4. Yellow and Red Flag Criteria for Maternal Early Obstetric Warning System (MEOWS)

	Yellow Trigger	Red Trigger
Respiratory rate (breaths/minute)	21-30	<10 or >30
Oxygen saturation (%)		<90
Heart rate (BPM)	100-120 or 30-40	<30 or >120
Systolic blood pressure (mm Hg)	80-90 or 150-160	<80 or >160
Diastolic blood pressure (mm Hg)	80-90	>90
Lochia		Heavy/foul smell
Proteinuria		>+2
Colour of liquor		Green
Nonresponse	Voice	Unresponsive, pain
General condition	Looks unwell	

BPM = beats per minute.

Information from Singh S, McGlennan A, England A, Simons R. A validation study of the CEMACH recommended modified early obstetric warning system (MEOWS). Anaesthesia. *2012;67(1):12-18*

Obstetric Early Warning Score

The Obstetric Early Warning Score (OEWS) is a standardized scoring system for women who are pregnant that was developed to classify the severity of the disease and identify those at risk of clinical decompensation. This tool incorporates a color scheme as well as a numeric measure of illness severity. Color coding is used to provide a visual reminder during identification of abnormal vital signs. Applied on admission, the OEWS score allows for the identification of the risk of death and unscheduled ICU admission.[82] This system has been validated in low-resource settings with a good discrimination capability, especially in women with direct pathologies, including maternal hypertensive disorders; maternal hemorrhage; maternal abortion, miscarriage, and ectopic pregnancy; maternal obstructed labor and uterine rupture; maternal sepsis and other maternal infections (see *Table 5*).[56]

Table 5. Obstetric Early Warning Score (OEWS)

	3	2	1	Normal	1	2	3
Systolic blood pressure, mm Hg	<80	80-90		90-139	140-149	150-159	>160
Diastolic blood pressure, mm Hg				<90	90-99	100-109	>110
Respiratory rate/minute	<10			10-17	18-24	25-29	>30
Heart rate/minute	<60			60-110		111-149	>150
% O_2 required to maintain SpO_2 >96%				Room air	24-39%		>40%
Temperature, degrees C	<34		34-35	35.1-37.9	38-38.9		>39
Conscious level				Alert			Not alert

Adapted from Paternina-Caicedo A, Miranda J, Bourjeily G, et al. Performance of the Obstetric Early Warning Score in critically ill patients for the prediction of maternal death. Am J Obstet Gynecol. *2017;216(1):58.e1-58.e8.*

Finally, a multidisciplinary working group convened by the National Partnership for Maternal Safety used a consensus-based approach to define the Maternal Early Warning Criteria, a list of abnormal parameters that indicate the need for urgent bedside evaluation by a provider with the capacity to escalate care as necessary in order to follow diagnostic and therapeutic interventions. The Maternal Early Warning Criteria were drawn from the MEOWS "Red Triggers", but two parameters were deleted: temperature and pain. Fever was eliminated because it is common, accompanied by other vital sign abnormalities, and unlikely to be missed or dismissed in routine clinical care. Pain was eliminated because of the poor relationship between pain and severe morbidity (see *Table 6*).[55]

Maternal Early Warning Trigger

The Maternal Early Warning Trigger (MEWT) is a tool developed from a retrospectively collected population of women who received maternity care at seven hospitals in the United States. Patients admitted to the ICU were compared with control patients who were not admitted.[57] As a result, implementation of the MEWT resulted in a significant reduction in patient morbidity and mortality, although there was no difference in frequency of ICU admission.[54]

Table 6. Maternal Early Warning Criteria (MEWC)

Systolic blood pressure; mm Hg	<90 or >160
Diastolic blood pressure; mm Hg	>100 <50 or >120
Heart rate; BPM	<10 or >30
Respiratory rate; breaths/minute	<95
Oxygen saturation; % room air, sea level	<30 mL/hour for 2 hours
Oliguria	
Maternal agitation, confusion, or unresponsiveness	
Patient with hypertension reporting a nonremitting headache or shortness of breath	

BPM = beats per minute.

Adapted from Mhyre JM, D'Oria R, Hameed AB, et al. The maternal early warning criteria: a proposal from the national partnership for maternal safety. J Obstet Gynecol Neonatal Nurs. *2014;43(6):771-777.*

Sepsis in Obstetric Score

The Sepsis in Obstetrics Score (SOS) was designed to identify women who present to the emergency department and are at high risk of clinical deterioration and subsequent admission to the ICU for sepsis.[83] The SOS creates a sepsis scoring system using physiologic data adjusted for changes in pregnancy. A single-center retrospective study found that the SOS could effectively identify women with sepsis who ultimately required ICU admission. More recently, a prospective validation study shows a score of 6 or greater had a sensitivity of 64%, specificity of 88%, positive predictive value of 15%, and negative predictive value of 98.6% for ICU admission. Women with a score of 6 or greater were more likely to be admitted to the ICU (15% compared with 1.4%, $P < .01$). They were also more likely to be admitted to a telemetry unit (37.3% compared with 7.2%, $P < .01$), and to have antibiotic therapy initiated (90% compared with 72.9%, $P < .01$), and have therapy initiated sooner (3.2 compared with 3.7 hours, $P = .03$).[84] This score has not been validated in low-resource settings.

Rationale of Hour 0 Bundle

A practical approach to understand Time 0 might be the following:

- **Option A:** If laboratory, arterial blood gases, and/or lactate testing resources are available and infection with a SIRS of 2 or greater or a qSOFA score of 2 or greater is present, then a SOFA score should be obtained. A SOFA score of 2 or greater is considered sepsis.
- **Option B:** If laboratory, arterial blood gases, and/or lactate testing resources are not available, infection with a SIRS of 2 or greater or a qSOFA score of 2 or greater plus an OEWS within high risk of deterioration (ie, OEWS of 6 or greater) is strongly suggestive of sepsis.[4]

Beyond these two scenarios, the diagnosis of septic shock should be considered in the presence of sepsis if a vasopressor is needed to elevate mean arterial pressure (MAP) levels to at least 65 mm Hg and/or lactate remains greater than 18 mg/dL (2 mmol/L) despite adequate fluid resuscitation. Fetal well-being should be considered in the assessment of women with severe sepsis and

septic shock. Maternal infection can quickly reach the fetus because uteroplacental circulation does not have mechanisms of self-regulation. Thus, fetal perfusion and oxygenation are dependent on maternal oxygenation and hemodynamic stability. For this reason, an abnormal fetal heart rate (fetal tachycardia or bradycardia, decreased variability, and recurrent late decelerations) should alert the provider, given that all may be present in the setting of maternal sepsis.[85]

Management of Sepsis and Septic Shock

Early goal-directed therapy (EGDT) has been recommended in international guidance for the resuscitation of patients presenting with early septic shock. However, recent evidence from multicenter RCTs across the United States, Australasia, and England has indicated that EGDT is not superior to typical resuscitation measures (ie, treatment based on clinical judgment).[86]

Early detection and intervention may improve outcomes and survival in women with sepsis and septic shock. Urgent initiation of therapy according to standardized protocols has been shown to reduce mortality, hospital costs, and length of hospital stay in randomized studies that did not include women who were pregnant.[87] Prompt and appropriate therapy requires the coordination of a multidisciplinary team, including physicians, nurses, and pharmacy and administrative staff. Many of women with sepsis and sepsis shock may require ICU admission.

Initial Resuscitation (From Time 0-3 Hours)

Sepsis 1-Hour Bundle: Urgent Interventions

Early hemodynamic resuscitation is the principal goal of therapy. Treatment of women with sepsis who are pregnant follows the same principles for all patients: resuscitation, identification and treatment of the source of infection, management of complications (eg, hypotension, tissue hypoxia), and the application of organ protection strategies.[85] This includes conducting a series of interventions:[88,89]

1. Administer high-flow oxygen
2. Obtain blood cultures and consider infective source
3. Administer IV antibiotics
4. Administer IV fluid resuscitation
5. Check hemoglobin and serial lactates
6. Measure urine output.

Administer high-flow oxygen. Oxygen supplementation must be provided immediately according to pulse oximetry and assessment of arterial blood gases. SpO_2 in pregnancy should not be less than 95% and/or PaO_2 of 70 mm Hg.[90,91] Oxygen supplementation therapy is no longer recommended if there is no evidence of respiratory distress, even if some guidelines advocate the use of oxygen for all patients with major trauma or obstetric emergencies.[90]

Laboratory evaluation. Cultures should be collected (including blood cultures) from probable sources of infection. Laboratory evaluation includes a complete blood count, metabolic assessment including serum lactate, coagulation studies and arterial blood gases, and urine analysis.

Administer intravenous antibiotics. Broad-spectrum antibiotic therapy should be initiated within the first hour of clinical identification. Guideline recommendations show that shorter delays indicate better outcomes.[92]

Sepsis 3-Hour Bundle: Hemodynamic Management and Fluid Resuscitation

The Surviving Sepsis Campaign (SSC) recommends that up to 30 mL/kg of crystalloid fluid be rapidly administered in patients with sepsis and evidence of hypoperfusion. However, fluid overload can lead to pulmonary edema.[74] Most young healthy women can handle IV fluid boluses, but the risk of pulmonary edema with fluid resuscitation will be higher in the setting of preeclampsia.[93]

In patients with sepsis plus hypotension and/or hypoperfusion, fluid resuscitation with rapid infusions of warm fluids (4 mL/kg bolus every 15 minutes with a target of 30 mL/kg during the first 3 hours of treatment) is recommended to optimize cardiac preload, afterload, and contractility.[94]

The Protocolized Care for Early Septic Shock (ProCESS) trial did not find any improvement with outcomes with protocols.[95]

A checklist should be performed within the first 3 hours of management (initial resuscitation phase).[74] This checklist includes:

1. Measure lactate level
2. Obtain blood cultures before administration of antibiotics
3. Administer broad-spectrum antibiotics
4. Administer 30 mL/kg of crystalloid if hypotension or lactate level is 36 mg/dL (4 mmol/L)

or greater (18 mg/dL [2 mmol/L] according to current recommendations).

Fluid administration as proposed in Sepsis Six recommendations should only be considered for patients with hypotension in addition to clinical or subclinical evidence of hypoperfusion. Hypotension may be present in the setting of sepsis without other clinical or laboratory signs suggesting hypoperfusion.[96] Studies in nonpregnant human and animal models have associated liberal administration of fluids in sepsis with worse outcomes.[97] Better outcomes have been observed when fluid administration is conservative, rather than liberal.[98] Evidence of fluid resuscitation in the general population also applies to women who are pregnant.

A practical approach to fluid resuscitation in sepsis is the Rescue, Optimization, Stabilization, and Deescalation (ROS-D) protocol.[99] This protocol recommends the administration of a fluid bolus (4 mL/kg over 15 minutes) for immediate management of life-threatening conditions associated with impaired tissue perfusion. During the *R*escue phase, hemodynamic monitoring is based on clinical parameters, using vital signs and pulse oximetry, without complex hemodynamic assessment. During the *O*ptimization phase, the patient is no longer in immediate life-threatening danger but is in a stage of compensated shock (but at high risk of decompensation), thus close observation and monitoring of the patient's hemodynamic status is required to prevent life-threatening over- or undertreatment.[99] During this phase, fluids should be monitored using noninvasive dynamic methods such as echocardiography, bedside cardiovascular ultrasound, and bioreactance cardiac output monitoring.[100] After optimization, the *S*tabilization phase proposes maintenance fluids (1-2 mL/kg/hour) for ongoing maintenance in a setting of normal fluid losses. The main difference of this phase from the prior two is the absence of shock. Finally, during *D*eescalation, fluids will be discontinued and the goal is typically to promote a negative fluid balance.

Sepsis 6-Hour Bundle: Reassessment

The first two phases of fluid management (rescue and optimization) are cornerstones of the 3- to 6-hour bundle. However, patients with persistent hemodynamic deficit following fluid resuscitation, including those with persistent high lactate levels or low urine output, require more invasive strategies for ongoing resuscitation. The time required for this phase is not clear and should be tailored to every patient; however, as in the 3-hour bundle, a checklist must be completed to confirm that all required actions have been taken. During the first 6 hours of resuscitation the physiologic perfusion objectives include applying vasopressors, reassessing volume status and tissue perfusion, and remeasuring lactate levels if initial levels are elevated.

Apply vasopressors. Apply vasopressors for hypotension that does not respond to initial resuscitation with fluids to maintain a MAP level of 65 mm Hg or greater. If, despite adequate IV fluid resuscitation, treatment does not improve hypoperfusion or in patients with profound hypotension at admission (MAP less than 50 mm Hg), vasopressor therapy is necessary. Time to initiation of vasopressors may prove to be an independent predictor of mortality. Recent evidence in patients with septic shock found that mortality rates increased with increasing time to norepinephrine initiation.[101]

Reassess volume status and tissue perfusion. In the case of persistent arterial hypotension despite resuscitation with fluid (septic shock) or initial lactate levels of 36 mg/dL (4 mmol/L) or greater (18 mg/dL [2 mmol/L] according to the new sepsis definition), reassess volume status and tissue perfusion with some of the following:

- Blood pressure/heart rate response
- Urine output
- Bedside cardiovascular ultrasound
- Central venous pressure and central venous oxygen saturation (ScvO2) if central venous access is in place
- Pulse pressure variation (patients receiving ventilation)
- Dynamic assessment of fluid responsiveness with passive leg raise or fluid challenge.[102]

The goal of this therapy is to restore effective tissue perfusion and normalize cellular metabolism. Norepinephrine is the first-line vasoactive drug used for septic shock and acts to increase MAP. Although norepinephrine can reduce uterine blood flow, in septic shock with hypotension not responding to initial resuscitative efforts, the benefits outweigh the risks.[102]

A common concern during treatment of these patients is the administration of these drugs by a peripheral route. However, a recent systematic

review found that, if necessary, use of a peripheral route using a vein line in the antecubital fossa, with needles larger than 20G (ie, 16-18G) and assessment of the puncture site every 2 hours, minimizes the risk of major complications such as skin necrosis. Nonetheless, peripheral administration of vasoactive drugs is not recommended for an extended period.[103]

Remeasure lactate levels if initial levels are elevated. A decrease of lactate levels to less than 18 mg/dL (2 mmol/L) and/or lactate clearance (a decrease of at least 10% from the initial value) are useful targets in patients with septic shock as defined by Sepsis-3 guidelines. Serum lactate level at 6 hours can be an effective tool for obtaining prognosis in patients with septic shock who were treated with protocol-driven resuscitation bundle therapy.[104]

Sepsis 12-Hour Bundle: Final Assessment

After blood pressure, heart rate, urine output and lactate levels return to normal following fluid resuscitation, a management plan should be documented, including timings of planned clinical review and escalation criteria. Attention should focus on urgent ongoing resuscitation and wider management including control of any source amenable to drainage or removal within 12 hours.

Many women with sepsis who are receiving maternity care will have multiple organ dysfunction or high OEWSs. In these cases, escalation of care to intermediate care (if a single organ system requires support) or even intensive care units (if invasive respiratory support or support for more than one organ system is required) should be conducted at senior nursing and medical levels in consultation with the woman and her family as appropriate. Whenever possible, these decisions should be made and documented before deterioration of the woman's condition.

Patient Safety Bundle: Preventing Retained Vaginal Sponges After Birth

In 2017, the Council on Patient Safety in Women's Healthcare published the Prevention of Retained Vaginal Sponges After Birth Patient Safety Bundle (see pages 17 and 18). The bundle has four action domains for standardizing management: Readiness, Recognition, Response, and Reporting, which can be applied to every patient and maternity care department.

Global Perspective

Disease Burden

Even though sepsis-related maternal mortality and other maternal infection rates have decreased in the past 25 years, they remain a major public health problem in many low- and middle-income countries (LMIC).[105]

Prevention Strategies for Maternal Sepsis

Making family planning methods readily accessible, acceptable, and affordable has a significant effect on the absolute number of maternal deaths, including maternal sepsis.[46] Routine antibiotic prophylaxis after uncomplicated childbirth is controversial, particularly in women who are at higher risk of puerperal infectious morbidities.

Infections During Pregnancy and Postpartum: Sites That Commonly Lead to Sepsis and Etiologic Organisms

In low-resource countries, the maternal mortality rate due to postpartum sepsis is approximately 33%. Secondary deaths are 2 to 2.7 times higher in Africa, Asia, Latin America, and the Caribbean than in developed countries.[106]

A Practical Approach to Managing Maternal Sepsis

Critical decision making during sepsis treatment in LMIC settings is mainly based on clinical parameters instead of serum lactate measurements. In places where this valuable tool is not available, the use of other clinical severity systems with the capacity to identify women who are at risk of dying of infection should be considered.

The OEWS has been validated for use in low-resource settings, especially in patients with direct pathologies, including maternal hypertensive disorders; maternal hemorrhage; abortion, miscarriage, and ectopic pregnancy; obstructed labor and uterine rupture; maternal sepsis and other maternal infections. The SOS has not been validated in low-resource settings. Recent studies have shown that capillary refill and mottling skin areas are clinical expression of skin hypoperfusion and are closely correlated with septic shock and poor outcomes.[107,108]

As a rationale of the Hour 0 Bundle, if laboratory, arterial blood gases, and/or lactate level testing resources are not available, infection with an SIRS of 2 or greater or a qSOFA score of 2 or

greater plus a OEWS with a high risk of deterioration (ie, OEWS of 6 or greater) is strongly suggestive of sepsis.[4]

Many women with sepsis who are receiving maternity care will need escalation of care to intermediate care or even intensive care. These decisions should be made and documented before the woman's condition deteriorates; however, this is seldom feasible in LMIC.

More information about burden of other maternal emergencies and a rationale for low- and middle-income settings is available at www.aafp.org/globalalso.

Nursing Considerations: Maternal Sepsis

- Advocate for prevention of sepsis by modeling infection control techniques, including hand hygiene, use of sterile technique, and correct instrument and sponge counts during procedures
- Educate women regarding hand hygiene, perineal hygiene (wipe front to back), benefits of influenza vaccination, and avoidance of sick contacts
- Early detection is key. Communicate abnormal clinical findings immediately during pregnancy or postpartum periods
- If symptomatic, think oxygen supplementation to maintain O_2 saturation, IV access, and strict fluid intake and urine output documentation

IV = intravenous.

COUNCIL ON PATIENT SAFETY IN WOMEN'S HEALTH CARE

safe health care for every woman

PATIENT SAFETY BUNDLE

Prevention of Retained Vaginal Sponges After Birth

READINESS

Every unit

- Educate all members of the health care team on the importance of preventing retained vaginal sponges.
- Educate all members of the health care team on proper counting and documentation techniques.
- Establish a process for preventing retained vaginal sponges in every birth setting that includes role assignments for all members of the health care team. Use sponge detection system (e.g. pelvic x-ray with radiopaque sponges or radio-frequency identification) when available.

RECOGNITION & PREVENTION

Every patient

- Perform opening count of all vaginal sponges and record the count in the birth record and in a location visible by all members of the health care team. *
- Place all used sponges into a separate receptacle or area of table for ease of retrieval during closing count.
- Perform closing count of all vaginal sponges and record the count in the birth record.
- Confirm absence of sponges in the vagina through validation of correct closing count and visual examination/inspection of the vagina and document in the birth record.

*In the event of a precipitous birth, the initial count should be performed immediately after birth before items on table are disturbed (except items immediately necessary for birth).

January 2018

COUNCIL ON PATIENT SAFETY IN WOMEN'S HEALTH CARE

safe health care for every woman

PATIENT SAFETY BUNDLE

Prevention of Retained Vaginal Sponges After Birth

RESPONSE

To an incorrect closing count

- Conduct recount of used sponges, carefully search room (all drapes, kick buckets, and linens), and explore vagina, paying attention to vaginal fornicies to identify missing sponges.
  - If missing sponge is located, record correct closing count in birth record.
  - If missing sponge remains unaccounted for, utilize sponge detection system to rule out retained sponge.
    - If missing sponge is located, record correct closing count in birth record.
    - If missing sponge is not located, or in settings where sponge detection systems are unavailable, record the closing count as incorrect in the birth record and inform the patient of discrepancy in count.

REPORTING/SYSTEMS LEARNING

Every unit

- Establish a culture of safety and accountability in every birth setting.
- Develop a process for effectively documenting the sponge count for every birth and informing patient of discrepancies in count.
- Conduct multidisciplinary review of cases of retained vaginal sponge.
- Monitor outcome and process metrics.

This bundle is not intended for patients who are transferred to the operating room nor patients who have intentional, documented vaginal sponge/pack left in place. Organizations are encouraged to have institutional policies for monitoring, documenting, and accounting for these situations

Standardization of health care processes and reduced variation has been shown to improve outcomes and quality of care. The Council on Patient Safety in Women's Health Care disseminates patient safety bundles to help facilitate the standardization process. This bundle reflects emerging clinical, scientific, and patient safety advances as of the date issued and is subject to change. The information should not be construed as dictating an exclusive course of treatment or procedure to be followed. Although the components of a particular bundle may be adapted to local resources, standardization within an institution is strongly encouraged.

The Council on Patient Safety in Women's Health Care is a broad consortium of organizations across the spectrum of women's health for the promotion of safe health care for every woman.

January 2018

Reprinted from Council on Patient Safety in Women's Health Care. AIM-Supported Patient Safety Bundles. Prevention of Retained Vaginal Sponges After Birth. Available at https://safehealthcareforeverywoman.org.

References

1. Centers for Disease Control and Prevention. Pregnancy Mortality Surveillance System. 2018. Available at https://www.cdc.gov/reproductivehealth/maternalinfanthealth/pregnancy-mortality-surveillance-system.htm.
2. World Health Organization. Statement on maternal sepsis. 2017. Available at https://www.who.int/reproductivehealth/publications/maternal_perinatal_health/maternalsepsis-statement/en/.
3. Barton JR, Sibai BM. Severe sepsis and septic shock in pregnancy. *Obstet Gynecol*. 2012;120(3):689-706.
4. National Institute for Health and Care Excellence. Sepsis: recognition, diagnosis and early management. 2019. Available at https://www.nice.org.uk/guidance/ng51.
5. World Health Organization. The WHO near-miss approach for maternal health. *Bull World Health Organ*. 2011;87(10):29.
6. Hershey TB, Kahn JM. State sepsis mandates - a new era for regulation of hospital quality. *N Engl J Med*. 2017;376(24):2311-2313.
7. Seymour CW, Liu VX, Iwashyna TJ, et al. Assessment of clinical criteria for sepsis: for the Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3). *JAMA*. 2016;315(8):762-774.
8. Levy MM, Fink MP, Marshall JC, et al. SCCM/ESICM/ACCP/ATS/SIS. 2001 SCCM/ESICM/ACCP/ATS/SIS International Sepsis Definitions Conference. *Crit Care Med*. 2003;31(4):1250-1256.
9. Bauer ME, Bauer ST, Rajala B, et al. Maternal physiologic parameters in relationship to systemic inflammatory response syndrome criteria: a systematic review and meta-analysis. *Obstet Gynecol*. 2014;124(3):535-541.
10. Say L, Chou D, Gemmill A, et al. Global causes of maternal death: a WHO systematic analysis. *Lancet Glob Health*. 2014;2(6):e323-e333.
11. Bauer M, Bateman B, Bauer S, et al. Maternal sepsis mortality and morbidity during hospitalization for delivery: temporal trends and independent associations for severe sepsis. *Anesth Analg*. 2013;117(4):944-950.
12. Abir G, Akdagli S, Butwick A, Carvalho B. Clinical and microbiological features of maternal sepsis: a retrospective study. *Int J Obstet Anesth*. 2017;29:26-33.
13. Glaser AP, Schaeffer AJ. Urinary tract infection and bacteriuria in pregnancy. *Urol Clin North Am*. 2015;42(4):547-560.
14. Committee on Obstetric Practice. Committee Opinion no. 717: sulfonamides, nitrofurantoin, and risk of birth defects. *Obstet Gynecol*. 2017;130(3):e150-e152.
15. Trampuz A, Widmer AF. Hand hygiene: a frequently missed lifesaving opportunity during patient care. *Mayo Clin Proc*. 2004;79(1):109-116.
16. Lumbiganon P, Thinkhamrop J, Thinkhamrop B, Tolosa JE. Vaginal chlorhexidine during labour for preventing maternal and neonatal infections (excluding Group B Streptococcal and HIV). *Cochrane Database Syst Rev*. 2014;(9):CD004070.
17. Smaill FM, Grivell RM. Antibiotic prophylaxis versus no prophylaxis for preventing infection after cesarean section. *Cochrane Database Syst Rev*. 2014;(10):CD007482.
18. Mackeen AD, Packard RE, Ota E, et al. Timing of intravenous prophylactic antibiotics for preventing postpartum infectious morbidity in women undergoing cesarean delivery. *Cochrane Database Syst Rev*. 2014;(12):CD009516.
19. Committee on Practice Bulletins-Obstetrics. ACOG Practice Bulletin no. 199: use of prophylactic antibiotics in labor and delivery. *Obstet Gynecol*. 2018;132(3):e103-e119.
20. Nabhan AF, Allam NE, Hamed Abdel-Aziz Salama M. Routes of administration of antibiotic prophylaxis for preventing infection after caesarean section. *Cochrane Database Syst Rev*. 2016;(6):CD011876.
21. Tita AT, Szychowski JM, Boggess K, et al. C/SOAP Trial Consortium. Adjunctive azithromycin prophylaxis for cesarean delivery. *N Engl J Med*. 2016;375(13):1231-1241.
22. Bonet M, Ota E, Chibueze CE, Oladapo OT. Routine antibiotic prophylaxis after normal vaginal birth for reducing maternal infectious morbidity. *Cochrane Database Syst Rev*. 2017;11:CD012137.
23. Chongsomchai C, Lumbiganon P, Laopaiboon M. Prophylactic antibiotics for manual removal of retained placenta in vaginal birth. *Cochrane Database Syst Rev*. 2014;(10):CD004904.
24. Bonet M, Ota E, Chibueze CE, Oladapo OT. Antibiotic prophylaxis for episiotomy repair following vaginal birth. *Cochrane Database Syst Rev*. 2017;11:CD012136.
25. Buppasiri P, Lumbiganon P, Thinkhamrop J, Thinkhamrop B. Antibiotic prophylaxis for third- and fourth-degree perineal tear during vaginal birth. *Cochrane Database Syst Rev*. 2014;(10):CD005125.
26. Valent AM, DeArmond C, Houston JM, et al. Effect of post-cesarean delivery oral cephalexin and metronidazole on surgical site infection among obese women: a randomized clinical trial. *JAMA*. 2017;318(11):1026-1034.
27. Cantwell R, Clutton-Brock T, Cooper G, et al. Saving mother's lives: reviewing maternal deaths to make motherhood safer: 2006-2008. The eighth report of the confidential inquiries into maternal deaths in the United Kingdom. *BJOG*. 2011;118(Suppl 1):1-203.
28. Melzer M, Welch C. Does the presence of a urinary catheter predict severe sepsis in a bacteraemic cohort? *J Hosp Infect*. 2017;95(4):376-382.
29. Faro S, Phillips LE, Baker JL, et al. Comparative efficacy and safety of mezlocillin, cefoxitin, and clindamycin plus gentamicin in postpartum endometritis. *Obstet Gynecol*. 1987;69(5):760-766.
30. Timezguid N, Das V, Hamdi A, et al. Maternal sepsis during pregnancy or the postpartum period requiring intensive care admission. *Int J Obstet Anesth*. 2012;21(1):51-55.
31. Udagawa H, Oshio Y, Shimizu Y. Serious group A streptococcal infection around delivery. *Obstet Gynecol*. 1999;94(1):153-157.
32. Burrows L, Meyn L, Weber A. Maternal morbidity associated with vaginal versus cesarean delivery. *Obstet Gynecol*. 2004;103(5 Pt 1):907-912.
33. Morgan J, Roberts S. Maternal sepsis. *Obstet Gynecol Clin North Am*. 2013;40(1):69-87.
34. Mackeen AD, Packard RE, Ota E, Speer L. Antibiotic regimens for postpartum endometritis. *Cochrane Database Syst Rev*. 2015;(2):CD001067.
35. Hyder JA, Wakeam E, Arora V, et al. Investigating the "Rule of W," a mnemonic for teaching on postoperative complications. *J Surg Educ*. 2015;72(3):430-437.
36. Deutscher M, Lewis M, Zell ER, et al; Active Bacterial Core Surveillance Team. Incidence and severity of invasive Streptococcus pneumoniae, group A Streptococcus, and group B Streptococcus infections among pregnant and postpartum women. *Clin Infect Dis*. 2011;53(2):114-123.
37. Locksmith GJ, Duff P. Assessment of the value of routine blood cultures in the evaluation and treatment of patients with chorioamnionitis. *Infect Dis Obstet Gynecol*. 1994;2(3):111-114.
38. Chebbo A, Tan S, Kassis C, et al. Maternal sepsis and septic shock. *Crit Care Clin*. 2016;32(1):119-135.

39. Tarai B, Jain D, Das P, Budhiraja S. Paired blood cultures increase the sensitivity for detecting pathogens in both inpatients and outpatients. *Eur J Clin Microbiol Infect Dis*. 2018;37(3):435-441.
40. Martens MG, Faro S, Hammill HA, et al. Sulbactam/ampicillin versus metronidazole/gentamicin in the treatment of post-cesarean section endometritis. *Diagn Microbiol Infect Dis*. 1989;12(4): 189S-194S.
41. Sweet RL, Roy S, Faro S, et al. Piperacillin and tazobactam versus clindamycin and gentamicin in the treatment of hospitalized women with pelvic infection. The Piperacillin/Tazobactam Study Group. *Obstet Gynecol*. 1994;83(2):280-286.
42. Dotters-Katz SK, Smid MC, Grace MR, et al. Risk factors for postpartum septic pelvic thrombophlebitis: a multicenter cohort. *Am J Perinatol*. 2017;34(11):1148-1151.
43. Klima DA, Snyder TE. Postpartum ovarian vein thrombosis. *Obstet Gynecol*. 2008;111(2 Pt 1):431-435.
44. Garcia J, Aboujaoude R, Apuzzio J, Alvarez JR. Septic pelvic thrombophlebitis: diagnosis and management. *Infect Dis Obstet Gynecol*. 2006;2006:15614.
45. Falagas ME, Vardakas KZ, Athanasiou S. Intravenous heparin in combination with antibiotics for the treatment of deep vein septic thrombophlebitis: a systematic review. *Eur J Pharmacol*. 2007;557(2-3):93-98.
46. World Health Organization. The prevention and management of unsafe abortion. Report of a Technical Working Group. Available at http://whqlibdoc.who.int/hq/1992/WHO_MSM_92.5.pdf.
47. Haddad L, Nour N. Unsafe abortion: unnecessary maternal mortality. *Rev Obstet Gynecol*. 2009;2(2):122-126.
48. Labandera A, Gorgoroso M, Briozzo L. Implementation of the risk and harm reduction strategy against unsafe abortion in Uruguay: From a university hospital to the entire country. *Int J Gynaecol Obstet*. 2016;134(Suppl 1):S7-S11.
49. Briozzo L. From risk and harm reduction to decriminalizing abortion: The Uruguayan model for women's rights. *Int J Gynaecol Obstet*. 2016;134(Suppl 1):S3-S6.
50. Meites E, Zane S, Gould CC. sordellii Investigators. Fatal Clostridium sordellii infections after medical abortions. *N Engl J Med*. 2010; 363(14):1382-1383.
51. Savaris RF, de Moraes GS, Cristovam RA, Braun RD. Are antibiotics necessary after 48 hours of improvement in infected/septic abortions? A randomized controlled trial followed by a cohort study. *Am J Obstet Gynecol*. 2011;204(4):301.e1-301.e5.
52. Jowett M. Safe motherhood interventions in low-income countries: an economic justification and evidence of cost effectiveness. *Health Policy*. 2000;53(3):201-228.
53. American College of Obstetricians and Gynecologists. Committee Opinion no. 712 summary: intrapartum management of intraamniotic infection. *Obstet Gynecol*. 2017;130(2):490-492.
54. Chapman E, Reveiz L, Illanes E, Bonfill Cosp X. Antibiotic regimens for management of intra-amniotic infection. *Cochrane Database Syst Rev*. 2014;(12):CD010976.
55. Verani JR, McGee L, Schrag SJ; Division of Bacterial Diseases, National Center for Immunization and Respiratory Diseases, Centers for Disease Control and Prevention (CDC). Prevention of perinatal group B streptococcal disease—revised guidelines from CDC, 2010. *MMWR Recomm Rep*. 2010;59(RR-10):1-36.
56. Rouse DJ, Landon M, Leveno KJ, et al; National Institute of Child Health And Human Development, Maternal-Fetal Medicine Units Network. The Maternal-Fetal Medicine Units cesarean registry: chorioamnionitis at term and its duration-relationship to outcomes. *Am J Obstet Gynecol*. 2004;191(1):211-216.
57. Czikk MJ, McCarthy FP, Murphy KE. Chorioamnionitis: from pathogenesis to treatment. *Clin Microbiol Infect*. 2011;17(9):1304-1311.
58. Edwards RK, Duff P. Single additional dose postpartum therapy for women with chorioamnionitis. *Obstet Gynecol*. 2003;102(5 Pt 1): 957-961.
59. Wing DA, Fassett MJ, Getahun D. Acute pyelonephritis in pregnancy: an 18-year retrospective analysis. *Am J Obstet Gynecol*. 2014;210(3):219.e1-219.e6.
60. Smaill FM, Vazquez JC. Antibiotics for asymptomatic bacteriuria in pregnancy. *Cochrane Database Syst Rev*. 2015;(8):CD000490.
61. Schneeberger C, Geerlings SE, Middleton P, Crowther CA. Interventions for preventing recurrent urinary tract infection during pregnancy. *Cochrane Database Syst Rev*. 2015;(7):CD009279.
62. Farkash E, Weintraub AY, Sergienko R, et al. Acute antepartum pyelonephritis in pregnancy: a critical analysis of risk factors and outcomes. *Eur J Obstet Gynecol Reprod Biol*. 2012;162(1):24-27.
63. Dotters-Katz SKD, Heine RP, Grotegut CA. Medical and infectious complications associated with pyelonephritis among pregnant women at delivery. *Infect Dis Obstet Gynecol*. 2013;2013:124102.
64. Hooton TM, Bradley SF, Cardenas DD, et al; Infectious Diseases Society of America. Diagnosis, prevention, and treatment of catheter-associated urinary tract infection in adults: 2009 International Clinical Practice Guidelines from the Infectious Diseases Society of America. *Clin Infect Dis*. 2010;50(5):625-663.
65. Vazquez JC, Abalos E. Treatments for symptomatic urinary tract infections during pregnancy. *Cochrane Database Syst Rev*. 2011;(1): CD002256.
66. Moon J, Prasad S, Egan M. Treating UTIs in reproductive-age women-proceed with caution. *J Fam Pract*. 2010;59(4):220-222.
67. Crider KS, Cleves MA, Reefhuis J, et al. Antibacterial medication use during pregnancy and risk of birth defects: National Birth Defects Prevention Study. *Arch Pediatr Adolesc Med*. 2009;163(11): 978-985.
68. Juan YS, Wu WJ, Chuang SM, et al. Management of symptomatic urolithiasis during pregnancy. *Kaohsiung J Med Sci*. 2007;23(5): 241-246.
69. Kamei J, Nishimatsu H, Nakagawa T, et al. Risk factors for septic shock in acute obstructive pyelonephritis requiring emergency drainage of the upper urinary tract. *Int Urol Nephrol*. 2014;46(3): 493-497.
70. Shu T, Green JM, Orihuela E. Renal and perirenal abscesses in patients with otherwise anatomically normal urinary tracts. *J Urol*. 2004;172(1):148-150.
71. Chen YH, Keller J, Wang IT, et al. Pneumonia and pregnancy outcomes: a nationwide population-based study. *Am J Obstet Gynecol*. 2012;207(4):288.e1-288.e7.
72. American College of Obstetricians and Gynecologists. Practice Bulletin no. 151: cytomegalovirus, parvovirus B19, varicella zoster, and toxoplasmosis in pregnancy. *Obstet Gynecol*. 2015;125(6): 1510-1525.
73. American College of Obstetricians and Gynecologists. ACOG Committee Opinion no. 732: influenza vaccination during pregnancy. *Obstet Gynecol*. 2018;131(4):e109-e114.

74. Dellinger RP, Levy MM, Rhodes A, et al. Surviving Sepsis Campaign Guidelines Committee including The Pediatric Subgroup. Surviving Sepsis Campaign: international guidelines for management of severe sepsis and septic shock, 2012. *Intensive Care Med*. 2013; 39(2):165-228.
75. Mathieu JE, Heffner TS, Goodwin GF, et al. The influence of shared mental models on team process and performance. *J Appl Psychol*. 2000;85(2):273-283.
76. Vincent JL, Moreno R, Takala J, et al. The SOFA (Sepsis-related Organ Failure Assessment) score to describe organ dysfunction/ failure. On behalf of the Working Group on Sepsis-Related Problems of the European Society of Intensive Care Medicine. *Intensive Care Med*. 1996;22(7):707-710.
77. Singer M, Deutschman C, Seymour C, et al. The Third International Consensus definitions for sepsis and septic shock (Sepsis-3). *JAMA*. 2016;315(8):801-810.
78. Serafim R, Gomes JA, Salluh J. Póvoa P. A comparison of the Quick-SOFA and Systemic Inflammatory Response Syndrome Criteria for the diagnosis of sepsis and prediction of mortality: a systematic review and meta-analysis. *Chest*. 2018;153(3):646-655.
79. Mikkelsen ME, Miltiades AN, Gaieski DF, et al. Serum lactate is associated with mortality in severe sepsis independent of organ failure and shock. *Crit Care Med*. 2009;37(5):1670-1677.
80. Kim CJ, Romero R, Chaemsaithong P, et al. Acute chorioamnionitis and funisitis: definition, pathologic features, and clinical significance. *Am J Obstet Gynecol*. 2015;213(4)(Suppl):S29-S52.
81. Romero R, Dey SK, Fisher SJ. Preterm labor: one syndrome, many causes. *Science*. 2014;345(6198):760-765.
82. Keelan JA, Newnham JP. Recent advances in the prevention of preterm birth. *F1000 Res*. 2017;6:1139-1150.
83. Albright CM, Ali TN, Lopes V, et al. The Sepsis in Obstetrics Score: a model to identify risk of morbidity from sepsis in pregnancy. *Am J Obstet Gynecol*. 2014;211(1):39.e1-39.e8.
84. Albright CM, Has P, Rouse DJ, Hughes BL. Internal validation of the Sepsis in Obstetrics Score to identify risk of morbidity from sepsis in pregnancy. *Obstet Gynecol*. 2017;130(4):747-755.
85. Lucas DN, Robinson PN, Nel MR. Sepsis in obstetrics and the role of the anaesthetist. *Int J Obstet Anesth*. 2012;21(1):56-67.
86. Mouncey PR, Osborn TM, Power GS, et al. Protocolised Management In Sepsis (ProMISe): a multicentre randomised controlled trial of the clinical effectiveness and cost-effectiveness of early, goal-directed, protocolised resuscitation for emerging septic shock. *Health Technol Assess*. 2015;19(97):i-xxv, 1-150.
87. Seymour CW, Gesten F, Prescott HC, et al. Time to treatment and mortality during mandated emergency care for sepsis. *N Engl J Med*. 2017;376(23):2235-2244.
88. Daniels R, Nutbeam T, McNamara G, Galvin C. The sepsis six and the severe sepsis resuscitation bundle: a prospective observational cohort study. *Emerg Med J*. 2011;28(6):507-512.
89. Bhat A, Asghar M, Raulia G, Mandal A. Improving multidisciplinary severe sepsis management using the Sepsis Six. *Clin Med (Lond)*. 2016;16(Suppl 3):s1.
90. Nesterenko TH, Acun C, Mohamed MA, et al. Is it a safe practice to administer oxygen during uncomplicated delivery: a randomized controlled trial? *Early Hum Dev*. 2012;88(8):677-681.
91. Langford E, Khwanda A, Langford K. Oxygen saturation response to exercise in healthy pregnant women: a simple protocol and normal range. *Obstet Med*. 2010;3(2):65-68.
92. Pruinelli L, Westra BL, Yadav P, et al. Delay within the 3-Hour Surviving Sepsis Campaign guideline on mortality for patients with severe sepsis and septic shock. *Crit Care Med*. 2018;46(4):500-505.
93. Anthony J, Schoeman L. Fluid management in pre-eclampsia. *Obstet Med*. 2013;6(3):100-104.
94. Aya HD, Rhodes A, Chis Ster I, et al. Hemodynamic effect of different doses of fluids for a fluid challenge: a quasi-randomized controlled study. *Crit Care Med*. 2017;45(2):e161-e168.
95. Yealy DM, Kellum JA, Huang DT, et al. ProCESS Investigators. A randomized trial of protocol-based care for early septic shock. *N Engl J Med*. 2014;370(18):1683-1693.
96. Hernandez G, Castro R, Romero C, et al. Persistent sepsis-induced hypotension without hyperlactatemia: is it really septic shock? *J Crit Care*. 2011;26(4):435.e9-435.e14.
97. Sadaka F, Juarez M, Naydenov S, O'Brien J. Fluid resuscitation in septic shock: the effect of increasing fluid balance on mortality. *J Intensive Care Med*. 2014;29(4):213-217.
98. Smith SH, Perner A. Higher vs. lower fluid volume for septic shock: clinical characteristics and outcome in unselected patients in a prospective, multicenter cohort. *Crit Care*. 2012;16(3):R76.
99. Hoste EA, Maitland K, Brudney CS, et al; ADQI XII Investigators Group. Four phases of intravenous fluid therapy: a conceptual model. *Br J Anaesth*. 2014;113(5):740-747.
100. Rhodes A, Evans LE, Alhazzani W, et al. Surviving Sepsis Campaign: International Guidelines for Management of Sepsis and Septic Shock: 2016. *Intensive Care Med*. 2017;43(3):304-377.
101. Bai X, Yu W, Ji W, et al. Early versus delayed administration of norepinephrine in patients with septic shock. *Crit Care*. 2014;18(5):532.
102. Marty P, Roquilly A, Vallée F, et al. Lactate clearance for death prediction in severe sepsis or septic shock patients during the first 24 hours in intensive care unit: an observational study. *Ann Intensive Care*. 2013;3(1):3.
103. Loubani OM, Green RS. A systematic review of extravasation and local tissue injury from administration of vasopressors through peripheral intravenous catheters and central venous catheters. *J Crit Care*. 2015;30(3):653.e9-653.e17.
104. Ryoo SM, Lee J, Lee Y-S, et al. Lactate level versus lactate clearance for predicting mortality in patients with septic shock defined by Sepsis-3. *Crit Care Med*. 2018;46(6):e489-e495.
105. GBD 2015 Maternal Mortality Collaborators. Global, regional, and national levels of maternal mortality, 1990-2015: a systematic analysis for the Global Burden of Disease Study 2015. *Lancet*. 2016; 388(10053):1775-1812.
106. Khan KS, Wojdyla D, Say L, et al. WHO analysis of causes of maternal death: a systematic review. *Lancet*. 2006;367(9516):1066-1074.
107. Ait-Oufella H, Bourcier S, Alves M, et al. Alteration of skin perfusion in mottling area during septic shock. *Ann Intensive Care*. 2013;3(1): 31.
108. Lara B, Enberg L, Ortega M, et al. Capillary refill time during fluid resuscitation in patients with sepsis-related hyperlactatemia at the emergency department is related to mortality. *PLoS One*. 2017;12(11): e0188548.

Third- and Fourth-Degree Perineal Lacerations

Learning Objectives

1. Understand the anatomy of the perineum and pelvic floor.
2. Explain the long-term consequences of occult and apparent pelvic floor trauma.
3. Outline repair strategies for rectal mucosa and rectal sphincter lacerations.
4. Outline repair techniques for complex/stellate vaginal and cervical lacerations.

Introduction

The risk of perineal lacerations varies depending on patient characteristics, birth environment, and delivery methods. Between 53% and 79% of women will experience some type of perineal laceration during vaginal delivery.[1] Third- and fourth-degree perineal lacerations can be a complication of vaginal delivery, especially in the presence of a midline episiotomy or an assisted vaginal delivery. Correct assessment of the laceration and proper repair is necessary to reduce the risk of complications including anal incontinence and rectovaginal fistula. Lacerations are classified by degree, which determines the method of repair.

Table 1. Factors Associated With Third- and Fourth-Degree Lacerations

Asian or Pacific Islander ethnicity
Assisted vaginal delivery[a]
Delivery with stirrups (delivery table, lithotomy)
Epidural analgesia
Increased fetal weight
Nulliparity
Occiput transverse or occiput posterior positions
Patient age <21 years
Prolonged second stage of labor
Routine episiotomy[b]
Use of oxytocin

[a]Higher incidence with use of forceps compared with vacuum devices

[b]Higher incidence with midline episiotomy compared with mediolateral episiotomy

Information from various sources.

Epidemiology

Third- and fourth-degree lacerations can occur after any type of vaginal delivery. Numerous studies have shown that midline episiotomy has the strongest association with a third- or fourth-degree laceration.[2] Restrictive episiotomy policies have many benefits compared with liberal episiotomy policies. With restrictive episiotomy, there is less posterior perineal trauma, suturing, and pain, but an increased risk of anterior perineal trauma.[3] Genital trauma has not been shown to affect postpartum urinary continence at 12 weeks' postpartum, but the long-term effects are uncertain.[4]

Other factors have been shown to predispose women to perineal laceration. These are listed in *Table 1*. A Cochrane review of 32 trials showed that, compared with forceps, a vacuum extractor is less likely to be associated with a third- or fourth-degree laceration.[5] Factors not associated with perineal laceration include body mass index, gestational age, and weight gain in pregnancy.[4,6]

Anatomy

The perineum is composed of multiple layers, including an epithelium and several muscular layers. These layers are disrupted when a third- or fourth-degree laceration occurs. A detailed understanding of the anatomy of the perineum is essential for proper repair.

A laceration involves the squamous epithelium of the perineum and the vaginal mucosa. Directly beneath the squamous epithelium at the posterior margin of the introitus is the perineal body. The perineal body is the central tendon of the perineum where the bulbocavernosus (also called the bulbospongiosus), transverse perineal, and external anal sphincter muscles join. The perineal body is triangular when visualized in the sagittal plane. The base is at the perineum, and the apex is at the top of the vagina. Superficial and deep to these muscles are lay-

ers of fascial tissue. Beneath the deep layer of fascia lies the deep transverse perineal muscle and the levator ani muscles, respectively, as a superior dissection is made superiorly. Posterior to and originating inferiorly from the levator ani muscle is the internal anal sphincter. Directly beneath the internal anal sphincter is the rectovaginal septum (also known as the perineal membrane, Denonvilliers fascia, or rectovaginal fascia) and the rectal mucosa.

The internal anal sphincter may not be a familiar anatomic structure for many providers because it was not well described in older obstetrical texts. It is a smooth muscle and provides most of the resting anal sphincter tone and continence. There is evidence that a laceration involving both the external and internal anal sphincters has a higher likelihood of postpartum fecal incontinence.[7] The presence of a defect in the internal anal sphincter on endoanal ultrasonography is associated with anal incontinence in some but not all studies.[7,8]

Classification

A classification system establishes a frame of reference for evaluation, research, and discussion of the degree of damage, as well as the proper means for repair. A four-level classification system is used in the United States, whereas a three-level system is used in Europe (the European third level is equivalent to the US fourth level)[9] (*Table 2*).

Table 2. Four-Level Laceration Classification Used in the United States

Degree of Laceration	Description
First-degree	Superficial laceration of the vaginal mucosa or perineal body
Second-degree	Laceration of the vaginal mucosa and/or perineal skin and deeper subcutaneous tissues
Third-degree	Involves anal sphincter complex
3A	<50% through external anal sphincter
3B	>50% through external anal sphincter with internal sphincter intact
3C	Involves external and internal anal sphincter
Fourth-degree	Anal sphincter complex laceration extending into the anal (rectal) mucosa

Adapted from American College of Obstetricians and Gynecologists. ACOG Practice Bulletin no. 198: Prevention and management of obstetric lacerations at vaginal delivery. Obstet Gynecol. *2018;132:e87–e102.*

Table 3. Preventive Strategies

- Allow time for adequate perineal stretching
- Avoid an assisted vaginal delivery[a]
- Avoid episiotomy and use mediolateral technique if needed
- Assume maternal lateral birth position
- Recommend perineal massage during the weeks before delivery in nulliparous women
- Apply perineal warm packs during the second stage of labor

[a]*Forceps use has a higher incidence of anal sphincter tears than vacuum extractor use.*

Information from various sources.

Prevention

Avoiding assisted vaginal delivery and midline episiotomy is the best way to prevent a third- or fourth-degree laceration. If an assisted vaginal delivery is required, the use of a vacuum extractor rather than forceps appears to reduce maternal morbidity.[5] Several studies have evaluated other preventive strategies with mixed results. A systematic review showed that perineal massage started weeks before delivery increased the likelihood of an intact perineum with a first vaginal delivery, but not in women with a previous vaginal birth.[10] A randomized controlled trial (RCT) showed that although a perineal warm pack did not reduce the likelihood of nulliparous women requiring suturing, it significantly reduced the third- and fourth-degree lacerations and pain during delivery.[11] Another RCT did not show any difference in the risk of genital trauma with the use of warm compresses, massage with lubricant, or no touching of the perineum.[12]

The literature is mixed on the best birth position, but the lateral position has the strongest evidence for an increased likelihood of an intact perineum after delivery, especially in the presence of regional anesthesia.[13,14] An RCT showed that perineal support at delivery did not decrease the frequency or degree of perineal lacerations compared with a *hands-off* approach (no perineal support or pressure on the fetal head),[15] whereas another RCT showed that a *hands-on* approach (perineal support with pressure on the fetal head) increased the risk of episiotomy but decreased perineal pain on the tenth postpartum day.[16] Preventive strategies are summarized in *Table 3*.

Surgical Repair

Evaluation

Before beginning the repair, the provider should obtain the necessary equipment and assistance for proper exposure (*Tables 4* and *5*). It is essential that the full extent of the laceration be known with specific attention given to the presence of extension into the internal anal sphincter and rectal mucosa and how superior the laceration extends. Even experienced providers commonly miss the presence of a third-degree laceration.[17]

A rectal examination is recommended for deeper second-degree lacerations. In fourth-degree lacerations, it is important to check for the so-called *buttonhole* defect in the rectal mucosa. This describes the fourth-degree laceration in which the middle portion of the rectal mucosa is spared. The provider misses the upper injury and repairs only the more inferior laceration. Therefore, a high rate of fistula and infectious morbidity occurs. The provider should perform a detailed rectal examination as described below before beginning the repair.

- The examiner gently elevates the anterior rectal wall into the vagina.
- The examiner or an assistant sponges and retracts the labia so that optimal visualization is made of the defect and the surrounding tissues.

Table 5. Equipment List for Third- and Fourth-Degree Laceration Repair

- Allis clamp (at least two)
- Gelpi retractor
- Irrigation supplies
- Local anesthetic
- Needle holder
- Needle/sutures[a]
  - 2-0 polydioxanone sulfate or polyglycolic acid on a large taper needle for external anal sphincter
  - 2-0 polyglycolic acid on a large taper needle for internal anal sphincter and vagina/perineum repair
  - 2-0 or 3-0 polyglycolic acid on a large taper needle for repair of vaginal and perineal muscle lacerations
  - 3-0 or 4-0 polyglycolic acid on a small taper needle for perineal skin, labial, periurethral, and periclitoral lacerations
  - 3-0 or 4-0 polyglycolic acid on a small taper needle for rectal mucosa repair
- Sharp tooth tissue forceps
- Suture scissors
- Sponges
- Syringe and needle for injection of local anesthesia with addition of Iowa trumpet needle guide for pudendal block
- Vaginal packing materials

[a]*Sutures may vary depending on provider.*

Table 4. Suture Materials

Suture	Brand Name Examples	Composition	Qualities	Duration
Polyglycolic acid	Vicryl	Braided synthetic of a glycolic acid homopolymer	Better and longer tensile strength than chromic. Mild tissue response. Decreases short-term pain	Predictable absorption by hydrolysis. Completely absorbed at 60 to 90 days
Polyglycolic acid and trimethylene	Maxon	Monofilament composed 1:3 of the noted polymers	Excellent tensile strength. Smooth and resists kinking	Absorbed completely by hydrolysis at 180 days
Polydioxanone sulfate	PDS Duracryl	Monofilament long lasting, delayed absorbable	Excellent tensile strength. Smooth and pliable	Absorbed completely by hydrolysis at 180 days
Chromic gut	Chromic	Twisted lengths of proteinaceous sheep or beef intestinal lining	Good tensile strength. Strong inflammatory response	Absorption by phagocytosis, which is unpredictable Maintains tensile strength from 7 to 10 days. Faster absorption in infected tissue

Information from Kettle C, Dowswell T, Ismail KM. Absorbable suture materials for primary repair of episiotomy and second degree tears. Cochrane Database Syst Rev. *2010;(6):CD000006.*

- The examiner can easily identify the length of the laceration, the presence of buttonholes, the condition of the surrounding anatomical structures, and any bleeding points.
- Before continuing with the repair, change the rectal glove.
- Copious irrigation is encouraged to minimize the risk of infection from colonic bacteria.

The choice of suture material for obstetric perineal laceration repair has historically been at the discretion of the provider; however, evidence suggests that an absorbable synthetic suture is the best choice. A Cochrane review comparing absorbable synthetic sutures with plain or chromic catgut sutures for perineal repair included 18 studies. The review showed that synthetic sutures were associated with less pain in the first 3 days after delivery, less need for analgesia, and less suture dehiscence.[18] There was no significant difference in long-term pain or the amount of dyspareunia experienced by the women. Suture removal was significantly more common with synthetic sutures. There was minimal difference between standard synthetic and rapidly absorbing synthetic sutures, except for the increased risk of suture removal. One trial comparing monofilament with standard polyglycolic sutures showed no difference in most short-term outcomes.[19]

Analgesia

Local anesthesia should provide adequate analgesia for most repairs. Infusing a local anesthetic into the vaginal mucosa, perineum, and anal sphincter should provide enough analgesia to complete the repair and may be supplemented with a pudendal block or regional anesthesia for more extensive lacerations. This can be administered before undertaking an episiotomy, or after the delivery and before the repair. The choice of anesthetic can be at the discretion of the provider; commonly used drugs include lidocaine, chloroprocaine, and bupivacaine. Lidocaine transfers quickly to the fetus and must be used sparingly if injected before delivery.[20] Chloroprocaine is rapidly metabolized in the woman and fetus and has poor placental transfer. Bupivacaine is more protein-bound and has a lower transfer rate to the fetus, but it has potential for maternal cardiac toxicity.[21]

Pudendal anesthesia is an ideal regional block for the repair of a third- or fourth-degree laceration when epidural analgesia is not in place. It provides excellent anesthesia of the central perineum and lower vagina by blocking the dorsal nerve of the clitoris, labial nerves, and inferior rectal nerve.[20] The pudendal regional block can be administered using a transvaginal approach with the assistance of an Iowa trumpet needle guide and a 20-gauge spinal needle. Local anesthetic is injected bilaterally just below the ischial spines.[22] The pudendal nerve is near the pudendal artery and vein. This increases the likelihood of intravascular injection, vessel laceration, and rapid circulatory absorption of the anesthetic. Therefore, the provider should aspirate before injection to verify the needle is not within a blood vessel.[20]

Women who are unable to tolerate the repair with an adequate pudendal block may be best treated with inhalation or intravenous analgesia. Women with epidural analgesia in place can have it re-dosed to provide the necessary analgesia. Postpartum spinal anesthesia is another option because it is important for the woman to be cooperative and comfortable to achieve good exposure and an adequate repair. Regional anesthesia (epidural or spinal) also will relax the internal anal sphincter and facilitate repair.

Laceration Repair

After adequate analgesia has been achieved, the repair can be started. The following describes a fourth-degree laceration repair, which includes a description of a third-degree laceration repair (beginning after closure of the rectal mucosa).

The most important step before beginning the repair is adequate exposure and visualization of the laceration. Several techniques can be used to achieve this. Many providers use one or two assistants to provide retraction, and vaginal packing placed into the upper vagina to stop the flow of blood into the surgical field. Some providers use sponges when vaginal packing material is unavailable. When an assistant is not available, the provider can use a Gelpi retractor. The Gelpi retractor is an adjustable, self-retaining retractor that can be used to open and separate the vaginal walls.

After good visualization is obtained, a 3-0 or 4-0 absorbable synthetic suture on a small tapered needle is used to reapproximate the rectal mucosa using an imbricating method (tissue sewn over the cut edges instead of edge to edge) to avoid placement of sutures into the rectal lumen. An anchor stitch should be placed at least 0.5 cm above the

apex of the incision followed by running unlocked sutures, placing each stitch approximately 0.5 cm apart. This suture should be carried beyond the anal verge onto the perineum by one stitch. Some providers use interrupted sutures.[23] There are no data to support one method over the other.[23]

Before closure of the external anal sphincter, the internal anal sphincter should be identified and reapproximated. It is typically seen as a pale to white, longitudinal fibrous layer between the rectal mucosa and the external anal sphincter, and it may be retracted laterally. It can vary in thickness and can be difficult to distinguish from the rectal mucosa. An Allis clamp can help with identification when the internal sphincter is retracted. After being identified, it should be reapproximated with a running unlocked or interrupted 2-0 absorbable synthetic suture placed approximately 0.5 cm apart.

The ends of the external anal sphincter must be clearly identified. They typically retract into the capsule laterally and will need to be grasped and brought anteriorly and medially. This can be done using an Allis clamp. The Allis clamp is used to grasp the muscle with its associated posterior and anterior capsules.[23] Historically, there have been two techniques recommended to reapproximate the sphincter: the end-to-end repair and the overlap repair. A 2010 RCT showed that an end-to-end repair was associated with lower rates of anal incontinence compared with the overlap technique. The primary outcome was flatal incontinence at 6 months, and this finding suggests end-to-end repair may be preferable to the overlap technique.[7] A 2013 Cochrane review of six RCTs showed the overlap technique was associated with lower risks for fecal urgency and anal incontinence symptoms.[24] However, there was considerable heterogeneity among the studies, and the experience of the surgeon was not addressed in these trials. The review was unable to recommend one technique over the other.

The external anal sphincter should be reapproximated using several carefully placed 2-0 polyglycolic acid or polydioxanone sulfate interrupted sutures on a large tapered needle. The first suture should be placed at the bottom, which is the most posterior portion of the sphincter. It is important to grasp at least 1 cm of the fascial capsule because this is the most supportive tissue. This should be followed by interrupted sutures at the inferior, superior, and anterior quadrants. Other sutures can be placed as clinically necessary for complete reapproximation of the sphincter. Many providers recommend avoiding figure-of-eight sutures because of the theoretical risk of tissue strangulation with resultant devascularization and poor healing.[23]

At this point, a rectal examination should be performed to check the integrity of the closure. If any defects are detected, they can be closed with interrupted sutures. If sutures are felt in the lumen then the management is controversial and lacking guiding evidence. As one approach to rectal mucosa repair is to tie the knots in the lumen, the presence of a suture from mucosal repair is acceptable. The suture is more problematic if it is a 2-0 PDS suture extending from the external anal sphincter repair because this slowly absorbable thicker suture may be present for 6 months and associated with infection, fistula development, and pain. Therefore, suture removal is recommended. Clinical judgment will be needed in other scenarios such as when 2-0 polyglycolic acid suture is used to repair the internal anal sphincter.

Then the remaining laceration should be examined for depth. To prevent future rectocele development, the rectovaginal fascia (also called the perineal membrane, Denonvilliers fascia, or the rectovaginal septum) should be repaired with interrupted or running 2-0 or 3-0 absorbable synthetic suture. This layer helps with posterior pelvic floor support and extends to the levator ani muscle, sacrum, and the perineal body. Care should be taken to prevent entry into the rectal lumen. Some providers use a running 2-0 absorbable synthetic suture closure. There are no data to support one method over the other. In a third- or fourth-degree laceration, much of the rectovaginal fascia has been exposed and can be repaired as another layer after the internal sphincter. In more superficial vaginal lacerations, the rectovaginal fascia may be repaired as part of the running suture that reapproximates the vaginal mucosa, and the inferior margin of the rectovaginal fascia may be reattached to its insertion on the perineal body muscles.

The remainder of the closure can be performed by the usual technique of a midline episiotomy repair. The vaginal mucosa can be closed with a variety of techniques and sutures, but an absorbable synthetic suture material is recommended.[18] The apex of the incision should be identified, and

the suture should be placed at least 1 cm above the apex to reach the retracted blood vessels to improve hemostasis and reduce risk of hematoma. The vaginal mucosa is then closed using a running or running lock to the level of the hymeneal ring. The suture should have adequate depth to incorporate the rectovaginal septum, but not deep enough to enter the rectum.

The running suture can be brought into the perineal body by driving the needle down through the vaginal mucosa internal to the hymenal ring and coming back out into the perineal body. Alternatively, the running suture can be tied inside the vagina proximal to the hymenal ring if the perineal body will be repaired with interrupted sutures for the transverse perineal and bulbocavernosus muscles. If the running suture approach is used, the option of using this suture to place the *crown stitch* can be considered. The crown stitch is used to reapproximate the perineal body at the attachments of the bulbospongiosus muscles. It is intended to restore the introitus and bring the labia together. Care should be taken to not overly restrict or tighten the introitus, which can lead to dyspareunia.[23] If the single running suture approach is used, deeper interrupted sutures may be necessary to close any open space and restore the perineal anatomy. If interrupted sutures are the primary method of repairing the perineal body, deeper sutures will typically not be needed because the interrupted sutures may be placed deeper and reapproximate a greater amount of perineal muscle.

The suture from the vaginal mucosal closure, or the crown stitch, can be used to close the perineal skin in a subcuticular fashion, or a new stitch of 3-0 or 4-0 absorbable synthetic sutures also can be used. A 2012 Cochrane review that evaluated continuous versus interrupted skin sutures for perineal repair showed that a continuous subcuticular technique was associated with less pain for up to 10 days' postpartum, a reduction in analgesia use, a reduction in dyspareunia, and a decrease in the need for suture removal. There were no differences in the need for resuturing of the wound or long-term pain. There was a greater reduction in pain when continuous suturing was used for all layers.[25]

Several trials of suturing of the perineal skin and/or muscles have been undertaken to determine if there is a difference in healing and postpartum pain. In 2013, the SUNS trial evaluated the outcomes of primiparous women with first- and second-degree perineal lacerations by comparing those with repair of the muscle and skin with those without. The trial showed no difference between the two groups regarding pain. However, difficulty in recruiting limited the study to a small sample size of 74 subjects rather than the 340 required by the power analysis. There was a statistically significant difference in wound closure at 6 weeks' postpartum, with 84% of the sutured group showing complete approximation versus 44% for the unsutured group (95% confidence interval [CI] = 16.5-56.9; $P = 0.001$). However, it is unknown if the delay in achieving complete skin closure had any clinical relevance.[26]

The 1-year follow-up from the Ipswich Childbirth Study comparing a 2-layer closure (leaving the skin edges unsutured) with a 3-layer closure showed a decreased likelihood of the perineum feeling *different* than before childbirth in the unsutured group.[27] A 2003 Nigerian study of 823 women compared a 2-layer closure to a 3-layer closure. That study showed that women with a 2-layer closure had less perineal pain at 48 hours (57% versus 65%; relative risk [RR] = 0.87; 95% CI = 0.78-0.97) and 14 days (22% versus 28%; RR = 0.77; 95% CI = 0.61-0.98), less need for suture removal (6% versus 10%; RR = 0.62; 95% CI = 0.39-0.99), and less dyspareunia at 3 months (6% versus 12%; RR = 0.52; 95% CI = 0.33-0.81).[28] Rates of wound healing were similar. A 2015 systematic review of six studies including 2,922 postpartum women also showed decreased short-term pain when leaving the skin unsutured. Skin separation was not greater in the unsutured group if skin adhesives were used.[29]

After the repair, care should be taken to ensure all vaginal packing, sponges, and instruments have been removed from the vagina. A retained sponge can increase the risk of postoperative infection and repair breakdown. Removal can be accomplished by a simple postprocedure vaginal examination ensuring the final count of all needles, laparotomy pads and sponges, and instruments are correct. Performing a final count will help ensure that all foreign bodies have been removed from the vagina. When removal of a sponge is difficult, an Allis clamp can be passed along a finger and the sponge grasped. A postoperative rectal examination is recommended to ensure that all rectal mucosal defects have been repaired and that the anal sphincter mass feels adequate. Optimally,

these examinations are performed after the repair of the rectal mucosa and external anal sphincter is complete. The repair should not proceed if any layer has not been adequately reapproximated.

After repair of an anal sphincter laceration the physician should document a comprehensive operative note including a detailed description of the laceration, a summary of the steps taken to repair it, a statement about the postoperative repair examination, and confirmation that the final operative count was correct. An example of a concluding statement is: *Postoperative examination revealed the vaginal and perineal laceration to be well approximated without active bleeding or hematoma. The rectal examination revealed good sphincter approximation and no palpable sutures or rectal defects. Final operative count was confirmed to be correct.*

The Complicated Repair

No two lacerations are identical. Some have lateral extensions, and some have multidirectional extensions. Others may extend into the lateral vascular bundles and cause heavy bleeding. Some women may experience extreme pain and be unable to tolerate the repair under local or regional anesthesia. A urinary catheter should be placed before suturing periurethral lacerations that extend close to the urethral opening. The provider must be prepared to request a consultation if having difficulty with the repair or lacking the necessary expertise.

In addition, it is important to address heavy bleeding in a timely manner. Women can quickly lose large volumes of blood through perineal lacerations, and it may be difficult for the provider to estimate the blood loss because of the absorption by drapes, vaginal packing, and sponges. At the end of the procedure a quantitative blood loss estimate can be obtained by weighing each blood-soaked item and subtracting the dry weights of the items. The provider should first expose the laceration and gain hemostasis. A figure-of-eight suturing with a 3-0 absorbable synthetic suture may stop an arterial bleed. If the upper limit of a sulcus laceration cannot be visualized, place a suture as far into the vagina as possible and retract downward. In some instances, applying pressure may help. When blood loss is excessive, consider transfer of the woman to the operating room for improved lighting and pain control, consultation, intravenous fluids, blood transfusion, and evaluation for disseminated intravascular coagulation. Additional information is available in the *Postpartum Hemorrhage* and *Maternal Resuscitation and Trauma* chapters. After hemostasis is obtained, the repair can be completed as previously discussed.

For lacerations that extend in multiple directions, the provider should first concentrate on those that are bleeding. The extensions should be closed in order of depth, with the deeper lacerations closed first and the superficial closed last. The provider should attempt to close the rectal mucosa and anal sphincter as soon as hemostasis is obtained and deep lacerations are closed. Care must be taken not to close the laceration and restrict exposure to the rectal mucosa and/or anal sphincter when addressing hemostasis before repair of the anal sphincter complex.

Antibiotics

One trial in a Cochrane review of antibiotic prophylaxis found that a single, intravenous dose of a second-generation cephalosporin helped to prevent perineal wound complications.[30] However, the review stated that the results should be interpreted with caution because the findings were based on one trial with a high loss to follow-up. After the Cochrane review, a retrospective study and a prospective study showed that intrapartum antibiotics were protective against wound complications in patients with an anal sphincter injury and repair.[31,32] A 2018 ACOG Practice Bulletin states that a single dose of antibiotics for prophylaxis for anal sphincter lacerations is reasonable but that more research is needed.[1]

Complications

It has been difficult to quantify the rates of complication from third- and fourth-degree lacerations because of the lack of uniformity in the literature in describing complications. The risks of wound infection and wound breakdown after laceration repair were observed to be 20% and 25%, respectively.[1] The most common complications are listed in *Table 6*.

The factors that lead to complications are numerous, but the most common is tissue breakdown secondary to infection.[33] Poor approximations due to inadequate surgical technique or postoperative hematoma also can result in severe complications. A previous vaginal delivery has been shown to be protective against breakdown of a perineal laceration repair (odds ratio = 0.14; 95% CI

Table 6. Complications of Third- and Fourth-Degree Laceration Repair

Short Term:	Long Term:
Dehiscence	Anal incontinence
Hematoma	Dyspareunia
Infection (superficial cellulitis, necrotizing fasciitis)	Rectocutaneous fistula
Perineal abscess	Rectovaginal fistula

Information from American College of Obstetricians and Gynecologists. ACOG Practice Bulletin no. 198: Prevention and management of obstetric lacerations at vaginal delivery. Obstet Gynecol. *2018;132:e87–e102; Homsi R, Daikoku NH, Littlejohn J, Wheeless CR Jr. Episiotomy: risks of dehiscence and rectovaginal fistula.* Obstet Gynecol Surv. *1994;49(12):803-808; Smith LA, Price N, Simonite V, Burns EE. Incidence of and risk factors for perineal trauma: a prospective observational study.* BMC Pregnancy Childbirth. *2013;13:59.*

Table 7. Factors Contributing to Third- and Fourth-Degree Repair Complications

Anemia	Infection
Blunt or penetrating trauma	Inflammatory bowel disease
Cigarette smoking	Malnutrition
Connective tissue disease	Obesity
Constipation	Poor perineal hygiene
Endometriosis	Poor tissue approximation (poor surgical technique)
Forceful coitus	Prior pelvic radiation
Hematologic disease	
Hematoma	

Information from American College of Obstetricians and Gynecologists. ACOG Practice Bulletin no. 198: Prevention and management of obstetric lacerations at vaginal delivery. Obstet Gynecol. *2018;132:e87–e102; Homsi R, Daikoku NH, Littlejohn J, Wheeless CR Jr. Episiotomy: risks of dehiscence and rectovaginal fistula.* Obstet Gynecol Surv. *1994;49(12):803-808; Oboro VO, Tabowei TO, Loto OM, Bosah JO. A multicentre evaluation of the two-layered repair of postpartum perineal trauma.* J Obstet Gynaecol. *2003;23(1):5-8.*

= 0.05-0.3).[34] Stool softeners are recommended for at least 2 weeks after an anal sphincter laceration. Discharge instructions should encourage rapid follow-up if infection is suspected. Women may be reluctant to discuss anal incontinence, therefore a routine 2-week follow-up for all third- and fourth-degree lacerations may be considered. *Table 7* lists contributing factors for repair complications.

Necrotizing fasciitis is a severe infection due to multiple bacterial pathogens, particularly anaerobes. Although rare, necrotizing fasciitis of the perineum has a high rate of morbidity and mortality and it occurs most often in women with insulin-dependent diabetes, cancer, or an immunosuppressive disorder. It manifests as a cyanotic discoloration of the wound and a loss of sensation. This infection should be aggressively managed with surgical debridement and broad-spectrum antibiotics.

Summary

Third- and fourth-degree perineal lacerations are common complications of all types of vaginal deliveries but are significantly increased after an assisted vaginal delivery or in the presence of a midline episiotomy. These lacerations typically heal well when repaired appropriately in the absence of infection, but a substantial proportion of women will have long-term anal incontinence symptoms. Complications can occur, with the most severe being rectovaginal fistula and necrotizing fasciitis. Women who sustain these lacerations require close observation and aggressive treatment of wound infections. Most treatment is based on expert opinion, but there is some evidence to guide the evaluation and treatment of these lacerations (*Table 8*).[18,25-29]

Table 8. Summary of Evidence

Outcome	% of Women with Outcome		Odds Ratio or Relative Risk (95% Confidence Interval)
	Vacuum	**Forceps**	
Significant maternal injury	9.8%	20.3%	0.41 (0.33, 0.50)
	Synthetic	**Catgut**	
Pain at 3 days	54.3%	65.4%	0.62 (0.54, 0.71)
Analgesic use	18.4%	24.2%	0.63 (0.52, 0.77)
Suture dehiscence	2.9%	5.4%	0.45 (0.29, 0.70)
Suture removal	18%	10.1%	2.01 (1.56, 2.58)
	Continuous	**Interrupted**	
Short-term pain	20.3%	27.3%	0.68 (0.53, 0.86)
Suture removal	26%	36.8%	0.61 (0.46, 0.80)
	2-layer[a]	**3-layer**	
Perineal pain at 24 to 48 hours	57%	65%	0.87 (0.78, 0.97)
Perineal pain at 14 days	22%	28%	0.77 (0.61, 0.98)
Suture removal	6%	10%	0.62 (0.39, 0.99)
Dyspareunia at 3 months	6%	12%	0.52 (0.33, 0.81)

[a]A 2-layer closer leaves the skin unsutured.

NNH = number needed to harm; NNT = number needed to treat.

Information from various sources.

Nursing Considerations: Third- and Fourth-Degree Perineal Lacerations

- Advocate for preventive measures during labor and delivery per patient preferences, including maternal positions
- Encourage skin-to-skin contact with neonate during repair process, as appropriate
- Facilitate adequate pain relief
- Be familiar with the equipment necessary for repairs
- Provide adequate lighting for visualization
- Educate patients on the necessity of good perineal hygiene, good nutrition, preventing constipation, not smoking, and nothing in the vagina to decrease postpartum complications

References

1. Committee on Practice Bulletins-Obstetrics. ACOG Practice Bulletin no. 198: prevention and management of obstetric lacerations at vaginal delivery. *Obstet Gynecol*. 2018;132(3):e87-e102.
2. Pergialiotis V, Vlachos D, Protopapas A, et al. Risk factors for severe perineal lacerations during childbirth. *Int J Gynaecol Obstet*. 2014; 125(1):6-14.
3. Jiang H, Qian X, Carroli G, Garner P. Selective versus routine use of episiotomy for vaginal birth. *Cochrane Database Syst Rev*. 2017;2: CD000081.
4. Rogers RG, Leeman LM, Migliaccio L, Albers LL. Does the severity of spontaneous genital tract trauma affect postpartum pelvic floor function? *Int Urogynecol J Pelvic Floor Dysfunct*. 2008;19(3): 429-435.
5. O'Mahony F, Hofmeyr GJ, Menon V. Choice of instruments for assisted vaginal delivery. *Cochrane Database Syst Rev*. 2010;(11): CD005455.
6. Klein MC, Gauthier RJ, Robbins JM, et al. Relationship of episiotomy to perineal trauma and morbidity, sexual dysfunction, and pelvic floor relaxation. *Am J Obstet Gynecol*. 1994;171(3):591-598.
7. Farrell SA, Gilmour D, Turnbull GK, et al. Overlapping compared with end-to-end repair of third- and fourth-degree obstetric anal sphincter tears: a randomized controlled trial. *Obstet Gynecol*. 2010; 116(1):16-24.
8. Mahony R, Behan M, Daly L, et al. Internal anal sphincter defect influences continence outcome following obstetric anal sphincter injury. *Am J Obstet Gynecol*. 2007;196(3):217.e1-217.e5.
9. Daniilidis A, Markis V, Tzafetas M, et al. Third degree perineal lacerations — how, why and when? A review analysis. *Open J Obstet Gynecol*. 2012;2:304-310.
10. Eason E, Labrecque M, Wells G, Feldman P. Preventing perineal trauma during childbirth: a systematic review. *Obstet Gynecol*. 2000;95(3):464-471.
11. Dahlen HG, Homer CS, Cooke M, et al. Perineal outcomes and maternal comfort related to the application of perineal warm packs in the second stage of labor: a randomized controlled trial. *Birth*. 2007;34(4):282-290.
12. Albers LL, Sedler KD, Bedrick EJ, et al. Midwifery care measures in the second stage of labor and reduction of genital tract trauma at birth: a randomized trial. *J Midwifery Womens Health*. 2005;50(5): 365-372.
13. Soong B, Barnes M. Maternal position at midwife-attended birth and perineal trauma: is there an association? *Birth*. 2005;32(3): 164-169.
14. Shorten A, Donsante J, Shorten B. Birth position, accoucheur, and perineal outcomes: informing women about choices for vaginal birth. *Birth*. 2002;29(1):18-27.
15. de Souza Caroci da Costa A, Gonzalez Riesco ML. A comparison of "hands off" versus "hands on" techniques for decreasing perineal lacerations during birth. *J Midwifery Womens Health*. 2006;51(2): 106-111.
16. McCandlish R, Bowler U, van Asten H, et al. A randomised controlled trial of care of the perineum during second stage of normal labour. *Br J Obstet Gynaecol*. 1998;105(12):1262-1272.
17. Andrews V, Sultan AH, Thakar R, Jones PW. Occult anal sphincter injuries—myth or reality? *BJOG*. 2006;113(2):195-200.
18. Kettle C, Dowswell T, Ismail KM. Absorbable suture materials for primary repair of episiotomy and second degree tears. *Cochrane Database Syst Rev*. 2010;(6):CD000006.
19. Dencker A, Lundgren I, Sporrong T. Suturing after childbirth—a randomised controlled study testing a new monofilament material. *BJOG*. 2006;113(1):114-116.
20. Anderson D. Pudendal nerve block for vaginal birth. *J Midwifery Womens Health*. 2014;59(6):651-659.
21. Wheeler AS. Fetal toxicity of local anethetics. *AANA J*. 1985;53(4): 332-337.
22. World Health Organization. Section 3: Procedures. In: *Managing Complications in Pregnancy and Childbirth: A guide for midwives and doctors*. Geneva, Switzerland: World Health Organization; 2000.
23. Yeomans ER, Hoffman BI, Gilstrap LC, Cunningham FG. Episiotomy and Obstetric Anal Sphincter Lacerations. In: Yeomans ER, Hoffman BI, Gilstrap LC, Cunningham FG, eds. *Cunningham and Gilstrap's Operative Obstetrics*. 3rd ed. New York, NY: McGraw-Hill Education, 2017; 320-334.
24. Fernando RJ, Sultan AH, Kettle C, Thakar R. Methods of repair for obstetric anal sphincter injury. *Cochrane Database Syst Rev*. 2013; (12):CD002866.
25. Kettle C, Dowswell T, Ismail KM. Continuous and interrupted suturing techniques for repair of episiotomy or second-degree tears. *Cochrane Database Syst Rev*. 2012;11:CD000947.
26. Fleming VE, Hagen S, Niven C. Does perineal suturing make a difference? The SUNS trial. *BJOG*. 2003;110(7):684-689.
27. Grant A, Gordon B, Mackrodat C, et al. The Ipswich childbirth study: one year follow up of alternative methods used in perineal repair. *BJOG*. 2001;108(1):34-40.
28. Oboro VO, Tabowei TO, Loto OM, Bosah JO. A multicentre evaluation of the two-layered repair of postpartum perineal trauma. *J Obstet Gynaecol*. 2003;23(1):5-8.
29. Seijmonsbergen-Schermers AE, Sahami S, Lucas C, Jonge Ad. Nonsuturing or skin adhesives versus suturing of the perineal skin after childbirth: a systematic review. *Birth*. 2015;42(2):100-115.
30. Buppasiri P, Lumbiganon P, Thinkhamrop J, Thinkhamrop B. Antibiotic prophylaxis for third- and fourth-degree perineal tear during vaginal birth. *Cochrane Database Syst Rev*. 2014;(10):CD005125.
31. Stock L, Basham E, Gossett DR, Lewicky-Gaupp C. Factors associated with wound complications in women with obstetric anal sphincter injuries (OASIS). *Am J Obstet Gynecol*. 2013;208(4):327.e1-327.e6.
32. Lewicky-Gaupp C, Leader-Cramer A, Johnson LL, et al. Wound complications after obstetric anal sphincter injuries. *Obstet Gynecol*. 2015;125(5):1088-1093.
33. Homsi R, Daikoku NH, Littlejohn J, Wheeless CR Jr. Episiotomy: risks of dehiscence and rectovaginal fistula. *Obstet Gynecol Surv*. 1994;49(12):803-808.
34. Jallad K, Steele SE, Barber MD. Breakdown of perineal laceration repair after vaginal delivery: a case-control study. *Female Pelvic Med Reconstr Surg*. 2016;22(4):276-279.

Diagnostic Ultrasound in Labor and Delivery

Learning Objectives

1. Identify indications for diagnostic ultrasound during labor and delivery.
2. Demonstrate point-of-care ultrasound techniques commonly used in the care of women receiving maternity care.
3. Interpret diagnostic imaging results and render treatment accordingly.
4. Accurately document findings using an ultrasound report in compliance with nationally recognized standards.

Introduction

Diagnostic ultrasonography is an essential tool in pregnancy management. Every maternity care provider can benefit from familiarity with ultrasound scanning. Bedside ultrasound can facilitate point-of-care clinical decisions including evaluation for fetal demise; determining fetal number, presentation, and position; assessing amniotic fluid status; and evaluating placental location. Practice is essential before using ultrasound for patient care. With additional training and practice, basic biometry can be learned to determine fetal gestational age, which is a particularly useful skill when a woman who has not received prenatal care presents in labor. Advanced applications such as an anatomic survey, cervical length measurements and umbilical Doppler ultrasound require a considerable amount of additional training and practice and are beyond the scope of this chapter.

Indications

The majority of women who are pregnant will develop one or more recognized indications for diagnostic ultrasonography during pregnancy. Indications for medical use of diagnostic ultrasound were first developed by a National Institutes of Health Consensus Development Conference in 1984 and have been reaffirmed over time by professional organizations, including the American Institute of Ultrasound in Medicine (AIUM), American College of Radiology (ACR), and American College of Obstetricians and Gynecologists (ACOG).[1-3] First-trimester ultrasound indications include: confirmation of intrauterine pregnancy, estimation of gestational age, evaluation of fetal viability, fetal number, suspected ectopic pregnancy, vaginal bleeding, pelvic pain, suspected gestational trophoblastic disease, pelvic masses or uterine anomalies, and adjunct to procedure (eg, chorionic villus sampling, intrauterine device removal). Second- and third-trimester ultrasound indications include: assessment of fetal anatomy, screening for anomalies, estimation of gestational age, evaluation of fetal number, cervical length, fetal growth, size dates discrepancy, fetal presentation, fetal viability, antenatal fetal assessment, amniotic fluid quantity, vaginal bleeding, abdominal or pelvic pain, placental location, suspected gestational trophoblastic disease, pelvic masses or uterine anomalies, and adjunct to procedures (eg, external cephalic version, amniocentesis).[2]

Types of Ultrasound Examinations During Pregnancy

Professional organizations also have agreed on the types of ultrasound examinations and the standard content, nomenclature, and required documentation.[2,3] The term *standard examination* has replaced the term *complete examination*. The terms *Level I* and *Level II examination* are also no longer used. A first trimester standard examination should document evaluation of the embryo/fetus, uterus, and adnexa. A second- or third-trimester standard examination should document biometry, anatomic survey, placental position, amniotic fluid assessment, and fetal number, viability, and presentation. The cervix and adnexa are evaluated when possible and appropriate. A *limited examination* is focused on answering a specific question such as fetal presentation, amniotic fluid assessment or fetal viability. *Specialized examinations* include fetal echocardiography and umbilical Doppler ultrasound and are for certain indications but are not a part of routine standard examinations.[3] By definition, scans performed in labor and delivery to answer specific clinical questions are limited examinations.

This chapter divides applications of limited examinations into *basic* and *advanced* based on how readily providers can typically learn them. Practicing providers with a base of knowledge in maternal-fetal anatomy and physiology often master the basic applications in a 1-day workshop. Significant additional study and supervised practice is necessary to learn the advanced applications.[4-6] Knowledge of ultrasound diagnosis by maternity care providers in labor and delivery is especially valuable in areas with limited access to hospital-based sonographers.

Liability

Ultrasound scanning at the point of care can provide answers needed to guide clinical management, improve patient care, and decrease liability. Providers who use ultrasound should be familiar with the available equipment and have training in scanning and interpretation of findings. Consultation should be obtained when the diagnosis or management plan is in doubt. Some institutions or practices may require specific credentialing for point-of-care ultrasound scanning.

When a limited scan is performed, the woman should be counseled on the limitations of the examination. Verbal and/or written consent should be obtained and documented.

To avoid missing major anomalies, if a woman has not undergone a standard examination/anatomic survey before a limited scan, one can be obtained afterward, assuming the woman has not already delivered.

Technical Considerations

Maternity care providers should have a basic understanding of ultrasound technology and its limitations and be familiar with the equipment available in their respective labor and delivery department. Essential baseline knowledge includes selection of the proper transducer and frequency, operation of gain and depth controls, performance of measurements, and, when required, storage of images. *Table 1* defines basic ultrasound terms and concepts. Written documentation should include indication and limited scope of the scan, findings, and a management plan. The *Appendix* presents a sample form for recording findings of a labor and delivery scan that can be customized based on local protocols.

The labor and delivery scan is a limited scan. A standard examination would also include documentation of an anatomic survey commenting on:

- Head, face, and neck: Lateral cerebral ventricles, choroid plexus, midline falx, cavum septi pellucidi, cerebellum, cisterna magna, and upper lip
- Heart: Four-chamber view, heart size, and position; left ventricular outflow tract; right ventricular outflow tract; and 3-vessel view and 3-vessel trachea view, if technically feasible
- Abdomen: Stomach (presence, size, and situs), kidneys, urinary bladder, umbilical cord insertion site into the fetal abdomen, and umbilical cord vessel number
- Spine: Cervical, thoracic, lumbar, and sacral spine
- Extremities: Presence of legs and arms and presence of hands and feet
- External genitalia: In multiple gestations and when medically indicated.[2]

Scanning Techniques

This chapter provides background information on common labor and delivery ultrasound applications. Scanning techniques for the basic applications are introduced in the corresponding optional workshop portion of this ALSO course. Advanced applications require additional training, techniques, and practice.

Transabdominal scanning can be performed in any trimester and during labor. Limitations include fetal position, decreased amniotic fluid (oligohydramnios) with loss of acoustic window, fetal crowding at advanced gestational age, low fetal station, or maternal obesity.

1. Position the woman for comfort; a left lateral tilt can decrease the incidence of supine hypotension. Discuss the reason for and scope of the examination.
2. Turn on the ultrasound machine and enter the woman's name, date, and other required identifying information according to local protocols.
3. Apply scanning gel to the transducer scanning surface and/or the woman's abdomen and hold the transducer so that the image is oriented properly on the video screen. By convention, scanning is performed with the examiner on the woman's right side. Transducer position and image orientation are described relative to the maternal body, rather than the body of the

Table 1. Diagnostic Ultrasound Terms and Physical Concepts

Transducers and Image Formation

Transducers are constructed from piezoelectric crystals. When a driving voltage is applied to a piezoelectric crystal, it vibrates and emits sound energy. The same crystals detect returning, reflected sound energy and convert it to tiny electrical impulses. Computation of the timing and intensity of these impulses is made to display an image. Sequential firing of the crystals creates a two-dimensional *slice* and produces a moving *real-time* image when rapidly updated

Scanning or Transducer Frequency

Transducers for transabdominal scanning in the third trimester are 3 or 3.5 MHz. Many modern transducers are variable frequency, which permits scanning at 5 MHz in earlier pregnancy and 3 to 3.5 MHz later in pregnancy. A 3 MHz transducer may be used in early pregnancy for overweight patients when more depth of view is needed. Transvaginal transducers use higher frequencies (5 to 10 MHz) for very early pregnancy scanning and assessment of the cervix and endocervical area. Higher frequency transducers produce improved resolution but have shallower depth of view

Power

Diagnostic ultrasound is sound energy. The energy output of diagnostic ultrasound scanners may be fixed or variable and is regulated by the United States Food and Drug Administration. If the power is variable, the lowest energy output that produces a readable image should be used

Gain and Time Gain Compensation

Returning echoes are weak and must be amplified in general (overall gain) and selectively by depth within the image TGC to produce a visible image. The gain controls are different from *brightness* controls on the video monitor. Gain controls must typically be readjusted between patients and when scanning different areas of the same patient to optimize the image

Acoustic Windows

Sound waves must reach the object of interest before echoes can be reflected to produce an image. Fluids, such as urine and amniotic fluid, allow sound waves to pass freely and act as *acoustic windows* to the structures beneath or within them

Acoustic Shadows

When adjacent tissues differ widely in density, strong echoes are produced and no sound is transmitted. Tissue-bone and gas-tissue interfaces are examples of areas that act like mirrors, reflecting approximately all the sound energy that strikes them and producing a shadow that conceals underlying structures. No amount of increased gain or power will enable the operator to see through such a mirror; instead the transducer position must be changed to avoid the obstruction. Using sufficient gel can avoid air-tissue shadows. Filling the umbilicus with gel can avoid an artifact when it is above the area being scanned

B Mode

Brightness mode is the normal mode in which two-dimensional, real-time scanning is performed. Echoes from structures within the body are displayed with varying degrees of brightness with depth on the vertical axis and width on the horizontal axis of the image

M Mode

This mode displays a single line of image on the vertical axis and time on the horizontal axis. It is useful to document motion, especially fetal cardiac motion

Doppler Velocimetry

This modality measures the velocity of blood flow and displays it as a waveform. It is useful for measuring blood flow in uterine and fetal vessels, including the umbilical cord. The use of Doppler ultrasound in high-risk pregnancies appears to improve many obstetric care outcomes and appears promising in helping to reduce perinatal mortalities

Color Doppler Imaging

This modality displays velocity and direction of blood flow within the image as color. An important example of application of this technique is the detection of fetal cardiac defects. Because color corresponds to whether the flow is towards or away from the transducer, arteries may appear blue and veins red, or vice versa

Power Doppler Imaging

This modality displays the volume of blood flow within the image as color without regard to its velocity or direction. This technique is useful for distinguishing one tissue from another and identifying umbilical cord within amniotic fluid

TGC = time gain compensation.

Information from Norton M. Callen's Ultrasonography in Obstetrics and Gynecology. 6th ed. Elsevier; 2016; Alfirevic Z, Stampalija T, Gyte GM. Fetal and umbilical Doppler ultrasound in high-risk pregnancies. Cochrane Database Syst Rev. *2013;11:CD007529.*

fetus. With transabdominal scanning in a sagittal plane, the woman's head is off the left side of the screen and her feet are off the right side of the screen. While scanning in a transverse plane, the woman's right is off the left side of the screen and her left is off the right side of the screen, viewed as if standing at her feet and looking up towards her head. This convention is the same as that used for other cross-sectional imaging techniques such as computed tomography and magnetic resonance imaging.

4. Make necessary gain and depth adjustments while performing a series of longitudinal and transverse sweeps of the maternal abdomen and viewing all quadrants of the uterus to note:
   - Fetal cardiac motion
   - Number of fetuses
   - Fetal lie, presentation, and position

- Quantity of amniotic fluid: measuring largest vertical pocket or four quadrant amniotic fluid index
- Placental location.

5. Print labeled images of significant findings for the medical record. When finished, record the findings and plan and discuss these with the woman.

Transvaginal scanning is essential during the first trimester to adequately visualize the uterus, adnexa, cul-de-sac, early gestational sac, and embryo (see the *First-Trimester Pregnancy Complications* chapter). It is particularly useful for estimating gestational age and setting the estimated due date, especially when the woman is unsure of her last menstrual period. Transvaginal scanning also is useful during the second and third trimesters to visualize the cervix and endocervical area in cases of preterm labor, incompetent cervix, and placenta previa. Skill in transvaginal scanning is not considered to be a basic skill that can be learned in this chapter's corresponding workshop.

The following features apply to transvaginal scanning:

1. After discussion of the reason for and scope of the examination, the woman is placed in lithotomy position
2. Scan gel is applied to the tip of the transvaginal transducer, which is then covered with a clean condom, examination glove, or commercial transducer sheath. Lubricating gel is applied over the cover and the transducer tip is introduced into the vagina. Sonographic visualization starts as soon as the transducer is introduced. Inserting the transducer tip too far can cause the examiner to miss seeing the cervix and lower uterine segment as the transducer tip reaches the posterior or anterior fornix
3. Gain and depth controls are adjusted, and the transducer tip is positioned to obtain whatever sagittal, coronal, or oblique views are necessary to visualize the structures. The vaginal opening acts as a fulcrum, making it necessary to move the transducer handle toward the opposite side of the body that the examiner wants to visualize. For example, to view the left adnexal area, the transducer handle is moved to the woman's right, and to look toward the bladder, the handle is lowered toward the woman's rectum. Fetal heart rate (FHR) is measured and documented using M mode. It is not recommended for operators to use spectral Doppler in the first trimester, as the energy levels delivered to the embryo are higher than B or M mode scanning. The use of the lowest gain settings and time spent scanning to obtain satisfactory diagnostic images are important aspects of the imaging safety principle of ALARA (as low as reasonably achievable)[2]
4. Transvaginal sagittal images are oriented like transabdominal images, with the woman's head off the left of the screen and feet to the right. In coronal planes, the woman's right shoulder is off the left of the screen and left shoulder is off the right side of the screen.

Scanning by the transperineal route is a useful alternative to transvaginal scanning during the second and third trimesters to visualize the cervix and endocervical area. This can be particularly helpful in cases of preterm labor, incompetent cervix, and suspected placenta previa, because the vagina is not entered. When considering manual rotation or assisted vaginal delivery, transperineal ultrasound can occasionally help to identify occiput posterior position, brow presentation, and asynclitism when assessment via vaginal examination or abdominal ultrasound is difficult.[7] Although the same transducer is used in transperineal and transabdominal scans, a wider field of vision is obtained than when a transvaginal transducer is used.

To perform transperineal scanning, gel is applied to the scanning surface of a transabdominal transducer, which is then covered with a clean or sterile glove. Lubricating gel is applied over the glove, and the transducer face is placed against the introitus and perineum. Transperineal images are oriented the same as transvaginal images but have a lower resolution than transvaginal scans. Skill in transperineal scanning is not considered to be a basic skill that can be learned in the workshop associated with this ALSO course.

Basic Applications

Basic applications include determining that the fetus is alive by visualizing the heartbeat, diagnosing fetal number, visualizing fetal lie, making a quantitative assessment of amniotic fluid volume, and determining basic placental location.

Fetal Life

If a fetal heartbeat is not identified via auscultation or Doppler, determining fetal life is a critical first step in management. Cardiac activity can be

documented on a still image with the use of an M mode tracing. Sonographic characteristics of fetal demise are listed in *Table 2*. If the findings are in doubt, consultation should be obtained before a final diagnosis is made. When fetal demise is diagnosed, the approach to the woman and family must be individualized, while maintaining communication and avoiding medical jargon. During real-time scanning, the presence or absence of cardiac activity is typically apparent to the examiner and the woman. After stating simply and directly the diagnosis of fetal demise, it is best to interrupt further examination and allow the woman and family the opportunity to react. Offer them private time before proceeding with the collection of additional data, imaging, or explanations. The *Birth Crisis* chapter offers additional guidance on dealing with pregnancy loss.

Fetal Number

A surprise twin at delivery should be a rarity in high-resource settings. Although it may seem straightforward to determine fetal number with ultrasound, missing a twin does happen. Use a structured approach while scanning all four quadrants in longitudinal and sagittal planes. Two separate heads, spines, and heartbeats should be visualized before making the diagnosis of twins. The fetal spine should be followed from the head to the sacrum, and the fundus should be evaluated for fetal parts. The false diagnosis of twins can be equally problematic. This can occur when the same structure, such as head or heartbeat, is viewed from two different angles. If more than one fetus is seen, further examination is warranted to avoid missing higher order multiples.

Fetal Presentation, Lie, and Position

Abnormal fetal presentation, lie, or position are common in labor; additional information is available in the *Malpresentations, Malpositions, and Multiple Gestations* chapter. Leopold maneuvers and vaginal palpation can often confirm fetal presentation, but approximately 30% of breech presentations are missed with Leopold maneuvers.[8] Ultrasound can be more definitive, but care should be taken not to mistake every round object for the head. Visualizing orbits, the falx cerebri, and the spine leading to the head can verify that a round structure is indeed the fetal head. *Table 3* defines the terminology related to fetal orientation.

By making a series of longitudinal and transverse sweeps of the maternal abdomen, presentation, lie, and position are typically quite clear. If the fetal lie is transverse and the woman is in labor, it is helpful to know if the spine is up or down in relation to the lower uterine segment. The risk of cord prolapse increases if the spine is up, and cesarean delivery may be difficult when the spine is down. Identifying the position of the spine may affect the type of uterine incision chosen at the time of cesarean delivery or consideration of intraabdominal version to facilitate delivery in cephalic or breech presentation. Ultrasound can be used after manual rotation to confirm success or failure.

Some details of fetal presentation, including compound presentations, cord presentations, and nuchal cord, may be difficult or impossible to visualize. If cord presentation or vasa previa is suspected, color or power Doppler may be indicated to rapidly clarify the diagnosis because blood flow will be readily detected.

Table 2. Sonographic Features of Fetal Demise

Absence of cardiac motion
Absence of fetal movement
Hydropic changes (skin and subcutaneous edema, pleural and pericardial effusions, placental edema)
Abnormal lie
Overlapping skull bones
Oligohydramnios is common, but polyhydramnios also may be present depending on the underlying pathology

Information from Norton M. Callen's Ultrasonography in Obstetrics and Gynecology. *6th ed. Elsevier; 2016; American College of Obstetricians and Gynecologists. ACOG Practice Bulletin no. 200 summary: early pregnancy loss.* Obstet Gynecol. *2018;132(5):1311-1313.*

Table 3. Fetal Orientation Terminology

Presentation: Fetal part located over the maternal pelvic inlet (eg, cephalic, breech)
Lie: Orientation of the fetal spine to the maternal spine (eg, longitudinal, transverse, oblique)
Position: Orientation of presenting fetal part to the maternal pelvis (eg, occiput anterior, occiput posterior, sacrum anterior)

Basic Placental Location

The differential diagnosis of late pregnancy bleeding includes placenta previa and abruption. Additional information on vaginal bleeding is available in the *Late Pregnancy Bleeding* chapter. For women who present with bleeding, a rapid diagnosis is essential but should never delay delivery for an unstable woman or fetus, if indicated. Determining placental location is also important before cesarean delivery, particularly if low anterior placenta was previously diagnosed or suspected. Placenta previa or implantation over a previous scar significantly increases the likelihood of abnormal placental attachment (ie, placenta accreta, increta, percreta) and the woman's risk of severe hemorrhage and need for cesarean hysterectomy. The placenta is typically more echogenic than the myometrium, but uterine contractions can alter the apparent location, appearance, and thickness of the placenta in relation to the endocervical area. A distended bladder can lead to a false diagnosis of placenta previa. Crowding and oligohydramnios can make it difficult to visualize the placenta. Posterior locations are obscured by acoustic shadows of the fetus, and succenturiate lobes may be difficult to locate. Examining for placenta previa and abruption is difficult and not considered a basic application. However, it may be easy to discern that an anterior placenta ends distant from the cervix, excluding the diagnosis of placenta previa in the evaluation of second- or third-trimester bleeding.

Placental abruption also can manifest with vaginal bleeding and is an obstetric emergency that is primarily a clinical diagnosis. Ultrasound can be an adjunctive diagnostic tool, but as with placenta previa, ultrasound should never delay clinically indicated treatment. Even when used by experienced providers, ultrasound can miss more than 50% of placental abruptions. Ultrasound is used in the setting of second- and third-trimester bleeding to evaluate for placenta previa or placental abruption. If an abruption is seen, this may support the clinical diagnosis; however, because of its low sensitivity, even for large abruptions, ultrasound cannot rule out placental abruption.[9]

Amniotic Fluid Assessment

The ability to determine a subjective or objective adequacy of amniotic fluid has many applications. Amniotic fluid is a function of placental health and fetal well-being. Several obstetric complications as well as fetal anomalies can cause a high (polyhydramnios) or a low (oligohydramnios) amniotic fluid volume. Techniques of quantitative amniotic fluid assessment include an amniotic fluid index (AFI) or a single largest vertical pocket (LVP) determination. Other terms synonymous with LVP include: deepest vertical pocket, single deepest pocket, and maximum vertical pocket. *Table 4* includes definitions of quantitative amniotic fluid assessment.

Assessment of amniotic fluid volume is an essential part of biophysical testing and can provide ancillary evidence of prior membrane rupture. To perform an AFI, the LVP of amniotic fluid in each of the four quadrants of the uterus is measured and the sum is computed. The transducer should be perpendicular to the floor, and the measured pockets should not contain umbilical cord or fetal extremities. The operator may use color Doppler ultrasound to document absence of cord in a measured amniotic fluid pocket.

Measurement of the single LVP is performed by identifying the single largest pocket of fluid that is free of cord or fetal parts. The vertical dimension relative to the transducer should normally measure at least 2 cm and less than 8 cm. The LVP has become the preferred parameter because of higher specificity over AFI.[10]

Oligohydramnios in a pregnancy is an indication for further testing or delivery. Oligohydramnios

Table 4. Amniotic Fluid Quantitative Assessment Terminology

	Amniotic Fluid Index (cm)	Largest Vertical Pocket (cm)
Oligohydramnios	<5	<2
Borderline (equivocal)	5 to 8	
Normal	8 to 23	2-7
Polyhydramnios	≥24	>8

Information from Reddy UM, Abuhamad AZ, Levine D, Saade GR; Fetal Imaging Workshop Invited Participants. Fetal Imaging: Executive Summary of a Joint Eunice Kennedy Shriver National Institute of Child Health and Human Development, Society for Maternal-Fetal Medicine, American Institute of Ultrasound in Medicine, American College of Obstetricians and Gynecologists, American College of Radiology, Society for Pediatric Radiology, and Society of Radiologists in Ultrasound Fetal Imaging Workshop. Am J Obstet Gynecol. *2014;210(5):387-397; Committee on Practice Bulletins—Obstetrics and the American Institute of Ultrasound in Medicine. Practice Bulletin no. 175: ultrasound in pregnancy.* Obstet Gynecol. *2016;128(6):e241-e256.*

may be associated with other causes of placental insufficiency. When reflective of an anatomic anomaly, oligohydramnios is often associated with a renal system anomaly. Mild to moderate polyhydramnios is common, typically idiopathic, but strongly associated with gestational diabetes. More severe polyhydramnios is a potent indicator of fetal anomalies, including central nervous system defects; ear, nose, and throat anomalies; gastrointestinal defects; and skeletal dysplasia. Polyhydramnios also may be an indicator of congestive heart failure, sometimes secondary to alloimmune disease or the anemia caused by parvovirus.

Advanced Applications

Biophysical assessment, fetal biometry, fetal anatomic survey, placental assessment, and cervical assessment are advanced applications that require significant training and experience that are beyond the scope of this chapter's corresponding workshop.

Biophysical Assessment

There are many indications for assessing fetal well-being in pregnancy. As discussed in the *Intrapartum Fetal Surveillance* chapter, fetal surveillance can be accomplished using multiple techniques, including the nonstress test (NST), contraction stress test, modified biophysical profile (MBPP), and biophysical profile (BPP). The MBPP and the BPP require ultrasound skills. The NST is often the first-line method of fetal surveillance and is discussed in detail in the *Intrapartum Fetal Surveillance* chapter. The definition of reactive NST is based on National Institute of Child Health and Human Development criteria of a minimum of two episodes of acceleration greater than 15 beats per minute lasting more than 15 seconds in a period of 20 minutes.[11] The NST alone has a high false-positive rate, which can lead to unnecessary intervention.[12,13]

The first fetal biophysical parameter to become compromised in the presence of fetal acidemia is the last to manifest as the fetus matures. In other words, the most primitive brain functions are the last to be compromised. Fetal tone is the earliest (most primitive) biophysical parameter; it can be identified as early as 8 weeks' gestational age. Fetal body movement follows approximately 1 to 2 weeks later. Fetal breathing is detectable by 21 weeks' gestation, but FHR reactivity is not well established until 28 to 32 weeks' gestation. Compromised FHR reactivity is therefore the most sensitive marker of acute fetal acidosis. As the insult becomes more profound and chronic, fetal breathing, then body movement, and then fetal tone are compromised. Amniotic fluid volume is considered a marker of placental perfusion and, therefore decreased fluid is considered a marker of chronic vascular compromise. *Table 5* summarizes these concepts. Using these concepts and combining the NST with a MBPP or BPP can significantly improve the predictive value of the test for fetal well-being.[12,13]

Table 5. Biophysical Parameters Sensitivity to Acute Hypoxia

Component	Process Assessed
Fetal tone	CNS, cortex/subcortical
Fetal movement	CNS, fourth ventricle
Fetal breathing	CNS, posterior hypothalamus, medulla
Nonstress test	CNS, cortex/nuclei
Amniotic fluid quantity	Uteroplacental/fetal perfusion

CNS = central nervous system.

Information from Manning FA, Platt LD, Sipos L. Antepartum fetal evaluation: development of a fetal biophysical profile. Am J Obstet Gynecol. *1980;136(6): 787-795; Manning FA. Antepartum fetal testing: a critical appraisal.* Curr Opin Obstet Gynecol. *2009;21(4):348-352.*

Modified Biophysical Profile

A MBPP is an NST (indicator of acute fetal status) along with an amniotic fluid assessment (marker of chronic fetal status). A reactive NST combined with a single LVP of amniotic fluid of 2 cm or greater is reassuring of fetal well-being and compares favorably with the complete BPP.[12,13] However, if the NST does not meet criteria for reactive and/or if oligohydramnios is present, further fetal testing is indicated and delivery may be considered, especially near term.

Biophysical Profile

The BPP is another method of predicting antepartum fetal acidemia. Although the test can be used as a primary method of fetal surveillance, it can be especially helpful in cases where the

NST is not reactive. There is some evidence that the BPP may even be useful during labor as an adjunct to FHR monitoring.[11]

The five components of a BPP integrate fetal cardiac activity in the form of an NST, amniotic fluid assessment, fetal breathing, fetal movement, and fetal tone. Each component is scored 2 points if present or the NST is reactive, and 0 points if abnormal. The presence of fetal breathing is likely to be as reliable as the NST in predicting the absence of fetal acidosis.[14] A BPP can be performed as early as 26 to 28 weeks' gestation. The test can be completed in as few as 5 minutes if the fetus is active and awake, but can take as many as 30 minutes. Acoustic stimulation is appropriate to use to reduce the duration of the study. *Table 6* includes criteria for each component of the BPP.[11]

The maximum BPP score is 10. A score greater than or equal to 8 is considered reassuring, with a low risk of fetal asphyxia within 1 week of the test. A score of 6 is equivocal, and a score less than or equal to 4 is abnormal, with a significantly higher risk of fetal asphyxia within 1 week of the test. However, not all components of the BPP are considered equal.[11] Oligohydramnios, when not associated with ruptured membranes, is a marker for placental dysfunction and chronic fetal hypoxemia or acidemia and may require further action or more frequent surveillance regardless of the total BPP score. However, if the amniotic fluid volume is normal but the NST is not reactive, the presence of fetal breathing predicts reassuring fetal status.

Table 6. Biophysical Profile

Parameter	Score
Nonstress Test	
	2: ≥2 FHR accelerations in 20 minutes
	0: <2 FHR accelerations in 20 minutes
Amniotic Fluid Volume	
	2: Largest pocket ≥2 cm in vertical diameter
	0: Largest pocket <2 cm in vertical diameter, crowded fetal small parts
Fetal Breathing	
	2: ≥1 episode of fetal breathing lasting ≥30 seconds within 30 minutes
	0: None, fetal breathing lasting <30 seconds
Fetal Movement	
	2: ≥3 gross movements (trunk and limbs) in 30 minutes
	0: None or <3 gross movements in 30 minutes
Fetal Tone	
	2: One episode extension of extremity or spine with return to flexion, or hand opening and closing
	0: Extension only, or movement not followed by return to flexion, or open hand

FHR = fetal heart rate.

Information from American College of Obstetricians and Gynecologists. Practice Bulletin no. 145: antepartum fetal surveillance. Obstet Gynecol. *2014;124(1):182-192.*

Doppler Velocimetry

Measurement of vascular resistance in various parts of the fetal circulation has been shown to be useful in the investigation and management of pregnancy complications including, but not limited to, intrauterine growth restriction (IUGR) and fetal cardiac anomalies. Doppler velocimetry has not been shown to be efficacious in screening otherwise low-risk pregnancies,[15] but it reduces the risk of perinatal mortality and results in fewer obstetric interventions in pregnancies complicated by conditions including IUGR, preeclampsia, and hypertensive disorders.[16] Umbilical artery Doppler ultrasound in high-risk pregnancy was associated with fewer perinatal deaths (RR = 0.71; 95% CI = 0.52-0.98; 16 studies, 10,225 infants, 1.2% versus 1.7%; number needed to treat [NNT] = 203; 95% CI = 103-4,352; moderate-quality evidence), fewer inductions (RR = 0.89; 95% CI = 0.80-0.99; 10 studies, 5,633 women, random effects; moderate-quality evidence), and fewer cesarean deliveries (RR = 0.90; 95% CI = 0.84-0.97; 14 studies, 7,918 women; moderate-quality evidence).[16] A detailed discussion of Doppler velocimetry of the uteroplacental and fetal circulation is beyond the scope of this chapter.

Estimating Gestational Age (Basic Fetal Biometry)

Ultrasound measurement during the first trimester of pregnancy is the most accurate way to determine pregnancy dating.[17] Measurement of the gestational sac and embryo is discussed in the *First-Trimester Pregnancy Complications* chapter.

A crown-rump length is determined by measuring the maximal straight-line distance from the fetal head to the rump, and is used for pregnancy dating between 6 and 12 weeks' gestation. From 13 weeks' gestation, fetal age is best determined

by measuring the fetal biparietal diameter. After 14 weeks, the head circumference, abdominal circumference, and femur length also are used for pregnancy dating. The estimated date of conception is changed from the last menstrual period (LMP) date to the ultrasound date if the estimated date of conception calculated by the first ultrasound differs from the LMP date by more than 5 days through 8 6/7 weeks' gestation, by more than 7 days from 9 to 15 6/7 weeks' gestation, by more than 10 days from 16 to 21 6/7 weeks' gestation, by more than 14 days from 22 to 27 6/7 weeks' gestation, or by more than 21 days at 28 weeks' gestation and beyond.[17]

To be accurate, all fetal measurements must be made properly according to two criteria. First, the correct anatomic plane must be identified. Second, measurements must be made at the proper landmarks within that plane. The proper planes and landmarks are shown in the diagrams and images in this chapter's accompanying slide set. Fetal crowding, oligohydramnios, and low station of the head can make acquiring accurate data difficult in women who are in labor. Most ultrasound machines are programmed to calculate gestational age based on measured parameters. Published standard biometric tables also can be used.

Estimating Fetal Weight

Sonographic fetal weight estimates are calculated from biometric parameters using software programmed into the ultrasound machine or from nomograms in standard textbooks. Such estimates are often off by 500 g (approximately 1.1 lb) or more during the third trimester and commonly overestimate birth weight.[18] Using standard equations, the positive predictive value for diagnosis of fetal macrosomia in postdates pregnancies is only approximately 50%. Fetal weight estimation may be more critical in the treatment of women with diabetes who are pregnant; risk of shoulder dystocia increases with increasing fetal weight. ACOG guidelines recommend considering cesarean delivery when the ultrasound estimated fetal weight (EFW) is greater than 4,500 g (approximately 9.9 lb) in women with diabetes and 5,000 g (approximately 11 lb) in women without diabetes.[19]

Even in the setting of maternal diabetes, the ability to detect macrosomia in pregnancy is limited, with the best results being obtained via methods that use the abdominal circumference and femur length.[20] Because the positive predictive value of ultrasound is low in detecting macrosomia, its use should be discouraged as a means of determining the route or timing of delivery for term or postdates pregnancies in women who do not have diabetes. There is evidence that sonographic diagnosis of macrosomia influences physician behavior in the management of labor. The use of retrospective cohort studies comparing pregnancies with a false positive diagnosis of macrosomia (EFW greater than 4,000 g [approximately 8.8 lb]) and true negatives show significantly increased rates of cesarean delivery (up to 50%) and failed induction without significant reduction in shoulder dystocia.[21-24] Limited data from a single randomized controlled trial in 2017 suggest that induction of labor in women with gestational diabetes did not reduce the risk of fetal macrosomia nor shoulder dystocia.[25]

American College of Obstetricians and Gynecologists guidelines recommend consideration of the option of planned cesarean delivery when EFW is more than 4,500 g (approximately 9.9 lb) in women with diabetes or more than 5,000 g (approximately 11 lb) in women who do not have diabetes.[26]

Determining Cervical Length

Scanning by the transperineal or transvaginal route may be useful to assess cervical length, to detect funneling and dilatation of the cervix, and to detect bulging membranes in women with suspected preterm labor or cervical incompetence. Shortening of the cervix in the second trimester is associated with increased risk of preterm delivery; 2 to 2.5 cm is commonly cited as the threshold,[27] and the use of progesterone for short cervical length after screening has been shown to decrease the likelihood of preterm delivery.[28] This is discussed in more detail in the *Preterm Labor and Prelabor Rupture of Membranes* chapter. A Cochrane review of five randomized controlled trials evaluated the effectiveness of transvaginal ultrasound cervical length screening and concluded that there is insufficient evidence to recommend routine screening to prevent preterm birth in women who are symptomatic or asymptomatic.[29] However, a 2012 ACOG Practice Bulletin considers the practice of routine screening to be acceptable but not mandatory.[30]

Guidance for External Cephalic Version

External cephalic version (ECV) of the fetus from breech or transverse to cephalic presentation is discussed in the *Malpresentations, Malpositions, and Multiple Gestations* chapter, including a discussion of parameters that affect success. A complete ultrasound scan, including biometry and anatomic survey, should be performed before ECV because the incidence of anomalies doubles with breech presentation.[31] Ultrasound is useful during the ECV procedure to monitor the changing fetal position and to visualize the fetal heartbeat.

Evaluating for Placenta Previa

Placenta previa can be difficult to diagnose sonographically depending on the location of the presenting part, the quantity of amniotic fluid, and the amount of urine in the maternal bladder. If the maternal bladder is overdistended, the lower uterine segment may be compressed, creating a false impression of placenta previa. Ultrasound evaluation of the lower uterine segment for possible placenta previa is best performed first with the bladder partially filled and then with an empty bladder. If possible, it should be performed during an interval without uterine contractions.

The lower uterine segment can be effectively visualized by the transperineal route using a glove-covered 3 or 3.5 MHz transducer placed at the introitus or on the perineum if transvaginal ultrasound is not available. Careful transvaginal scanning with a 5 or 7.5 MHz transducer may also be undertaken.[32-34] Technical aspects of transvaginal and transperineal scanning are discussed in the Scanning Techniques section of this chapter.

Evaluating for Placental Abruption

Placental abruption may have a variable sonographic appearance depending on the age of the abruption. The amount of bleeding is often underestimated. Fresh hemorrhage may appear as a sonolucent area between the uterine wall and the placenta or in the substance of the placenta. Clotted blood may exhibit echogenicity similar to that of placental tissue. Nonpathologic venous lakes and marginal sinuses can have a similar appearance. Abruption also may present as an abnormal thickening or rounding of the placental edge, presumably from a marginal separation. The sonographic diagnosis of abruption should be incorporated into available clinical information to arrive at a management plan. The *Late Pregnancy Bleeding* chapter includes more discussion on clinical management options in cases of abruption. The failure to diagnose abruption sonographically in a suspected clinical setting does not exclude the diagnosis and the provider should not change the management plan based on a negative ultrasound scan alone.

Anatomic Survey for Anomalies

Skill in performance of the anatomic survey for fetal anomalies requires significant additional training and supervised experience beyond what can be accomplished in this chapter's corresponding ALSO workshop. The standard antepartum ultrasound examination includes a comprehensive survey of fetal anatomy as agreed on by several professional organizations.[2] Attention to the items in this survey will detect most, but not all, fetal anomalies depending on gestational age. It is common for the woman to ask, "Is my baby all right?" during a limited labor and delivery scan. Because limited labor and delivery scans do not include a fetal anatomic survey, the provider should routinely explain the goals and limitations of the examination and what can and cannot be diagnosed on the basis of the examination being performed. Often the woman will have undergone a previous examination that included a standard survey of fetal anatomy that provides reassurance. If clinical questions exist about the presence of fetal anomalies, a standard examination that includes an anatomic survey should be performed when time and conditions permit.

Intrapartum Twin Management

The intrapartum management of twins in the delivery room can be facilitated by assessing the initial presentation and lie of the fetuses. After delivery of the first twin, ultrasound is often needed to determine the presentation and monitor the heart rate of the second twin. If the second twin is nonvertex, ultrasound may be used during an ECV, internal podalic version, or breech extraction.[35]

Amniocentesis Guidance

Ultrasound guidance can facilitate third trimester amniocentesis for lung maturity testing or to rule out amnionitis. In cases of preterm labor, such information may guide decisions about transfer from hospitals without neonatal intensive care facilities. Sonographic guidance aids in locating a

pocket for sampling that is free of the umbilical cord, away from the fetal face, and not directly underneath the placenta.

Perspective on Routine Scanning During Pregnancy

This chapter has discussed specific indications and techniques for limited sonographic examination, mostly during late pregnancy and in the labor and delivery setting. It has not addressed the value of routine scanning for all women who are pregnant. A detailed exposition of this topic is beyond the scope of this chapter. This issue continues to be debated and is complicated by significant regional and international differences in practice patterns and patient populations. A variety of clinical benefits have been ascribed to routine ultrasound examination of all women who are pregnant, some of which are supported by high-quality evidence.[35-37]

Routine early pregnancy ultrasound examination before 24 weeks' gestation and subsequent adjustment of delivery date appear to reduce the incidence of postterm pregnancy and postdates induction and lead to earlier detection of multifetal pregnancy.[36] More accurate gestational dating can facilitate routine induction of labor at 41 weeks' gestation, which appears to reduce perinatal mortality.[35,37] A 2018 Cochrane review found that induction of women at or beyond term compared with expectant management resulted in lower risk of perinatal death (RR = 0.33; 95% CI = 0.14-0.78; 20 trials, 9,960 infants; moderate-quality evidence), lower risk of cesarean delivery (RR = 0.92; 95% CI = 0.85-0.99; 27 trials, 11,738 women; moderate-quality evidence) but a higher rate of assisted vaginal delivery (RR = 1.07; 95% CI = 0.99-1.16; 18 trials, 9,281 women; moderate-quality evidence).[37] Scanning pregnancies after 24 weeks' gestation in unselected and low-risk populations does not reduce perinatal mortality rates or confer other benefits.[38]

Routine second-trimester anatomic survey, although commonly performed in developed countries, has not been shown to improve perinatal outcomes. A detailed anatomic survey may be indicated for many reasons, including abnormal serum screening, advanced maternal age, history of fetal anomalies, or exposures to teratogenic agents. Detection of major anomalies or genetic conditions may help women and their families prepare emotionally for anomalies and plan the optimal delivery site. Identification of anomalies and genetic conditions in the second trimester allows the option of pregnancy termination.

A 2016 ACOG Practice Bulletin recommends ultrasonography for all women who are pregnant. ACOG guidelines suggest that, in the absence of other indications, 18 to 22 weeks' gestation is optimal timing for a single ultrasound for pregnancy dating and anatomic survey.[3]

Learning Strategy

A variety of learning methods are available to maternity care providers who would like to include diagnostic ultrasound in their practice. Start with self-study using textbooks and multimedia materials, and then follow up with formal hands-on courses or informal apprenticeship. Simulators using physical phantoms or electronically stored images also are available.[39]

Ultrasound in Low-Resource Settings

Some have suggested that "…the ultrasound machine will probably be the stethoscope of the 21st century."[40] However, the cost of ultrasound technology makes this unrealistic in many low-resource settings. Portability, affordability, maintenance, and repair are important issues for units produced for or imported to developing countries, and inexpensive compact machines offer promise but outcomes research is needed.[41]

Teleradiology may help expand valuable ultrasound services to low-resource areas.[42] Ultrasound may have a greater effect in developing countries than developed countries by allowing prevention and early intervention where resources are scarce. Improved diagnosis of ectopic pregnancy, IUGR, placenta previa, malpresentation, and multiple gestation may have lifesaving implications.[42,43] In a study in Rwanda, midwives performing ultrasound had 100% agreement with sonographers when evaluating for multiple gestation, malpresentation, and viability.[44] Of 542 ultrasounds obtained for suspected abnormal findings, 39% aided in diagnosis and 22% changed management.[44] Additional information on ultrasound in developing countries is available at www.aafp.org/globalalso.

Summary

All maternity care providers who deliver infants can benefit from learning the basic applications of diagnostic ultrasound. The ability to rapidly assess

fetal life, fetal number, fetal presentation, quantity of amniotic fluid, and placental location may be learned rather quickly and can have significant bearing on clinical management. Advanced applications require further study and practice. Frequent use of the basic applications will help maintain proficiency while keeping in mind the shortcomings, complicating factors, and potential pitfalls of ultrasound scanning.

Nursing Considerations: Diagnostic Ultrasound in Labor and Delivery

- Position women who are pregnant in a left lateral tilt during extended ultrasound examination
- Verify ultrasound machine location every shift and learn how to turn it on
- During a vaginal delivery of twins, facilitate the presence of an ultrasound machine at bedside

Appendix
Documentation of Labor and Delivery Ultrasound Scan

Patient name: _____ Date: _____ Patient I.D. number: _____

Attending physician: _____ Other physician: _____

Pregnancy data: Maternal age: _____ G: _____ P: _____ Ab: _____

Today's gestational age: _____ weeks + or - _____ weeks

Based on: last menstrual period of: _____ and/or previous ultrasound scan on: _____ (date)

Findings of today's fetal heart rate tracing: _____

Reason for labor and delivery ultrasound scan *(check all that apply)*:

☐ No prenatal care / unknown dates ☐ Prelabor rupture of membranes ☐ Pain
☐ Part of antepartum testing (amniotic fluid index) ☐ Bleeding ☐ Trauma
☐ Adjunct to amniocentesis ☐ Suspected breech presentation ☐ Preterm labor
☐ Adjunct to external version ☐ Preterm labor ☐ _____
☐ Fetal weight estimate (reason): _____)

Other coexisting problems: _____

Findings: (complete only relevant items) (attach photos to reverse side or new page)

Fetal heartbeat: ☐ Yes ☐ No Fetal number: _____

Fetal lie, presentation, and position: _____

Placental location: _____

Biophysical data:

Amniotic fluid index: _____ + _____ + _____ + = _____ cm. Or largest vertical pocket: _____ cm

Breathing: ☐ Yes ☐ No Tone: ☐ Yes ☐ No Body movement: ☐ Yes ☐ No

Biophysical score: _____ points out of possible _____points

Fetal biometry:

Biparietal diameter = _____ cm = _____ weeks' gestation + or - _____ weeks' gestation

Femur = _____ cm = _____ weeks' gestation + or - _____ weeks' gestation

Head Circ. = _____cm = _____ weeks' gestation + or - _____ weeks' gestation

Abd. Circ. = _____ cm = _____ weeks' gestation + or - _____ weeks' gestation

Composite gestational age = _____ weeks + or - _____ weeks

Estimated date of delivery: _____ + or - _____ weeks. Estimated fetal weight: _____ grams

Describe any abnormalities seen: _____

_____

_____

Consultative scan ordered: ☐ Yes ☐ No

Diagnosis related to reason for scan: _____

_____

Treatment plan: _____

_____

Signature(s): _____ Date and time _____

Note: This labor and delivery scan is a limited scan. If a patient has not had a standard examination/anatomic survey, one can be ordered afterwards, assuming the patient has not already delivered.

Courtesy of Mark Deutchman, MD

References

1. Diagnostic ultrasound imaging in pregnancy. National Institutes of Health Consensus Development Conference. *Natl Inst Health Consens Dev Conf Consens Statement*. 1984;5(1):17.
2. AIUM Practice Parameter for the Performance of Obstetric Ultrasound Examinations. AIUM-ACR-ACOG-SMFM-SRU Practice Parameter for the Performance of Standard Diagnostic Obstetric Ultrasound Examinations. *J Ultrasound Med*. 2018;37(11):E13-E24.
3. Committee on Practice Bulletins—Obstetrics and the American Institute of Ultrasound in Medicine. Practice Bulletin no. 175: ultrasound in pregnancy. *Obstet Gynecol*. 2016;128(6):e241-e256.
4. Hahn RG, Roi LD, Ornstein SM, et al. Obstetric ultrasound training for family physicians. Results from a multi-site study. *J Fam Pract*. 1988;26(5):553-558.
5. Smith CB, Sakornbut EL, Dickinson LC, Bullock GL. Quantification of training in obstetrical ultrasound: a study of family practice residents. *J Clin Ultrasound*. 1991;19(8):479-483.
6. Rodney WM, Deutchman ME, Hartman KJ, Hahn RG. Obstetric ultrasound by family physicians. *J Fam Pract*. 1992;34(2):186-194, 197-200.
7. Bellussi F, Ghi T, Youssef A, et al. Intrapartum ultrasound to differentiate flexion and deflexion in occipito-posterior rotation. *Fetal Diagn Ther*. 2017;42(4):249-256.
8. Hemelaar J, Lim LN, Impey LW. The impact of an ECV service is limited by antenatal breech detection: a retrospective cohort study. *Birth*. 2015;42(2):165-172.
9. Glantz C, Purnell L. Clinical utility of sonography in the diagnosis and treatment of placental abruption. *J Ultrasound Med*. 2002;21(8):837-840.
10. Nabhan AF, Abdelmoula YA. Amniotic fluid index versus single deepest vertical pocket as a screening test for preventing adverse pregnancy outcome. *Cochrane Database Syst Rev*. 2008;(3):CD006593.
11. American College of Obstetricians and Gynecologists. Practice Bulletin no. 145: antepartum fetal surveillance. *Obstet Gynecol*. 2014;124(1):182-192.
12. Manning FA, Platt LD, Sipos L. Antepartum fetal evaluation: development of a fetal biophysical profile. *Am J Obstet Gynecol*. 1980;136(6):787-795.
13. Manning FA. Antepartum fetal testing: a critical appraisal. *Curr Opin Obstet Gynecol*. 2009;21(4):348-352.
14. Ohno Y, Tsuji M, Fujibayashi H, et al. Assessment of fetal heart rate variability with abdominal fetal electrocardiogram: changes during fetal breathing movement. *Asia Oceania J Obstet Gynaecol*. 1986;12(2):301-304.
15. Alfirevic Z, Stampalija T, Medley N. Fetal and umbilical Doppler ultrasound in normal pregnancy. *Cochrane Database Syst Rev*. 2015;4(4):CD001450.
16. Alfirevic Z, Stampalija T, Dowswell T. Fetal and umbilical Doppler ultrasound in high-risk pregnancies. *Cochrane Database Syst Rev*. 2017;6:CD007529.
17. Committee on Obstetric Practice. the American Institute of Ultrasound in Medicine, and the Society for Maternal-Fetal Medicine. Committee Opinion no 700: methods for estimating the due date. *Obstet Gynecol*. 2017;129(5):e150-e154.
18. Delpapa EH, Mueller-Heubach E. Pregnancy outcome following ultrasound diagnosis of macrosomia. *Obstet Gynecol*. 1991;78(3 Pt 1):340-343.
19. Committee on Practice Bulletins—Obstetrics. Practice Bulletin no. 178: shoulder dystocia. *Obstet Gynecol*. 2017;129(5):e123-e133.
20. Pollack RN, Hauer-Pollack G, Divon MY. Macrosomia in postdates pregnancies: the accuracy of routine ultrasonographic screening. *Am J Obstet Gynecol*. 1992;167(1):7-11.
21. McLaren RA, Puckett JL, Chauhan SP. Estimators of birth weight in pregnant women requiring insulin: a comparison of seven sonographic models. *Obstet Gynecol*. 1995;85(4):565-569.
22. Levine AB, Lockwood CJ, Brown B, et al. Sonographic diagnosis of the large for gestational age fetus at term: does it make a difference? *Obstet Gynecol*. 1992;79(1):55-58.
23. Parry S, Severs CP, Sehdev HM, et al. Ultrasonographic prediction of fetal macrosomia. Association with cesarean delivery. *J Reprod Med*. 2000;45(1):17-22.
24. Blackwell SC, Refuerzo J, Chadha R, Carreno CA. Overestimation of fetal weight by ultrasound: does it influence the likelihood of cesarean delivery for labor arrest? *Am J Obstet Gynecol*. 2009;200(3):340.e1-340.e3.
25. Biesty LM, Egan AM, Dunne F, et al. Planned birth at or near term for improving health outcomes for pregnant women with gestational diabetes and their infants. *Cochrane Database Syst Rev*. 2018;1(1):CD012910.
26. American College of Obstetricians and Gynecologists' Committee on Practice Bulletins—Obstetrics. Practice Bulletin no. 173: fetal macrosomia. *Obstet Gynecol*. 2016;128(5):e195-e209.
27. Iams JD, Goldenberg RL, Meis PJ, et al; National Institute of Child Health and Human Development Maternal Fetal Medicine Unit Network. The length of the cervix and the risk of spontaneous premature delivery. *N Engl J Med*. 1996;334(9):567-572.
28. Hassan SS, Romero R, Vidyadhari D, et al. PREGNANT Trial. Vaginal progesterone reduces the rate of preterm birth in women with a sonographic short cervix: a multicenter, randomized, double-blind, placebo-controlled trial. *Ultrasound Obstet Gynecol*. 2011;38(1):18-31.
29. Berghella V, Baxter JK, Hendrix NW. Cervical assessment by ultrasound for preventing preterm delivery. *Cochrane Database Syst Rev*. 2013;1(1):CD007235.
30. Committee on Practice Bulletins—Obstetrics, The American College of Obstetricians and Gynecologists. Practice Bulletin no. 130: prediction and prevention of preterm birth. *Obstet Gynecol*. 2012;120(4):964-973.
31. Mostello D, Chang JJ, Bai F, et al. Breech presentation at delivery: a marker for congenital anomaly? *J Perinatol*. 2014;34(1):11-15.

32. Adeyomoye A, Ola E, Arogundade R, Awosanya G, Abudu O. Comparison of the accuracy of trans-abdominal sonography (TAS) and transperineal sonography (TPS) in the diagnosis of Placenta Praevia. *Niger Postgrad Med J*. 2006;13(1):21-25.
33. Hertzberg B, Bowie J, Carroll B, et al. Diagnosis of placenta previa during the third trimester: role of transperineal sonography. *AJR Am J Roentgenol*. 1992;159(1): 83-87.
34. Petpichetchian C, Pranpanus S, Suntharasaj T, et al. Comparison of transabdominal and transvaginal sonography in the diagnosis of placenta previa. *J Clin Ultrasound*. 2018;46(6):386-390.
35. Fox NS, Silverstein M, Bender S, et al. Active second-stage management in twin pregnancies undergoing planned vaginal delivery in a U.S. population. *Obstet Gynecol*. 2010;115(2 Pt 1):229-233.
36. Whitworth M, Bricker L, Mullan C. Ultrasound for fetal assessment in early pregnancy. *Cochrane Database Syst Rev*. 2015;7(7):CD007058.
37. Middleton P, Shepherd E, Crowther CA. Induction of labour for improving birth outcomes for women at or beyond term. *Cochrane Database Syst Rev*. 2018;5: CD004945.
38. Bricker L, Medley N, Pratt JJ. Routine ultrasound in late pregnancy (after 24 weeks' gestation). *Cochrane Database Syst Rev*. 2015;6(6):CD001451.
39. Dresang LT, Rodney WM, Dees J. Teaching prenatal ultrasound to family medicine residents. *Fam Med*. 2004;36(2):98-107.
40. Rodney WM. More on the use of ultrasonography in the emergency department. *West J Med*. 1995;163(4): 393-394.
41. Harris RD, Marks WM. Compact ultrasound for improving maternal and perinatal care in low-resource settings: review of the potential benefits, implementation challenges, and public health issues. *J Ultrasound Med*. 2009;28(8):1067-1076.
42. Stanton K, Mwanri L. Global maternal and child health outcomes: the role of obstetric ultrasound in low resource settings. *World J Prev Med*. 2013;1(3):22-29.
43. Sippel S, Muruganandan K, Levine A, Shah S. Review article: Use of ultrasound in the developing world. *Int J Emerg Med*. 2011;4(4):72.
44. Stein W, Katunda I, Butoto C. A two-level ultrasonographic service in a maternity care unit of a rural district hospital in Tanzania. *Trop Doct*. 2008;38(2):125-126.

First-Trimester Pregnancy Complications

Learning Objectives

1. Determine etiology of first-trimester vaginal bleeding.
2. Assess the value of human chorionic gonadotropin levels and sonographic discriminatory criteria in diagnosing first-trimester pregnancy complications.
3. Outline management options for women with ectopic pregnancy, gestational trophoblastic disease, or miscarriage.
4. Using shared decision-making and while providing emotional support, counsel patients on therapeutic options to manage early pregnancy loss.

Introduction

Complications during the first trimester of pregnancy are common. Approximately 15% of clinically recognized pregnancies result in spontaneous miscarriage, and estimates of miscarriage before clinical recognition are as high as 50%.[1] In addition to miscarriage, vaginal bleeding can be associated with ectopic pregnancy, trophoblastic disease, or cervical bleeding from causes unrelated to pregnancy, or bleeding may occur in pregnancies that proceed without further complications.

Normal First-Trimester Pregnancy Progress

Pregnancy is clinically dated from the first day of the last normal menstrual period, which is an observable event, instead of the conception date. Conception occurs approximately 2 weeks later. All gestational landmarks in this chapter are based on menstrual dating, assuming a 28-day cycle; embryology textbooks commonly use conception dating, which is 2 weeks less.

The placenta produces human chorionic gonadotropin (hCG) after implantation. Implantation occurs at approximately 23 menstrual days, which is approximately 8 days after conception. Commonly available over-the-counter urine pregnancy tests are approximately 100% sensitive and specific for detecting the beta subunit of hCG at levels of 25 mIU/mL, which may allow detection of pregnancy around the time of the first missed period.[2,3] Serum tests can detect hCG as low as 5 mIU/mL. The rate of increase in quantitative serum hCG levels may be used to monitor women with pain or bleeding whose initial ultrasound examination did not yield a definitive diagnosis. A large study of such women with pregnancies of less than 10 weeks' gestation showed that a viable intrauterine pregnancy (IUP) with hCG levels greater than 5,000 mIU/mL had an hCG level increase of 53% in 48 hours. However, women with a miscarriage or an ectopic pregnancy also can have an increase within this range. Therefore, an adequately rising hCG level does not rule out nonviable pregnancy.[3-5]

The gestational sac first becomes visible on transvaginal ultrasound during the fifth menstrual week as a 0.2- to 0.5-cm sonolucent area surrounded by an echogenic ring of chorionic villi. This early gestational sac is only visible using a high-frequency transducer (5 MHz or greater) and the transvaginal scanning route. A small sonolucent fluid collection, or pseudogestational sac, can also be present in cases of ectopic pregnancy. Therefore, additional features of a normal gestational sac can be sought, particularly the eccentric location of the gestational sac indicating that it is implanted within the endometrium rather than the endometrial stripe. The yolk sac appears during transvaginal scanning during the sixth menstrual week and provides clear evidence of an IUP. By the end of the sixth menstrual week, the fetal pole becomes visible during transvaginal scanning as a 0.2- to 0.8-cm pole with embryonic cardiac activity. These sonographic landmarks are visible with transabdominal scanning approximately 1 week later than with transvaginal scanning.[6]

Embryologic, clinical, hCG, and sonographic findings are closely correlated and are shown in *Table 1*.[7,8] Ultrasonography is so valuable that, when it is readily available, it is the preferred primary tool in evaluating first-trimester complications,[9] leaving serum hCG testing as a secondary tool used only if sonographic findings are equivocal such as when no IUP is seen and ectopic pregnancy is suspected. A routine first-trimester ultra-

Table 1. Early Pregnancy Landmarks

Menstrual Age (Weeks)	Embryologic Event/ Sonographic/hCG Correlation
3 to 4	Implantation site – decidual thickening
4	Trophoblast – peritrophoblastic flow on color flow Doppler
4 to 5	Gestational sac typically visible when hCG reaches 1,000 to 3,000 mIU/mL
5 to 6	Yolk sac usually appears
5 to 6	Embryo and cardiac activity

hCG = human chorionic gonadotropin.

Information from Paspulati RM, Bhatt S, Nour SG. Sonographic evaluation of first-trimester bleeding. Radiol Clin North Am. *2004;42(2):297-314. Erratum in* Radiol Clin North Am. *2008;46(2):437.*

sound in early pregnancy appears to enable better assessment of gestational age and earlier detection of multifetal pregnancies. However, the benefits for other substantive outcomes are less clear.[10]

The use of specific sonographic findings as discriminatory criteria to diagnose early pregnancy loss is presented in detail in the Early Pregnancy Loss: Pathophysiology, Discriminatory Criteria, Clinical Course, and Prognosis section of this chapter.

After the embryo is sonographically visible, first-trimester menstrual age is determined using crown-rump length, which is calculated by various equations that are included in software of all modern ultrasound equipment.

Early Pregnancy Loss: Pathophysiology, Discriminatory Criteria, Clinical Course, and Prognosis

Common terms applied to early pregnancy loss are defined in *Table 2*.

Spontaneous Abortion (Miscarriage)

The cause of spontaneous abortion is rarely determined in clinical practice, but it is known that approximately half are because of major genetic abnormalities, typically trisomy, triploidy, or monosomy.[11] Environmental factors linked to spontaneous abortion include uterine anomalies and fibroids, incompetent cervix, progesterone deficiency, advanced maternal age, exposure to occupational chemicals, infections, and exposure to radiation, alcohol, tobacco, and cocaine.

Spontaneous abortion can manifest clinically in several different ways. Most commonly, vaginal bleeding and cramping are present. But occasionally, regression of pregnancy symptoms or lack of Doppler ultrasound-detected fetal heart tones by 10 to 12 weeks' gestation are the first clinical signs in the setting of anembryonic pregnancy or embryonic demise. Spontaneous abortion may also be discovered incidentally when women who are asymptomatic undergo early ultrasound examinations for other reasons such as pregnancy dating or genetic screening.

Clinical examination should include palpation of the abdomen and pelvis, noting the size and position of the uterus, the location of any tenderness, the presence of rebound tenderness, and the presence of masses. Adnexal tenderness and any masses should raise suspicion for ectopic pregnancy, although a normal corpus luteum cyst can also be the cause of that tenderness or of masses. If the woman's last menses was at least 9 to 10 weeks prior and ultrasound is not readily available, consider listening for the fetal heartbeat during the bimanual pelvic examination while elevating the uterus with the intravaginal hand.

A speculum examination will reveal nonuterine causes of bleeding, the degree of cervical dilation and, if present, tissue being passed. The quantity of blood in the vault and the source of bleeding (from the os versus other sites) should be noted. If an intact gestational sac, an embryo, or the characteristic fronds of chorionic villi have passed through the cervix, miscarriage is proven and ectopic pregnancy is virtually ruled out, except in the rare case of heterotopic pregnancy. Heterotopic pregnancy can occur in naturally conceived pregnancies (1:4,000 to 1:30,000), but women undergoing in vitro fertilization are at significant increased risk (1:100).[12] If there is doubt about the origin of expelled tissue, an examination for chorionic villi can be performed. To examine for chorionic villi, rinse and float the tissue in saline. Low magnification, backlighting, and teasing the tissue can help. Passed tissue can be submitted for pathologic examination, which is definitive in questionable cases.

If products of conception are seen at the cervical os, ring forceps can be used to gently remove the tissue. More aggressive attempts to remove

partially expelled tissue should be preceded by discussion with the woman, informed consent, and administration of analgesia or sedation.

When the diagnosis is not clear based on clinical findings, transvaginal scanning is essential for accurate diagnosis. Specific sonographic characteristics of the gestational sac, yolk sac, and embryo can be reliably used to make an accurate and timely diagnosis. *Table 3* presents guidelines for use of sonographic findings when discriminating between viable and failed early pregnancy. Pregnancy failure can be reliably diagnosed if the gestational sac is 25 mm or greater without an embryo, if an embryo fails to appear by 11 days after a yolk sac appears, or if an embryo of crown-rump length greater than 7 mm does not show a heartbeat.[13]

When ultrasound reveals a fetal heartbeat in a woman with bleeding, the probability of miscarriage is only 2.1% in women younger than 35 years, but increases to 16.1% in women older than 35 years.[14]

Complete spontaneous miscarriage might result in an empty uterus with a bright endometrial stripe as a result of the uterine walls collapsing against each other. Echogenic material within the endometrial cavity commonly creates an endometrial stripe greater than 15 mm after treatment of early pregnancy failure with misoprostol.[15] Therefore, endometrial thickness alone is not an indication of the need for surgical intervention after medical management of miscarriage with misoprostol.

Table 2. Terms Applied to Early Pregnancy Loss

Anembryonic pregnancy — presence of a gestational sac >25 mm without evidence of embryonic tissues (ie, yolk sac, embryo). This term is preferred to the older and less accurate phrase blighted ovum

Ectopic pregnancy — pregnancy outside of the uterine cavity, most commonly located in the fallopian tube but can occur in the broad ligament, ovary, cervix, or elsewhere in the abdomen

Embryonic demise — an embryo with a crown-rump length >7 mm without cardiac activity

Gestational trophoblastic disease, or hydatidiform mole — complete mole: placental proliferation in the absence of a fetus. Most have a 46, XX chromosomal composition, all derived from paternal source. Partial mole: molar placenta occurring together with a fetus. Most are genetically triploid (69, XXX)

Heterotopic pregnancy — simultaneous intrauterine and ectopic pregnancy. Incidence is rare, thought to occur in 1/30,000 spontaneous pregnancies, but occurs in 1.5/1,000 pregnancies involving assisted reproductive techniques

Recurrent pregnancy loss — more than two consecutive pregnancy losses

Spontaneous abortion — spontaneous loss of a pregnancy before 20 weeks' gestation. Can be further described as:

- Incomplete — occurs when some but not all the products of conception have passed
- Complete — all products of conception have passed through the external cervical os
- Septic — incomplete abortion associated with ascending infection of the endometrium, parametrium, adnexa, or peritoneum
- Inevitable — bleeding in the presence of a dilated cervix, indicating that passage of the conceptus is unavoidable
- Missed — the fetus or embryo is deceased, but no tissue has been passed. The cervix is closed. These patients often present with no growth in uterine size or no audible fetal heart tones

Subchorionic hemorrhage — ultrasonographic finding of blood between the chorion and uterine wall, typically seen in the setting of vaginal bleeding

Threatened abortion — bleeding before 20 weeks' gestation in the presence of an embryo with cardiac activity or a gestational/yolk sac and a closed cervix

Vanishing twin — A multifetal pregnancy is identified, and one or more fetuses later disappear. Identified more commonly with early ultrasound scanning. If this occurs early in pregnancy, the embryo is often reabsorbed. Later occurrence results in a compressed (ie, *mummified*) fetus or amorphous material

Information from Chen BA, Creinin MD. Contemporary management of early pregnancy failure. Clin Obstet Gynecol. 2007;50(1): 67-88; Doubilet PM, Benson CB, Bourne T, et al; Society of Radiologists in Ultrasound Multispecialty Panel on Early First Trimester Diagnosis of Miscarriage and Exclusion of a Viable Intrauterine Pregnancy. Diagnostic criteria for nonviable pregnancy early in the first trimester. N Engl J Med. 2013;369(15):1443-1451.

Table 3. Guidelines for Transvaginal Ultrasonographic Diagnosis of Pregnancy Failure in Women With Intrauterine Pregnancies of Uncertain Viability

Findings Diagnostic of Pregnancy Failure	Findings Suspicious for, but not Diagnostic of, Pregnancy Failure[a]
Crown-rump length of >7 mm and no heartbeat	Crown–rump length of <7 mm and no heartbeat
Mean sac diameter of >25 mm and no embryo	Mean sac diameter of 16–24 mm and no embryo
Absence of an embryo with heartbeat >2 weeks after a scan that showed a gestational sac without a yolk sac	Absence of embryo with heartbeat 7–13 days after a scan that showed a gestational sac without a yolk sac
Absence of embryo with heartbeat >11 days after a scan that showed a gestational sac with a yolk sac	Absence of embryo with heartbeat 7–10 days after a scan that showed a gestational sac with a yolk sac
	Absence of embryo >6 weeks after last menstrual period
	Empty amnion (amnion seen adjacent to yolk sac, with no visible embryo)
	Enlarged yolk sac (>7 mm)
	Small gestational sac in relation to the size of the embryo (<5 mm difference between mean sac diameter and crown-rump length)

Criteria are from the Society of Radiologists in Ultrasound Multispecialty Consensus Conference on Early First Trimester Diagnosis of Miscarriage and Exclusion of a Viable Intrauterine Pregnancy, October 2012.

[a]*When there are findings suspicious for pregnancy failure, follow-up with ultrasonography in 7 to 10 days to assess the pregnancy for viability is generally appropriate.*

Reprinted from Doubilet PM, Benson CB, Bourne T, et al; Society of Radiologists in Ultrasound Multispecialty Panel on Early First Trimester Diagnosis of Miscarriage and Exclusion of a Viable Intrauterine Pregnancy. Diagnostic criteria for nonviable pregnancy early in the first trimester. N Engl J Med. *2013;369(15):1443-1451.*

Septic abortion should be presumed when the woman is febrile, has excessive uterine or adnexal tenderness, or signs of peritonitis. The provider should ask about a history of attempted therapeutic abortion or medically unassisted abortion that might have left tissue behind or perforated the uterus. Septic abortion is a potentially life-threatening condition requiring prompt resuscitation, uterine evacuation, and broad-spectrum antibiotic treatment.[16,17] For more information, see the *Venous Thromboembolism in Pregnancy* chapter.

Sonography may reveal a hematoma between the chorion and uterine wall in subchorionic hemorrhage, and a gestational sac and embryo will be present. When subchorionic hemorrhage is visible on ultrasound, the likelihood of miscarriage averages approximately 10% even when a heartbeat is detected, but varies by maternal age, size of the hematoma, and gestational age.[18] Therefore, the woman should be advised to expect bleeding.

The quantity of bleeding predicts pregnancy loss only when it is heavy. A prospective analysis of 4,510 women who were monitored in the early first trimester showed that 1,204 (27%) had some bleeding or spotting. There was no increase in miscarriage risk in early pregnancy when there was spotting or light bleeding. However, miscarriage risk increased significantly in the 8% of women who reported heavy bleeding. This was the only group to have an increased risk of miscarriage (adjusted odds ratio 2.84; 95% confidence interval [CI] = 1.82-4.43).[19]

Management of Spontaneous Abortion

If ultrasound examination reveals an IUP with cardiac activity, the woman should be monitored with cautious optimism and told that there are no known interventions to prevent miscarriage.

When the provider's examination reveals incomplete miscarriage, the woman must choose between expectant, medical, or surgical (uterine aspiration) management. Most first-trimester miscarriages occur completely and spontaneously without intervention. Although surgical intervention in the form of uterine aspiration has traditionally been used liberally, expectant management and medical treatment are valid options. Women with excessive bleeding, pain, or infection benefit from medical or surgical intervention.[20]

In an observational study of 451 women, 91% of those with incomplete miscarriage, 76% of those with missed miscarriage, and 66% of those with anembryonic pregnancy completed the miscarriage without uterine aspiration. Overall, 70% of women completed the miscarriage within 14 days of classification.[21] Women who have not completed the miscarriage for an emotionally uncomfortable period may prefer medical or surgical intervention. The woman's emotional state and personal preferences are important in choosing the course of action. Ultimately, the decision should be primarily driven by the woman's desires after being informed of her options.[22]

Clinical trials comparing expectant management with misoprostol and misoprostol with uterine aspiration have shown:[23-25]

- In incomplete miscarriage, expectant and medical management with misoprostol are highly successful
- In missed abortion, medical management with misoprostol and surgical (uterine aspiration) treatment are more effective than expectant treatment
- Typical misoprostol doses are 600 mcg orally or 600 to 800 mcg vaginally or buccally
- Women treated with misoprostol have more bleeding but less pain than those treated surgically
- Women treated expectantly have more outpatient visits than those treated with misoprostol
- Uterine aspiration is associated with more trauma and infection complications than misoprostol treatment
- Misoprostol has fewer gastrointestinal adverse effects when administered vaginally than when administered orally.

The trials conducted on the medical management of miscarriage with misoprostol, support offering this option.[26] It is also reasonable for a woman to change treatment course and mix the three treatment options. Women commonly choose a period of expectant management followed by medical therapy with misoprostol if they no longer desire to wait. This could also be followed by uterine aspiration if medical therapy is not successful (*Appendix*). A 2018 randomized controlled trial showed the addition of mifepristone, a progesterone antagonist, 24 hours prior to misoprostol has been shown to increase the likelihood of gestational sac expulsion by day 8 to 89.2% compared with 74.5% for misoprostol alone (risk ratio 1.20; 95% CI = 1.07-1.33).[27] Availability of mifepristone is limited by the Food and Drug Administration mandated Risk Evaluation and Mitigation Strategies (REMS), which do not permit direct patient access through pharmacies.[28]

There is no evidence supporting the use of prophylactic antibiotics in early pregnancy failure,[29] although there is such evidence for uterine aspiration for induced abortion. The administration of a single prophylactic dose of doxycycline 200 mg 1 hour prior to uterine aspiration appears optimal, but adding a course of postprocedural antibiotics is not recommended.[30] Metronidazole 500 mg is an option for women with doxycycline allergy. When misoprostol is used for medical abortion, the incidence of infection complications may be reduced by administering the drug via the buccal route and by administering doxycycline 100 mg twice/day for 7 days. However, the evidence in this study was low-quality because it compares two different periods.[31] It is unclear whether it is the use of antibiotics or the change in the route of misoprostol administration that resulted in the lowered incidence of infection. It is also unclear whether this association with infection also holds true when misoprostol with or without mifepristone is used to manage early pregnancy loss.

After a miscarriage, it is customary to recommend a brief period of contraception before attempting another pregnancy. However, this practice is not supported in the literature. A prospective study showed no statistically significant difference of recurrent miscarriage in women who had interpregnancy intervals of less than 6 months versus those who had longer intervals.[32] For women desiring long-term contraception and in whom there is no evidence of infection or ongoing significant bleeding, an intrauterine device placed immediately after spontaneous or induced first-trimester abortion is safe and effective. This is considered a best practice even though there is a small increased incidence of device expulsion.[33,34]

Ectopic Pregnancy

Pathophysiology and Risk Factors

Ectopic pregnancy typically occurs in the fallopian tube (greater than 90%) but can also occur in the ovary (1% to 3%), a scar from a prior cesarean delivery (1% to 3%), the cervix (1%), or the abdomen (1%). Rarely, intrauterine and ectopic pregnancies occur simultaneously (heterotopic

pregnancy). Pregnancy implantation outside the uterus increases maternal morbidity due to delayed diagnosis and treatment.[12] Ectopic pregnancy can result in impairment or loss of fertility and, because of internal hemorrhage, remains a significant cause of maternal mortality.[12] Early diagnosis is the key to preventing morbidity and mortality and preserving fertility.

All maternity care providers should have a working knowledge of ectopic pregnancy and a high index of suspicion in any woman who presents with bleeding and/or pain during early pregnancy. Many ectopic pregnancies occur in women without risk factors, but risk factors include previous tubal pregnancy, previous tubal surgery, history of tubal infection including pelvic inflammatory disease, endometriosis, intrauterine device contraception, and cigarette smoking.[35]

Signs, Symptoms, and Diagnosis

Pain and vaginal bleeding are the hallmark symptoms of ectopic pregnancy. Pain is almost universal; it is typically unilateral and in the lower abdomen. Bleeding is also common after a short period of amenorrhea. A tender adnexal mass may manifest in ectopic pregnancy, but it is not a reliable factor for diagnosis when used alone given that the corpus luteum can be tender on examination in a normal IUP. Finally, signs and symptoms of hemoperitoneum and shock can occur, including a distended, silent, doughy abdomen, shoulder pain, bulging cul-de-sac into the posterior fornix of the vagina, and hypotension.

Hearing the fetal heart rate on Doppler ultrasound is not sufficient to exclude ectopic pregnancy. A cornual ectopic pregnancy may not rupture until approximately 13 weeks' gestation; the fetal heart rate may be heard as early as 8 weeks' gestation by handheld Doppler ultrasound.

In ectopic pregnancy, the serum hCG level initially increases but then typically plateaus or decreases. Transvaginal ultrasound scanning is a key diagnostic tool and can aid in rapidly making these diagnoses:

Ectopic pregnancy is ruled out by the presence of an IUP with the exception of rare heterotopic pregnancy

Ectopic pregnancy is proven when a gestational sac and an embryo with a heartbeat are seen outside of the uterus

Ectopic pregnancy is highly likely if any adnexal mass distinct from the corpus luteum or a significant amount of free pelvic fluid is seen.[36]

Ultrasound findings associated with ectopic pregnancy include: no mass or free fluid (20%), any free fluid (71%), echogenic mass (85%), moderate to large amount of fluid (95%), and echogenic mass with fluid (100%).[37]

When ultrasound findings are not definitive, the location of the pregnancy is unknown. *Table 4* provides guidelines for using hCG measurements in combination with transvaginal ultrasound findings to establish a diagnosis and avoid interrupting a potentially viable pregnancy. When transvaginal ultrasound shows no intrauterine fluid (gestational sac) and the hCG level is above a discriminatory zone threshold, viable IUP is unlikely; the provider should have a high index of suspicion for ectopic pregnancy. This threshold for hCG is in question; some sources use 3,000 mIU/mL,[15] others use 3,510 mIU/mL.[38,39] The threshold is dependent on the quality of the ultrasound equipment and the sonographer performing the ultrasound. In women who are stable, repeat hCG testing and transvaginal ultrasound are prudent before diagnosing and treating for ectopic pregnancy.[13,38]

In some cases of ectopic pregnancy, a small fluid collection within the uterus can be mistaken for a true gestational sac. However, this pseudogestational sac lacks a surrounding echogenic ring of chorionic villi, a yolk sac or fetal pole, and the eccentric location of a normal gestational sac. An unruptured corpus luteum cyst can be mistaken for an ectopic gestational sac. A ruptured corpus luteum cyst can produce free pelvic fluid suggesting a ruptured ectopic pregnancy. Culdocentesis can be helpful in differentiating the thin pink fluid of a ruptured ovarian cyst, which can be managed expectantly, from the frank hemorrhage due to ruptured ectopic pregnancy. However, improved use of ultrasound and highly sensitive hCG testing have decreased the need for this procedure. The presence of any cul-de-sac fluid suggests ectopic pregnancy until proven otherwise.

When hCG levels are not rising normally, and ultrasound cannot confirm pregnancy location, a uterine aspiration may yield chorionic villi or a gestational sac. This proves a failed IUP and treatment for an ectopic pregnancy is avoided. A decrease in hCG levels by less than 50% at 12 to 24 hours after uterine aspiration should raise

suspicion for ectopic pregnancy.[40] When suspicion for ectopic pregnancy is high but cannot be confirmed with noninvasive testing, laparoscopy can confirm the diagnosis and accomplish surgical treatment. Alternatively, methotrexate can be administered if a viable IUP has been definitively excluded.

Management

With early diagnosis, the management of ectopic pregnancy occurs most frequently in the outpatient setting by a provider with experience and confidence in ectopic management. Current treatment options favor medical and laparoscopic surgical management with expectant management reserved for cases with a declining quantitative hCG level less than 1,000 mIU/mL.[41,42]

Open surgical management is limited to tubal rupture and hemoperitoneum if a surgeon with laparoscopic training is available. Surgical management via laparoscopy or open laparotomy can involve complete removal of the fallopian tube (salpingectomy) or removal of the ectopic pregnancy and conservation of the tube (salpingostomy). Ectopic pregnancies located in the tubal cornua, interstitial area, or uterine cervix are dangerous and difficult to manage. *Table 5* includes criteria for surgical management.

Expectant or medical management are options for hemodynamically stable women who have a nonviable pregnancy and are carefully selected and informed according to the criteria listed in *Table 5*.[42-45] The hCG level is the best predictor of successful management with methotrexate. A systematic review showed that failure with single-dose methotrexate occurred 3.7% of the time when hCG levels were below 5,000 mIU/mL versus 14.3% when above this cut-off level. Thus, methotrexate is used only in special circumstances when hCG levels exceed 5,000 mIU/mL.[46] Single and multiple-dose methotrexate protocols are available; multiple-dose protocols are more commonly used in cases of high hCG levels.[39]

Expectant management is used most often when the location of the pregnancy cannot be determined. Medical management with methotrexate,

Table 4. Diagnostic and Management Guidelines Related to the Possibility of Viable Intrauterine Pregnancy in Women With Pregnancies of Unknown Location

Finding	Key Points
No intrauterine fluid collection and normal (or near-normal) adnexa on ultrasonography [a]	A single measurement of hCG, regardless of its value, does not reliably distinguish between ectopic and intrauterine pregnancy (viable or nonviable) If a single hCG measurement is <3,500 mIU/mL, presumptive treatment for ectopic pregnancy with the use of methotrexate or other pharmacotherapy or surgical means should not be undertaken, in order to avoid the risk of interrupting a viable intrauterine pregnancy If a single hCG measurement is >3,500 mIU/mL, a viable intrauterine pregnancy is possible but unlikely. However, the most likely diagnosis is a nonviable intrauterine pregnancy, so it is generally appropriate to obtain at least one follow-up hCG measurement and a follow-up ultrasound before undertaking treatment for ectopic pregnancy
Ultrasonography not yet performed	The hCG levels in women with ectopic pregnancies are highly variable, often <1,000 mIU/mL, and the hCG level does not predict the likelihood of ectopic pregnancy rupture. Thus, when the clinical findings are suspicious for ectopic pregnancy, transvaginal ultrasonography is indicated even when the hCG level is low

Note: Criteria are from the Society of Radiologists in Ultrasound Multispecialty Consensus Conference on Early First Trimester Diagnosis of Miscarriage and Exclusion of a Viable Intrauterine Pregnancy, October 2012.

[a] Near-normal (ie, inconsequential) adnexal findings include corpus luteum, a small amount of free pelvic fluid, and paratubal cyst.

hCG = human chorionic gonadotropin.

Reprinted from Doubilet PM, Benson CB, Bourne T, et al; Society of Radiologists in Ultrasound Multispecialty Panel on Early First Trimester Diagnosis of Miscarriage and Exclusion of a Viable Intrauterine Pregnancy. Diagnostic criteria for nonviable pregnancy early in the first trimester. N Engl J Med. *2013;369(15):1443-1451.*

Table 5. Criteria for Ectopic Pregnancy Management Options

	Expectant Management	Medical Management With Methotrexate	Surgical Management With Laparoscopy or Open Laparotomy
Vital signs	Stable	Stable	Unstable
Bleeding	Minimal	Minimal to low	Hemoperitoneum
Reliability of follow-up	Requires reliable follow-up	Requires reliable follow-up	Best management when follow-up is not reliable
Tubal/Adnexal Characteristics	Adnexal mass <3 cm, No cardiac activity hCG level <1,000 mIU/mL and decreasing No signs of tubal rupture	Adnexal mass <4 cm No cardiac activity hCG level <5,000 to 10,000 mIU/mL No signs of tubal rupture No contraindications to methotrexate use	Advanced ectopic (elevated hCG levels, large mass, cardiac activity) OR uncertain diagnosis OR methotrexate contraindicated

Note: The decision for any of the above management plans depends on overall clinical, laboratory, and radiologic data as well as patient choice after informed decision-making process [Level A].

hCG: human chorionic gonadotropin.

Information from Cohen MA, Sauer MV. Expectant management of ectopic pregnancy. Clin Obstet Gynecol. *1999;42(1): 48-54, quiz 55-56; Mashiach S, Carp HJ, Serr DM. Nonoperative management of ectopic pregnancy. A preliminary report.* J Reprod Med. *1982;27(3):127-132; Adoni A, Milwidsky A, Hurwitz A, Palti Z. Declining beta-HCG levels: an indicator for expectant approach in ectopic pregnancy.* Int J Fertil. *1986;31(1):40-42; Garcia AJ, Aubert JM, Sama J, Josimovich JB. Expectant management of presumed ectopic pregnancies. Fertil Steril. 1987;48(3):395-400; American College of Obstetricians and Gynecologists. ACOG Practice Bulletin 193: tubal ectopic pregnancy.* Obstet Gynecol. *2018;131:e91-103.*

a folic acid antagonist, is appropriate for properly selected women and has been shown in randomized trials to be safe and effective. Methotrexate can be less costly and result in equal or better subsequent fertility than conservative surgical treatment.[39,47,48]

Monitoring the hCG level until it is negative after an abortion, spontaneous miscarriage, misoprostol use, or dilation and curettage for early pregnancy loss will help avoid missing a molar pregnancy or heterotopic pregnancy;[49] however, this may not be routinely needed when a prior ultrasound showed a normal IUP. If the hCG plateaus or increases, further investigation is indicated.

Gestational Trophoblastic Disease

Pathophysiology and Risk Factors

Gestational trophoblastic disease, or molar pregnancy, is an occasional cause of first-trimester bleeding in the United States (approximately 1:1,000 pregnancies), but is more common in Southeast Asia.[50]

A complete mole consists of placental proliferation in the absence of a fetus. Risk factors include extremes of age and previous molar pregnancy. The placental villi are swollen and often resemble clusters of grapes. Most complete moles have a 46, XX chromosomal composition, all derived from paternal sources. Mole recurrence may progress to metastatic choriocarcinoma. A partial mole is a molar placenta occurring together with a fetus, which is typically nonviable. Genetic testing usually reveals triploidy (69, XXY). Partial mole is less common than a complete mole and has a lower risk of recurrence.[50]

Signs, Symptoms, and Diagnosis

The signs and symptoms of gestational trophoblastic disease are listed in *Table 6*.

Treatment

Prompt evacuation of the uterus is the primary treatment. After evacuation of a complete mole, all women should undergo serial monitoring of hCG levels for 6 months to 1 year and use a highly reliable method of contraception.[51] If the hCG level plateaus or increases, then recurrence must be assumed, investigated, and treated with chemotherapy (methotrexate). Because of the relative rarity of this condition and the many possible complications, consultation is recommended when

the hCG level is not decreasing appropriately. Theca-lutein ovarian cysts (functional ovarian cysts which are typically bilateral and caused by elevated hCG levels) do not require management and will resolve after evacuation of the molar tissue. The risk of recurrence is 1:100 after one complete mole and 1:4 after two complete moles.[52]

Grief and Psychological Management of Early Pregnancy Loss

Miscarriage has different significance for different women. It often represents a major loss to the woman and her family. The grief reaction that follows can be similar in intensity to that experienced after other major losses, though women experience and describe it in varied ways. Although healing will occur, the time to recovery also varies. The feelings of loss tend to be strongest in the first 6 months after the miscarriage but can be persistent and pervasive enough to cause long-term symptoms or even affect the woman's next pregnancy. Women at risk of a stronger grief reaction include those who experience a missed abortion, loss at a later gestational age, and longer time to conception of their next pregnancy as well as those who have a critical self-perception.[53,54] A prospective study showed higher depression scores in younger women and those with a history of mental illness before or during pregnancy.[55]

Partners may also experience grief with pregnancy loss. Because partners are often a primary social support for women who are pregnant and attend many post-loss visits, it is important for the health care professional to include them in the care plan. Available evidence suggests that although the vast majority of women desire support from their health care professional, many do not receive the quantity or quality of support they desire.[56] The types of interventions that are most effective in managing psychological symptoms are uncertain, but the following approaches may be practical ways to mitigate the normal grief after early pregnancy loss.

Acknowledge and attempt to dispel guilt. Many women think that some action on their part caused or contributed to the miscarriage. This guilt can revolve around sexual activity, food, alcohol, tobacco, illicit drugs, medications, minor trauma, physical activity, religious or cultural beliefs, or emotional stress. When losses can be ascribed to a definite cause, women have lower levels of anxiety and grief. Therefore, evaluation of available tissue for chromosomal anomalies is recommended when possible.[57]

Even when a definitive cause cannot be ascertained, reassurance that the woman did nothing to cause the loss is appropriate. This reassurance may need to be repeated several times. Women should be counseled that genetic or developmental errors likely occurred early in the pregnancy, and there was no possibility of the pregnancy progressing to produce a live infant. The post-loss follow-up visit is not the time to focus on modifiable risk factors that might have contributed to the loss (ie, alcohol or tobacco use). Addressing these issues before future pregnancies is indicated, but is better performed after the acute trauma of loss diminishes. The woman's religious belief system can be called on during this counseling.

Acknowledge and legitimize grief. Allowing women to discuss their emotions surrounding their loss might be the most important aspect of psychological care. One study suggests that women who had a medical follow-up visit at which they were not provided an opportunity to discuss their feelings had more anxiety and depression than those who had no follow-up.[58] Women and their partners should be allowed to cry or feel sad. Minimizing their feelings can isolate them and decrease the medical professional's credibility. Legitimize their feelings by confirming that miscarriage is the death of an infant. Comments

Table 6. Trophoblastic Disease Signs and Symptoms

Uterus is larger than expected for gestational age
Absent fetal heartbeat
Higher than expected hCG levels (except in cases of a partial mole)
Hyperemesis, pregnancy-induced hypertension at an early gestational age, and/or thyrotoxicosis
Ovarian enlargement, caused by theca-lutein cysts resulting from ovarian hyperstimulation because of high hCG levels
Vaginal bleeding in the first or early second trimester, which is often dark and may cause anemia
Grapelike vesicles are passed in cases that progress into the second trimester

hCG = human chorionic gonadotropin

such as "you can try again" or "at least it happened early" are inappropriate. Simple measures that validate grief should not be underestimated. Listening to the woman, holding her hand, or telling her how sad you feel for her can help her through this traumatic period. The woman should be seen within 1 to 2 weeks in the office or called on the phone a few days after the miscarriage.

Reassure the woman about the future. Grief will diminish with time. Most women have an excellent likelihood of a subsequent normal pregnancy. With fewer than three miscarriages, the risk of miscarriage in future pregnancies is no greater than usual. It is important to explain that the next pregnancy will not need to be managed differently because of the miscarriage. This is an excellent time to encourage the initiation of a prenatal vitamin.[53,54]

Counsel the woman about how to tell family and friends about the miscarriage. If family members and friends knew about the pregnancy, a designated individual can inform them of the loss. This allows them to express their sympathy and provide emotional support and may avoid embarrassing encounters in which others assume the pregnancy is progressing. If the pregnancy was unknown to family and friends, they may recognize and be concerned about external signs of grief or distress. A decision about whether to tell them must be made. Informing other children in the family can also be difficult. However, families often find comfort in allowing children to share in the grieving and remembering process. Parents should be encouraged to discuss the loss in honest and developmentally appropriate ways, just as they would the death of another family member.

Warn women of the anniversary phenomenon. A recurrence of feelings of grief on their due date or the anniversary of the miscarriage can occur. This can also occur at the birth of a friend's infant or during the woman's subsequent pregnancy. Posttraumatic stress disorder should be considered in women experiencing prolonged grieving, anxiety, or other symptoms that affect their general or reproductive functioning.

Include the partner in psychological care. Partners often feel the pain of loss and should be included in counseling and decision making. Compared with women's reactions to pregnancy loss, men's reactions are more strongly influenced by the status of the marital relationship. Offering couples counseling and including partners in the healing process can speed resolution for both partners.[53,54]

Assess the level of grief and adjust counseling accordingly. Many women are ambivalent or distressed by the pregnancy and may experience mixed feelings or profound relief at the loss. A history of abortion, failed birth control, or rape may further complicate the emotional response. Allowing the woman to express emotions in a supportive and nonjudgmental atmosphere is always an appropriate intervention.

Rh Prophylaxis and Future Conception After Pregnancy Loss

Several follow-up issues must be addressed after any type of pregnancy loss. Women who are Rh-negative who have a miscarriage during the first trimester should receive a minimum of 50 mcg of anti-D immune globulin.[59] Those who have received instrumentation are at higher risk of Rh sensitization. Women with miscarriages that occur after 12 weeks' gestation should receive 300 mcg of anti-D immune globulin. Anti-D immune globulin is also recommended for women who are Rh negative who have been treated for molar pregnancy.[59] Providers should refer to current guidelines in special cases.[59] Contraception should be discussed and started immediately if conception is not desired; all methods are equally safe immediately after spontaneous abortion or ectopic pregnancy. There is no good evidence suggesting an ideal interpregnancy interval.[60] Folic acid supplementation before future conception attempts substantially reduces the risk of neural tube defects.[61]

First-Trimester Complications in Low-Resource Settings

First-trimester miscarriage, ectopic pregnancy, and complications from unsafe abortion are major causes of maternal morbidity in developing nations.[62] The diagnostic schema presented in the ALSO syllabus relies heavily on the availability of highly sensitive quantitative beta hCG measurements and first-trimester ultrasound. Diagnosing the etiology of first-trimester bleeding without these technologies necessitates accurate physical examination skills, experience with medical interventions appropriate for low-resource settings, and the knowledge of when to refer for acute surgical or diagnostic evaluation. Culdocentesis, while rarely used in high-resource settings, may provide

valuable information regarding cul-de-sac fluid in low-resource settings.

The inability to identify women who are Rh negative and administer anti-D immune globulin may have severe consequences to the fetus in the subsequent pregnancy. Many women in developing countries will lack access to the facilities and physicians required to optimally manage an alloimmunized pregnancy.

Unsafe abortion occurs predominantly (95%) in developing countries and accounts for approximately 13% of maternal deaths worldwide.[63] The World Health Organization estimates that 20% to 30% of the 20 million unsafe abortions performed each year result in reproductive tract infections, and that 2% of women are infertile due to undergoing unsafe abortion.[64] Access to contraception can decrease unwanted pregnancies and unsafe abortion.

When a woman presents with septic abortion, typically from attempted unsafe abortion or less commonly from incomplete miscarriage, the recommendation is to simultaneously treat with broad-spectrum antibiotics and to promptly evacuate the uterus by suction curettage with manual vacuum or electric suction. Uterine evacuation should not be delayed to initiate antibiotics, as septic abortion cannot be adequately treated until the uterus is emptied.[63]

Additional information is available at www.aafp.org/globalalso.

Summary

First-trimester pregnancy complications are common, and the differential diagnosis includes life-threatening conditions such as ectopic pregnancy. Knowledge and application of discriminatory criteria can significantly aid in distinguishing among normal early pregnancy, miscarriage, and ectopic pregnancy. Medical treatment of ectopic pregnancy is possible in properly selected cases. In incomplete miscarriage, nonsurgical management has a high likelihood of success depending on the diagnosis. In embryonic demise or anembryonic pregnancy, misoprostol or surgical management are significantly more effective than expectant management.[23-25]

Because there is a lack of clear superiority of expectant versus medical or surgical management of miscarriage, the woman's preference should play a dominant role in decision-making.[65] When the choice is made to manage early pregnancy failure by other than expectant means, vaginal misoprostol is highly effective, safe, and well-accepted by women, with fewer gastrointestinal adverse effects than the oral route.[23-25] Evidence does not support the use of antibiotics in all women with incomplete abortion. After any type of first-trimester pregnancy loss, women who are Rh-negative should receive a minimum of 50 mcg of anti-D immune globulin.[59] Acknowledgement of grief and demonstrations of empathy and reassurance are useful techniques in counseling women after miscarriage.

Nursing Considerations: First-Trimester Pregnancy Complications

- Identify blood type and Rh status — evaluate for anti-D immune globulin
- Educate patients regarding normal blood loss and signs and symptoms to report
- Advocate for a contraceptive plan after a first-trimester pregnancy loss
- Acknowledge and validate the grief process for patients and partners, meeting individual expressed needs and level of support, regardless of time lapsed since the loss occurred

References

1. Wilcox AJ, Weinberg CR, O'Connor JF, et al. Incidence of early loss of pregnancy. *N Engl J Med*. 1988;319(4):189-194.
2. Cole LA, Sutton-Riley JM, Khanlian SA, et al. Sensitivity of over-the-counter pregnancy tests: comparison of utility and marketing messages. *J Am Pharm Assoc*. 2005;45(5):608-615.
3. Ehrenkranz JR. Home and point-of-care pregnancy tests: a review of the technology. *Epidemiology*. 2002;13(Suppl 3):S15-S18.
4. Barnhart KT. Clinical practice. Ectopic pregnancy. *N Engl J Med*. 2009;361(4):379-387.
5. Seeber BE, Barnhart KT. Suspected ectopic pregnancy. *Obstet Gynecol*. 2006;107(2 Pt 1):399-413. Erratum in *Obstet Gynecol*. 2006;107
6. Laing FC, Frates MC, Benson CB. Ultrasound Evaluation During the First Trimester of Pregnancy. In: Callen PW, ed. *Ultrasonography in Obstetrics and Gynecology*. 4th ed. W.B. Saunders Co; 2000.
7. Paspulati RM, Bhatt S, Nour SG. Sonographic evaluation of first-trimester bleeding. *Radiol Clin North Am*. 2004;42(2):297-314. Erratum in *Radiol Clin North Am*. 2008;46
8. Kadar N, Bohrer M, Kemmann E, Shelden R. The discriminatory human chorionic gonadotropin zone for endovaginal sonography: a prospective, randomized study. *Fertil Steril*. 1994;61(6):1016-1020.
9. Committee on Practice Bulletins—Gynecology. The American College of Obstetricians and Gynecologists Practice Bulletin no. 150. Early pregnancy loss. *Obstet Gynecol*. 2015;125(5):1258-1267.
10. Whitworth M, Bricker L, Mullan C. Ultrasound for fetal assessment in early pregnancy. *Cochrane Database Syst Rev*. 2015;(7):CD007058.
11. Goddijn M, Leschot NJ. Genetic aspects of miscarriage. *Best Pract Res Clin Obstet Gynaecol*. 2000;14(5):855-865.
12. Committee on Practice Bulletins—Gynecology. ACOG Practice Bulletin no. 191: tubal ectopic pregnancy. *Obstet Gynecol*. 2018;131(2):e65-e77.
13. Doubilet PM, Benson CB, Bourne T, et al.; Society of Radiologists in Ultrasound Multispecialty Panel on Early First Trimester Diagnosis of Miscarriage and Exclusion of a Viable Intrauterine Pregnancy. Diagnostic criteria for nonviable pregnancy early in the first trimester. *N Engl J Med*. 2013;369(15):1443-1451.
14. Smith KE, Buyalos RP. The profound impact of patient age on pregnancy outcome after early detection of fetal cardiac activity. *Fertil Steril*. 1996;65(1):35-40.
15. Creinin MD, Harwood B, Guido RS, et al.; NICHD Management of Early Pregnancy Failure Trial. Endometrial thickness after misoprostol use for early pregnancy failure. *Int J Gynaecol Obstet*. 2004;86(1):22-26.
16. Eschenbach DA. Treating spontaneous and induced septic abortions. *Obstet Gynecol*. 2015;125(5):1042-1048.
17. Stubblefield PG, Grimes DA. Septic abortion. *N Engl J Med*. 1994;331(5):310-314.
18. Bennett GL, Bromley B, Lieberman E, Benacerraf BR. Subchorionic hemorrhage in first-trimester pregnancies: prediction of pregnancy outcome with sonography. *Radiology*. 1996;200(3):803-806.
19. Hasan R, Baird DD, Herring AH, et al. Association between first-trimester vaginal bleeding and miscarriage. *Obstet Gynecol*. 2009;114(4):860-867.
20. Chen BA, Creinin MD. Contemporary management of early pregnancy failure. *Clin Obstet Gynecol*. 2007;50(1):67-88.
21. Luise C, Jermy K, May C, et al. Outcome of expectant management of spontaneous first trimester miscarriage: observational study. *BMJ*. 2002;324(7342):873-875.
22. Allison JL, Sherwood RS, Schust DJ. Management of first trimester pregnancy loss can be safely moved into the office. *Rev Obstet Gynecol*. 2011;4(1):5-14.
23. Zhang J, Gilles JM, Barnhart K, et al.; National Institute of Child Health Human Development (NICHD) Management of Early Pregnancy Failure Trial. A comparison of medical management with misoprostol and surgical management for early pregnancy failure. *N Engl J Med*. 2005;353(8):761-769.
24. Bagratee JS, Khullar V, Regan L, et al. A randomized controlled trial comparing medical and expectant management of first trimester miscarriage. *Hum Reprod*. 2004;19(2):266-271.
25. Weeks A, Alia G, Blum J, et al. A randomized trial of misoprostol compared with manual vacuum aspiration for incomplete abortion. *Obstet Gynecol*. 2005;106(3):540-547.
26. Winikoff B. Pregnancy failure and misoprostol—time for a change. *N Engl J Med*. 2005;353(8):834-836.
27. Schreiber CA, Creinin MD, Atrio J, et al. Mifepristone pretreatment for the medical management of early pregnancy loss. *N Engl J Med*. 2018;378(23):2161-2170.
28. Raymond EG, Blanchard K, Blumenthal PD, et al.; Mifeprex REMS Study Group. Sixteen years of overregulation: time to unburden Mifeprex. *N Engl J Med*. 2017;376(8):790-794.
29. May W. Gülmezoglu AM, Ba-Thike K. Antibiotics for incomplete abortion. *Cochrane Database Syst Rev*. 2007;(4):CD001779.
30. American College of Obstetricians and Gynecologists. ACOG Practice Bulletin no. 195. Prevention of infection after gynecologic procedures. *Obstet Gynecol*. 2018;131(6):e172-e189.
31. Fjerstad M, Trussell J, Sivin I, et al. Rates of serious infection after changes in regimens for medical abortion. *N Engl J Med*. 2009;361(2):145-151.
32. Buss L, Tolstrup J, Munk C, et al. Spontaneous abortion: a prospective cohort study of younger women from the general population in Denmark. Validation, occurrence and risk determinants. *Acta Obstet Gynecol Scand*. 2006;85(4):467-475.
33. Okusanya BO, Oduwole O, Effa EE. Immediate postabortal insertion of intrauterine devices. *Cochrane Database Syst Rev*. 2014;(7):CD001777.

34. Committee on Practice Bulletins-Gynecology, Long-Acting Reversible Contraception Work Group. Practice Bulletin no. 186: long-acting reversible contraception: implants and intrauterine devices. *Obstet Gynecol*. 2017; 130(5):e251-e269.
35. Bouyer J, Coste J, Shojaei T, et al. Risk factors for ectopic pregnancy: a comprehensive analysis based on a large case-control, population-based study in France. *Am J Epidemiol*. 2003;157(3):185-194.
36. Sawyer E, Jurkovic D. Ultrasonography in the diagnosis and management of abnormal early pregnancy. *Clin Obstet Gynecol*. 2007;50(1):31-54.
37. Mahony BS, Filly RA, Nyberg DA, Callen PW. Sonographic evaluation of ectopic pregnancy. *J Ultrasound Med*. 1985;4(5):221-228.
38. Connolly A, Ryan DH, Stuebe AM, Wolfe HM. Reevaluation of discriminatory and threshold levels for serum β-hCG in early pregnancy. *Obstet Gynecol*. 2013;121(1): 65-70.
39. American College of Obstetricians and Gynecologists. ACOG Practice Bulletin no. 193: Tubal ectopic pregnancy. *Obstet Gynecol*. 2018;131(3):e91-e103.
40. ACOG Committee on Practice Bulletins—Gynecology. ACOG Practice Bulletin no. 104: antibiotic prophylaxis for gynecologic procedures. *Obstet Gynecol*. 2009; 113(5):1180-1189.
41. van Mello NM, Mol F, Ankum WM, et al. Ectopic pregnancy: how the diagnostic and therapeutic management has changed. *Fertil Steril*. 2012;98(5):1066-1073.
42. Cohen MA, Sauer MV. Expectant management of ectopic pregnancy. *Clin Obstet Gynecol*. 1999;42(1):48-54, quiz 55-56.
43. Mashiach S, Carp HJ, Serr DM. Nonoperative management of ectopic pregnancy. A preliminary report. *J Reprod Med*. 1982;27(3):127-132.
44. Adoni A, Milwidsky A, Hurwitz A, Palti Z. Declining beta-HCG levels: an indicator for expectant approach in ectopic pregnancy. *Int J Fertil*. 1986;31(1):40-42.
45. Garcia AJ, Aubert JM, Sama J, Josimovich JB. Expectant management of presumed ectopic pregnancies. *Fertil Steril*. 1987;48(3):395-400.
46. Menon S, Colins J, Barnhart KT. Establishing a human chorionic gonadotropin cutoff to guide methotrexate treatment of ectopic pregnancy: a systematic review. *Fertil Steril*. 2007;87(3):481-484.
47. Stovall TG, Ling FW. Single-dose methotrexate: an expanded clinical trial. *Am J Obstet Gynecol*. 1993;168(6 Pt 1):1759-1762, discussion 1762-1765.
48. Lipscomb GH, McCord ML, Stovall TG, et al. Predictors of success of methotrexate treatment in women with tubal ectopic pregnancies. *N Engl J Med*. 1999;341(26): 1974-1978.
49. Dresang LT. A molar pregnancy detected by following beta-human chorionic gonadotropin levels after a first trimester loss. *J Am Board Fam Pract*. 2005;18(6): 570-573.
50. Lurain JR. Gestational trophoblastic disease I: epidemiology, pathology, clinical presentation and diagnosis of gestational trophoblastic disease, and management of hydatidiform mole. *Am J Obstet Gynecol*. 2010;203(6): 531-539.
51. Berkowitz RS, Goldstein DP. Clinical practice. Molar pregnancy. *N Engl J Med*. 2009;360(16):1639-1645.
52. Eagles N, Sebire NJ, Short D, et al. Risk of recurrent molar pregnancies following complete and partial hydatidiform moles. *Hum Reprod*. 2015;30(9):2055-2063.
53. Brier N. Grief following miscarriage: a comprehensive review of the literature. *J Womens Health (Larchmt)*. 2008;17(3):451-464.
54. Murphy FA, Lipp A, Powles DL. Follow-up for improving psychological well being for women after a miscarriage. *Cochrane Database Syst Rev*. 2012;3(3):CD008679.
55. Mann JR, McKeown RE, Bacon J, et al. Predicting depressive symptoms and grief after pregnancy loss. *J Psychosom Obstet Gynaecol*. 2008;29(4):274-279.
56. Nikcevic AV, Tunkel SA, Nicolaides KH. Psychological outcomes following missed abortions and provision of follow-up care. *Ultrasound Obstet Gynecol*. 1998;11(2): 123-128.
57. Athey J, Spielvogel AM. Risk factors and interventions for psychological sequelae in women after miscarriage. *Prim Care Update Ob Gyns*. 2000;7(2):64-69.
58. Lee C, Slade P, Lygo V. The influence of psychological debriefing on emotional adaptation in women following early miscarriage: a preliminary study. *Br J Med Psychol*. 1996;69(Pt 1):47-58.
59. Committee on Practice Bulletins-Obstetrics. Practice Bulletin no. 181: Prevention of Rh D Alloimmunization. *Obstet Gynecol*. 2017;130(2):e57-e70.
60. Wong LF, Schliep KC, Silver RM, et al. The effect of a very short interpregnancy interval and pregnancy outcomes following a previous pregnancy loss. *Am J Obstet Gynecol*. 2015;212(3):375.e1-375.e11.
61. Cordero AM, Crider KS, Rogers LM, et al. Optimal serum and red blood cell folate concentrations in women of reproductive age for prevention of neural tube defects: World Health Organization guidelines. *MMWR Morb Mortal Wkly Rep*. 2015;64(15):421-423.
62. Khan KS, Wojdyla D, Say L. et al. WHO analysis of causes of maternal death: a systematic review. *Lancet*. 2006;367(9516):1066-1074.
63. Warriner IK, Shah IH, eds. *Preventing Unsafe Abortion and Its Consequences: Priorities for Research and Action*. Guttmacher Institute; 2006.
64. Grimes DA, Benson J, Singh S, et al. Unsafe abortion: the preventable pandemic. *Lancet*. 2006;368(9550): 1908-1919.
65. Nanda K, Lopez LM, Grimes DA, et al. Expectant care versus surgical treatment for miscarriage. *Cochrane Database Syst Rev*. 2012;3(3):CD003518.

Appendix
Uterine Aspiration for Miscarriage: Electric Suction Dilation and Curettage or Manual Vacuum Aspiration

Most first-trimester miscarriages occur completely and spontaneously without intervention. And when intervention is selected, medical management is highly effective. Uterine aspiration by electric suction or manual vacuum aspiration may be indicated when:

1. Heavy bleeding is present (greater than one pad per hour)
2. The woman is clinically stable (no bleeding or cramping), but pregnancy loss is shown conclusively, and the woman prefers intervention to expectant management
3. Ectopic pregnancy needs to be ruled out. In certain situations, a clinical distinction cannot be made between an ectopic and an intrauterine pregnancy (IUP). If tissue from a dilation and curettage procedure contains chorionic villi, the pregnancy was intrauterine. Rarely, an intrauterine and an ectopic pregnancy (heterotopic pregnancy) may coexist, creating a confusing and dangerous clinical situation.

Contraindications to Uterine Aspiration

1. Medical contraindications are rare but include active pelvic infection.
2. Pregnancy loss is not proven to the woman's satisfaction, the physician's satisfaction, or both.
3. The woman prefers to await spontaneous completion for any reason (eg, religious beliefs, cost, desire to avoid a procedure or drug).

Uterine aspiration is not appropriate if the spontaneous abortion appears to be complete based on the following criteria:

- The uterus is small and firm
- Scant or no bleeding is occurring
- Tissue has been passed and is available for inspection and appears complete
- The woman is reliable for follow-up
- Ultrasound examination (preferably transvaginal) shows an empty uterus.

Procedure for Uterine Aspiration Performed Under Local Anesthesia

1. Place an intravenous (IV) line if the woman is bleeding heavily or if IV medications will be used.
2. An Rh factor test should be obtained in women whose status is unknown. A hematocrit or hemoglobin should be obtained if there is suspicion of anemia or excessive blood loss. A white blood cell count, prothrombin time, partial thromboplastin time, fibrin split products, and blood type and screen may be obtained depending on clinical circumstances (eg, heavy bleeding).
3. Sedation and analgesia should be administered. Two to 5 mg IV midazolam and 50 to 100 mcg IV fentanyl are commonly used.[1,2] Alternatively, other opioids or benzodiazepines may be used. In many situations, the woman's partner or another support individual may be present during the procedure.
4. The size and position of the uterus should be identified by bimanual examination. If the fetal measurements and uterine size are greater than those for 14 weeks' gestation, a dilation-and-extraction procedure is indicated, which requires advanced training beyond that required for first-trimester aspiration. If a procedure is required beyond 14 weeks' gestation, consider adding 20 units of oxytocin to IV fluids.
5. The cervix should be exposed with a speculum. A medium Graves speculum typically suffices. The cervix and posterior fornix are cleansed with an antiseptic solution. The anterior lip of the cervix is then grasped with a single-toothed tenaculum.
6. A paracervical block can be achieved with 20 cc of 1% lidocaine or another local anesthetic agent via a 20-gauge spinal needle. One-fourth of the amount of the block is administered at 2:00, 4:00, 8:00, and 10:00 positions; or one-half the amount of the block at 4:00 and 8:00 where the cervix meets the vagina. A superficial wheal is raised, and

the syringe is aspirated before injecting to avoid intravascular injection. Several variants of the paracervical block exist, and all are equally satisfactory.[3]

7. If the cervix is closed, or insufficiently dilated to easily admit the required suction curette, it can be progressively dilated using Hegar cervical dilators. Dilation should occur to the number of millimeters that is equivalent to the estimated gestational age in weeks or 1 mm less (eg, dilate to 9 or 10 mm to aspirate a missed abortion at 10 weeks' gestation). Control is indicated for this portion of the procedure, as dilators and uterine sounds cause the largest number of uterine perforations. If the woman is clinically stable, overnight dilation with laminaria is an option. Another means of dilation that has been successful but is not approved by the Food and Drug Administration, is the buccal, sublingual, or vaginal administration of misoprostol 400 mcg 2 to 3 hours before the procedure.[4]

8. If the os is open, ring forceps can be used to remove any loose tissue that is encountered.

9. The suction curette should be equivalent to the size of the uterus in weeks' gestation (eg, a number 10 curette for a uterus of 10 weeks' gestation in size) is appropriate. A curved curette is used if the uterus is anteflexed or retroflexed. A straight curette can be used if the uterus is in midposition. The suction curette is inserted along the previously determined axis of the uterus, until slight resistance is felt, while exerting slight traction on the tenaculum to stabilize the cervix and straighten the cervico-vaginal angle. The curette should never be forced after passing the internal os because perforation is the most serious potential complication of the procedure. A "pencil grip" may help avoid using excessive force.

10. After the curette is in place near the fundus, the suction hosing is attached and the suction machine is turned on. Then the suction valve on the handle of the hose. Sixty cm of mercury (Hg) or greater must be achieved for adequate suction.

11. With the suction activated, the curette is rotated several times in one direction, then several times in the other direction, with a slight in-and-out motion. Most of the pressure should be maintained laterally, and forceful jabbing at the uterine fundus should be avoided because perforation is a risk. The amount and nature of tissue that appears in the plastic curette should be observed carefully. Products of conception often appear tan or grey, admixed with blood and clots. Yellowish fluid can be noted. The curette is withdrawn slowly, avoiding the vaginal side wall while the suction is operating.

12. The suction and rotation sequence can be repeated after reinserting the curette into the uterus.

13. Manual vacuum aspiration is accomplished with a simple handheld plastic syringe that generates its own suction mechanically. This device is inexpensive, easy to use, and does not require electricity. It is particularly appropriate for completions of early gestations (eg, less than 8 to 10 weeks' menstrual age). It can be used in the office setting where a suction machine is not accessible. It is also appropriate in developing countries where electricity is not available.

14. A light, sharp curettage of the uterus can be performed to determine that it is empty, followed by one more pass of the suction curette. This is no longer routinely indicated because of the increased pain and increasing use of postprocedure vaginal ultrasound to confirm completion in association with tissue examination.

15. After operator examination of the tissue, the tissue should be sent to the pathology department for examination and confirmation of diagnosis. To confirm an IUP, chorionic villi must be identified. The tissue must be sent to a laboratory for pathology unless the presence of villi or an embryo is conclusively confirmed by the physician performing the uterine aspiration.

16. After the uterine aspiration is completed, the woman should be monitored for excessive bleeding. Misoprostol can be administered by the rectal, buccal, or sublingual route in a dose of 400 to 800 mcg. Methergine 0.2 mg may be administered intramuscularly or orally. Transfusions are rarely required.[5]

17. If the woman is Rh negative, a minimum of a 50 mcg (ie, *mini-dose*) of anti-D immune globulin should be administered.[6]

18. Doxycycline 200 mg is administered orally within 1 hour before the procedure to decrease the likelihood of endometritis.[7] Metronidazole 500 mg orally is an option if the woman has doxycycline allergy.[8]

Complications of Uterine Aspiration

Complications of uterine aspiration (suction dilation and curettage) can occur. Poor outcomes

can be prevented with careful performance of the procedure, consultation with more experienced physicians when needed, and a high index of suspicion for identifying complications.

Perforation

Perforation occurs when an instrument passes through the uterine wall. The diagnosis is typically apparent when a sound or dilator passes through the cervix to a significantly greater depth than expected. Occasionally, a suction curette or a sharp curette will draw maternal abdominal contents such as omentum or bowel out through the cervix. Heavy bleeding, signs of peritonitis, or evidence of intra-abdominal bleeding also can help identify perforation. If perforation occurs with a blunt instrument, such as a uterine sound, and if the dilation and curettage has been completed, observation alone may suffice.[9,10] If perforation has occurred with a sharp instrument, such as a curette or with a suction curette, laparoscopy or laparotomy may be indicated. If the uterine aspiration has not been completed at the time the perforation is identified, it can be completed under ultrasonic or laparoscopic guidance. Broad-spectrum antibiotics should be considered for any perforation.[11]

Incomplete Evacuation

Incomplete evacuation is identified by continued bleeding and cramping after the procedure, or ultrasound evidence of retained tissue or endometritis. Incomplete evacuation can be managed by repeating the procedure. Ultrasonic guidance or general anesthesia is often helpful. Antibiotics are recommended if the second procedure occurs more than a few hours after the first. Routine use of intraoperative ultrasonography can minimize this risk.

Vaginal Bleeding

The differential diagnosis of bleeding includes perforation, incomplete evacuation with retained tissue, cervical or uterine injury, or a bleeding disorder. Methylergonovine 0.2 mg by mouth four times a day for 2 days is commonly administered to women with more than average bleeding during and after a procedure. An alternative is misoprostol 200 mcg by mouth four times a day for 2 days. This is an off-label use of misoprostol, but is effective in practice because of its powerful uterotonic effect.[12]

Infection

Infection may be referred to as septic abortion, endometritis, paraendometritis, or pelvic peritonitis. It is diagnosed by the presence of fever, uterine and parauterine tenderness, peritonitis, and an elevated leukocyte count. Management is with antibiotics. In women who are ill and require hospitalization, IV third-generation cephalosporin or triple antibiotics (ampicillin, gentamycin, and clindamycin or metronidazole) may be required.[11] Women with less severe infection can be treated in the outpatient setting. Clear guidelines for antibiotic regimens are lacking. When tissue is found to be retained, repeating the uterine evacuation may be necessary. Oxytocic drugs should be administered as described previously in the Vaginal Bleeding section. Rarely, in women with more severe infection, hospitalization and hysterectomy may be necessary.

Late Sequelae

Intrauterine synechiae (Asherman syndrome) is often discussed but rarely seen. It is most likely to occur when suction dilation and curettage is performed in the presence of infection, a prolonged missed abortion, or postpartum. Incompetent cervix can occur rarely because of cervical injury. The most common late sequelae to suction dilation and curettage are depression and related psychological reactions to the loss of the pregnancy.

References

1. Cunningham FG, Leveno KJ, Bloom SL, et al., eds. *Williams Obstetrics*. 24th ed. McGraw-Hill Education/Medical; 2014.
2. Westfall JM, Sophocles A, Burggraf H, Ellis S. Manual vacuum aspiration for first-trimester abortion. *Arch Fam Med*. 1998;7(6):559-562.
3. Renner RM, Nichols MD, Jensen JT, Li H, Edelman AB. Paracervical block for pain control in first-trimester surgical abortion: a randomized controlled trial. *Obstet Gynecol*. 2012;119(5):1030-1037.
4. Ngai SW, Tang OS, Lao T, Ho PC, Ma MK. Oral misoprostol versus placebo for cervical dilatation before vacuum aspiration in first trimester pregnancy. *Hum Reprod*. 1995;10(5):1220-1222.
5. American College of Obstetricians and Gynecologists' Committee on Practice Bulletins—Gynecology. ACOG Practice Bulletin no. 200: early pregnancy loss. *Obstet Gynecol*. 2018;132(5):e197-e207.
6. Committee on Practice Bulletins-Obstetrics. Practice Bulletin no. 181: prevention of Rh D alloimmunization. *Obstet Gynecol*. 2017;130(2):e57-e70.
7. American College of Obstetricians and Gynecologists. ACOG Practice Bulletin no. 195. Prevention of infection after gynecologic procedures. *Obstet Gynecol*. 2018;131(6):e172-e189.
8. Achilles SL, Reeves MF; Society of Family Planning. Prevention of infection after induced abortion: release date October 2010: SFP Guideline 20102. *Contraception*. 2011;83(4):295-309.
9. Aydeniz B, Gruber IV, Schauf B, et al. A multicenter survey of complications associated with 21,676 operative hysteroscopies. *Eur J Obstet Gynecol Reprod Biol*. 2002;104(2):160-164.
10. Ben-Baruch G, Menczer J, Shalev J, et al. Uterine perforation during curettage: perforation rates and postperforation management. *Isr J Med Sci*. 1980;16(12):821-824.
11. Shakir F, Diab Y. The perforated uterus. *TOG*. 2013;15(4):256-261.
12. Weeks A, Alia G, Blum J, et al. A randomized trial of misoprostol compared with manual vacuum aspiration for incomplete abortion. *Obstet Gynecol*. 2005;106(3):540-547.

Cesarean Delivery

Learning Objectives

1. Develop a plan to obtain advanced procedural training, competence, and experience in performing cesarean deliveries.
2. Outline management strategies for complications associated with cesarean delivery, indications for consultation.
3. Determine local privileging requirements, including necessary documentation, to perform cesarean deliveries.

Introduction

The Recommended Curriculum Guidelines for Family Medicine Residents: Maternity Care, which is endorsed by the American Academy of Family Physicians (AAFP) describe core and advanced obstetric training for family physicians including cesarean delivery.[1] A 2018 AAFP position paper, *Cesarean Delivery in Family Medicine*, details scope of practice for family physicians, training methods, testing, demonstrated proficiency, and documentation, as well as credentialing and privileges.[2] A consensus guideline for family medicine maternity care training describes a scope of advanced maternity care training that includes operative and high-risk obstetric care.[3]

Family physicians performing cesarean deliveries are essential in maintaining rural maternity care services.[4] This chapter will review cesarean delivery within the contexts of family physicians, obstetric and gynecology residents, and certified nurse midwives who are consulting, first assisting, operating as primary surgeon, performing resuscitative hysterotomy, and attending women planning labor after cesarean (LAC).

History

The origin of the term *cesarean* is not entirely clear.[5] One possible origin of the term is the Latin verb *caedere*, which means to cut. Others think the term originated from the Roman custom, Lex Cesare, which mandated postmortem operative delivery when women died during childbirth, so the woman and infant could be buried separately.

Epidemiology

Frequency

The cesarean delivery rate was 31.9% of US births in 2018.[6] The cesarean delivery rate in the United States increased from 4.5% in 1965 to 20.7% in 1996.[6] The rate peaked in 2009 at 32.9% after increasing every year since 1996.[6] The increase is a result of the increased rate of primary cesarean delivery and the decreased rate of vaginal birth after cesarean delivery (VBAC).

Safe Prevention of the Primary Cesarean Delivery

The rapid increase in cesarean delivery rates from 1996 to 2011 without clear evidence of a concomitant decrease in maternal or neonatal morbidity or mortality has raised concern that cesarean delivery is overused.[7] In 2014, ACOG and the Society for Maternal-Fetal Medicine (SMFM) developed recommendations for the safe prevention of primary cesarean delivery (*Table 1*).[7] The consensus statement gave several examples of interventions which can contribute to the safe lowering of the primary cesarean delivery rate, including revisiting the definition of labor dystocia in the first and second stage of labor; improving and standardizing fetal heart rate (FHR) interpretation and management; increasing access to continuous labor support; attempting external cephalic version (ECV) for malpresentation; and attempting a trial of labor in women with twin gestations.[7]

Morbidity and Mortality

The maternal mortality rate for elective repeat cesarean delivery is 13.4 per 100,000 births in the United States.[8] Half of these mortalities are related to intraoperative complications, and the others are related to anesthetic and postoperative complications. In recent years, there has been a shift in the etiology of mortalities from hemorrhage and infection to thromboembolic events (see Technical Pitfalls section).[8]

Table 1. Recommendations for the Safe Prevention of Primary Cesarean Delivery

Recommendations	Grade of Recommendations
First stage of labor	
A prolonged latent phase (eg, greater than 20 hours in nulliparous women and greater than 14 hours in multiparous women) should not be an indication for cesarean delivery	1B Strong recommendation, moderate-quality evidence
Slow but progressive labor in the first stage of labor should not be an indication for cesarean delivery	1B Strong recommendation, moderate-quality evidence
Cervical dilation of 6 cm should be considered the threshold for the active phase of most women in labor. Thus, before 6 cm dilation is achieved, standards of active phase progress should not be applied	1B Strong recommendation, moderate-quality evidence
Cesarean delivery for active phase arrest in the first stage of labor should be reserved for women at or beyond 6 cm dilation with ruptured membranes who fail to progress despite 4 hours of adequate uterine activity, or at least 6 hours of oxytocin administration with inadequate uterine activity and no cervical change	1B Strong recommendation, moderate-quality evidence
Second stage of labor	
A specific absolute maximum length of time spent in the second stage of labor beyond which all women should undergo operative delivery has not been identified	1C Strong recommendation, low-quality evidence
Before diagnosing arrest of labor in the second stage, if the maternal and fetal conditions permit, allow for the following: • At least 2 hours of pushing in multiparous women • At least 3 hours of pushing in nulliparous women. Longer durations may be appropriate on an individualized basis (eg, with the use of epidural analgesia or with fetal malposition) if progress is being documented	1B Strong recommendation, moderate-quality evidence
Operative vaginal delivery in the second stage of labor by experienced and well-trained physicians should be considered a safe, acceptable alternative to cesarean delivery. Training in, and ongoing maintenance of, practical skills related to operative vaginal delivery should be encouraged	1B Strong recommendation, moderate-quality evidence
Manual rotation of the fetal occiput in the setting of fetal malposition in the second stage of labor is a reasonable intervention to consider before moving to operative vaginal delivery or cesarean delivery. To safely prevent cesarean deliveries in the setting of malposition, it is important to assess the fetal position in the second stage of labor, particularly in the setting of abnormal fetal descent	1B Strong recommendation, moderate-quality evidence
Fetal heart rate monitoring	
Amnioinfusion for repetitive variable fetal heart rate decelerations may safely reduce the rate of cesarean delivery	1A Strong recommendation, high-quality evidence
Scalp stimulation can be used as a means of assessing fetal acid–base status when abnormal or indeterminate (formerly, nonreassuring) fetal heart patterns (eg, minimal variability) are present and is a safe alternative to cesarean delivery in this setting	1C Strong recommendation, low-quality evidence

continued

Indications

The most common indication for overall cesarean delivery is a repeat procedure, whereas the most common indications for primary cesarean delivery include labor dystocia (34%), abnormal or indeterminate (previously, nonreassuring) FHR tracing (23%), fetal malpresentation (17%), multiple gestation (7%), and suspected fetal macrosomia (4%).[7] Other indications are listed in *Table 2*.

Contraindications

There are few contraindications for cesarean delivery. A guiding principle is, what is best for the woman is best for the fetus. If the woman

Table 1. Recommendations for the Safe Prevention of Primary Cesarean Delivery
(continued)

Recommendations	Grade of Recommendations
Induction of labor	
Before 41 weeks' gestation, induction of labor generally should be performed based on maternal and fetal medical indications. Inductions at 41 weeks' gestation and beyond should be performed to reduce the risk of cesarean delivery and the risk of perinatal morbidity and mortality	1A Strong recommendation, high-quality evidence
Cervical ripening methods should be used when labor is induced in women with an unfavorable cervix	1B Strong recommendation, moderate-quality evidence
If the maternal and fetal status allow, cesarean deliveries for failed induction of labor (up to 24 hours or longer) in the latent phase can be avoided by allowing longer durations of the latent phase and requiring that oxytocin be administered for at least 12 to 18 hours after membrane rupture before deeming the induction a failure	1B Strong recommendation, moderate-quality evidence
Fetal malpresentation	
Fetal presentation should be assessed and documented beginning at 36 weeks' gestation to allow for external cephalic version to be offered	1C Strong recommendation, low-quality evidence
Suspected fetal macrosomia	
Cesarean delivery to avoid potential birth trauma should be limited to estimated fetal weights of at least approximately 11 lb (5,000 g) in women without diabetes and at least approximately 9.9 lb (4,500 g) in women with diabetes. The prevalence of birth weight of approximately 11 lb (5,000 g) or more is rare, and patients should be counseled that estimates of fetal weight, particularly late in gestation, are imprecise	2C Weak recommendation, low-quality evidence
Excessive maternal weight gain	
Women should be counseled about the IOM maternal weight guidelines in an attempt to avoid excessive weight gain	1B Strong recommendation, moderate-quality evidence
Twin gestations	
Perinatal outcomes for twin gestations in which the first twin is in cephalic presentation are not improved by cesarean delivery. Thus, women with either cephalic/cephalic-presenting twins or cephalic/noncephalic presenting twins should be counseled to attempt vaginal delivery	1B Strong recommendation, moderate-quality evidence
Other	
Individuals, organizations, and governing bodies should work to ensure that research is conducted to provide a better knowledge base to guide decisions regarding cesarean delivery and to encourage policy changes that safely lower the rate of primary cesarean delivery	1C Strong recommendation, low-quality evidence

IOM = Institute of Medicine.

Reprinted from American College of Obstetricians and Gynecologists; Society for Maternal-Fetal Medicine. Obstetric care consensus no. 1: safe prevention of the primary cesarean delivery. Obstet Gynecol. *2014;123(3):693-711.*

is medically unstable and the fetus is nonviable, then the maternal condition should be stabilized regardless of fetal consideration. Alternatively, if the fetus is of a gestational age consistent with viability, the maternal condition should be stabilized first and then cesarean delivery considered for obstetric or fetal indications. The exception to this dictum is resuscitative hysterotomy.

Timing of Elective Cesarean Delivery

Elective cesarean delivery is typically not performed before 39 weeks' gestation secondary to the risk of neonatal complications due to fetal immaturity. Despite this risk, at one facility, over one-third of elective cesarean deliveries completed between 1999 and 2002 were performed before 39 weeks' gestation; delivery before 39 weeks' gestation was

Table 2. Common Indications for Cesarean Delivery

Fetal	Maternal-Fetal	Maternal
Category II or III FHR tracing	Failure to progress in labor:	Repeat cesarean delivery
Malpresentation:	Arrest of descent	Contracted pelvis:
Transverse lie	Arrest of dilation	Congenital
Breech	Placental abruption	Fracture
Brow	Placenta previa	Obstructive tumors
Face/mentum posterior	Conjoined twins	Abdominal cerclage
Cord prolapse	Perimortem	Reconstructive vaginal surgery
HIV with viral load >1,000/mL		Medical conditions (eg,cardiac, pulmonary, thrombocytopenia)
Active herpes virus or prodromal symptoms		
Congenital anomalies		
Vasa previa		

FHR = fetal heart rate.

associated with increased rates of respiratory issues, sepsis, hypoglycemia, and prolonged hospitalization or requirement for increased level of care.[9] Therefore, cesarean delivery should not be performed before 39 weeks' gestation unless there are medical indications for the woman or fetus.

For women with complicating factors (eg, placenta previa or prior classical uterine incision), the risk of maintaining the pregnancy to 39 weeks' gestation may outweigh the risks to the woman and fetus if delivery occurred before 39 weeks' gestation. Similarly, women with planned repeat cesarean deliveries may have obstetric complications with indications for delivery before 39 weeks' gestation such as gestational hypertension, preeclampsia, or intrauterine growth restriction with abnormal Doppler ultrasound study results.

Anatomy and Physiology

Many alterations in maternal cardiovascular physiology during pregnancy are relevant to cesarean delivery. More information is available in the *Maternal Resuscitation and Trauma* chapter. These physiologic changes increase maternal blood volume and flow in the pelvic organs, rendering the woman more susceptible to serious hemorrhage during cesarean delivery. The surgical anatomy is described in this chapter with each aspect of the procedure. An understanding of pelvic blood supply is essential for physicians performing a cesarean delivery.

Uterine Artery

The aorta bifurcates into bilateral common iliac arteries at the level of the fourth lumbar vertebra. The common iliac divides into external and internal iliac arteries. The internal iliac or hypogastric artery courses medio-inferiorly along the border of the psoas muscle and divides into anterior and posterior divisions. The anterior division has parietal and visceral branches of variable origin.

The uterine artery, a main visceral branch of the anterior division of the hypogastric artery, descends for a short distance, enters the base of the broad ligament, and turns medially to the lateral aspect of the uterus. The relationship between the uterine artery and ureter is surgically significant. Approximately 2 cm lateral to the cervix, the uterine artery crosses over the ureter. The ureter can be injured in the process of clamping and ligating the uterine vessels in the setting of postpartum hemorrhage (PPH) or during hysterectomy. A common way to remember this is that the uterine vessels form *a bridge over running water*.

The inferior branch of the uterine artery supplies the upper vagina and the lower cervix, and the marginal branch traverses the lateral aspect of the uterus before dividing into three terminal branches: ovarian, tubal, and fundal. Near the upper lateral portion of the uterus, the ovarian artery anastomoses with the ovarian branch of the uterine artery. Throughout its length, the marginal branch is a convoluted vessel with numerous branches penetrating the body of the uterus, including one large branch that extends to the upper portion of the cervix.

Ovarian Artery

The ovarian artery is a direct branch of the aorta and enters the broad ligament through the infun-

dibulopelvic ligament. At the ovarian hilum, the ovarian artery divides into ovarian branches and a main branch that traverses the broad ligament.

Uterine and Ovarian Veins

The lateral uterus is composed largely of venous sinuses. These sinuses coalesce into arcuate veins that unite to form the uterine vein. Several large uterine veins accompany the uterine artery and empty into the hypogastric vein, which empties into the common iliac vein. The ovarian vein collects blood from the upper part of the uterus through a large pampiniform plexus in the broad ligament. The right ovarian vein empties into the vena cava, and the left ovarian vein empties into the left renal vein.

Vaginal Blood Supply

The vagina receives blood from the inferior extension of the uterine artery along the lateral sulci of the vagina and from a vaginal branch of the hypogastric artery. These form an anastomotic arcade along the lateral aspect of the vagina at the 3 o'clock and 9 o'clock positions. Branches of these vessels also merge along the anterior and posterior vaginal walls.

Physical Findings and Diagnosis

History

History should be obtained that is pertinent to impending surgery, including medical, surgical, obstetric, gynecologic, family, tobacco and drug habits, transfusion, medications, allergies, and thrombotic and anesthetic complications. Information about the current obstetric indication is required (eg, length of labor and duration of ruptured membranes, day of operation, time of last food and drink intake).

Physical Examination

The physical examination for cesarean delivery should address major medical, obstetric, and anesthetic concerns. The operating team must be cognizant that regional anesthesia may be converted to general anesthesia at any time during the procedure. A vaginal examination should be performed in most women who are in labor just before surgical draping to ensure that sufficient progress in labor has not occurred that would permit vaginal delivery. Women with a history of herpes should undergo a careful examination for active lesions.

Ancillary Tests

Preoperative laboratory evaluation should include a complete blood count, blood type, and screen. A blood clot tube should be present in the blood bank for cross matching if transfusion is required. Prenatal HIV status should be known in all women so that measures can be taken to decrease the risk of vertical transmission of infection. A Kleihauer-Betke test to quantify the amount of fetal-maternal hemorrhage may be indicated in women who are Rh negative in situations such as major trauma.

Procedure

Preoperative and Nonsurgical Considerations

The woman should be prepared in the same manner as for any other major abdominal procedure including skin preparation, vaginal preparation, and surgical *time out* (*Table 3*) (see the *Safety in Maternity Care* chapter). Additional fluids are necessary because of peripheral vasodilation caused by regional anesthesia, increased insensible loss with labor, blood loss and intraoperative loss of 1,000 cc/hour because of exposed viscera.[10,11]

Fluid administration before epidural or spinal analgesia in women who are normotensive typically involves a 1,000-cc bolus of isotonic fluids. Isotonic fluids are effective first-line agents in the event of excessive bleeding, but blood product replacement is often necessary for blood loss greater than 1,000 mL. Use of prophylactic antibiotics has been shown to decrease the incidence of fever, endometritis, wound infection, urinary tract infection, and serious postoperative infection after cesarean delivery.[12] Ampicillin and first-generation cephalosporins have similar efficacy in reducing postoperative endometritis. There does not appear to be added benefit in using a broader spectrum agent or a multiple-dose regimen.[13]
All women undergoing cesarean delivery should receive intravenous (IV) antibiotics before skin incision, unless the woman is already receiving appropriate antibiotics (eg, for chorioamnionitis) or there is not adequate time because of an emergency cesarean delivery.[14,15] Pre-incision prophylaxis is advantageous for the woman and not harmful to the newborn, therefore the practice of administering antibiotics after cord clamping has been discontinued.[16] Consider clindamycin 900 mg IV in women with a penicillin allergy and

with a history of anaphylaxis, urticaria, or other life-threatening reaction. If a procedure exceeds 4 hours, then re-dosing should be considered. In addition, data suggests that in women with obesity (body mass index of 30 kg/m^2 or greater, or body weight of 176 lb [80 kg] or greater), the cefazolin dose should be increased from a 1- or 2-g IV dose to a 3-g IV dose.[17]

A multicenter, randomized trial including over 2,000 women showed that administering azithromycin 500 mg IV before skin incision, in addition to preoperative cefazolin, resulted in a 50% reduction in the composite outcome of endometritis (3.8% versus 6.1%), wound infection (2.4% versus 6.6%), and other infection compared with placebo without affecting the frequency of adverse neonatal outcomes.[18] The trial only included women who underwent cesarean delivery during labor or at least 4 hours after rupture of membranes. Azithromycin is not recommended for scheduled cesarean deliveries.[18]

Infective endocarditis prophylaxis is no longer recommended for vaginal or cesarean delivery in the absence of infection, regardless of the type of maternal cardiac lesion.[19] Abdominal hair removal often is not necessary. If hair is removed, it should be removed in the operating room and not the evening before the procedure. The hair should be clipped and not shaved to decrease the risk of infection.

Table 3. Preoperative Preparation/Orders

Vital signs and nonstress test on admission
Anesthesia consultation
Nothing per mouth (except nonparticulate citrate antacid)
IV: Lactated Ringer solution at 125 cc/hour; if regional anesthetic, then bolus of IV fluids per anesthesia
Nonparticulate citrate antacid 30 cc per mouth 1 hour preoperatively, or on-call to OR
Cefazolin 1 to 3 g IV to be administered 15 to 60 minutes before skin incision, dose by weight
Azithromycin 500 mg IV, if during labor or at least 4 hours after rupture of membranes
Place woman in left lateral decubitus position
Insert bladder catheter
Clip lower abdominal hair as needed, remove metal jewelry
Sequential compression devices on lower extremities
Laboratory tests: Complete blood count, blood type, and screen
Patient education: Cesarean delivery

IV = intravenous; OR = operating room.

Information from Tita AT, Szychowski JM, Boggess K, et al; C/SOAP Trial Consortium. Adjunctive azithromycin prophylaxis for cesarean delivery. N Engl J Med. *2016;375(13):1231-1241; Lim SK, Elegbe EO. The use of single dose of sodium citrate as a prophylaxis against acid aspiration syndrome in obstetric patients undergoing caesarean section.* Med J Malaysia. *1991;46(4):349-355; Swank ML, Wing DA, Nicolau DP, McNulty JA. Increased 3-gram cefazolin dosing for cesarean delivery prophylaxis in obese women.* Am J Obstet Gynecol. *2015;213(3):415.e1-8.*

Informed Consent

The surgeon should thoroughly discuss the risk and benefits of the procedure in medical and nonmedical terminology with the woman and a family member, if available. The counseling is best documented in narrative form, though a preprinted form can be used. Preoperative documentation should be signed and dated by the woman and surgeon. Documentation should include diagnosis, procedure, common and important risk factors, alternatives to the proposed procedure, and other potential procedures anticipated by the surgeon (eg, tubal ligation or oophorectomy for known adnexal mass). The risk factors can be simplified to bleeding, infection, internal organ damage, anesthesia risk, hysterectomy, injury to fetus, need for additional procedures, and risk of maternal mortality.

Cesarean Delivery Procedure

The primary surgeon and assistant typically focus their conversation before a cesarean delivery on variations of the technique and the increased risk of complications based on the clinical scenario (*Table 4*). A *pre-op* or *surgical* huddle may occur prior to transporting the woman to the operating room. A *time-out* should occur before incision to ensure the woman's name band matches the electronic medical record or paperwork, preoperative consent documentation is in place, and the care team understands the procedure. Time-outs should also address any specific obstetric, neonatal, or anesthesia risks and whether intrauterine device (IUD) placement or tubal ligation will occur during the surgery.

Abdominal Wall Incision

Options for the abdominal wall incision include the modified Pfannenstiel, Joel-Cohen, and midline vertical incisions, plus several variants of these incisions (*Figure 1*).[20] The midline vertical incision was traditionally considered the quickest abdominal wall incision, but most experienced surgeons

can perform the Joel-Cohen or the modified Pfannenstiel incision just as quickly.[20]

Pfannenstiel

The modified Pfannenstiel incision is made 3 cm (two-finger breadths) above the pubic symphysis. The incision is extended beyond the lateral borders of the rectus muscles in a curvilinear fashion to within 2 to 3 cm inferior and medial of the anterior superior iliac crests. The incision may be placed under the pannus in women with obesity, but this area is heavily colonized with bacteria and may be difficult to prepare surgically, keep dry, and inspect in the postoperative period.

The subcutaneous tissues are completely separated from the fascia and a transverse incision is made through the fascia. The fascial sheath is then grasped with Kocher clamps and completely separated from the underlying rectus muscles by blunt and sharp dissection to the umbilicus and caudad until the pubis is palpable. Blood vessels perforating through the muscles can be ligated with electrocautery or cut and clamped as required for hemostasis. The peritoneum is elevated and sharply or bluntly opened longitudinally in the midline.

Joel-Cohen (Misgav Ladach Modification)

The Joel-Cohen abdominal wall incision, modified by the Misgav Ladach Hospital in Jerusalem, emphasizes stretching tissue within existing planes, rather than sharp dissection (*Table 5*).[21]

Joel-Cohen-based cesarean delivery compared with Pfannenstiel cesarean delivery was associated with reduced blood loss, operating time, time to

Table 4. Cesarean Delivery Techniques

Prepare the woman

Informed consent

Nothing by mouth except nonparticulate antacid

Anesthesia

Bladder catheter

Clip hair, cleanse skin, left lateral decubitus position

Chlorhexidine-alcohol for skin antiseptic

Povidone-iodine for vaginal antiseptic

Cefazolin intravenously within 60 minutes before skin incision

Azithromycin intravenously within 60 minutes before skin incision if in labor or ruptured membranes more than 4 hours

Sequential compression devices on lower extremities

Abdominal wall incision

Joel-Cohen (Misgav Ladach modification)

Pfannenstiel

Midline vertical

Others: Maylard, Cherney

Fascial incision

Joel-Cohen: small midline fascial incision, stretch tissue

Pfannenstiel: long transverse incision, separate rectus muscle/sheath, stretch rectus muscles apart

Peritoneal incision

Parietal: longitudinal; transverse (Joel-Cohen)

Visceral: transverse vesicouterine; ±develop bladder flap

Uterine incision

Low transverse

Classical

Low vertical

Elevate the fetal presenting part

Elevate presenting part, maintain flexion if cephalic

Second assistant to dislodge presenting part vaginally, if deeply seated

Apply fundal pressure

Administer oxytocin after delivery of presenting part

Clamp and cut umbilical cord

Obtain cord blood for:

Type and direct Coombs, if clinically indicated

Cord pH from loop of cord obtained before cord blood sample, if clinically indicated

Delivery of placenta

Assisted spontaneous

Manual

Cleanse uterine cavity

Uterine closure

Place ring forceps at apices of uterine incision (optional)

Externalize uterus (optional)

Inspect for possible extensions

Two layers of absorbable suture

Inspect pelvic and abdominal contents

Remove foreign material from peritoneal cavity

Sponge and needle count

Peritoneal closure (optional)

Visceral (bladder flap)

Parietal

Fascial closure

Single nonlocking

Two lines of suture meeting in the midline, nonlocking (optional)

Subcutaneous (if tissue >2 cm)

Close dead space if >2 cm

Irrigate subcutaneous tissue

Skin closure: subcuticular, staples, widely spaced mattress sutures

Apply sterile dressing

Information from Mathai M, Hofmeyr GJ, Mathai NE. Abdominal surgical incisions for caesarean section. Cochrane Database Syst Rev*. 2013;5(5): CD004453.*

Figure 1. Abdominal Wall Incisions

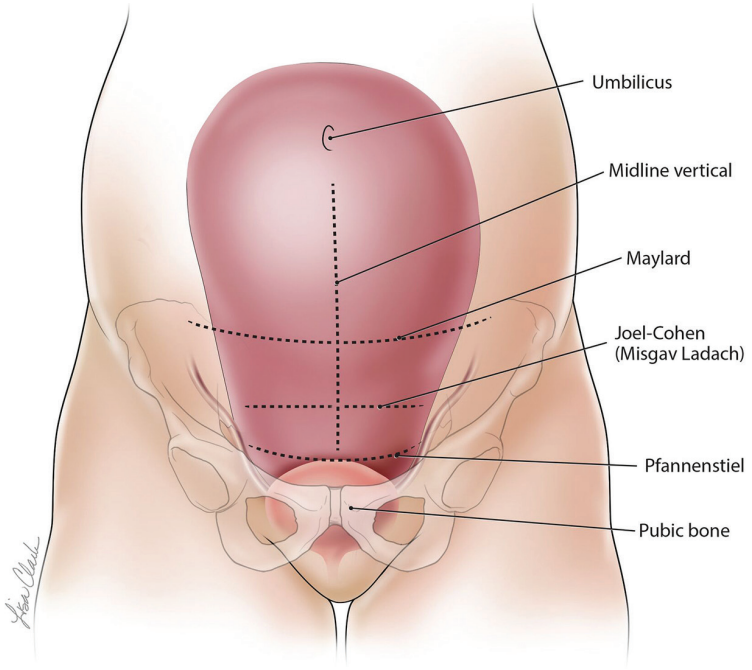


Table 5. Misgav Ladach Method of Cesarean Delivery

1. Perform abdominal incision using a modified Joel-Cohen approach
2. Use index fingers to open the parietal peritoneum transversely
3. Use a small lower uterine transverse incision and extend laterally using index fingers
4. Close the uterus with a single continuous nonlocked layer of absorbable monofilament
5. Do not close the visceral or parietal peritoneum
6. Close the fascia with a single nonlocking continuous closure of polyglactin
7. Skin is closed with several widely spaced skin stitches and the margins between sutures are approximated with Allis clamps for 5 minutes

Information from Holmgren G, Sjöholm L, Stark M. The Misgav Ladach method for cesarean section: method description. Acta Obstet Gynecol Scand. *1999; 78(7):615-621.*

oral intake, fever, duration of postoperative pain, analgesic injections, and time from skin incision to birth of the newborn.[20]

Available evidence suggests that the Joel-Cohen-based techniques (Joel-Cohen, Misgav Ladach, and modified Misgav Ladach) have advantages over Pfannenstiel and traditional cesarean delivery techniques in relation to short-term outcomes. There is no evidence in relation to long-term outcomes.[20]

This technique has advantages in remote or rural areas because it requires fewer instruments than other methods for opening the abdominal wall and can be performed quickly. The modified Joel-Cohen incision begins with a transverse incision, 15 to 17 cm long, made 3 cm below the anterior superior iliac crests. The skin is opened superficially, followed by sharp dissection of the subcutaneous fat to open the fascia in the midline only.

The fascial incision is extended sharply 2 to 3 cm under the intact subcutaneous tissue. After opening the fascia, the remaining subcutaneous tissue, fascia, and rectus muscles are dissected bluntly. The fascia is best opened with cephalad and caudad pressure followed by transverse pressure on the rectus muscles laterally. The incision is rapid and results in less blood loss than other techniques. There is a decreased need for transfusion and less risk of HIV transmission because the technique simply stretches tissues transversely. Decreased tissue damage also leads to less postoperative analgesia and early resumption of feeding and activity.

Midline Vertical

The midline-vertical skin incision extends from the pubic symphysis to within 2 cm of the umbilicus. The fascia is elevated and sharply dissected from the pubis to the umbilicus. This midline-vertical abdominal wall incision can be performed rapidly and provides excellent exposure of the pelvis and sidewalls. Pfannenstiel and Joel-Cohen-based skin incisions are most commonly used in the United States, but a vertical skin incision is commonly used in other countries (eg, Mexico). The presence of a vertical skin incision does not indicate that a vertical uterine incision has occurred. A surgeon performing a repeat cesarean delivery may elect to use a prior vertical skin incision or perform a Pfannestiel and should discuss this decision with the woman prior to surgery.

Other Incisions

The transverse Maylard rectus-cutting incision begins with a curvilinear skin incision that extends 18 to 19 cm between the anterior superior iliac crests. The Maylard incision offers maximal exposure for abnormal lie, multiple gestation, or macrosomia. In the transverse Cherney incision, the rectus muscles are detached from their insertion at the pubic symphysis. The transversalis fascia and peritoneum are incised transversely in

the Cherney, as opposed to the longitudinal Pfannenstiel technique.

Peritoneal Incision

Parietal Peritoneum

The parietal peritoneum should be entered at the highest position possible to avoid inadvertent bladder injury, especially in repeat procedures. The parietal peritoneum is sharply incised or entered with blunt digital dissection and then stretched open.

Urinary Bladder

The urinary bladder can be divided into two portions, the dome and base. The base of the bladder, which rests on the upper vagina and cervix, contains the trigone and is contiguous with the muscle of the vesical neck and urethra. The muscular dome of the bladder is relatively thin when distended. The bladder base is thicker and varies less with distention. The bladder is encountered twice before delivery. Initially, the surgeon visualizes the bladder when opening the peritoneum, and it is encountered again when dissecting the bladder flap off the lower uterine segment. The extent of the bladder can be confirmed by palpation of the catheter bulb.

Visceral Peritoneum

Omission of the bladder flap provides short-term advantages such as reduction of operating time and incision-delivery interval.[22] However, one study found no difference for bladder injury, total operating time, blood loss, or duration of hospitalization.[22] Long-term effects still need to be evaluated. In cases in which a cesarean hysterectomy is planned, a bladder flap should be created.

If deemed necessary, the vesicouterine peritoneum is elevated and opened transversely 1 cm above the bladder reflection onto the lower uterine segment. The bladder flap is bluntly and sharply developed transversely 10 to 12 cm, then inferiorly to the level of the bladder's apposition to the cervix. If a bladder flap is developed, the bladder blade is repositioned to lower and protect the bladder. With some repeat cesarean deliveries, scarring makes developing a bladder flap difficult or impossible.

Uterine Incision

Cesarean delivery is performed via one of several uterine incisions. The most common is a low transverse, or Kerr incision. A less common surgical approach is the classical or vertical uterine incision. Both can be performed through any abdominal incision.

Uterus

As the uterus enlarges, it reaches almost to the liver and displaces the intestines laterally and superiorly. The uterine musculature is arranged in three layers. The muscle cells in the middle layer are interlaced such that when they contract after delivery, they constrict the perforating blood vessels. When a woman who is pregnant is supine, her uterus falls back to rest on the vertebral column and the great vessels, especially the aorta and the inferior vena cava. With ascent from the pelvis as pregnancy progresses, the uterus undergoes dextrorotation, resulting in the left margin facing anteriorly.

Low Transverse Incision

The low transverse incision is made in the inactive or noncontractile lower uterine segment. Most cesarean deliveries use a low transverse uterine incision because of the ease of delivery and low rate of immediate and subsequent wound dehiscence. The low incidence of dehiscence occurs because the low transverse incision avoids the active uterine segment. It also requires less surgical repair, results in less blood loss, and is less likely to result in the formation of adhesions to the bowel or omentum.[23]

The lower uterine segment is delicately scored in the median aspect with a scalpel 1 to 2 cm from the upper margin of the bladder while taking care to avoid injury to the fetus. If the lower uterine segment is thin, fetal laceration injury, which occur in between 0.7% and 1.9% of cesarean deliveries,[24] can be avoided by elevating the lower uterine segment with Allis clamps. Another method involves cutting to within a few cells of the uterine cavity, then tapping the closed blades of a pair of scissors against the incision. Opening and closing the blunt tips of the scissors will not hurt the fetus nor enter the amniotic cavity but will penetrate the remaining layers of lower segment.

The uterine incision is extended bluntly using two fingers. Blunt expansion of the uterine incision by separating the fingers in a cephalad-caudad direction results in less unintended extension and blood loss than expansion in a transverse direction.[25-28] The incision should extend approximately 10 cm transversely and slightly cephalad in a curvilinear fashion. If the uterine wall is

thickened or there is need for an extension, then bandage scissors should be used. In this case, care should be taken to avoid injury to the fetus or umbilical cord. The incision should be large enough to avoid fetal injury and to avoid inadvertent extension into lateral vessels. If it is necessary to extend the uterine incision, then the first superior curvilinear incision should be to the right to avoid the lateral vessels because of the dextrorotation. Some surgeons place laparotomy sponges in the peritoneal cavity to minimize contamination with chorioamnionitis or thick meconium. All laparotomy sponges placed in the abdomen should have radio-opaque tails. The surgeon should know the location of every sponge in the abdomen, and not simply rely on the operating team's count.

Classical/Vertical Incision

The classical uterine incision is made vertically into the active myometrium. The classical incision is indicated in significant prematurity with a poorly developed narrow lower uterine segment, dense adhesions, or structural uterine abnormalities (eg, myoma in the lower uterine segment or Bandl's contractile uterine ring). The classical incision may be used in some cases of anterior placenta previa and malpresentation (eg, back-down transverse lie, preterm breech, interlocking twins).

Low Vertical Incision

The low vertical incision begins as inferiorly as possible to avoid the active uterine segment. The incision is typically made approximately 2 cm above the bladder and continued as far cephalad as necessary to allow facile delivery. This may be performed completely in the lower uterine segment and repair may be as strong as the low transverse incision.

There is less data on low vertical incisions, but the limited studies have found a rupture rate of 1% to 2%.[29] The major disadvantage of the low vertical incision is the possibility of extension cephalad into the uterine fundus or caudally into the bladder, cervix, or vagina. It is also difficult to determine if the low vertical incision is truly low, as the separation between lower and upper uterine segments is not easily identifiable clinically.

Anterior Placenta Management

If an anterior placenta is present, it should be dissected or separated from the uterine wall facilitating exposure of the fetus. There is a short-term risk of fetal hemorrhage unless delivery is rapid. If the placenta is lacerated, the operator should cut through the rest of the placenta, quickly deliver the infant, and clamp the cord. A vertical incision may be necessary. A preoperative ultrasound for placental location may be helpful. In addition, if the woman has undergone a previous cesarean delivery, the possibility of placenta accreta should be considered and the operating team should be prepared for the potential of hemorrhage and need for hysterectomy (see Placenta Accreta section).

Delivery of the Fetus

Cephalic Presentation

To deliver a fetus in a cephalic presentation, remove the retractors and manually elevate the presenting part. The assistant then applies transabdominal pressure to the uterine fundus. If the presenting part is deeply applied to the cervix or vagina, then gently insinuate the hand into the uterus with side-to-side motions to break the suction and act as a lever to elevate the presenting part. The operator should avoid using the uterine incision as a fulcrum for elevating the presenting part to avoid extension of the uterine incision. The assistant applies fundal pressure when the operator feels the presenting part is elevated enough that the force will push the presenting part up and out of the incision instead of deeper into the pelvis. Flexion is desirable in occiput posterior or anterior positions. Too much manipulation of a thin lower uterine segment may lead to a deep cervical laceration. If the presenting part is deeply seated in the pelvis, an additional assistant may need to reach under the operative drapes to manually dislodge the presenting part via the vagina.

If the presenting part is high, then a vacuum device or single forceps blade may be helpful. Deliver the torso by gently working the shoulders out one at a time with continued fundal pressure. Alternatively, the fetal feet may be delivered first. The newborn is transferred to an attendant after the umbilical cord is clamped and cut.

Breech Presentation

The presenting part should be confirmed preoperatively with ultrasound. A breech presentation may require a slightly larger abdominal wall and uterine incision for adequate exposure. A vertical uterine incision may be necessary if the lower

uterine segment is not well developed (eg, very preterm). The techniques for a breech cesarean delivery are similar to those used in a breech vaginal delivery. Additional information on breech deliveries is available in the *Malpresentations, Malpositions, and Multiple Gestations* chapter. The abdominal and uterine incisions can be extended if delivery of the fetal head is difficult. The uterine incision can be extended vertically into the active myometrium, perpendicular to the transverse uterine incision in an inverted T shape or extended perpendicularly to the uterine vessels in a J shape. These extensions should be noted in the operative report and the woman informed that repeat cesarean delivery is indicated in future pregnancies because a T incision is generally considered a contraindication for future LAC due to increased risk of uterine rupture.[30]

After Delivery

Cord blood is obtained and may be sent for infant blood type and Rh status, Coombs testing, HIV and/or rapid plasma reagin testing based on facility guidelines.[31] In addition, a 10 to 15 cm segment of umbilical cord may be saved for blood gas measurement. To obtain a sufficient amount of arterial pH specimen, the cord should be clamped close to the placenta. Delayed cord clamping for 1 minute may be used if the infant is vigorous and the bleeding from uterine incision is not excessive and is particularly beneficial for preterm infants.[31]

Delivery of the Placenta

An infusion of oxytocin 10 to 40 units in 1 L of isotonic crystalloid is administered immediately on delivery.[32] Assisted spontaneous delivery of the placenta involves fundal massage and gentle traction on the umbilical cord. Manual extraction of the placenta may be necessary on occasion but assisted spontaneous delivery of the placenta with gentle cord traction is preferred.

Assisted spontaneous delivery of the placenta with fundal massage and cord traction at cesarean delivery has advantages compared with manual removal. Assisted spontaneous delivery results in fewer instances of endometritis, less blood loss, less decrease in hematocrit levels postoperatively, and shorter duration of hospitalization. It also does not add significantly to operative time.[28]

The uterine cavity should be inspected and cleansed with a laparotomy sponge. Routine manual/instrumental cervical dilatation before closing the uterus in an elective cesarean delivery is unnecessary. A randomized controlled trial (RCT) showed that this practice does not improve blood loss and postoperative infectious morbidity.[33]

Repair of the Uterus

If a woman has requested and consented to IUD placement, the device may be inserted prior to repair of the uterus or deferred until the first layer of the uterine repair is approximately 50% completed. When an IUD is placed immediately postpartum, its strings are left untrimmed until after the uterus has returned to its prepregnancy size.[34] In one study, IUD use at 6 months was higher in women who received their IUD intraoperatively versus those who received it at least 6 weeks postpartum (40/48 [83%] versus 32/50 [64%]; RR 1.3; 95% CI = 1.02-1.66); however, the same study showed that intraoperative placement increased expulsion rates of the IUD (4/48 [8%] versus 1/50 [2%]; RR 4.2; 95% CI = 0.48-35.95).[35]

The uterus can be repaired within the peritoneal cavity or while externalized. A 2004 Cochrane review did not definitively conclude which method of uterine closure is preferred.[36] Externalization offers increased exposure of the uterus and adnexa, plus ease of fundal massage. No differences in complication rates were shown between extraabdominal and intraabdominal repair at cesarean delivery.[37] In addition, there was no difference in rates of intraoperative nausea/vomiting among those who underwent cesarean delivery with regional anesthesia.[37] A 2015 meta-analysis of randomized trials of extraabdominal (exteriorized) versus intraabdominal (in situ) repair showed no clinically significant differences in blood loss, intraoperative nausea, vomiting, or pain between the two approaches.[38] Uterine repair by exteriorization may reduce blood loss and the associated decrease in hemoglobin, but the difference may not be clinically relevant. In situ repair may be associated with an earlier return of bowel function.[38,39]

Significant bleeding vessels should be clamped with ring forceps and the fundus of the uterus covered with a moist sponge. The surgical assistant can apply fundal massage and assess uterine tone. The margins of the uterine incision should be identified. The uterine incision is initially closed

with a single layer of No. 0 or No. 1 absorbable running suture. The running suture should be locked if significant bleeding is occurring. Otherwise, a locked or unlocked suture is acceptable.[40-43]

The surgeon traditionally sutures toward herself/himself. To ensure that each apex is closed, a suture should be placed just beyond each apex while taking care to avoid lateral vessels. A second imbricating suture layer is indicated if a woman may become pregnant again but is not necessary if she is undergoing tubal ligation, unless performed for hemostasis. There is conflicting evidence regarding the advantage of the second suture layer.[27] Evidence based on a systematic review of RCTs does not support a specific type of uterine closure for optimal maternal outcomes and is insufficient to conclude on the risk of uterine rupture.[40] Single-layer closure and locked first layer are possibly coupled with thinner residual myometrium thickness.[40] For women who would consider LAC, a two-layer uterine closure is recommended to prevent the possibility that she will not be considered a candidate for LAC based on having had a single-layer closure, although data to support this is not conclusive.[27] Closure of a vertical incision requires a two- or three-layered closure, using a No. 0 or No. 1 absorbable suture.

Closure of Peritoneal, Fascial, Subcutaneous, and Skin Layers

After exploration of the pelvis and abdomen, all foreign material should be removed from the pelvis. It is essential to confirm that needle and sponge counts are correct. Closure of the peritoneum offers no advantage and increases operative time; febrile morbidity; rates of cystitis, narcotic use, antibiotic use; and length of stay in the hospital. There was improved short-term postoperative outcome if the peritoneum was not closed.[44]

The fascia is closed with a No. 0 or No. 1 nonlocked continuous long-lasting absorbable suture (eg, polyglactin 910). Sutures should be placed at 1-cm intervals approximately 1.5 cm from the margin of the cut fascia. Some surgeons close the fascia with two lines of suture that meet in the midline. Many surgeons perform a Smead-Jones mass closure with a No. 0 polydioxanone sulfate (PDS) suture for vertical incisions at high risk of dehiscence. At least six throws for each knot is recommended for PDS suture due to increased risk of dehiscence.[45,46]

Suture closure of subcutaneous fat during cesarean delivery results in a 34% decrease in risk of wound disruption in women with fat thickness greater than 2 cm.[45,46] The skin can be closed with staples, subcuticular 4-0 absorbable sutures, skin adhesive, or with widely spaced mattress sutures. A 2015 meta-analysis of RCTs showed that closure of the transverse skin incision with a subcutaneous suture rather than staples significantly decreases wound morbidity, specifically wound separation, without significant differences in pain, patient satisfaction, or cosmesis.[47] Compared with absorbable sutures, removal of staples on day three is associated with an increased incidence of skin separation and the need for reclosure, which requires an additional postoperative visit for women discharged before postoperative day three.[48] Compared with absorbable subcutaneous sutures, nonabsorbable staples are associated with an increased risk of skin separation and need for reclosure.[48] Suture placement took 7 minutes longer than staples. Skin closure using glue or a monofilament synthetic suture had similar results. Both methods were shown to be safe and successful for skin closure after a scheduled cesarean delivery.[49]

Instrument and Soft Material Count

Accurate counts of instrument and soft material should be executed as soon as closure starts. In the case of an emergent cesarean delivery indication, accurate counts before the procedure often could not be performed. In that case, an x-ray evaluation should be facilitated before surgical closing. In addition, radiofrequency sponges, barcoded sponges, or other identification systems should be used to prevent retained surgical sponges.[50]

Tips for Surgical Assistance at Cesarean Delivery

The surgical assistant has a key role in cesarean delivery (*Table 6*). The key elements are maintaining excellent exposure, maintaining the flow of the procedure, and being prepared for the unexpected (see the Situational Awareness section of the *Safety in Maternity Care* chapter).

Technical Pitfalls

Closing the Uterine Incision

A common error is placement of sutures beyond the uterine incision. This may result in increased bleeding from the lateral uterine vessels and

increases ureteral injury. An inexperienced operator, or one operating with inadequate exposure, may inadvertently suture incorrect tissue. The edge of the uterus, uterine incision, and any extensions should be carefully identified before closure.

Malpresentation Deliveries

Some experienced providers consider converting a breech or transverse presentation to cephalic presentation after opening the abdomen but before the uterine incision. Intraoperative version before uterine incision may prevent a traumatic delivery, a classical uterine incision, an inverted T incision, or an extension. Adequate abdominal wall and uterine exposure is critical for atraumatic delivery of a malpresentation. A skilled assistant must be available to avoid hyperextension and assist with fetal head flexion when performing a cesarean delivery of a fetus in breech presentation.

Postoperative Care

Although it is customary for fluids and/or food to be withheld for a time after abdominal operations, there is no evidence from randomized trials reviewed to justify a policy of withholding oral fluids after uncomplicated cesarean delivery.[51] Early oral intake within 6 to 8 hours after cesarean delivery improves the return of gastrointestinal function and does not increase the occurrence of gastrointestinal complications.[52]

Choice of Procedure

Randomized controlled studies have shown that many aspects of the traditional cesarean delivery practiced in the United States are unnecessary. The modified Joel-Cohen cesarean delivery avoids these steps and is associated with less operative time, fewer complications, and shorter length of stay.[20]

Uterine closure with single-layer continuous nonlocking suture has short-term benefit.[40] Single- and double-layer closure of the uterine incision after cesarean delivery are associated with a similar incidence of cesarean scar defects as well as uterine dehiscence and rupture in subsequent pregnancies.[43,53] Locked closure has been associated with higher occurrences of surrogate markers of scar weakness (ie, thinner myometrial thickness, bell-shaped uterine wall defects) and dehiscence/rupture.[40-42] Inclusion of the decidua/endometrium (full thickness suturing technique) appears to be another factor that increases scar strength.[54]

Although no clear benefits of one method over another have been shown, the choice may be influenced by the clinical setting. For example, in a resource-constrained environment where large numbers of cesarean deliveries are performed, a cost-effective choice may be spinal analgesia and Joel-Cohen-based surgical methods, which require only two lengths of suture material for the operation and double-layer closure of the uterus.[20]

Table 6. Tips for Surgical Assistance at Cesarean Delivery

Exposure

Lateral aspects of the following on opening and closing: Rectus fascia, vesicouterine peritoneum, and uterus

Rectus fascia (with Pfannenstiel incision)

Elevate fascia with Kocher clamps

Blunt dissection of rectus muscle from fascia

Provide counter traction on muscle while surgeon dissects fascia from muscle

Re-examine under fascia for bleeding or defects before closure

Uterine incision

Suction blood and fluid from incision as surgeon scores uterus

Delivery

Apply fundal pressure when requested

Assist with clamping and cutting umbilical cord

Obtain cord blood samples

If uterus is externalized after delivery

Hold tension on fundus with a moist lap while keeping the uterine incision dry for visualization of repair

Retract anterior abdominal wall for better visualization

First assistant should create exposure with bladder blade

Uterine closure

Follow holding suture

If surgeon is locking sutures, then loop suture over needle each pass

Knot tying

Three loops or throws for chromic gut suture

Four loops or throws for polyglactin 910, with first being a double throw or surgeon's knot

Summary of Cesarean Delivery Techniques

1. No preoperative hair removal. No clipping or depilatory creams on the day of surgery or the preceding day (no shaving).
2. Chlorhexidine-alcohol solution should be used for skin and povidone-iodine for vaginal antiseptic before draping.[10,11]
3. Antibiotic prophylaxis with ampicillin or a first-generation cephalosporin. Azithromycin if in labor or ruptured membranes greater than 4 hours.[13,18]
4. Double gloving is advised in areas with high rates of bloodborne infections to achieve fewer perforations in inner glove and prevent needlestick injuries.[20]
5. Transverse lower abdominal wall opening and uterine opening using Joel-Cohen-based methods.[21]
6. Bladder peritoneum may be reflected downward or not.[55]
7. Placental removal with fundal massage and cord traction.[28]
8. Intraabdominal or extraabdominal repair of the uterus. [36,38,39]
9. Uterine closure with interrupted, single-layer or double-layer continuous full-thickness closure.[40,42,43,54] In women who may become pregnant again, double-layer closure should be strongly considered.[27,40,41,43,53]
10. Nonclosure of peritoneal layers.[44]
11. Closure of the subcutaneous tissues if greater than 2-cm thickness. [27,45,46]
12. No routine drainage of the subcutaneous tissues.[56]
13. Skin closure with subcuticular sutures or staples.[47]
14. No withholding of oral fluids after surgery.[51]

Poor Documentation in Operative Reports

Accurate documentation of the operative procedure can prevent confusion and complications in the future. In particular, the surgeon should take care in describing the uterine incision. For example, repeat low transverse cesarean delivery might be ambiguous. It could mean:

- The prior incision was a classical or low vertical and the current procedure was a repeat cesarean delivery performed via a low transverse incision.
- The previous operation was a low transverse incision and the same procedure was used in the current procedure.

In the first example, the woman should never undergo LAC, whereas the woman in the second example is a LAC candidate. A better description of the procedure would be, *low transverse uterine incision, repeat cesarean delivery*. When describing indications for the cesarean delivery, the type of previous scar should be listed. It is also important to properly document the extent of active uterine segment involved in a low vertical incision, the extent of an inverted T incision, and the nature of any uterine lacerations. The dictation should contain a brief description of the ovaries, tubes, and surrounding structures. If the cesarean delivery technique is such that the woman should not undergo LAC in the future, this should be clearly documented in the operative report and the woman should be informed.

Intraoperative Complications

Intraoperative injuries are uncommon, but they can still occur despite careful attention to technique. The operative team is responsible for identifying and repairing injuries or seeking appropriate assistance.

Hemorrhage

Uterine atony is the most common cause of hemorrhage during cesarean delivery. The first management steps are uterine massage and pharmacotherapy, then surgical management. Pharmacotherapy should proceed in a stepwise fashion from oxytocin 10 to 40 U/L IV, to methylergonovine 0.2 mg intramuscularly (unless the woman is hypertensive), to 15-methyl-prostaglandin F2 0.25 mg intramuscularly or intramyometrially. 15-methyl-prostaglandin F2 can be repeated every 15 minutes to a maximum dose of 2 mg. More information on hemorrhage is available in the *Postpartum Hemorrhage* chapter. Misoprostol can be used as an alternative to 15-methyl-prostaglandin F2. In a woman who is awake and alert, 400 mcg may be administered subingually.[57,58] Oral misoprostol peak levels are lower than with sublingual administration and decline rapidly over 2 hours because of hepatic metabolism.[57] It takes longer to achieve peak concentration via rectal administration (800 mcg) compared with oral or sublingual administration but has a longer duration of action than oral/sublingual routes (4 hours versus 2 to 3 hours).[57]

Based on the dosing regimen used in the World

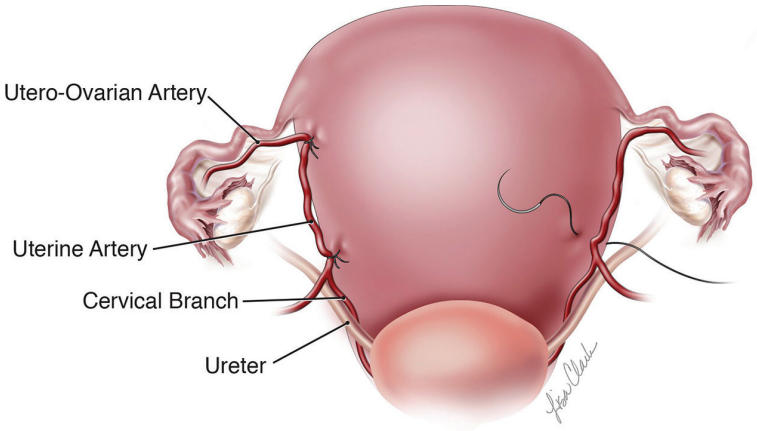
Maternal Antifibrinolytic Trial (WOMAN) trial,[59] the World Health Organization (WHO) recommends administering tranexamic acid (TXA) at a fixed dose of 1 g (100 mg/mL) IV at 1 mL/minute (ie, administered over 10 minutes), with a second dose of 1 g IV if bleeding continues after 30 minutes.[60] The WOMAN trial defined clinically diagnosed PPH as clinically estimated blood loss of more than 500 mL after a vaginal birth or 1,000 mL after cesarean delivery, or any blood loss sufficient to compromise hemodynamic stability.[59] Based on evidence from the trial, the reference point for the start of the 3-hour window for TXA administration is time of birth. As most deaths due to PPH occur within the first 2 to 3 hours after birth, it is critical that TXA is administered as soon as possible to achieve clinical benefits.[60] ACOG guidelines state that TXA should be considered in the setting of obstetric hemorrhage when initial medical therapy fails.[61] ACOG concurs that earlier use is likely to be superior to delayed treatment, given that in the stratified analysis it appeared that the benefit was primarily in women treated sooner than 3 hours from the time of delivery.[61]

The surgical management of hemorrhage should proceed in a stepwise fashion depending on the woman's hemodynamic status. The first step is unilateral or bilateral O'Leary sutures of the uterine arteries. These No. 0 or No. 1 absorbable sutures are placed in the lateral aspect of the uterus just cephalad to the ureter. A second step to decrease uterine bleeding if the O'Leary sutures are not sufficient is bilateral ligation of the uterine vessels just medial to the ovaries (*Figure 2*).

Next, uterine compression sutures are an effective method for reducing PPH and avoiding hysterectomy. Limited follow-up of women who received a uterine compression suture suggests that there are no adverse effects on future pregnancies. The B-Lynch suture envelops and compresses the uterus, similar to the result achieved with manual uterine compression (*Figure 3*).[62,63] In case reports and small series, it has been highly successful in controlling uterine bleeding from atony when other methods have failed.[64]

Bilateral ligation of the internal iliac arteries (hypogastric arteries) was often used in the past to control uterine hemorrhage by reducing pulse pressure of blood flowing to the uterus. The technique is difficult, especially with a large uterus, a small transverse incision, a pelvis full of blood, and a surgeon who rarely operates in the pelvic retroperitoneal space. A case series of 19 women showed that the majority (58%) still went on to undergo hysterectomy despite bilateral internal iliac ligation.[65] For these reasons, uterine artery ligation and use of compressive uterine sutures has largely replaced this procedure.[66]

Figure 2. Ligation of the Uterine Artery



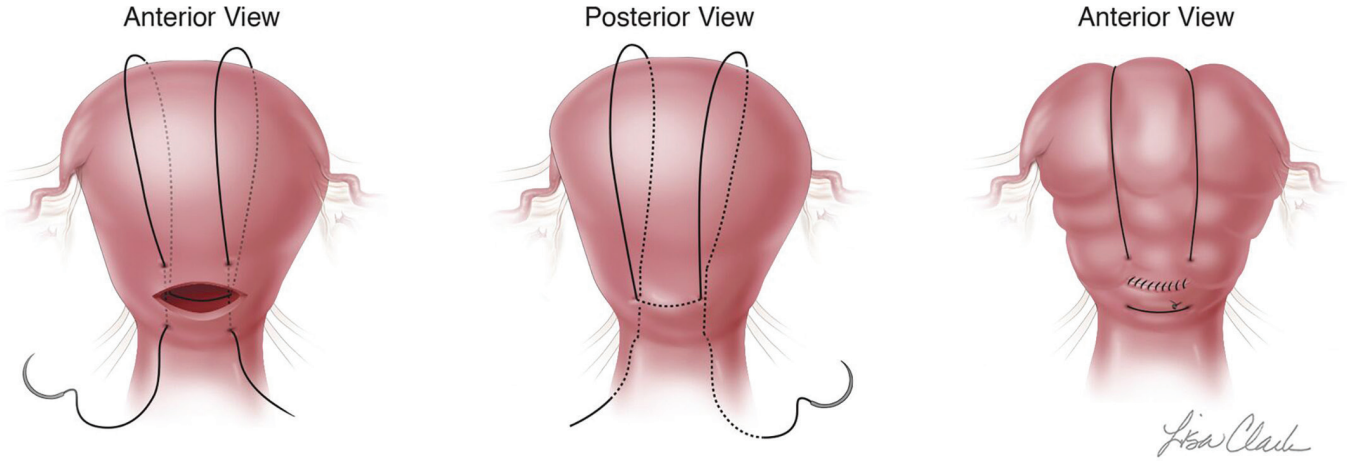
If the hemorrhage continues after atony is resolved and the woman is hemodynamically stable, then placement of one or more No. 30 French Foley catheters with a full 30 cc balloon through the cervix into the uterine cavity may tamponade the bleeding. The Bakri tamponade balloon and Ebb double balloon tamponade system were specifically designed for uterine tamponade to control postpartum bleeding (*Figure 4*).[67] The Bakri is a silicone balloon with a capacity of 500 mL of saline and strength to withstand a maximum internal and external pressure of 300 mm Hg. The balloon is filled until bleeding is controlled or 300 to 500 mL.[68]

These temporizing measures may allow time for correction of reversible conditions such as coagulopathy or thrombocytopenia. An IV fluid bag can be attached to the catheter as it exits the vagina to provide traction.

Another modality to stop uterine bleeding is selective arterial embolization.

If these efforts fail, a hysterectomy may be necessary. In the setting of severe PPH, obstetric surgeons must balance the maternal risks of attempting to avoid hysterectomy (including massive transfusion and even mortality) compared with the loss of desired fertility.

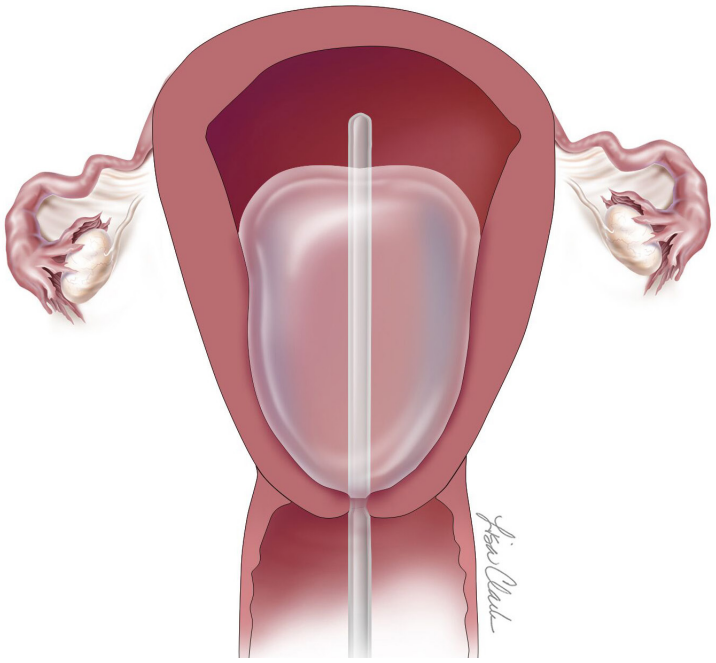
Figure 3. Anterior Uterine Wall With B-Lynch Suture in Place



Cesarean Hysterectomy

Indications for cesarean hysterectomy are uterine hemorrhage unresponsive to treatment, uterine rupture that would result in an unstable repair, placenta accreta, laceration of major pelvic vessels, and advanced cervical dysplasia or carcinoma. Complications of cesarean hysterectomy are more common during emergent procedures and include increased blood loss and anesthesia time, plus infection, blood transfusion, and the psychological effects of unanticipated sterility. Each facility should have a predetermined plan for urgent consultation with surgeons with advanced skills (eg, gynecologic oncologists, urogynecology subspecialists) or transfer to a higher level of care if necessary.

Figure 4. Uterine Tamponade Balloon



Extensions of the Uterine Incision

Extension of the uterine incision is more common with malpresentations, macrosomia, or attenuated lower uterine segment such as one that occurs in a woman with a preterm delivery. If the lower uterine segment is thin, then laceration can sometimes be avoided if the uterine incision is made slightly higher than normal. Other common extensions are into the broad ligament and vagina. To achieve a satisfactory repair, the full extent of the laceration must be exposed and visualized. The first suture should be placed just beyond the apex of laceration. The remaining sutures can be placed in a locked or interrupted fashion. If ureteral injury is suspected, cystoscopy should be performed. Lateral extensions into the broad ligament or inferior extensions into the cervix and vagina can be technically difficult to repair but are not a contraindication to a future labor and vaginal birth. Vertical extensions into the uterine contractile muscle (eg, inverted T or J incisions) are similar to prior classical cesarean deliveries in being a strict contraindication to future planned labor.

Urinary Tract Injury

Bladder injury is more common with repeat cesarean delivery, uterine rupture, and cesarean hyster-

ectomy. The ureter is most often injured during efforts to control bleeding from lateral uterine lacerations.

Lower urinary tract injury is shown to occur in 0.3% of cesarean deliveries compared with a rate of 3% in cesarean hysterectomies.[69,70] In a study of 107,429 deliveries, 54% were full-thickness bladder, 3% ureteral, and 43% were partial-thickness bladder injuries.[69] The risk of cystotomy is higher for cesarean deliveries performed in the second stage than in the first stage and in repeat cesarean deliveries.[71,72]

Before initiating a cesarean delivery, the surgeon should inspect the Foley catheter bag for the presence of blood. If none exists before initiation but develops during delivery, bladder injury should be suspected. If a bladder injury is suspected, instillation of methylene blue, indigo carmine, or sterile milk through the Foley catheter can assist in identification of injury if they are found to leak into the surgical field.

The dome of the bladder can be repaired with two layers of 2-0 absorbable suture. If the base or trigone of the bladder is involved, then consultation is suggested. The ureters should be cannulated to facilitate their identification during the repair. A urethral catheter should remain in place for 10 to 14 days after cystotomy.[73]

Ureteral injury may go unrecognized, but if suspected, it is necessary to dissect the length of the ureter to ensure that ureteral peristalsis is present. Cystoscopy should be performed to demonstrate the presence of bilateral ureteral flow into the bladder. Ureteral repair will require consultation from urology, urogynecology, or gynecological oncology surgeons. If the ureter is transected, a No. 8 French ureteral catheter should be threaded directly into the ureteral orifice by cystoscopy. Another approach is to cannulate the ureter through a cystotomy in the bladder dome. The cystotomy can be closed with two layers of 2-0 absorbable suture.

Gastrointestinal Injury

Gastrointestinal injuries occur in 0.04% to 0.08% of cesarean deliveries and are more common in women with adhesions from prior surgical procedures.[74,75] The risk of bowel injury can be minimized by limiting sharp dissection to transparent peritoneum, lysis of adhesions, and sharp dissection with the scissors pointed away from the bowel. Full-thickness defects of less than 1 cm are repaired in a double-layered transverse closure of a longitudinal laceration to avoid bowel lumen narrowing. The mucosa is repaired with a 3-0 absorbable suture in an interrupted fashion. The muscular and serosal layers are closed with a 3-0 silk suture in an interrupted fashion. Larger or complex lacerations may require consultation and assistance from a general or colorectal surgeon.

If fecal contamination of the operative field occurs, then copious irrigation and broad-spectrum antibiotics with gram-negative aerobic and anaerobic coverage are needed. Appropriate antibiotics include cefoxitin 1 to 2 g IV every 6 hours or cefotetan 1 to 2 g IV every 12 hours with or without gentamicin sulfate 1.5 mg/kg every 8 hours.[76] If the colon is involved, consider adding metronidazole 0.5 to 1 g IV or ampicillin/sulbactam 3 g IV to the cephalosporin.[76] Prophylactic wound drainage is rarely needed outside the settings of morbid obesity or a wet wound. Significant contamination may require secondary closure, especially in women with obesity.

Anesthetic Complications

Despite the advances in anesthesia and increased use of regional anesthesia, the number of mortalities caused by anesthesia has not decreased.[77] These mortalities are frequently attributed to the inability to intubate or ventilate the woman and are more common in women with obesity. Other complications are aspiration, inadequate ventilation, respiratory failure, cardiac arrest, local anesthetic toxicity, high spinal/epidural-related hypotension, overdosage, and spinal headache. Because of these risks, the American Society of Anesthesiologists Task Force on Obstetric Anesthesia recommends that neuraxial anesthesia (spinal or epidural) is preferred for most cesarean deliveries.[78] Of interest, a Cochrane review showed no evidence to support that regional anesthesia is superior to general anesthesia in terms of major maternal or neonatal outcomes.[79]

Although typically quite safe, anesthetic techniques are associated with various adverse effects such as systemic local anesthetic toxicity or spinal headache are caused by technical factors and/or dosing, such as inadvertent IV injection or unrecognized dural puncture. Spinal hematoma is a rare complication and is more likely in women receiving anticoagulants. Women receiving a therapeutic dosage of low-molecular-weight heparin (LMWH)

should discontinue anticoagulation at least 12 to 24 hours before labor induction or scheduled cesarean delivery, and women taking a once-daily prophylactic dose of LMWH should receive 50% of their dose on the morning of the day before delivery (see the *Venous Thromboembolism* chapter).[80-82] Meticulous attention to proper technique reduces the risk of these complications. Should they occur, prompt treatment is indicated.

Other common adverse effects are caused by known pharmacologic effects of the analgesic drugs, such as hypotension, pruritus, nausea and vomiting, and respiratory depression. Hypotension may be treated by administering fluids and/or a vasopressor. Opioid-induced adverse effects are optimally managed by administering small doses of an opioid antagonist. Itching from opioid spinal anesthesia responds better to naloxone than to antihistamines.[83]

Table 7. Postoperative Orders

1. Vital signs and fundal status every hour for 4 hours, every 4 hours for 24 hours, then every 8 hours
2. Uterine massage per schedule above; report extra lochia
3. Intake and output monitoring every 4 hours for 24 hours
4. Activity ad lib, encourage ambulation 3 times/day. Assistance at first
5. Cough, deep breathing, and incentive spirometry every hour when awake
6. Foley catheter to closed drainage; discontinue catheter first postoperative morning, or when ambulating well
7. Diet as tolerated after nausea resolved
8. D5 lactated Ringer solution with oxytocin 20 units/L at 125 cc/hour times two bags, then D5 lactated Ringer solution at 125 cc/hour. Convert to heparin lock when tolerating oral well
9. Morphine sulfate 2 to 8 mg IV every 2 hours as needed for pain
10. NSAIDs may be used for pain management, but are discouraged beyond 24 hours postpartum in setting of hypertension
11. Droperidol 1.25 to 5 mg every 4 hours IV as needed for nausea (or promethazine 25 to 50 mg every 4 hours intramuscularly as needed or ondansetron 4 to 8 mg orally every 12 hours)
12. Oxycodone 5 mg once or twice every 3 to 4 hours as needed after tolerating oral intake
13. Laboratory testing: first postoperative day — hemoglobin/hematocrit
14. Administer Rh_o(D) immune globulin if indicated by infant cord blood Rh status
15. Administer rubella, hepatitis, varicella, and Tdap vaccines at discharge if indicated

IV = intravenous; NSAID = nonsteroidal anti-inflammatory drug; Tdap = tetanus, diphtheria, and pertussis

Information from Sutton CD, Carvalho B. Optimal pain management after cesarean delivery. Anesthesiol Clin. 2017;35(1):107-124.

A variety of other adverse effects have been attributed to regional analgesic techniques, such as long-term backache, effects on the progress and outcome of labor, and effects on breastfeeding success. Although an association between regional analgesia and these adverse effects may exist, a cause-and-effect relationship has not been established. The risk of potential adverse effects must be weighed against the unparalleled pain relief these techniques provide.

Studies have shown that women who receive regional anesthesia for cesarean delivery have decreased blood loss and less of a postoperative decrease in hematocrit levels compared with those who receive general anesthesia.[79]

Postoperative Care

The care required after cesarean delivery is similar to that of any major abdominal surgery (*Table 7*).

Wound dressings should be removed in 24 hours and the wound monitored daily. Staples, when used, can be removed in 4 days for transverse skin incisions and 7 to 10 days for vertical skin incisions; with both, extra support can be obtained via Steri-Strips and tincture of benzoin or other topical skin adhesive when staples are removed. Breastfeeding should be encouraged in all women. The postoperative hemoglobin level will determine need for iron replacement. Discharge can typically be accomplished in 2 to 4 days with a gradual return to full activity based on the woman's comfort level. Early ambulation is encouraged. Fertility planning should be discussed before discharge and/or at clinic visits.

Postoperative Patient Instructions

Postcesarean delivery patient instructions are similar to those for any major gynecologic or abdominal surgery:

- Call the office for any issues, including increased abdominal pain, spreading redness, wound dehiscence, fever, concerning vaginal bleeding, or depression
- The abdominal wound should be kept dry and is best treated with minimal dressing. The area can be cleansed with warm water and mild soap. The woman should notify her maternity care provider if she notices redness or increased warmth, drainage, fluid under the skin, or a temperature greater than 100.4° F (38° C)
- Any unanswered questions a woman has after a cesarean delivery should be discussed with her

physician or case manager. When she is feeling well, an unhurried conversation with her physician will help with resolving her questions

• Women should receive information regarding whether they would currently be eligible for LAC in a future pregnancy and the potential increased risk of uterine rupture when a trial of labor occurs within 18 months of a prior cesarean delivery.[84] Women with a vertical uterine incision or extension should be informed that current recommendations support repeat cesarean delivery at 36 to 37 weeks' gestation in women with a history of classical cesarean delivery.[85] It is reasonable to provide the woman with a copy of the operative report for presentation to her maternity care provider during a subsequent pregnancy

• The current epidemic of opioid use disorder and overdose deaths requires physicians providing postoperative care to balance adequately treating pain with the understanding that 1 in 300 women taking opioids after a cesarean delivery become persistent users.[86] Most women can be discharged with a reasonable amount of opioids to manage pain that is not controlled with acetaminophen and nonsteroidal antiinflammatory drugs.[87] Codeine should be avoided in women who are breastfeeding because of the risk of potential neonatal toxicity in women who are *ultra-rapid metabolizers*.[88] If codeine is used, the woman must be warned about the possibility of infant sedation. However, codeine use within the first 48 hours after discharge is less concerning due to smaller quantities being transmitted to the newborn prior to lactogenesis.

Recommendations for activity after obstetric and gynecologic procedures remain based on tradition and anecdote. The available data do not support many of the recommendations currently provided.[89] Restrictions on lifting and climbing stairs should likely be abandoned. Guidance on driving should focus on concerns regarding cognitive function and analgesic drug use rather than concerns of wound separation/dehiscence. Given the effect of these recommendations on daily life events, the use of consistent, evidence-based advice on when and how women can safely resume exercise, driving, working, and sexual intercourse is critical (*Table 8*).[89]

Early Postoperative Complications

The most common early complications after cesarean delivery are infection-related. Maternal wound infection occurs in 68 to 97 per 1,000 deliveries, maternal endometritis occurs in 39 to 160 per 1,000 deliveries, and serious maternal infectious complications occur in 25 per 1,000 deliveries without the use of prophylactic antibiotics.[12] A single dose of a first-generation cephalosporin or ampicillin is as effective as other regimens, including multiple doses or lavage techniques reducing the risk of maternal wound infection by 60%, maternal endometritis by 62%, and maternal serious infectious complications by 69%.[12,13] Atelectasis is a common source of fever and can lead to pneumonitis. Septic shock, pelvic abscess, and septic thrombophlebitis occur in less than 2% of cases. Consider using the 5 Ws mnemonic (**W**aves [electrocardiogram waves], **W**ind [pneumonia], **W**ater [urinary tract], **W**ound [superficial surgical site infection and deep superficial surgical site infection], and **W**alking [venous thromboembolism]) plus a sixth W for the postcesarean delivery setting (**W**omb [endomyometritis]) to assess for possible complications (see the *Maternal Sepsis* chapter).[12,13]

Endomyometritis

Endomyometritis is a clinical diagnosis that presents with uterine or parametrial tenderness, fever (two postoperative temperatures greater than or equal to 100.4° F [38° C] beyond 24 hours), and leukocytosis. The leukocyte count is normally elevated in labor and the early puerperium, averaging 14,000 to 16,000/mm^3 and may not help in distinguishing an infectious etiology. Cultures of the lochia are often misleading. Blood cultures are frequently negative. Some women will develop septic thrombophlebitis, parametrial phlegmon, pelvic abscess, and peritonitis. Treatment for endomyometritis should include antibiotics with coverage for penicillin-resistant anaerobic bacteria, such as IV clindamycin and gentamycin. Ampicillin/sulbactam 3 g IV every 6 hours is another acceptable regimen. After symptoms resolve, additional oral antibiotics are not warranted.[90]

Wound Separation/Infection

Wound separation or opening is a common surgical complication after cesarean delivery, occurring in approximately 5% of cases. Of those wounds that open, approximately two-thirds are infected.[91]

Wound infection manifests as erythema and tenderness, and purulence and fever may develop.

Wound infection is a clinical diagnosis with laboratory data serving as an adjunct. The leukocytosis is variable and wound cultures are often misleading. Ultrasound of the abdominal wall may be helpful to localize an abscess. Treatment includes broad-spectrum antibiotics and vigorous wound care. The wound may need to be probed, opened, irrigated, and packed, and necrotic tissue debrided if the wound infection does not respond quickly to antibiotics. The woman and caregiver should be

Table 8. Evidence Supporting Post-Obstetric Surgery Advice

Advice	Evidence	Recommendations	Future Research
Lifting	Lifting increases intraabdominal pressure much less than the Valsalva maneuver, forceful coughing, or rising from supine to erect position	1. Patients should continue lifting patterns as before surgery 2. Patients need an adequate postoperative analgesic regimen 3. Preprocedure and postprocedure recommendations should be consistent	1. Prospective cohort study of patients encouraged to resume regular exercise program 2. Trial in which women are randomly assigned to lift weights lighter than before surgery or lift the same weights as before surgery
Climbing stairs	Climbing stairs increases intraabdominal pressure much less than Valsalva, forceful coughing, or rising from supine to erect position	1. Patients should continue climbing stairs as before surgery 2. Patients need an adequate postoperative analgesic regimen 3. Preprocedure and postprocedure recommendations should be consistent	Prospective cohort study of patients encouraged to resume regular exercise program, including climbing stairs
Driving	No retrospective or prospective evidence	1. Patients need an appropriate postoperative analgesic regimen that does not cause a clouded sensorium when driving 2. Patients may resume driving when comfortable with hand and foot movements required for driving 3. Preprocedure and postprocedure recommendations should be consistent	Prospective cohort study of women encouraged to resume normal activities, including driving
Exercise	Limited retrospective and prospective evidence. Forceful coughing increases intraabdominal pressure as much as jumping jacks.	1. Patients need an appropriate postoperative analgesic regimen 2. Patients may resume preprocedure exercise level 3. Exercise program may need to be tailored for postpartum women 4. Preprocedure and postprocedure recommendations should be consistent	Prospective interventional studies to encourage women to resume exercise programs, as well as build strength and cardiovascular health
Vaginal intercourse	No consistent retrospective evidence; no prospective evidence	1. Women and their partners should make the decision to resume intercourse mutually 2. Women should use vaginal lubricants and sexual positions permitting the woman to control the depth of vaginal penetration 3. Women should use appropriate contraception after childbirth 4. Preprocedure and postprocedure recommendations should be consistent	Prospective interventional studies aimed to help women resume sexual intimacy after gynecologic surgery; such studies should capture data on incidence of vaginal vault dehiscence and its associated factors
Returning to work	No consistent prospective or retrospective evidence	1. Women should be encouraged to return to work relatively soon postprocedure 2. Consider graded return to work 3. Preprocedure and postprocedure recommendations should be consistent	Prospective studies evaluating the optimal strategies to permit women to return to effective work

Reprinted from Minig L, Trimble EL, Sarsotti C, et al. Building the evidence base for postoperative and postpartum advice. Obstet Gynecol. *2009; 114(4):892-900.*

instructed about ongoing home care. The decision about delayed secondary closure versus healing by secondary intention will be influenced by the size of the wound and the logistics of follow-up care.

Fascial dehiscence occurs in approximately 6% of open wounds.[91] The fascia in all open cesarean delivery wounds should be probed for integrity. Fascial dehiscence may present with copious discharge followed by protrusion of bowel through the surgical wound. If this occurs, the bowel should be covered with a moist sterile gauze pad and consultation obtained immediately. The wound should be explored, cleansed, debrided, and closed with retention sutures or a mass closure (eg, Smead-Jones closure), using long-term absorbable suture.

Urinary Tract Infection

Urinary tract infections are often associated with use of an indwelling urethral catheter. Treatment should be initiated with broad-spectrum antibiotics, and subsequent antibiotic therapy based on urine culture and sensitivity results.

Gastrointestinal Complications

An ileus manifests as abdominal distention, nausea, vomiting, and failure to pass flatus. Physical examination may reveal the absence of bowel sounds. X-ray studies show distended loops of small and large bowel, with gas typically present in the colon. Treatment involves withholding oral intake, awaiting the return of bowel function, and providing adequate fluids and electrolytes.

In contrast, obstruction has high-pitched bowel sounds and peristaltic rushes. X-ray studies show single or multiple loops of distended bowel, typically in the small bowel, with air-fluid levels. The woman may need nasogastric suctioning or a duodenal/jejunal tube. Surgical consultation and possible lysis of adhesions may be needed if an obstruction persists.

Thromboembolic Complications

Deep venous thrombosis (DVT) is about twice as common after cesarean delivery than vaginal delivery.[92] DVT can progress to pulmonary embolus if untreated. Pulmonary embolism risk is increased 2.5 to 20 times in the setting of a cesarean delivery. DVT typically manifests as leg tenderness, swelling, or a palpable cord. Additional information is available in the *Venous Thromboembolism in Pregnancy* chapter.

The American College of Chest Physicians practice guidelines recommend early mobilization in postpartum women with no risk factors for DVT other than the postpartum state and the operative delivery.[93] For women with at least one additional risk factor, the guidelines suggest pharmacotherapy thromboprophylaxis (prophylactic LMWH or unfractionated heparin) or mechanical prophylaxis while the woman is in the hospital. For women with multiple risk factors for thromboembolism, guidelines suggest pharmacotherapy thromboprophylaxis combined with graduated compression stocking and/or intermittent pneumatic compression. ACOG recommends mechanical thromboprophylaxis before and after cesarean delivery.[94,95] ACOG notes that weight-based dosage for venous thromboembolism thromboprophylaxis may be more effective than dosage strategies based on body mass index in women with Class III obesity after cesarean delivery.[94,95]

Septic Thrombophlebitis

Septic thrombophlebitis is a diagnosis of exclusion. Persistent and unexplained fever is often the only symptom of septic thrombophlebitis, although some women report pelvic pain. Physical examination, ultrasound, and computed tomography scan are frequently negative. Continued fever without a known origin despite several days of antibiotic therapy suggests septic thrombophlebitis. Antibiotic coverage should include agents against streptococci, Enterobacteriaceae, and anaerobes. The ideal length of therapy is unknown, though some providers continue administering antibiotics until the woman has clinically improved and been without fever for at least 48 hours, and leukocytosis has resolved. Four to 8 days have been described for other abdominal infectious processes to occur, with up to 14 days being observed for septic emboli or positive blood cultures.[96] Anticoagulation is thought to prevent further thrombosis and reduce the spread of septic emboli, though evidence for its benefit needs further study.[97,98]

Delayed Postoperative Complications

Uterine Dehiscence and/or Rupture

Dehiscence and rupture of a uterine scar are uncommon complications that are diagnosed during a subsequent pregnancy. They are discussed in detail in the Counseling for Labor After Cesarean section.

Placenta Accreta

With a placenta previa and one previous cesarean delivery, the risk of placenta accreta is 24%; this risk continues to increase to approximately 67% with a placenta previa and four or more cesarean deliveries.[99] There is a significant increased risk of placenta previa, placenta accreta, placenta previa with accreta, and the need for gravid hysterectomy after a woman's second cesarean delivery.[99-101] One in 4 women who undergo repeat cesarean delivery because of placenta previa will require cesarean hysterectomy for hemorrhage caused by placenta accreta.[99-101] This complication increases with the number of prior uterine incisions.[102] Composite maternal morbidity in women with placenta previa and zero, one, two, or three prior cesarean deliveries was 15%, 23%, 59%, and 83%, respectively, and almost all of the excess composite maternal morbidity in women with a prior cesarean delivery was related to complications associated with placenta accreta.[103] In focal placenta accreta, the placental bed can be curetted and oversewn with interrupted sutures placed around the area of hemorrhage. If unsuccessful, then complete hysterectomy may be necessary because supracervical hysterectomy may not control the hemorrhage (see the Cesarean Hysterectomy section).

Repeat Cesarean Delivery

A major complication of cesarean delivery is that, based on 2019 birth data, 87% of patients will undergo cesarean delivery with subsequent pregnancies.[8] Repeat surgeries may involve adhesions and subfertility, chronic pain syndromes, and keloid formation. Repeat cesarean delivery is chosen over LAC for several reasons.[104] Some women express fear of a possible 'prolonged' labor and schedule cesarean delivery prophylactically to avoid anticipated psychological or physical harm. Training of health care providers could be examined in a variety of contexts to ascertain whether providers enable vaginal delivery, or at least labor onset, rather than encourage women to schedule a cesarean delivery.[104]

Controversies

Electronic Fetal Monitoring

The widespread use of electronic fetal monitoring (EFM) and the increased rate of cesarean delivery in response to fetal heart patterns detected with EFM have neither decreased acidosis-related newborn morbidity, nor decreased the incidence of cerebral palsy. Additional information is available in the *Intrapartum Fetal Surveillance* chapter.

Breech Presentation

American College of Obstetricians and Gynecologists guidelines recommend that the decision regarding mode of delivery of a fetus in breech presentation should depend on the experience of the maternity care provider. Cesarean delivery will be the preferred mode for most physicians because of the diminishing expertise in vaginal breech delivery.[105] Planned vaginal delivery of a term singleton in breech presentation may be reasonable under hospital-specific protocol guidelines for eligibility and labor management.[106] Before a vaginal breech delivery is planned, women should be informed that the risk of perinatal or neonatal mortality or short-term serious neonatal morbidity may be higher than if a cesarean delivery is planned,[107] and the woman's informed consent should be documented.[105]

Women with a fetus in breech presentation at 37 weeks' gestation should be encouraged to undergo ECV if they do not have a contraindication.[108,109] When LAC or vaginal breech delivery are not offered by the woman's maternity care provider or planned facility, referral to a provider and/or facility that does allow LAC or vaginal breech delivery should be offered.[110] Compared with planned vaginal delivery, planned cesarean delivery reduced perinatal or neonatal mortality as well as the composite outcome mortality or serious neonatal morbidity, although maternal morbidity increased somewhat. In a subset with 2-year follow-up, infant medical conditions were increased after planned cesarean delivery and no difference was shown in long-term neurodevelopmental delay or "death or neurodevelopmental delay" outcomes.[107] Additional information is available in the *Malpresentations, Malpositions, and Multiple Gestations* chapter.

Incidental Procedures

Some providers choose to perform cesarean delivery in women who are pregnant and near term if the woman has another indication for surgery (eg, desires sterilization). Several simple methods that result in tubal occlusion are available. Performance of an elective cesarean delivery because of the second surgical procedure should be discour-

aged, however, because of the increased morbidity and hospital stay related to cesarean versus vaginal delivery.

The surgeon's primary responsibility is safe operative delivery, even when pathology is found. Removal of adnexal abnormalities should be reserved for obvious malignancy, or lesions susceptible to torsion. Most leiomyomas regress after pregnancy and are highly vascular, hence removal should not be attempted unless an accessible pedicle and torsion is anticipated. Such lesions should be cross-clamped and Heaney transfixion ligated with an absorbable suture. A woman's lifetime risk of acute appendicitis is approximately 6.7%.[111] Thus, routine elective removal of the appendix at the time of cesarean delivery is not indicated.

Macrosomia

Although the diagnosis of fetal macrosomia is imprecise, prophylactic cesarean delivery may be considered for suspected fetal macrosomia with estimated fetal weights greater than approximately 11 lb (5,000 g) in women without diabetes and greater than approximately 9.9 lb (4,500 g) in women with diabetes.[112] A review of the available literature on the sonographic detection of macrosomia (greater than or equal to approximately 8.8 lb [4,000 g]) in general obstetric populations showed widely varying results: sensitivity of 12% to 75%, specificity of 68% to 99%, and posttest probability after a positive test of 17% to 79%; results for populations with a high prevalence of macrosomia were at the upper end of these ranges.[113] The diagnosis of macrosomia defined as greater than or equal to approximately 9.9 lb (4,500 g) was even less accurate, and there were no data on the ability to identify fetuses greater than approximately 11 lb (5,000 g). Hence 3,695 cesarean deliveries would have to be performed at an additional cost of $8.7 million to prevent one permanent brachial plexus injury in fetuses greater than approximately 9.9 lb (4,500 g) in women without diabetes.[114]

Most brachial plexus injuries resolve spontaneously and can occur in fetuses weighing less than approximately 8.8 lb (4,000 g) born by cesarean delivery. The results of the single RCT comparing elective delivery with expectant management at term in women with insulin-requiring diabetes who were pregnant show that induction of labor reduces the risk of macrosomia.[115] Induction of labor for suspected fetal macrosomia in women who do not have diabetes has not been shown to alter the risk of maternal or neonatal morbidity.[116]

Litigation

Concerns over liability risk have a major effect on the willingness of physicians and health care institutions to offer a trial of labor.[8] These concerns derive from the perception that catastrophic events associated with a trial of labor could lead to compensable claims with large verdicts or settlements for fetal/maternal injury, regardless of the adequacy of informed consent. Clearly, these medical malpractice issues affect practice patterns among providers and facilities, and they have a role in the genesis of the immediately available standard set by the 1999 ACOG guideline.[117]

Studies have attempted to model the effect of tort reform on primary and repeat cesarean delivery rates and have shown that modest improvements in the medico-legal climate may result in increases in VBAC and reductions in cesarean deliveries.[8] These analyses suggest that caps on noneconomic damages and reductions in physician malpractice premiums would result in fewer cesarean deliveries.

Many health care professionals incorrectly assume that performing a cesarean delivery helps avoid malpractice litigation. Performance of a cesarean delivery offers no protection against allegations of malpractice if an unhealthy infant is born. The plaintiff's legal team may shift to other issues, such as the cesarean delivery not being performed sooner, or a perceived lack of antenatal testing or prenatal care.

Family-Centered, Gentle Cesarean Delivery

The hospital-based procedures accompanying normal vaginal delivery have undergone many changes over the past few decades to support greater participation of family and to support early skin-to-skin contact in the delivery room. A 2008 study in England described a natural cesarean delivery where the surgical drape was lowered to allow the woman to observe her infant slowly emerging from the abdominal incision followed by immediate placement on the maternal chest.[118] Various additional modifications have been described in hospitals in the United States since 2013, including the use of a clear drape to facilitate observation,

delayed cord clamping, additional support individuals with the woman to assist with helping latch on during the cesarean delivery, and environmental changes including dimmed ambient light and a quieter operating room.

The family-centered cesarean delivery is appropriate for scheduled cesarean deliveries including elective repeat procedures and fetal malpresentations as well as nonemergent cesarean deliveries for labor dystocia between 37 and 41 weeks' gestation. Emergency cesarean deliveries occurring for indications such as concerning FHR monitoring, cord prolapse, placental abruption, or requiring general anesthesia would not be appropriate. Although RCTs or cohort studies have not been performed, a case series of 144 gentle cesarean deliveries showed good outcomes.[119] There is evidence that does not support the need for the presence of a pediatrician at a term cesarean delivery occurring for maternal indications.[120] Evidence supports the benefits of immediate skin-to-skin contact and delayed cord clamping for vaginal deliveries.[121,122] Safely introducing a gentle cesarean delivery approach requires collaborative planning between the obstetric surgeons, newborn care team, nursing, and anesthesiology.

Resuscitative Hysterotomy

Cesarean delivery has come full circle from its ancient origins as a postmortem procedure to the current recommendation that all appropriately skilled physicians should be able to perform a perimortem cesarean delivery that could save two lives.[123] *Perimortem cesarean delivery* or *emergency hysterotomy* has been renamed *resuscitative hysterotomy* by some to reflect the mutual optimization of resuscitation efforts that would potentially provide earlier and more substantial benefits to the woman and the infant based on the maternal cardiac rhythm.[124]

Since the 1990s, the American Heart Association has recommended resuscitative hysterotomy in women who are pregnant who have not benefitted from resuscitative efforts.[125] Uterine evacuation can increase cardiac output by 17% to 18% by relief of aortocaval compression. If promptly performed, resuscitative hysterotomy improves infant and maternal survival. The best survival rates are obtained when resuscitative hysterotomy is performed within 4 minutes of ineffective maternal circulation. It is still worthwhile to pursue delivery after 4 minutes because fetal mortality is 100% if no action is taken. It is not necessary to obtain consent from family members before performing the procedure.[126] Additional information is available in the *Maternal Resuscitation and Trauma* chapter.

Emergency hysterotomy is indicated when all of these requirements are met:

- Personnel with appropriate skill and equipment to perform the procedure are involved
- The woman does not benefit with a return of spontaneous circulation within 4 minutes
- Singleton gestation of 20 weeks' gestation or greater
- Appropriate facilities and personnel are available to care for the woman and infant after the procedure.

Counseling for Labor After Cesarean Delivery

Another possible approach in the nomenclature is to use the term *labor after cesarean* as recommended by ACOG instead of *trial of labor after cesarean*.[127] *Planned vaginal birth after cesarean* is a more optimistic way to refer to LAC. Decreasing the number of cesarean deliveries for labor dystocia and malpresentation as well as encouraging LAC can decrease cesarean rates without compromising outcomes.[128]

The decision by a woman who is pregnant to attempt a vaginal birth after LAC or plan a repeat cesarean delivery involves a balancing of maternal and neonatal risks for each woman, as well as personal preference.[29] The rate of perinatal mortality associated with LAC is similar to the perinatal mortality rate for infants born to nulliparous women in labor—1.3 per 1,000 deliveries compared with 0.5 per 1,000 deliveries in women choosing repeat cesarean delivery.[129]

During the informed consent process, at least three basic issues need to be addressed:

- What is the woman's plan for future family size?
- What is the likelihood of a successful VBAC?
- What are the safety concerns?

Future Family Size

Although there is no difference between planned cesarean delivery and planned vaginal delivery in risk of peripartum hysterectomy in a woman's first delivery, there is a significant increased risk of placenta previa, placenta accreta, placenta previa

with accreta, and the need for gravid hysterectomy after a woman's second cesarean delivery.[101] This emphasizes the need to consider the woman's total number of planned or expected pregnancies if cesarean delivery on maternal request is discussed during her first pregnancy, realizing that many pregnancies are unplanned.[130] These are also factors that may be influenced by parity and planned family size. Uterine scars put women at increased risk of uterine rupture in subsequent pregnancies.[129,131]

For those considering larger families, LAC may avoid potential future maternal consequences of multiple cesarean deliveries[130] such as hysterectomy, bowel or bladder injury, transfusion, infection, and abnormal placentation such as placenta previa and invasive placenta.[102]

Possibility of Successful Vaginal Birth

Most women with one previous cesarean delivery with a low transverse incision are candidates for and should be counseled about VBAC and offered LAC.[130]

Approximately 75% of women who attempt LAC will be successful; this rate varies depending on the clinical situation that led to the first cesarean delivery.[8] The VBAC rate is highest in women with a previous successful LAC, previous vaginal delivery, previous cesarean delivery for nonvertex presentation, and women with spontaneous onset of labor. Based on 43 US studies, 74% of women attempting a LAC deliver vaginally.[8] Prior vaginal delivery increases the likelihood to approximately 94% and a prior cesarean delivery for labor dystocia decreases the likelihood to approximately 54%.[8,132]

The studies of women with twin gestations who attempt VBAC have consistently shown that their outcomes are similar to those of women with singleton gestations who attempt VBAC with regard to the likelihood of success and the risk of uterine rupture or maternal or perinatal morbidity complications.[133] Women with one previous low transverse cesarean delivery, who are otherwise appropriate candidates for twin vaginal delivery, may be considered candidates for LAC.[29,130]

Use of the currently available VBAC calculators is discouraged. They often underestimate the likelihood of successful VBAC.[134] In reality, the underestimation of success will be even greater as the definition of active labor has changed from 4 cm to 6 cm since this validation study was completed.[7] VBAC calculators are most accurate when they predict a likely successful VBAC and when performed after admission to the hospital.[134] The VBAC calculators can distract from the most important part of counseling: risk of uterine rupture versus risks of repeat cesarean delivery. Why use calculators in women who have had a cesarean delivery when they are not used in women who have not?[135] By underestimating LAC success rates and giving an apparently objective number, VBAC calculators in their current form likely contribute to the low number of women in the United States choosing LAC; the current VBAC rate is 8%, but it could be 74% or higher if all women with a prior cesarean delivery chose LAC.[136]

Safety Concerns

Women with previous cesarean delivery have two options for delivery during subsequent pregnancies; they may have an elective repeat cesarean delivery or they may undergo LAC. Sixty percent to 80% of women with previous cesarean delivery can have a successful vaginal delivery.[129] The benefit of this is decreased maternal risk associated with vaginal delivery (ie, decreased mortality, blood loss, risk of transfusion, risk of thromboembolism, risk of infection) and a quicker recovery period with decreased length of hospitalization. The *Appendix* provides a sample informed consent form for conveying risks and benefits to women considering LAC.

Planned elective repeat cesarean delivery and planned VBAC for women with a previous cesarean delivery are associated with benefits and harms. The two major risks of LAC are uterine dehiscence and/or rupture. Because of the risk of uterine rupture, fetal mortality has been shown to increase with LAC versus repeat elective cesarean delivery. Evidence for the risks and benefits of LAC versus repeat cesarean delivery are predominantly from retrospective cohort studies because of the inability to conduct RCTs.[137]

Uterine Dehiscence and/or Rupture

Dehiscence and rupture of a uterine scar are uncommon complications that are diagnosed during a subsequent pregnancy. The overall rate of uterine rupture during a subsequent LAC is 0.7%.[131] On average, the incremental risk of rupture with LAC compared with elective repeat cesarean delivery is 2.7 per 1,000.[129]

The term *uterine dehiscence* is commonly applied to asymptomatic scar separation that does not

penetrate the serosa and does not produce hemorrhage. Dehiscence occurs in 0 to 19 per 1,000 LACs (mean weighted average 12.6 per 1,000 LACs). This rate is comparable to that in women undergoing elective repeat cesarean delivery.[129] Dehiscence presents as a serosal window and is often discovered unexpectedly during a repeat cesarean delivery. Rupture of lower segment scars typically occurs during labor, but may occur antepartum, particularly with classical uterine scars.[138]

Rupture

In contrast to dehiscence, uterine rupture is a through-and-through scar separation that is clinically symptomatic and requires surgical intervention. Uterine rupture occurs in approximately 0.7% of women with a prior cesarean delivery.[131]

The risk of perinatal mortality or hypoxic-ischemic encephalopathy during LAC is approximately 1 in 2,000 deliveries.[139] This risk is similar to that for infants of women during their first delivery.[140]

Fetal bradycardia is the most common and characteristic clinical manifestation of uterine rupture, occurring in 33% to 70% of symptomatic cases.[141] Variable or late decelerations may precede the bradycardia, but there is no FHR pattern pathognomonic of rupture. Perinatal mortality/morbidity is higher in fetuses that experience complete extrusion into the maternal abdomen.[142]

Maternal presentations are variable. In women with known uterine scarring or trauma, uterine rupture should always be strongly considered if constant abdominal pain and signs of intraabdominal hemorrhage are present. Vaginal bleeding is not a cardinal symptom, because it may be modest, despite major intraabdominal hemorrhage. Other clinical manifestations include maternal tachycardia, hypotension ranging from subtle to severe (hypovolemic shock), cessation of uterine contractions, loss of station of the fetal presenting part, uterine tenderness, and change in uterine shape. Postpartum uterine rupture is characterized by pain and persistent vaginal bleeding despite use of uterotonic agents. Hematuria may occur if the rupture extends into the bladder.

Treatment of symptomatic uterine rupture is largely dependent on the woman's hemodynamic status and desire for future fertility. In some cases, a layered closure of the myometrium with absorbable suture will suffice, though hysterectomy may be necessary.

Previous Uterine Incision

The preponderance of evidence suggests that most women with one previous cesarean delivery with a low transverse incision are candidates for and should be counseled about VBAC and offered LAC.[143,144]

A National Institute of Child Health and Human Development Maternal-Fetal Medicine Units Network study with sufficient size to control for confounding variables showed no increased risk of uterine rupture (0.7% versus 0.9%; P = 0.37) in women with one versus multiple prior cesarean deliveries.[143] The adverse maternal outcomes of a trial of vaginal delivery after two previous cesarean deliveries are comparable to a third repeat cesarean delivery.[144] Given the available data of success rate (71.1%) and uterine rupture rate (1.36%), and the comparable risk of maternal morbidity with repeat cesarean delivery, providers should appropriately counsel women requesting a trial of vaginal delivery after two cesarean deliveries.[144] In addition, the likelihood of achieving VBAC appears to be similar for women with one or more cesarean deliveries. Given the overall data, it is reasonable to consider women with two previous low transverse cesarean deliveries to be candidates for LAC and to counsel them based on the combination of other factors that affect their probability of achieving a successful VBAC.[130]

Women at high risk of complications (eg, those with previous classical or T-incision, prior uterine rupture, or extensive transfundal uterine surgery) and those in whom vaginal delivery is otherwise contraindicated (eg, those with placenta previa) are not typically candidates for planned LAC.[130]

Induction of Labor

Induction of labor for maternal or fetal indications remains an option in women undergoing LAC.[130]

Studies of specific prostaglandins are limited in size, but indicate that rupture risk may vary among these agents. Given the lack of data suggesting increased risk with mechanical dilation and transcervical catheters, such interventions may be an option for LAC candidates with an unfavorable cervix.[130]

Women who have had cesarean deliveries who undergo induction of labor, especially with prostaglandins, have an increased risk of uterine rupture.[145] Therefore, misoprostol should not be used for third-trimester cervical ripening or labor

induction in women who have had a cesarean delivery or major uterine surgery.[130] Oxytocin use appears to increase risk of uterine rupture, with higher risk of rupture at higher doses. However, rupture risk is lower than with prostaglandin use and LAC may be attempted if acceptable to the woman and provider. There is no established dosage limit for oxytocin during LAC.[130] One large RCT showed the risk of uterine rupture after cesarean delivery to be 1.1% when labor was induced with oxytocin compared with 0.4% with spontaneous labor.[139]

Regional Analgesia

Epidural analgesia for labor may be used as part of LAC, and adequate pain relief may encourage more women to choose LAC.[29,130] In addition, effective regional analgesia should not be expected to mask signs and symptoms of uterine rupture, particularly because the most common sign of rupture is fetal heart tracing abnormalities.

External Cephalic Version

Limited data regarding ECV for fetuses in breech presentation in a woman with a prior uterine incision suggest that ECV is not contraindicated if a woman is at low risk of adverse maternal or neonatal outcomes from ECV and LAC.[146] The likelihood of successful ECV has been shown to be similar in women with and without a prior cesarean delivery.[130]

Unknown Type of Previous Uterine Incision

The type of uterine incision performed at the time of a prior cesarean delivery cannot be confirmed in some women. Although some have questioned the safety of offering VBAC under these circumstances, two case series from large tertiary care facilities reported rates of VBAC success and uterine rupture similar to those from other contemporaneous studies of women with documented previous low transverse uterine incisions.[147] No significant association was shown with the presence of an unknown scar when evaluating risk factors for uterine rupture. The absence of an association may result from the fact that most cesarean incisions are low transverse, and the uterine scar type can often be inferred based on the indication for the prior cesarean delivery. Therefore, LAC is not contraindicated in women with one previous cesarean delivery with an unknown uterine scar type unless there is a high clinical suspicion of a previous classical uterine incision.[130]

The percentages listed in *Table 9* are estimates of the influence of a single factor's influence on the likelihood of uterine rupture. Because women rarely present with only one of these factors and evidence is limited on the additivity of multiple factors, providers must try to assess the effect of a series of influences to provide individualized guidance to women during prenatal care.

Level of Care

After consideration of the data from the National Institutes of Health Consensus Development Conference on Vaginal Birth After Cesarean: New Insights, ACOG stated that a trial of labor after previous cesarean delivery should be undertaken at facilities capable of performing emergency deliveries.[130]

Table 9. Factors Influencing Risk of Uterine Rupture

Decreased risk of uterine rupture (<1%)
Prior vaginal delivery
Low uterine segment incision from prior cesarean delivery
Preterm delivery
Two-layer closure of uterine incision (data suggestive but not conclusive)
Unknown uterine scar without high risk of prior classical incision
Mild increased risk of uterine rupture (1% to 2%)
Induction of labor with good Bishop score with oxytocin
One-layer uterine closure
Gestational age of more than 40 weeks
Low vertical uterine incision (limited data; could be increased to up to 5%)
Morbid obesity (BMI ≥40 kg/m^2)
Two or more prior uterine incisions without vaginal delivery
Induction of labor with poor Bishop score with oxytocin
Increased risk of uterine rupture (<2% to 4%)
Unknown scar in the setting of high risk of prior classical incision (eg, preterm abnormal lie, term transverse lie)
Classical or T-shaped uterine incision (4% to 9%)
Prior myomectomy, cornual resection, or other full-thickness uterine surgery
Prior uterine rupture

BMI = body mass index.

Information from Spong CY, Landon MB, Gilbert S, et al; National Institute of Child Health and Human Development (NICHD) Maternal-Fetal Medicine Units (MFMU) Network. Risk of uterine rupture and adverse perinatal outcome at term after cesarean delivery. Obstet Gynecol. *2007;110(4):801-807; Roberge S, Demers S, Berghella V, et al. Impact of single- vs double-layer closure on adverse outcomes and uterine scar defect: a systematic review and metaanalysis.* Am J Obstet Gynecol. *2014;211(5):453-460.*

Because of the risks associated with LAC and the unpredictability of uterine rupture and other complications, ACOG guidelines recommend that LAC be attempted in facilities that can provide emergency cesarean delivery for situations that are immediate threats to the life of the woman or fetus. When such resources are not available, ACOG guidelines recommend that women and obstetricians or other maternity care providers considering LAC should discuss the availability of obstetric, pediatric, anesthesiology, and operating room staff and other hospital resources.[130]

Furthermore, ACOG stated that after counseling, the ultimate decision to undergo LAC or a repeat cesarean delivery should be made by the woman in consultation with her maternity care provider.[130] The potential risks and benefits of LAC and elective repeat cesarean delivery should be discussed. Documentation of counseling and the management plan should be included in the medical record.

The AAFP guideline on planning for VBAC states, "All women desiring LAC/VBAC should be counseled about the capabilities of their specific delivery setting, and women determined to be at high risk of complications with labor and VBAC or repeat cesarean birth should be referred to facilities capable of effectively treating problems as they develop."[132,148]

Global Considerations for Cesarean Delivery

Although some low-resource settings have cesarean delivery rates that are too low, others have cesarean delivery rates that are too high. The WHO recommends a cesarean delivery rate of approximately 15%.[149]

Low cesarean delivery rates can lead to maternal and neonatal mortality and morbidity, including obstetric fistulas. In a WHO study, cesarean delivery was underused; in the study of 83,439 births, the average cesarean delivery rate was 8.8% and only 73% of facilities had cesarean delivery capability.[150] Even when cesarean delivery is feasible and available, another limiting factor may be the availability of stored safe blood supply or safe anesthesia. Emergent cesarean deliveries are associated with increased morbidity and mortality.[150]

Other low-resource areas have cesarean delivery rates that are too high, which is associated with increased maternal and neonatal morbidity and mortality. In a WHO study of Latin America, private hospitals had a mean cesarean delivery rate over 50%.[151] The higher cesarean delivery rates were associated with increased fetal mortality rates, higher preterm delivery rates, and longer neonatal intensive care unit stays.[151]

Wherever cesarean delivery is performed, it is important that the woman understands the indications for her cesarean delivery and any implications that the type of incision made may have on future delivery options, especially in areas with poor access to prenatal care. This education should be provided before discharge.

For additional details on cesarean delivery in developing countries, refer to the Global ALSO Program available at http://www.aafp.org/globalalso.

Summary

Cesarean delivery is the most common operative procedure in the United States and has accounted for approximately one-third of all deliveries.[9] Cesarean delivery can involve significant morbidity and mortality, both of which can be minimized by thoughtful preoperative, intraoperative, and postoperative care, and careful patient selection. All maternity care providers should be familiar with the diagnosis and management of postcesarean delivery complications. Efforts to lower the primary cesarean delivery rate and increase access to LAC are important to public health because of the increased morbidity and mortality of repeat cesarean delivery.

The application of evidence-based practice to cesarean delivery and support for patient safety in the operating room and postpartum settings can decrease operative morbidity.

Nursing Considerations: Cesarean Delivery

- Identify the location of additional instruments that may be necessary, including routine instruments, vacuum, forceps, and resuscitative hysterotomy kit
- Facilitate a time out before every surgery
- Communicate completion of all counts
- If a resuscitative hysterotomy is initiated, assist in facilitating the 4-minute rule by avoiding an ultrasound or FHR tracing, and by not transferring the woman

FHR = fetal heart rate.

Appendix
Sample Patient Informed Consent Form: Information About Labor and Vaginal Delivery After Cesarean Delivery

Many women in the United States deliver their babies by cesarean delivery, an operation where the baby is delivered through an incision, or cut, in the woman's abdomen and uterus. For years, physicians believed that if a woman underwent one cesarean delivery, she must undergo cesarean delivery for all subsequent pregnancies. Studies have shown that it is safe for most women who have previously undergone cesarean delivery to attempt a vaginal delivery. This is called a labor after cesarean delivery (LAC). If the woman is successful, she has had a vaginal birth after cesarean delivery (VBAC).

Although it is safe for most women to attempt LAC/VBAC, there are some women with risk factors that might make it unsafe. Your maternity care provider will review your history and records to determine if you would be a good candidate for LAC/VBAC. Many experts encourage women who do not have risk factors to attempt LAC/VBAC. At [INSERT FACILITY NAME HERE], we feel that it is the best choice for many women. Approximately 75% of women who attempt LAC will be successful.

There are some advantages and some risks to undergoing a repeat cesarean delivery or LAC/VBAC. Both options have some risks. The decision about whether to attempt LAC/VBAC is a personal one. This information sheet provides general facts about repeat cesarean delivery and LAC/VBAC. Please discuss your individual case with your provider to ensure the correct decision is made.

Advantages of LAC/VBAC

1. Less risk to the woman. Women who have a vaginal delivery have a reduced likelihood of getting an infection. Typically, there is less bleeding and less risk of needing a blood transfusion.

2. Shorter recovery time. Most women can leave the hospital 1 or 2 days after having a vaginal delivery. Most women stay at least 2 to 3 days after a cesarean delivery. After going home, women who have had a vaginal delivery typically return to normal activities sooner than those who have undergone cesarean delivery. There is typically less pain after a vaginal delivery.

3. More involvement in the birth. Many women think a vaginal delivery allows them to be more involved in the birth. After a vaginal delivery, the woman can usually hold the newborn right away and begin breastfeeding. After a cesarean delivery, the woman often cannot hold the newborn or breastfeed until the operation is complete and the effects of anesthesia have subsided. More than one family member may be in the room for a vaginal delivery if the woman wishes, whereas only one person is allowed in the room for a cesarean delivery. If the woman needs general anesthesia, no friends or family are allowed in the operating room.

4. Future pregnancies. Women who have a successful VBAC will have less risk of complications with future pregnancies compared with women having a repeat cesarean delivery.

Disadvantages of LAC

1. Unsuccessful labor. Not all women who attempt a vaginal delivery are successful. Women who need a repeat cesarean delivery after an unsuccessful labor may have more risk of infection, bleeding and blood transfusion, or injury to nearby organs (eg, bowel, bladder). The likelihood of a successful VBAC is higher in women with a previous vaginal delivery.

2. Uterine rupture. There is a small chance that the scar in the uterus from the previous cesarean delivery may rupture, or come apart, in labor. If this happens, an emergency cesarean delivery is required. There is a risk that the baby may be seriously injured or die. At [INSERT FACILITY

NAME HERE], there are staff in the hospital 24 hours a day who are capable of performing an emergency cesarean delivery. In most cases, the baby is delivered before it is harmed. Rupture of the uterus also increases the risk of injury to the woman's nearby tissues, such as the bladder or bowel. There is also a risk of needing a hysterectomy. The risk of rupture is higher in women who have undergone more than one previous cesarean delivery and lower in women who have had a previous vaginal delivery.

Other Issues

1. Pain. Many women think that labor may be painful and a cesarean delivery will mean they do not have to experience the pain of labor. Although labor is painful, there are several options for relieving the pain. Medicine may be administered intravenously and this works for many women. Epidural analgesia (a procedure that numbs the abdomen so that the woman does not feel labor pains) is available for women who would like to have it. Women who undergo a cesarean delivery typically have more pain for a longer time than women who have a vaginal delivery.

2. Labor induction. The drug oxytocin is used to start labor or assist if labor is not progressing normally. Many studies show that oxytocin does not increase the risk of problems for women undergoing LAC/VBAC if used to help a labor that has already begun. If oxytocin is used to start labor (induction), the likelihood of uterine rupture increases from about 1 in 200 to 1 in 100 women. At [INSERT FACILITY NAME HERE], we use oxytocin in women attempting LAC/VBAC if it is needed.

3. Monitoring. Women attempting LAC/VBAC at [INSERT FACILITY NAME HERE] receive continuous monitoring of the baby's heart rate and uterine contractions. Also, an intravenous line will be placed. This helps with identifying problems and performing a cesarean delivery rapidly if necessary.

This information should help you with choosing between LAC/VBAC or repeat cesarean delivery. Please talk to your provider about any questions you have so that you may get the information you need to help make this choice. Talking with family members may also be helpful. Regardless of which delivery method you choose, our goal at [INSERT FACILITY NAME HERE] is to provide you with the best possible care for a good outcome—a healthy mother and a healthy baby.

I have read the information sheet and have had the chance to discuss it with my provider. Any questions I had have been answered to my satisfaction.

❑ I choose a trial of labor and vaginal birth after cesarean delivery

❑ I choose to have a repeat cesarean delivery

Patient (please print)	Patient Signature	Date
Provider (please print)	Provider Signature	Date
Witness (please print)	Witness Signature	Date

References

1. American Academy of Family Physicians. Maternity and Gynecologic Care. 2014. Available at http://www.aafp.org/dam/AAFP/documents/medical_education_residency/program_directors/Reprint261_Maternity.pdf.
2. American Academy of Family Physicians. Cesarean Delivery in Family Medicine (Position Paper). 2018. Available at https://www.aafp.org/about/policies/all/cesarean-delivery.html.
3. Magee SR, Eidson-Ton WS, Leeman L, et al. Family Medicine Maternity Care Call to Action: moving toward national standards for training and competency assessment. *Fam Med*. 2017;49(3):211-217.
4. Dresang L, Koch P. The need for rural family physicians who can perform cesareans. *Am J Chin Med*. 2009;6(2):39-41.
5. Sewell JE. *Cesarean Section: a Brief History*. Washington, DC, American College of Obstetricians and Gynecologists; 1993.
6. Martin JA, Hamilton BE, Osterman MJK, et al. Births: final data for 2016. *Natl Vital Stat Rep*. 2018;67(1):1-55.
7. American College of Obstetricians and Gynecologists; Society for Maternal-Fetal Medicine. Obstetric care consensus no. 1: safe prevention of the primary cesarean delivery. *Obstet Gynecol*. 2014;123(3):693-711.
8. Martin JA, Hamilton BE, Osterman MJK. Births in the United States, 2018. *NCHS Data Brief*. 2019;(346):1-8.
9. Tita ATN, Landon MB, Spong CY, et al.; Eunice Kennedy Shriver NICHD Maternal-Fetal Medicine Units Network. Timing of elective repeat cesarean delivery at term and neonatal outcomes. *N Engl J Med*. 2009;360(2):111-120.
10. Tuuli MG, Liu J, Stout MJ, et al. A randomized trial comparing skin antiseptic agents at cesarean delivery. *N Engl J Med*. 2016;374(7):647-655.
11. Haas DM, Morgan S, Contreras K. Vaginal preparation with antiseptic solution before cesarean section for preventing postoperative infections. *Cochrane Database Syst Rev*. 2013;(1):CD007892.
12. Smaill FM, Grivell RM. Antibiotic prophylaxis versus no prophylaxis for preventing infection after cesarean section. *Cochrane Database Syst Rev*. 2014;10(10):CD007482.
13. Gyte GML, Dou L, Vazquez JC. Different classes of antibiotics given to women routinely for preventing infection at caesarean section. *Cochrane Database Syst Rev*. 2014;11(11):CD008726.
14. Sullivan SA, Smith T, Chang E, et al. Administration of cefazolin prior to skin incision is superior to cefazolin at cord clamping in preventing postcesarean infectious morbidity: a randomized, controlled trial. *Am J Obstet Gynecol*. 2007;196(5):455.e1-455.e5.
15. American College of Obstetricians and Gynecologists. ACOG Practice Bulletin no. 120: use of prophylactic antibiotics in labor and delivery. *Obstet Gynecol*. 2011;117(6):1472-1483.
16. Mackeen AD, Packard RE, Ota E, et al. Timing of intravenous prophylactic antibiotics for preventing postpartum infectious morbidity in women undergoing cesarean delivery. *Cochrane Database Syst Rev*. 2014;12(12):CD009516.
17. Committee on Practice Bulletins-Obstetrics. ACOG Practice Bulletin no. 199: use of prophylactic antibiotics in labor and delivery. *Obstet Gynecol*. 2018;132(3):e103-e119.
18. Tita AT, Szychowski JM, Boggess K, et al. C/SOAP Trial Consortium. Adjunctive azithromycin prophylaxis for cesarean delivery. *N Engl J Med*. 2016;375(13):1231-1241.
19. Wilson W, Taubert KA, Gewitz M, et al.; American Heart Association Rheumatic Fever, Endocarditis, and Kawasaki Disease Committee. American Heart Association Council on Cardiovascular Disease in the Young; American Heart Association Council on Clinical Cardiology; American Heart Association Council on Cardiovascular Surgery and Anesthesia; Quality of Care and Outcomes Research Interdisciplinary Working Group. Prevention of infective endocarditis: guidelines from the American Heart Association: a guideline from the American Heart Association Rheumatic Fever, Endocarditis, and Kawasaki Disease Committee, Council on Cardiovascular Disease in the Young, and the Council on Clinical Cardiology, Council on Cardiovascular Surgery and Anesthesia, and the Quality of Care and Outcomes Research Interdisciplinary Working Group. *Circulation*. 2007;116(15):1736-1754.
20. Hofmeyr JG, Novikova N, Mathai M, Shah A. Techniques for cesarean section. *Am J Obstet Gynecol*. 2009;201(5):431-444.
21. Mathai M, Hofmeyr GJ, Mathai NE. Abdominal surgical incisions for caesarean section. *Cochrane Database Syst Rev*. 2013;5(5):CD004453.
22. O'Neill HA, Egan G, Walsh CA, et al. Omission of the bladder flap at caesarean section reduces delivery time without increased morbidity: a meta-analysis of randomised controlled trials. *Eur J Obstet Gynecol Reprod Biol*. 2014;174:20-26.
23. Dahlke JD, Mendez-Figueroa H, Rouse DJ, et al. Evidence-based surgery for cesarean delivery: an updated systematic review. *Am J Obstet Gynecol*. 2013;209(4):294-306.
24. Alexander JM, Leveno KJ, Hauth J, et al.; National Institute of Child Health and Human Development Maternal-Fetal Medicine Units Network. Fetal injury associated with cesarean delivery. *Obstet Gynecol*. 2006;108(4):885-890.
25. Cromi A, Ghezzi F, Di Naro E, et al. Blunt expansion of the low transverse uterine incision at cesarean delivery: a randomized comparison of 2 techniques. *Am J Obstet Gynecol*. 2008;199(3):292.e1-292.e6.
26. Saad AF, Rahman M, Costantine MM, Saade GR. Blunt versus sharp uterine incision expansion during low transverse cesarean delivery: a metaanalysis. *Am J Obstet Gynecol*. 2014;211(6):684.e1-684.e11.
27. Dodd JM, Anderson ER, Gates S, Grivell RM. Surgical techniques for uterine incision and uterine closure at the time of caesarean section. *Cochrane Database Syst Rev*. 2014;7(7):CD004732.
28. Anorlu RI, Maholwana B, Hofmeyr GJ. Methods of delivering the placenta at caesarean section. *Cochrane Database Syst Rev*. 2008;(3):CD004737.
29. Sabol B, Denman MA, Guise JM. Vaginal birth after cesarean: an effective method to reduce cesarean. *Clin Obstet Gynecol*. 2015;58(2):309-319.

30. American College of Obstetricians and Gynecologists' Committee on Practice Bulletins—Obstetrics. ACOG Practice Bulletin no. 205: vaginal birth after cesarean delivery. *Obstet Gynecol*. 2019;133(2):e110-e127.

31. American Academy of Pediatrics Committee on Fetus and Newborn; American College of Obstetricians and Gynecologists Committee on Obstetric Practice. *Guidelines for Perinatal Care*. 8th ed. Washington, DC: American College of Obstetricians and Gynecologists; 2017.

32. American College of Obstetricians and Gynecologists. Postpartum hemorrhage from vaginal delivery. Patient safety checklist no. 10. *Obstet Gynecol*. 2013;121: 1151-1152.

33. Güngördük K, Yildirim G, Ark C. Is routine cervical dilatation necessary during elective caesarean section? A randomised controlled trial. *Aust N Z J Obstet Gynaecol*. 2009;49(3):263-267.

34. Chi IC, Zhou SW, Balogh S, Ng K. Post-cesarean section insertion of intrauterine devices. *Am J Public Health*. 1984;74(11):1281-1282.

35. Levi EE, Stuart GS, Zerden ML, et al. Intrauterine device placement during cesarean delivery and continued use 6 months postpartum: a randomized controlled trial. *Obstet Gynecol*. 2015;126(1):5-11.

36. Jacobs-Jokhan D, Hofmeyr G. Extra-abdominal versus intra-abdominal repair of the uterine incision at caesarean section. *Cochrane Database Syst Rev*. 2004;(4): CD000085.

37. Walsh CA, Walsh SR. Extraabdominal vs intraabdominal uterine repair at cesarean delivery: a metaanalysis. *Am J Obstet Gynecol*. 2009;200(6):625.e1-625.e8.

38. Zaphiratos V, George RB, Boyd JC, Habib AS. Uterine exteriorization compared with in situ repair for cesarean delivery: a systematic review and meta-analysis. *Can J Anaesth*. 2015;62(11):1209-1220.

39. Coutinho IC, Ramos de Amorim MM, Katz L, Bandeira de Ferraz AA. Uterine exteriorization compared with in situ repair at cesarean delivery: a randomized controlled trial. *Obstet Gynecol*. 2008;111(3):639-647.

40. Roberge S, Demers S, Berghella V, et al. Impact of single- vs double-layer closure on adverse outcomes and uterine scar defect: a systematic review and meta-analysis. *Am J Obstet Gynecol*. 2014;211(5):453-460.

41. Yasmin S, Sadaf J, Fatima N. Impact of methods for uterine incision closure on repeat caesarean section scar of lower uterine segment. *J Coll Physicians Surg Pak*. 2011;21(9):522-526.

42. Ceci O, Cantatore C, Scioscia M, et al. Ultrasonographic and hysteroscopic outcomes of uterine scar healing after cesarean section: comparison of two types of single-layer suture. *J Obstet Gynaecol Res*. 2012; 38(11):1302-1307.

43. Bennich G, Rudnicki M, Wilken-Jensen C, et al. Impact of adding a second layer to a single unlocked closure of a cesarean uterine incision: randomized controlled trial. *Ultrasound Obstet Gynecol*. 2016;47(4):417-422.

44. Bamigboye AA, Hofmeyr GJ. Closure versus non-closure of the peritoneum at caesarean section: short- and long-term outcomes. *Cochrane Database Syst Rev*. 2014;8(8):CD000163.

45. Gurusamy KS, Toon CD, Davidson BR. Subcutaneous closure versus no subcutaneous closure after non-caesarean surgical procedures. *Cochrane Database Syst Rev*. 2014;1(1):CD010425.

46. Anderson ER, Gates S. Techniques and materials for closure of the abdominal wall in caesarean section. *Cochrane Database Syst Rev*. 2004;(4):CD004663.

47. Mackeen AD, Schuster M, Berghella V. Suture versus staples for skin closure after cesarean: a metaanalysis. *Am J Obstet Gynecol*. 2015;212(5):621.e1-621.e10.

48. Mackeen AD, Berghella V, Larsen ML. Techniques and materials for skin closure in caesarean section. *Cochrane Database Syst Rev*. 2012;11:CD003577.

49. Daykan Y, Sharon-Weiner M, Pasternak Y, et al. Skin closure at cesarean delivery, glue vs subcuticular sutures: a randomized controlled trial. *Am J Obstet Gynecol*. 2017;216(4):406.e1-406.e5.

50. Regenbogen SE, Greenberg CC, Resch SC, et al. Prevention of retained surgical sponges: a decision-analytic model predicting relative cost-effectiveness. *Surgery*. 2009;145(5):527-535.

51. Mangesi L, Hofmeyr GJ. Early compared with delayed oral fluids and food after caesarean section. *Cochrane Database Syst Rev*. 2002;3(3):CD003516.

52. Hsu YY, Hung HY, Chang SC, Chang YJ. Early oral intake and gastrointestinal function after cesarean delivery: a systematic review and meta-analysis. *Obstet Gynecol*. 2013;121(6):1327-1334.

53. Di Spiezio Sardo A, Saccone G, McCurdy R, et al. Risk of cesarean scar defect following single- vs double-layer uterine closure: systematic review and meta-analysis of randomized controlled trials. *Ultrasound Obstet Gynecol*. 2017;50(5):578-583.

54. Yazicioglu F, Gökdogan A, Kelekci S, et al. Incomplete healing of the uterine incision after caesarean section: Is it preventable? *Eur J Obstet Gynecol Reprod Biol*. 2006;124(1):32-36.

55. Tuuli MG, Odibo AO, Fogertey P, et al. Utility of the bladder flap at cesarean delivery: a randomized controlled trial. *Obstet Gynecol*. 2012;119(4):815-821.

56. Gates S, Anderson ER. Wound drainage for caesarean section. *Cochrane Database Syst Rev*. 2013;12(12): CD004549.

57. Tang OS, Gemzell-Danielsson K, Ho PC. Misoprostol: pharmacokinetic profiles, effects on the uterus and side-effects. *Int J Gynaecol Obstet*. 2007;99(Suppl 2): S160-S167.

58. Winikoff B, Dabash R, Durocher J, et al. Treatment of post-partum haemorrhage with sublingual misoprostol versus oxytocin in women not exposed to oxytocin during labour: a double-blind, randomised, non-inferiority trial. *Lancet*. 2010;375(9710):210-216.

59. WOMAN Trial Collaborators. Effect of early tranexamic acid administration on mortality, hysterectomy, and other morbidities in women with post-partum haemorrhage (WOMAN): an international, randomised, double-blind, placebo-controlled trial. *Lancet*. 2017;389(10084): 2105-2116.

60. World Health Organization. WHO recommendation on tranexamic acid for the treatment of postpartum haemorrhage. 2017. Available at http://apps.who.int/iris/bitstream/handle/10665/259374/9789241550154-eng.pdf;jsessionid=B9A05DB8981A3BC1A493B96EAA14BFAE?sequence=1.
61. Committee on Practice Bulletins-Obstetrics. Practice Bulletin no. 183: postpartum hemorrhage. *Obstet Gynecol*. 2017;130(4):e168-e186.
62. B-Lynch C, Coker A, Lawal AH, et al. The B-Lynch surgical technique for the control of massive postpartum haemorrhage: an alternative to hysterectomy? Five cases reported. *Br J Obstet Gynaecol*. 1997;104(3):372-375.
63. Professor Christopher B-Lynch. Description of Technique. 1997. Available at http://www.cblynch.co.uk/description-of-technique/.
64. Kayem G, Kurinczuk JJ, Alfirevic Z, et al.; U.K. Obstetric Surveillance System (UKOSS). Uterine compression sutures for the management of severe postpartum hemorrhage. *Obstet Gynecol*. 2011;117(1):14-20.
65. Clark SL, Phelan JP, Yeh SY, et al. Hypogastric artery ligation for obstetric hemorrhage. *Obstet Gynecol*. 1985;66(3):353-356.
66. Joshi VM, Otiv SR, Majumder R, et al. Internal iliac artery ligation for arresting postpartum haemorrhage. *BJOG*. 2007;114(3):356-361.
67. Bakri YN, Amri A, Abdul Jabbar F. Tamponade-balloon for obstetrical bleeding. *Int J Gynaecol Obstet*. 2001;74(2):139-142.
68. Cook Medical. Bakri Postpartum Balloon. Available at https://www.cookmedical.com/data/resources/RH-D38426-EN-F_M3_1510939328515.pdf.
69. Oliphant SS, Bochenska K, Tolge ME, et al. Maternal lower urinary tract injury at the time of cesarean delivery. *Int Urogynecol J Pelvic Floor Dysfunct*. 2014;25(12):1709-1714.
70. Shellhaas CS, Gilbert S, Landon MB, et al.; Eunice Kennedy Shriver National Institutes of Health and Human Development Maternal-Fetal Medicine Units Network. The frequency and complication rates of hysterectomy accompanying cesarean delivery. *Obstet Gynecol*. 2009;114(2 Pt 1):224-229.
71. Alexander JM, Leveno KJ, Rouse DJ, et al.; National Institute of Child Health and Human Development (NICHD) Maternal-Fetal Medicine Units Network (MFMU). Comparison of maternal and infant outcomes from primary cesarean delivery during the second compared with first stage of labor. *Obstet Gynecol*. 2007;109(4):917-921.
72. Phipps MG, Watabe B, Clemons JL, et al. Risk factors for bladder injury during cesarean delivery. *Obstet Gynecol*. 2005;105(1):156-160.
73. Lee JS, Choe JH, Lee HS, Seo JT. Urologic complications following obstetric and gynecologic surgery. *Korean J Urol*. 2012;53(1):795-799.
74. Nielsen TFH. ökegård K-H. Cesarean section and intraoperative surgical complications. *Acta Obstet Gynecol Scand*. 1984;63(2):103-108.
75. Jones OH. Cesarean section in present-day obstetrics. Presidential address. *Am J Obstet Gynecol*. 1976;126(5):521-530.
76. Antimicrobial prophylaxis for surgery. *Treat Guidel Med Lett*. 2009;7(82):47-52.
77. Hawkins JL, Koonin LM, Palmer SK, Gibbs CP. Anesthesia-related deaths during obstetric delivery in the United States, 1979-1990. *Anesthesiology*. 1997;86(2):277-284.
78. American Society of Anesthesiologists Task Force on Obstetric Anesthesia. Practice guidelines for obstetric anesthesia: an updated report by the American Society of Anesthesiologists Task Force on Obstetric Anesthesia. *Anesthesiology*. 2007;106(4):843-863.
79. Afolabi BB, Lesi FE. Regional versus general anaesthesia for caesarean section. *Cochrane Database Syst Rev*. 2012;10:CD004350.
80. Horlocker TT, Wedel DJ, Rowlingson JC, Enneking FK. Executive summary: regional anesthesia in the patient receiving antithrombotic or thrombolytic therapy: American Society of Regional Anesthesia and Pain Medicine Evidence-Based Guidelines (Third Edition). *Reg Anesth Pain Med*. 2010;35(1):102-105. Erratum in *Reg Anesth Pain Med*. 2010;35(2):226.
81. Bates SM, Greer IA, Middeldorp S, et al. VTE, thrombophilia, antithrombotic therapy, and pregnancy: Antithrombotic Therapy and Prevention of Thrombosis, 9th ed: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines. *Chest*. 2012;141(2 Suppl):e691S-e736S.
82. Royal College of Obstetricians and Gynaecologists. Reducing the risk of venous thromboembolism during pregnancy and the puerperium. Green-top Guideline No. 37a. 2015. Available at https://www.rcog.org.uk/globalassets/documents/guidelines/gtg-37a.pdf.
83. Kumar K, Singh SI. Neuraxial opioid-induced pruritus: an update. *J Anaesthesiol Clin Pharmacol*. 2013;29(3):303-307.
84. Bujold E, Gauthier RJ. Risk of uterine rupture associated with an interdelivery interval between 18 and 24 months. *Obstet Gynecol*. 2010;115(5):1003-1006.
85. Spong CY, Mercer BM, D'alton M, et al. Timing of indicated late-preterm and early-term birth. *Obstet Gynecol*. 2011;118(2 Pt 1):323-333.
86. Bateman BT, Franklin JM, Bykov K, et al. Persistent opioid use following cesarean delivery: patterns and predictors among opioid-naïve women. *Am J Obstet Gynecol*. 2016;215(3):353.e1-353.e18.
87. ACOG Committee Opinion no. 742: postpartum pain management. *Obstet Gynecol*. 2018;132(1):e35-e43.
88. US Food and Drug Administration. FDA drug safety communication: FDA restricts use of prescription codeine pain and cough medicines and tramadol pain medicines in children; recommends against use in breastfeeding women. 2017. Available at https://www.fda.gov/Drugs/DrugSafety/ucm549679.htm.
89. Minig L, Trimble EL, Sarsotti C, et al. Building the evidence base for postoperative and postpartum advice. *Obstet Gynecol*. 2009;114(4):892-900.
90. Mackeen AD, Packard RE, Ota E, Speer L. Antibiotic regimens for postpartum endometritis. *Cochrane Database Syst Rev*. 2015;2(2):CD001067.

91. Martens MG, Kolrud BL, Faro S, et al. Development of wound infection or separation after cesarean delivery. Prospective evaluation of 2,431 cases. *J Reprod Med*. 1995;40(3):171-175.
92. James A; Committee on Practice Bulletins—Obstetrics. Practice Bulletin no. 123: thromboembolism in pregnancy. *Obstet Gynecol*. 2011;118(3):718-729.
93. Guyatt GH, Akl EA, Crowther M, et al.; American College of Chest Physicians Antithrombotic Therapy and Prevention of Thrombosis Panel. Executive summary: Antithrombotic therapy and prevention of thrombosis, 9th ed: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines. *Chest*. 2012; 141(2 Suppl):7S-47S.
94. Committee on Practice Bulletins—Gynecology, American College of Obstetricians and Gynecologists. ACOG Practice Bulletin no. 84: Prevention of deep vein thrombosis and pulmonary embolism. *Obstet Gynecol*. 2007; 110(2 Pt 1):429-440.
95. ACOG Practice Bulletin no. 156: obesity in pregnancy. *Obstet Gynecol*. 2015;126(6):e112-e126.
96. Sawyer RG, Claridge JA, Nathens AB, et al.; STOP-IT Trial Investigators. Trial of short-course antimicrobial therapy for intraabdominal infection. *N Engl J Med*. 2015;372(21):1996-2005.
97. Garcia J, Aboujaoude R, Apuzzio J, Alvarez JR. Septic pelvic thrombophlebitis: diagnosis and management. *Infect Dis Obstet Gynecol*. 2006;2006:15614.
98. Klima DA, Snyder TE. Postpartum ovarian vein thrombosis. *Obstet Gynecol*. 2008;111(2 Pt 1):431-435.
99. Clark SL, Koonings PP, Phelan JP. Placenta previa/accreta and prior cesarean section. *Obstet Gynecol*. 1985;66(1):89-92.
100. Miller DA, Chollet JA, Goodwin TM. Clinical risk factors for placenta previa-placenta accreta. *Am J Obstet Gynecol*. 1997;177(1):210-214.
101. Silver RM, Landon MB, Rouse DJ, et al.; National Institute of Child Health and Human Development Maternal-Fetal Medicine Units Network. Maternal morbidity associated with multiple repeat cesarean deliveries. *Obstet Gynecol*. 2006;107(6):1226-1232.
102. Ananth CV, Smulian JC, Vintzileos AM. The association of placenta previa with history of cesarean delivery and abortion: a metaanalysis. *Am J Obstet Gynecol*. 1997; 177(5):1071-1078.
103. Grobman WA, Gersnoviez R, Landon MB, et al.; National Institute of Child Health and Human Development (NICHD) Maternal-Fetal Medicine Units (MFMU) Network. Pregnancy outcomes for women with placenta previa in relation to the number of prior cesarean deliveries. *Obstet Gynecol*. 2007;110(6):1249-1255.
104. Tully KP, Ball HL. Misrecognition of need: women's experiences of and explanations for undergoing cesarean delivery. *Soc Sci Med*. 2013;85:103-111.
105. ACOG Committee Opinion no. 745: mode of term singleton breech delivery. *Obstet Gynecol*. 2018;132(2):e60-e63.
106. Kotaska A, Menticoglou S, Gagnon R, et al.; Society of Obstetricians and Gynaecologists of Canada. SOGC clinical practice guideline: Vaginal delivery of breech presentation: no. 226, June 2009. *Int J Gynaecol Obstet*. 2009;107(2):169-176.
107. Hofmeyr GJ, Hannah M, Lawrie TA. Planned caesarean section for term breech delivery. *Cochrane Database Syst Rev*. 2015;7(7):CD000166.
108. American College of Obstetricians and Gynecologists' Committee on Practice Bulletins—Obstetrics. Practice Bulletin no. 161: external cephalic version. *Obstet Gynecol*. 2016;127(2):e54-e61.
109. Hutton EK, Hannah ME, Ross SJ, et al.; Early ECV2 Trial Collaborative Group. The Early External Cephalic Version (ECV) 2 Trial: an international multicentre randomised controlled trial of timing of ECV for breech pregnancies. *BJOG*. 2011;118(5):564-577.
110. Leeman LM. Prenatal counseling regarding cesarean delivery. *Obstet Gynecol Clin North Am*. 2008;35(3): 473-495, ix.
111. Addiss DG, Shaffer N, Fowler BS, Tauxe RV. The epidemiology of appendicitis and appendectomy in the United States. *Am J Epidemiol*. 1990;132(5):910-925.
112. American College of Obstetricians and Gynecologists' Committee on Practice Bulletins—Obstetrics. Practice Bulletin no. 173: fetal macrosomia. *Obstet Gynecol*. 2016;128(5):e195-e209.
113. Chauhan SP, Grobman WA, Gherman RA, et al. Suspicion and treatment of the macrosomic fetus: a review. *Am J Obstet Gynecol*. 2005;193(2):332-346.
114. Rouse DJ, Owen J, Goldenberg RL, Cliver SP. The effectiveness and costs of elective cesarean delivery for fetal macrosomia diagnosed by ultrasound. *JAMA*. 1996; 276(18):1480-1486.
115. Boulvain M, Stan C, Irion O. Elective delivery in diabetic pregnant women. *Cochrane Database Syst Rev*. 2001; (2):CD001997.
116. Boulvain M, Irion O, Dowswell T, Thornton JG. Induction of labour at or near term for suspected fetal macrosomia. *Cochrane Database Syst Rev*. 2016;5(5): CD000938.
117. ACOG practice bulletin. Vaginal birth after previous cesarean delivery. Number 5, July 1999 (replaces practice bulletin number 2, October 1998). Clinical management guidelines for obstetrician-gynecologists. American College of Obstetricians and Gynecologists. *Int J Gynaecol Obstet*. 1999;66(2):197-204.
118. Smith J, Plaat F, Fisk NM. The natural caesarean: a woman-centred technique. *BJOG*. 2008;115(8):1037-1042, discussion 1042.
119. Magee SR, Battle C, Morton J, Nothnagle M. Promotion of family-centered birth with gentle cesarean delivery. *J Am Board Fam Med*. 2014;27(5):690-693.
120. Gordon A, McKechnie EJ, Jeffery H. Pediatric presence at cesarean section: justified or not? *Am J Obstet Gynecol*. 2005;193(3 Pt 1):599-605.
121. McDonald SJ, Middleton P, Dowswell T, Morris PS. Effect of timing of umbilical cord clamping of term infants on maternal and neonatal outcomes. *Cochrane Database Syst Rev*. 2013;7(7):CD004074.
122. Moore ER, Bergman N, Anderson GC, Medley N. Early skin-to-skin contact for mothers and their healthy newborn infants. *Cochrane Database Syst Rev*. 2016;11: CD003519.

123. Katz VL, Dotters DJ, Droegemueller W. Perimortem cesarean delivery. *Obstet Gynecol*. 1986;68(4):571-576.

124. Rose CH, Faksh A, Traynor KD, et al. Challenging the 4- to 5-minute rule: from perimortem cesarean to resuscitative hysterotomy. *Am J Obstet Gynecol*. 2015;213(5): 653-656, 653.e1

125. Jeejeebhoy FM, Zelop CM, Lipman S, et al.; American Heart Association Emergency Cardiovascular Care Committee, Council on Cardiopulmonary, Critical Care, Perioperative and Resuscitation, Council on Cardiovascular Diseases in the Young, and Council on Clinical Cardiology. Cardiac Arrest in Pregnancy: A Scientific Statement From the American Heart Association. *Circulation*. 2015;132(18):1747-1773.

126. Benson MD, Padovano A, Bourjeily G, Zhou Y. Maternal collapse: challenging the four-minute rule. *EBioMedicine*. 2016;6:253-257.

127. Kalok A, Zabil SA, Jamil MA, et al. Antenatal scoring system in predicting the success of planned vaginal birth following one previous caesarean section. *J Obstet Gynaecol*. 2018;38(3):339-343.

128. Dresang LT, Leeman L. Cesarean delivery. *Prim Care*. 2012;39(1):145-165.

129. Guise JM, McDonagh MS, Hashima J, et al. Vaginal Birth After Cesarean (VBAC): Summary. In: AHRQ Evidence Report Summaries. Rockville (MD): Agency for Healthcare Research and Quality (US);2003:71.

130. Committee on Practice Bulletins-Obstetrics. Practice Bulletin no. 184: vaginal birth after cesarean delivery. *Obstet Gynecol*. 2017;130(5):e217-e233.

131. Spong CY, Landon MB, Gilbert S, et al.; National Institute of Child Health and Human Development (NICHD) Maternal-Fetal Medicine Units (MFMU) Network. Risk of uterine rupture and adverse perinatal outcome at term after cesarean delivery. *Obstet Gynecol*. 2007;110(4): 801-807.

132. King VJ, Fontaine PL, Atwood LA, et al. Clinical practice guideline executive summary: labor after cesarean/planned vaginal birth after cesarean. *Ann Fam Med*. 2015;13(1):80-81.

133. Varner MW, Thom E, Spong CY, et al.; National Institute of Child Health and Human Development (NICHD) Maternal-Fetal Medicine Units Network (MFMU). Trial of labor after one previous cesarean delivery for multifetal gestation. *Obstet Gynecol*. 2007;110(4):814-819.

134. Costantine MM, Fox KA, Pacheco LD, et al. Does information available at delivery improve the accuracy of predicting vaginal birth after cesarean? Validation of the published models in an independent patient cohort. *Am J Perinatol*. 2011;28(4):293-298.

135. Dresang LT, Hampton A. Vaginal birth after cesarean (VBAC) calculator risks. *Birth*. 2015;42(4):379-380.

136. Deline J, Varnes-Epstein L, Dresang LT, et al. Low primary cesarean rate and high VBAC rate with good outcomes in an Amish birthing center. *Ann Fam Med*. 2012; 10(6):530-537.

137. Dodd JM, Crowther CA, Huertas E, et al. Planned elective repeat caesarean section versus planned vaginal birth for women with a previous caesarean birth. *Cochrane Database Syst Rev*. 2013;12(12):CD004224.

138. Halperin ME, Moore DC, Hannah WJ. Classical versus low-segment transverse incision for preterm caesarean section: maternal complications and outcome of subsequent pregnancies. *Br J Obstet Gynaecol*. 1988;95(10): 990-996.

139. Landon MB, Hauth JC, Leveno KJ, et al.; National Institute of Child Health and Human Development Maternal-Fetal Medicine Units Network. Maternal and perinatal outcomes associated with a trial of labor after prior cesarean delivery. *N Engl J Med*. 2004;351(25): 2581-2589.

140. Smith GC, Pell JP, Cameron AD, Dobbie R. Risk of perinatal death associated with labor after previous cesarean delivery in uncomplicated term pregnancies. *JAMA*. 2002;287(20):2684-2690.

141. Ridgeway JJ, Weyrich DL, Benedetti TJ. Fetal heart rate changes associated with uterine rupture. *Obstet Gynecol*. 2004;103(3):506-512.

142. Leung AS, Leung EK, Paul RH. Uterine rupture after previous cesarean delivery: maternal and fetal consequences. *Am J Obstet Gynecol*. 1993;169(4):945-950.

143. Landon MB, Spong CY, Thom E, et al.; National Institute of Child Health and Human Development Maternal-Fetal Medicine Units Network. Risk of uterine rupture with a trial of labor in women with multiple and single prior cesarean delivery. *Obstet Gynecol*. 2006;108(1):12-20.

144. Tahseen S, Griffiths M. Vaginal birth after two caesarean sections (VBAC-2)-a systematic review with meta-analysis of success rate and adverse outcomes of VBAC-2 versus VBAC-1 and repeat (third) caesarean sections. *BJOG*. 2010;117(1):5-19.

145. Guise JM, Eden K, Emeis C, et al. Vaginal birth after cesarean: new insights. *Evid Rep Technol Assess (Full Rep)*. 2010;(191):1-397.

146. Clock C, Kurtzman J, White J, Chung JH. Cesarean risk after successful external cephalic version: a matched, retrospective analysis. *J Perinatol*. 2009;29(2):96-100.

147. Leung AS, Farmer RM, Leung EK, et al. Risk factors associated with uterine rupture during trial of labor after cesarean delivery: a case-control study. *Am J Obstet Gynecol*. 1993;168(5):1358-1363.

148. American Academy of Family Physicians. Labor after cesarean/planned vaginal birth after cesarean. 2014. Available at https://www.aafp.org/dam/AAFP/documents/patient_care/clinical_recommendations/AAFP%20PVBAC%20guideline.pdf.

149. United Nations Children's Fund; World Health Organization. United Nations Population Fund. Guidelines for monitoring the availability and use of obstetric services. 1997. Available at https://www.unicef.org/health/files/guidelinesformonitoringavailabilityofemoc.pdf.

150. Shah A, Fawole B, M'imunya JM, et al. Cesarean delivery outcomes from the WHO global survey on maternal and perinatal health in Africa. *Int J Gynaecol Obstet*. 2009;107(3):191-197.

151. Villar J, Valladares E, Wojdyla D, et al. WHO 2005 global survey on maternal and perinatal health research group. Caesarean delivery rates and pregnancy outcomes: the 2005 WHO global survey on maternal and perinatal health in Latin America. *Lancet*. 2006;367(9525): 1819-1829.

Birth Crisis

Learning Objectives

1. Identify types of birth crises encountered in practice.
2. Describe the varying emotional responses to birth crises.
3. Describe an approach to handling a birth crisis utilizing the expanded Four Cs mnemonic.
4. Identify support groups and agencies to assist families with a birth crisis.
5. Identify resources for health professionals after a birth crisis.

Background

Birth crisis includes, but is not limited to, any unexpected adverse outcome of pregnancy including:

Fetal or neonatal circumstances

- Spontaneous pregnancy loss in first or second trimesters
- Stillbirth
- Neonatal death (death of the infant within the first 4 weeks of life)
- Diagnosis of abnormality compatible with life
- Diagnosis of abnormality incompatible with life
- Elective termination after diagnosis of abnormality incompatible with life

Maternal circumstances

- Critical or severe illness
- Emergency hysterectomy
- Maternal death during the peripartum period.

A *near miss* of any of these situations can create similar levels of emotion, and a *near loss* can have similar effects as a loss/death on women, families, and the health care team.

When women and their partners experience a birth crisis, the effects are often profound, causing long-lasting emotional trauma to the parent(s) and to family members.[1] Management of these crises by the health care team is important because parents may recall, even years after the event, the words spoken by and interactions with members of the team.[2]

Birth crises also affect the individuals who provide care to these women and their families. Maternity care providers can experience significant emotional trauma.[3,4] Although the depth and level of loss experienced by the provider may not be as profound as that experienced by the family, it can still have a significant effect on the provider, especially if multiple losses are experienced over time.

In caring for women and families who experience a birth crisis, maternity care providers must attempt to provide mindful, compassionate, and empathetic care. Accusations and blame are not helpful and may prevent or delay a positive resolution to the process of grieving and loss. Compassionate, humanistic psychosocial management of adverse birth outcomes is imperative for all maternity care providers.[5]

The ability of the health care team to provide care in this way may be hindered by the clinical detachment often required in the practice setting and the absence of emotional support from colleagues.[3,6] It is not unusual for members of the health care team to experience a stillbirth and a normal birth in the same day with no time to process the grief and loss and related feelings.

This chapter will address the needs of the parents and family after a birth crisis and recommend ways in which the health care team can support parents and provide effective care. Provider resources will also be offered and suggestions on how to better support each other during and after a birth crisis will be discussed.

Emotional Support in Birth Crisis

Perinatal bereavement necessitates support of the parents and family. However, the needs of the practitioners should not be overlooked. Despite the constancy of death and the inevitability of distress when a birth crisis occurs, members of the medical team are often uncertain about or inadequately prepared for how to proceed. Oftentimes, team members have anxiety about saying something that will cause further discomfort for the parents. Some team members can develop a routine that serves them well; however, others can struggle with or become overwhelmed by the enormity of parental grief and loss.

There is no universal approach to these situations. Maternity care providers should acknowledge this and attempt to empathize with parental grief to humanize the situation.[6] A Cochrane review attempted to determine the specific psychological support or counseling needs of parents and families after perinatal death, but the results were inconclusive.[7] The needs depend on the circumstances, context, and expressed needs of the family. Even after counseling, many families report receiving inadequate emotional support from their maternity care provider.[8] A Royal College of Obstetrics and Gynaecologists (RCOG) guideline on stillbirth and late intrauterine fetal death (IUFD) states that many strategies have been described for discussing bad news, and that a crucial component is determining the emotional feelings and needs of the woman and her support team.[9] The empathetic approach attempts to identify a woman's thoughts and wishes without trying to shape them.[10,11]

Women and their partners generally want support from their care providers. In a study about neonatal death, women felt sad when they perceived that they received insufficient support from professionals, particularly related to the time they could spend with their infant. They were also disappointed when those providing care neither acknowledged nor validated their motherhood. These women also felt hurt when maternity care providers lacked respect or when they felt abandoned by those who were meant to be providing care. These feelings turned to anger when they were treated with indifference or providers were callous toward their loss.[12]

Understanding how parents process and address grief can be useful for provideers. A model has been developed that helps describe the adaptation of parents to the birth of an infant with a congenital malformation. The feelings reported by these parents were identified by stages: shock; denial; sadness, anger, and anxiety; adaptation; and reorganization.[13] Most parents will experience these feelings at various times after a birth crisis, but the time to navigate the complex process of mourning will differ in each situation.[14] Many of the elements of this model are useful in perinatal death, as well.

Parents can have discordant grieving, where each parent's grief is equal but different. This can create tension and disruption in relationships after a birth crisis. Understanding discordant grieving is critically important, because the ability for the woman to talk to her partner about the stillborn infant in the postpartum period has been shown to reduce the risk of maternal depression.[15] Maternity care providers should attempt to recognize discordant grieving and, where possible, facilitate open and honest discussions with and between all those involved while remaining sensitive to all variations of the human condition that relate with loss within a couple.[16]

If the parents have other children, it can be difficult for them to know how to help their children cope with the death of a sibling. The parents may try to maintain a balance between mourning the loss of their child and maintaining the normalcy of everyday life for the sibling(s).[17] The profound parental grief can cause unintended neglect of the other children and failure to explain and comfort adequately, which can cause feelings of guilt in the older child(ren).[18] From an early age children can sense a change in the family dynamic, which may cause confusion and feelings of insecurity.[19] The grief a sibling experiences may be as intense as that of the parents, and yet most parents are unprepared and without resources to help them.[20] Unresolved grief can last for many years.[20]

Other family members, including grandparents, may also experience grief and mourn a perinatal death.[19] Grandparents may mourn the loss of their grandchild and also feel sadness and helplessness about their child's grief.[21] Grandparents may have different ideas than their adult children about how to process the grief, and may think that talking to children about death is harmful. Parents and their relatives may not know how to help each other, resulting in a disruption of relationships.[22]

Results of a survey showed that intergenerational perinatal bereavement programs can benefit all family members, including siblings and grandparents, to understand the mourning process and cope better with perinatal death.[19] The guidance and support offered by a professional team was positively received by grandparents.

The *Safety in Maternity Care* chapter describes the Five Cs mnemonic: **c**ompassion, **c**ommunication, **c**ompetence, **c**onfession, and **c**harting that are used when an adverse outcome or a medical error occurs.

Building on this approach, a provider's actions and care of the woman and her family after a birth crisis could be based on an expanded Four Cs mnemonic:

Coming Together. Ensuring the right time, place, and environment

Communication and Consideration. What is the goal of this encounter? What do I know, what can I do to help, how will I do it? What will I say and how best to say it? Maintain a mindful, empathetic, and caring manner and approach

Contact. We should not be afraid of making emotional or physical contact

Consultation. Do we need to discuss issues or seek guidance from others for advice and support? Do the parents and family need additional information, counseling, or support from internal or external agencies? Do we need similar support for ourselves?

Guidelines for initial management of a birth crisis are outlined in *Table 1*.[23]

An institutional standardized check list should be used to ensure all aspects of care are consistently and appropriately managed, including mementoes for a memory box, photographs taken, death certificates completed, funeral arrangements prepared (when appropriate), and appropriate testing obtained to attempt to establish the cause of the perinatal loss.[24]

Fetal Death, Stillbirth, and Neonatal Loss

Definitions

Fetal death. Fetal death is the spontaneous intrauterine death of a fetus of any gestational age.[25] In the United States, the National Center for Health Statistics reports fetal death data annually.[26] The 2013 US fetal death rate was 5.96 per 1,000 live births.[27] The fetal death rate for non-Hispanic black women (10.53) was more than twice that for non-Hispanic white women (4.88).[27]

Stillbirth. In the United States, the definition of stillbirth varies by state. These definitions are available from the Centers for Disease Control and Prevention (CDC) at www.cdc.gov/nchs/products/other/miscpub/statereq.htm. Most states define stillbirth as fetal death at 20 weeks' gestation or more or 350 g or more if the gestational age is not known.[28,29] Patient surveys in the United States find that parents prefer the term *stillbirth* over *fetal death*.[29]

Changing the denominator for calculating stillbirth is under consideration. To date, stillbirths are calculated as the number of stillbirths per 1,000 live births and stillbirths. If the denominator was changed to the number of women who

Table 1. Guidelines for Initial Management of Adverse Pregnancy or Birth Outcome

Meet with the woman and her partner as soon as possible
Share the information with both parents together, if possible
Express feelings for the parent's loss (eg, "I am sorry for the loss of your baby")
Remember that saying, "I am sorry this happened," in the case of neonatal asphyxia or birth trauma is not considered a confession of guilt
Involve family members, as appropriate, for psychosocial support and information sharing
Sit at eye level
Do not be afraid of physical contact if accepted by the parent
Do not be afraid to exhibit some of the emotion you are experiencing
Avoid using medical jargon
Enable and support parents to express their feelings
Recognize that guilt and self-blame are common
Review the facts but acknowledge their limits (eg, do not be afraid to say, "I don't know why")
Avoid assigning blame and or premature diagnostic labels
Recognize that most parents must attach before letting go and that parental grief may be equal but expressed differently (maternal reactions are based on degree of prenatal attachment whereas paternal reactions are based on connectedness to pregnancy, a sense of fatherhood, and image of the infant)
Encourage parents to see and hold the infant. If the infant is severely deformed, holding it wrapped completely in a blanket and exposing a foot or hand may be sufficient
Talk about and value the normal aspects of the infant
Allow the parent to take their time with this process
Offer mementos such as footprints, hair, or photographs. (If parents decline these initially, they may request them later. A safe storage process is necessary). It is important for photographs to be flattering; black and white photographs often work well. Photographs of the infant's hands and feet or of the parents holding the infant wrapped are often treasured by the family
Plan the timing of follow up meetings and enable family members to attend if the parents desire
Undertake ongoing assessments of the family's needs
Attend to the emotional concerns of the patient and family
If the parents have other children, involve social workers/counselors specialized in grief in children to help parents support these siblings
Provide resources and guidance for funeral arrangements/memorial service plans
Monitor maternal physical, social, and emotional health and make a referral, if necessary
Address financial issues and refer to social services, if necessary
Anticipate anniversary grief and explain to families that it is likely to occur

Information from Van Dinter MC, Graves L. Managing adverse birth outcomes: helping parents and families cope. Am Fam Physician. *2012;85(9):900-904.*

remain pregnant (the population at risk of stillbirth), stillbirth rates would allow calculation of a prospective risk of stillbirth at a given gestational age. Stillbirths would be calculated as the number of stillbirths per 1,000 live births and stillbirths at that gestational age or greater.[29]

The CDC subcategorizes still birth into:
Early stillbirth – 20-27 weeks' gestation
Late stillbirth – 28-36 weeks' gestation
Term stillbirth – 37 weeks' gestation or more.[30]

The World Health Organization (WHO) defines stillbirth as fetal death after 22 weeks' gestation. Legal definitions of stillbirth vary among countries.[31] In the United Kingdom, the accepted gestational age is 24 completed weeks' gestation.[32] In Australia, a stillborn is defined as an infant weighing more than 400 g (approximately 0.88 lb) or, if weight is unknown, more than 20 completed weeks' gestation. Birth must be registered for these infants.[33]

The lack of a clear definition and accurate global recording of stillbirth complicates and compromises data collection and research into the etiologies of and, thus, prevention of stillbirth.[34] One paper from a 2016 series on stillbirth highlighted the importance of a universal classification system of stillbirth. Recommendations were made for improved routine data collection for stillbirth and the need for antepartum and intrapartum stillbirths to be core indicators in national data sets.[35]

Neonatal loss. Neonatal loss is defined as the death of an infant after live birth in the first 28 days (4 weeks) of life.[36] The 2017 US neonatal mortality rate was 3.84 deaths per 1,000 live births.[37] The emotional responses of the family and provider to neonatal death occurring soon after birth as well as the causes of the death and medical evaluation have many similarities. Early neonatal death (within the first 7 days after birth) may reflect an unanticipated outcome of the birth process (eg, neonatal encephalopathy), may reflect postnatal events (eg, sepsis), or may occur secondary to lethal anomalies that may or may not have been known prior to death. Infant death includes a death after live birth in the first year of life. The 2017 US infant mortality rate was 5.79 deaths per 1,000 live births.[37]

Prevention

Understanding risk factors for stillbirth and which are modifiable is important in stillbirth prevention efforts. Maternity care providers should remain vigilant in diagnosing and addressing modifiable causal factors in women such as obesity; diabetes; hypertension and thyroid disease management; and alcohol, tobacco, methamphetamine, cocaine, and other illicit substance use disorders. Obesity is the leading modifiable cause of stillbirth. The American College Obstetrics and Gynecology (ACOG) recommends a hemoglobin A1c of less than 7 for women with diabetes who are planning pregnancy. Smoking and secondhand tobacco increase the risk of stillbirth. Other risk factors for stillbirth include: non-Hispanic black race, nulliparity, grand multiparity, maternal age less than 15 years or more than 35 years, having a pregnancy using assisted reproductive technology, multiple gestation, male fetal sex, unmarried status, lupus, renal disease, cholestasis of pregnancy, antiphospholipid antibody syndrome, and history of stillbirth.[29] Chromosomal analysis after a stillbirth may identify genetic causes of stillbirth which could repeat in a future pregnancy. Between 6 and 13% of stillbirths have an abnormal karyotype.[29] The most common chromosomal abnormalities found in stillbirth are trisomy 21 (31%), monosomy X (22%), trisomy 18 (22%), trisomy 13 (6%), and other chromosomal abnormalities (19%).[38]

Rates of stillbirth rise the further women surpass their due date. Compared with the risk of stillbirth at 37 weeks' gestation, the relative risk of stillbirth is 2.9 (95% CI = 2.6-3.2) at 41 weeks' gestation and 5.1 (95% CI = 4.4-6.0) at 42 weeks' gestation.[39] ACOG recommends induction by 42 weeks' gestation.[29]

Intrauterine growth restriction (IUGR) increases the risk of stillbirth, and is an indication for antenatal testing and early delivery when detected. IUGR is defined as an estimated fetal weight (EFW) less than 10% for a given gestational age. Those with an EFW less than 2.5% are at greatest risk of stillbirth.[40] Careful prenatal monitoring and use of ultrasound to identify IUGR may better identify a fetus at risk.[41,42] Antenatal testing including serial umbilical artery Doppler measurements improve fetal outcomes. Induction is recommended at 38 to 39 6/7 weeks' gestation with isolated IUGR and may be indicated earlier with additional risk factors such as oligohydramnios or abnormal antenatal testing.[40]

Placental abruption is an often unavoidable and unpredictable cause of stillbirth. Tobacco, cocaine,

and methamphetamine use are modifiable risk factors for stillbirth due to abruption.[29] Blood pressure control may help prevent abruption.

Infection is identified in 10% to 20% of stillbirths. Bacteria associated with stillbirth include group B Streptococcus, *Escherichia coli*, Listeria monocytogenes, and syphilis. Viruses which can cause stillbirth include cytomegalovirus, parvovirus, and Zika virus. Umbilical cord conditions which can cause stillbirth include vasa previa, cord entrapment, and evidence of occlusion and fetal hypoxia, prolapse, or stricture with thrombi.[43] Nuchal cord is not considered a cause of stillbirth as 24% of deliveries have a nuchal cord and 4% have multiple nuchal cords. Most true knots of the umbilical cord are found after live births.[29]

Evidence-Based Management

All maternity care providers are likely to encounter an unexpected adverse birth outcome during their careers. Diagnosis of IUFD may follow a maternal concern about decreased fetal movement, or it may occur unexpectedly when the fetal heartbeat is not detected during a routine examination. In countries with high- and middle-economies, ultrasound will typically be conducted to confirm the finding.

For women and families, the birth of a stillborn infant is a life-changing event. Research has shown that stillbirth can have devastating psychological, physical, and social costs, with ongoing effects on interpersonal relationships and subsequent born children.[44] However, parents who experience a stillbirth can develop resilience and new life skills and capacities,[44] and the care they receive around the time of the stillbirth and later can make a significant difference.[45]

A systematic review found that the type of care provided to women and their families around and after the time of diagnosis of a stillbirth affected the parents and providers.[46] In particular, the behaviors and actions of staff were shown to have a long-lasting effect on parents. In this review, parents reported distress caused by maternity care providers being afraid to closely interact with parents and avoiding interaction by busying themselves with activities and routine guidelines for care. Health care staff described using routine tasks and distancing as methods to cope with the stress of the situation. Important evidence-based strategies for institutions include ongoing education and training for staff within supportive systems and structures as well as continuity of care for families.[46] Culturally appropriate care is imperative, and staff and maternity care providers should always seek advice from the family about their cultural needs and desires.[45]

When a maternity care provider suspects an IUFD, it is not acceptable for the woman and partner to be left with uncertainty or a long wait until the diagnosis is made. Depending on their level of expertise with obstetric ultrasound, the provider performing an ultrasound that finds a lack of fetal heart motion may want to have a second examiner confirm the diagnosis. The woman should be informed in a private space, preferably with her partner or support person. She and her family will need time to come to terms with the news. Several meetings may be needed for the parents to understand what has happened.[24,47] The provider must be aware of the amount of information being conveyed and the amount being absorbed, and then be willing to repeat information several times to ensure full understanding. Adequate time must be allotted for the meetings to avoid being rushed and avoid the frequent perception of lack of support.[29]

When an IUFD occurs, labor induction will usually be offered in the next few days. In the absence of acute maternal illness there is no urgency and offering the option of induction or expectant management is reasonable and may be beneficial psychologically[48] by allowing the family time to process what has happened. If no medical contraindications exist, expectant management is a viable option for women and families.[49]

Dilation and evacuation (D&E) may be a preferred option for second-trimester IUFD if a physician is available who is skilled in the procedure.[49,50] Coagulopathy due to prolonged retention of a dead fetus is rare and not an indication for cesarean or expedited delivery.[29] Compared with D&E, induction of labor has more maternal morbidity (mainly infection requiring intravenous antibiotics), is less effective and leads to more complications in the setting of fetal demise between 14 and 24 weeks' gestation.[29] In the third trimester, induction of labor using local institutional guidelines is appropriate.[49] Cervical ripening may be required. Women need one-to-one care and support in labor to assist them with managing the pain of labor and the trauma associated with the impending delivery of a stillborn infant. Adequate analgesia/anesthesia

must be available, and the woman's wishes about the labor and delivery must be accommodated and respected. Providing continuity of care during this time is important.

Many women will experience anxiety about laboring and delivering a stillborn infant, and some may request a cesarean delivery. Avoiding cesarean delivery and the well-documented potential for maternal morbidity without fetal benefit is encouraged except in certain circumstances, such as having a prior vertical uterine incision[49,51] or more than two prior cesarean deliveries. Women at less than 28 weeks' gestation with one or two prior cesarean deliveries may be administered prostaglandins for cervical ripening and induction after careful counseling about the maternal risk.[49] Women with prior cesarean delivery and third-trimester fetal demise are best managed with a Foley catheter for cervical ripening followed by oxytocin, although misoprostol may be used with caution after counseling regarding uterine rupture.[52,53] Results from a study of delivery type in women with stillbirth found that although most delivered vaginally, there was a cesarean delivery rate of 15%.[29] In many of these cesarean deliveries, there was no documented obstetric indication.[51]

A neonatal death due to birth asphyxia or unanticipated neonatal illness will likely be preceded by urgent and emergency care for the infant. The parent(s) may witness a resuscitation attempt or be excluded when the infant is taken away for treatment. In the absence of a prenatal diagnosis of abnormality, this death will often be unexpected and, as in the case of stillbirth, compassion, adequate time with the infant, and understanding are the key components of immediate care.

Communication with the entire health care team including midwives, nurses, pastoral care, physicians (including residents, if involved), social workers, and neonatal intensive care unit (NICU) staff is important so that consistent and accurate information is provided to the family. A standardized protocol to provide consistent care is beneficial, especially in settings where stillbirth rarely is managed.[54] Using a standardized system to subtly mark the doors of women's rooms will help the health care team and ancillary staff be aware of the need for sensitivity when entering the room.[45]

Having adequate time to hold and spend with the infant has been shown to be beneficial in emotional recovery after a stillbirth after 37 weeks' gestation. The benefit in holding a stillborn infant between 28 and 37 weeks' gestation is uncertain and requires more study, although experience shows most parents benefit from this if it is undertaken sensitively and supportively.[55] One systematic review of 23 studies found positive outcomes for parents who saw or held their infant.[56] That review found that increased psychological morbidity was associated with choosing not to see the infant, having insufficient time with the infant, and/or having insufficient mementos. The importance of the role of maternity care providers in facilitating access and supporting families during this period was noted.

When families no longer are receiving care from the maternity care provider, it is important to recognize that the grief and loss will continue, in different ways for different families. Good evidence shows that stillbirth has life-long psychological and economic effects on families. A review showed that stillbirth was associated with substantial direct, indirect, and intangible costs for women, their partners, their families, their maternity care providers, the government, and the wider society.[1] Other studies have shown that this is also true for families who experience neonatal deaths and is likely to affect subsequent pregnancies.[57] This is important for providers to acknowledge to ensure women and families receive appropriate community-based support and referrals.

No internationally recognized, evidence-based guidelines exist for the evaluation for the cause of stillbirth. However, several professional organizations have positions on evaluation (*Table 2*).

The 2017 study conclusion by the Stillbirth Collaborative Research Network states, "The most useful tests were placental pathology and fetal autopsy followed by genetic testing and testing for antiphospholipid antibodies."[58] Perinatal autopsy is underused in many countries, including the United States. In Utah, for example, rates of 35% were found in tertiary centers and only 13.3% in community hospitals.[59] Reasons for the global underuse of perinatal autopsy may include lack of funding in low-resource countries, few pathologists with experience and expertise, lack of remuneration, and parental misunderstanding of the potential value.[59]

A study identified the need to refine postmortem examinations for stillborn infants in which the authors concluded, based on objective criteria, 30% to 60% of IUFDs remain unexplained.[60] A survey

found that emotional, practical, and psychosocial issues related to the consent process for perinatal postmortem examination can present real or perceived barriers for staff and parents. Providers need education and support to increase their knowledge to ensure accurate counseling and regard for the highly individual responses of parents.[61]

In 2020, the American College of Obstetrics and Gynecology (ACOG) and the Society of Maternal-Fetal Medicine (SMFM) published a consensus statement on the management of stillbirth.[29] The investigations recommended were amniocentesis; fetal autopsy; gross and histologic examination of the placenta, cord, and membranes; and genetic testing.[29]

If not performed earlier in pregnancy, amniocentesis is recommended because of its higher success rate in culturing cells than if performed after delivery. In one study, cells culture was 85% successful when amniocentesis was performed before delivery compared with 28% when samples were obtained after delivery.[38] The placental sample should be a 1-cm by 1-cm block below cord insertion site. The umbilical cord segment should be 1.5 cm. Internal fetal tissue can be obtained from the costochondral junction or patella but should not be skin. Collected samples should be placed in lactated Ringer's solution at room temperature and not in formalin.

Physical examination at time of stillbirth should note any anomalies and document, weight, height, and head circumference. If a woman declines an autopsy, options include external examination by a perinatal pathologist, imaging (x-ray, ultrasound and/or magnetic resonance imaging), and genetic analysis of the placenta, umbilical cord, and internal fetal tissue.

Laboratory testing should be individualized based on clinical history and guided by 2020 ACOG and Society of Obstetricians and Gynaecologists of Canada (SOGC) guidelines.[29,62] A complete blood count, blood type and antibody screen, hemoglobin A1c, Kleihauer-Betke test, and an antiphospholipid antibody syndrome panel (lupus anticoagulant, anticardiolipin [immunoglobulin M, immunoglobulin G], anti-Beta 2 glycoprotein 1 [immunoglobulin M, immunoglobulin G]) are appropriate for most cases of IUFD. Tests may include immunoglobulin M and immunoglobulin G testing for TORCH (**t**oxoplasmosis; **o**ther [syphilis, varicella-zoster, parvovirus B19], **r**ubella, **c**ytomegalovirus, **h**erpes) infection.

Other tests should be used selectively such as anti-Ro/LA (SSA-SSB) if there is evidence of fetal hydrops, endomyocardial fibroelastosis, or atrioventricular node calcification on autopsy, and hemoglobin electrophoresis, urine toxicology (eg, cocaine, amphetamine, other illicit drugs), and bile acids/liver enzymes if maternal symptoms of cholestasis are present.[62] Testing for inherited thrombophilias is not recommended as they are not associated with increased risk of stillbirth. Microarray analysis is preferable to traditional karyotyping. Specific genetic testing may be obtained based on family history and/or physical examination findings. Results should be shared with family and involved clinicians in a timely manner.[29,62]

The 2010 RCOG Late Intrauterine Death and Stillbirth Guideline provides recommendations for evaluation based on specific clinical scenarios.[9] The RCOG has also endorsed the use of bereavement midwives who are trained in the care of grieving parents and are available in all health districts. Additional studies need to be undertaken to assess and potentially develop this role.

In 2009, the Perinatal Society of Australia and New Zealand (PSANZ) developed a clinical guideline for the evaluation of potential causes of stillbirth; the guideline was updated in 2019.[63,64]

Table 2. Evaluation of Stillbirth

Fetal Studies	Maternal Studies
Amniotic fluid analysis	Prenatal genetic evaluation (amniocentesis at time of diagnosis of abnormalities or stillbirth)
Autopsy (with parental consent)	Thrombophilia evaluation
Photographs of fetus	Placental examination
Fetal karyotype analysis (cord blood, placenta and cord or fetal tissue)	
Internal fetal tissue specimen	
Placental block 1 cm by 1 cm taken from below cord insertion site	
Umbilical cord segment 1.5 cm	
Internal tissue specimen, such as costochondral junction or patella	
Specimens to be placed in a sterile tissue culture medium of lactated Ringer's solution kept at room temperature	

Information from Van Dinter MC, Graves L. Managing adverse birth outcomes: helping parents and families cope. Am Fam Physician. *2012;85(9):900-904; American College of Obstetricians and Gynecologists, Society for Maternal-Fetal Medicine. Management of Stillbirth: Obstetric Care Consensus No, 10.* Obstet Gynecol. *2020;135(3):e110-e132.*

In 2020, the SOGC updated its guideline on the investigation of stillbirth.[62] Maternity care providers should be familiar with their own national guidelines. The ACOG, RCOG, SOGC, and PSANZ guidelines remain current as of August 2020.

The provider should discuss options for evaluation with the parent(s) based on the guidelines recommended in their country and allow the parent(s) to decide how they want to proceed.

In addition to the studies described above, take photographs of the infant. Photographs should include whole body, frontal and profile views of face, extremities, and palms, and of specific abnormalities. It is important to document all findings, particularly abnormalities. The family may eventually read the descriptions, so documentation should be sensitively and respectfully worded.

Care of Women in Subsequent Pregnancy

Limited evidence exists to guide the care of women after an unexplained stillbirth. In women at low-risk of stillbirth after an unexplained stillbirth, the risk in a subsequent pregnancy after 20 weeks' gestation was found to be from 9 to 20 in 1,000 live births and stillbirths.[29] A systematic review of 16 studies found that the risk of stillbirth in subsequent pregnancies was higher in women who experienced a stillbirth in their first pregnancy, and this increased risk remained after adjusted analysis.[65]

The risks of early induction of labor for a woman with an uncomplicated subsequent pregnancy after a prior unexplained stillbirth before 39 weeks' gestation must be weighed against the real risks of an early or late preterm birth.[66] Although it has been a common practice to induce labor prior to 39 weeks' gestation with such a history, this is no longer recommended based on a 2011 workshop sponsored by National Institute of Child Health and Human Development and SMFM.[67]

It is reasonable to consider serial growth ultrasounds beginning at 28 weeks' gestation if there is lag in fundal height measurement or other concern about fetal growth, because IUGR is associated with a stillbirth risk of 6.7 per 1,000 live births.[68] ACOG recommends beginning fetal surveillance at 32 weeks' gestation or 1 to 2 weeks earlier than a prior stillbirth.[29] There is an estimated 1.5% rate of iatrogenic prematurity for intervention based on a false positive result during antenatal surveillance.[69]

Amniocentesis to assess fetal lung maturity to permit delivery before 39 weeks' gestation is not recommended because there are other organ systems to consider before initiating such an induction. However, if a provider and the woman choose delivery at or before 39 weeks' gestation, it may be considered.[67,68]

There is no good evidence to show that maternal monitoring of fetal kick counts prevents stillbirth.[70] More frequent antenatal visits, although not necessarily preventing recurrence, may offer reassurance. Listening to concerns and helping women manage their anxiety is important, especially toward the end of pregnancy. Again, continuity of care provided is likely to be beneficial.

Management of Second-Trimester Perinatal Death

Second-trimester perinatal death requires the same emotional care and support discussed in the previous section. Clinical management after diagnosis includes offering the option of labor induction or D&E.[29] Usually this will depend on maternal preference and the availability of a provider skilled in second-trimester D&E. Maternal grief resolution is not affected by the choice of termination if the woman self-selects the procedure.[50] In the United States, most second-trimester pregnancy terminations are completed by D&E, but in the late second-trimester, medical induction becomes the more common procedure for fetal anomalies.[71] The benefits of labor induction are the ability to better evaluate for fetal anomaly and the ability for parent(s) to see and hold the infant. Chromosomal analysis can be performed on products obtained from D&E.[71]

Misoprostol, as a single agent administered vaginally or sublingually, is an effective form of labor induction after a second-trimester fetal death.[52,72] Use of misoprostol is considered safe in women with a prior cesarean delivery, with a low transverse scar, and before 28 weeks' gestation. Recommended doses of misoprostol vary. One approach is 400 to 600 mcg every 3 to 6 hours before 28 weeks' gestation; after 28 weeks' gestation, normal induction protocols can be followed.[29] In women with a prior uterine scar, consider a lower initial dose and do not double after the first dose is administered.[52,72] Mifepristone 200 or 600 mg

orally 24 to 48 hours prior to the first misoprostol dose decreases time to delivery.[29]

Prenatal Diagnosis of Abnormality

Although there has been an increase in the amount of research into the effects of perinatal loss on parents and families, limited research is available about the psychological effects of the diagnosis of an abnormality and the subsequent decision to terminate the pregnancy. Without full disclosure and transparency, parents are not prepared to make an informed choice about whether to test or not test. Before proceeding with testing, parents need to be carefully counseled about the limitations of testing and the potential choices that may arise.

The same emotional support and counseling should be made available to parents who choose to terminate a pregnancy as to those who experience the spontaneous loss of a pregnancy.

The Infant With an Abnormality or Disability

Caring for a child with disabilities is stressful for the parents, and the best predictor of the level of stress is the severity of the disability.[73,74] Caregivers of children with intellectual disabilities are likely to experience depression.[75] The depression may be the result of the financial, social, and physical stressors experienced by the family.[76]

Tasks for parents of a child with a disability include accepting their child's condition, managing their child's activities of daily living, meeting their child's developmental needs, coping with ongoing stress, primarily depression, establishing a support system, and monitoring the magnitude of their grief.

Parents with an infant born with a disability or abnormality should be offered the same emotional support as described elsewhere in this chapter. There is some evidence that family centered care provides the best support for caregivers of these children.[77] A family physician is likely to be a caregiver for the family and, possibly, the child in the long term and, thus, is in a prime position to help diagnose neonatal and infant abnormalities and provide family support. Parents should be provided with information about community resources and directed to available support groups.

Maternal Critical Illness and Death

Although the focus of this chapter is on the experience of infant birth crisis, the critical illness or death of a woman in pregnancy or near childbirth is important to address. The principles of providing support to families who experience birth crisis are essentially the same for families and staff who experience a maternal death.

Maternal critical illness may be related to prior comorbidities such as obesity, coronary artery disease, or hypertension. It may also occur because of a pregnancy complication such as preeclampsia, venous thromboembolism, or hemorrhage. Prenatal recognition of a high-risk pregnancy is important to ensure that appropriate care is provided, usually with referral to a maternal-fetal medicine team. All facilities providing maternity care must be prepared to handle emergencies such as massive blood loss, maternal seizure, and sepsis. The implementation of evidence-based protocols can guide providers who work in facilities without immediate access to an intensive care team.[75,78] The regular practice of team-based drills to manage obstetric emergencies has been shown to improve outcomes.[76,79,80]

In developed countries, maternal death is rare and usually unexpected. The maternal mortality rate in the United States is no longer decreasing, perhaps due to the increase in obesity and cesarean deliveries. In the United States, the pregnancy-related mortality ratio was 17.2 deaths per 100,000 live births in 2015.[81] In Australia, maternal death is unexpected and the numbers of deaths are small, however, more than one woman dies every 2 weeks due to complications related to pregnancy or childbirth associated with childbearing.[78,82] Ninety four percent of maternal deaths occur in developing countries.[83] The maternal mortality ratio for Ethiopia, a country where ALSO is taught, has decreased more than 50% since 2000 but was still 401 per 100,000 live births in 2017.[84] Every maternal death has a significant effect on the family, the community, and the providers involved.[85]

A provider's experience of the death of a woman has been shown to be comparable to that of emergency personnel attending large-scale disasters, and most providers are unprepared.[85] Providers also report having flashbacks and memories of the death that affect personal and professional relationships.[85] More common than maternal death are near-miss events such as severe maternal hemorrhage. The reactions to these events can be processed by women, their families, and providers with support and time.

Maternity Care Providers

Maternity care providers may experience fear about having missed something that could have prevented the outcome or about contributing directly to the outcome. The amount of support that is needed to deal with their own grief or fear should not be underestimated.[3]

Several mostly qualitative studies have explored maternity care providers' experiences related to perinatal loss. One common theme is that providers often experience guilt because they think their role is about saving lives rather than being around death.[86] Guilt is also connected with blame and, sometimes, blaming one another for not doing enough to support the women and families.[86-88] Personal grief has been found to be common in obstetricians who have cared for a woman who experiences a stillbirth. Reactions frequently experienced included self-doubt and self-blame.[3]

Another common response is rationalization whereby staff compartmentalize the difficult and sad aspects of their role to manage later.[87] This is especially true when working in busy environments where there may not be adequate time to grieve, acknowledge the event, and assess the possible effects of the event.[87] Unresolved personal issues also affect the way maternity care providers cope, and these should be addressed.[87]

Elements for increasing the wellbeing of maternity care providers include reflection and review; emotional and practical support; access to counseling services; initial and ongoing education and training; and institutional policies and guidelines.[86]

Reflection and Review

Seeking out others to discuss the loss and its personal effect can be helpful. Formal reflection on the care of the woman during her pregnancy with colleagues is also important. Many hospitals or health care settings have regular perinatal mortality meetings to discuss the care of women who experience a stillbirth or neonatal loss. These meetings can provide a safe and confidential environment to reflect on the care provided and to determine whether anything could have been done differently. Changes in policy and practice to reduce the risk of similar events occurring in the future should be addressed.[45]

Fear of litigation is a concern of providers involved in maternity care.[88] It needs to be acknowledged so as not to affect future practice adversely.

Emotional and Practical Support

Emotional and practical support from colleagues is essential. Providers who work in a supportive environment that has a no-blame culture are more likely to provide better, more effective care and make practice changes to improve care.[86]

Time pressure has been identified by providers as a barrier to providing and receiving support. It is important for managers and others in leadership positions to seek out staff who have been involved in caring for families that have experienced perinatal loss and ensure that staff have adequate support. Supporting other members of the team also is necessary.

Midwives' satisfaction with working with families who experience perinatal loss was related to the level of support they received from the organization in which they worked.[86] This included being in a hospital that acknowledged loss and grieving, as well as being able to provide continuity of care to these families.[89]

Access to Counseling Services

In some health care settings, a bereavement counselor is offered, and this has been found to be useful.[87] This type of counseling may be provided in a group or one-on-one setting. Peer support groups, multidisciplinary team meetings, and formal debriefing with trained personnel are all strategies that can provide support for clinicians. Personal healing must take place to enable providers to move beyond the intense emotions after a perinatal loss.[88]

Initial and Ongoing Education and Training

Less experienced providers and staff may need mentoring to be able to care for families who experience perinatal loss. In one small qualitative study, midwives in their final year of education felt unprepared for caring for women with perinatal loss. It is likely that physicians' experiences are similar.[90] Families who have experienced perinatal loss should be included in the education and training programs for maternity care staff.[86,87,91] The elements of this training could include communication, role playing, breaking bad news, and expressing condolences appropriately.[90] Knowing what to say and being brave enough to say it should be included in training programs.[89]

The emotional and psychological aspects of perinatal loss and grief as well as the theoreti-

cal knowledge of grief should be emphasized in education.[87] Students are often protected from working with families experiencing perinatal loss and, although this can be appropriate at times, it means that these students may not have the necessary skills when they graduate. Students should have the opportunity, under supervision and with support, to develop the skills necessary for providing care for bereaving families.[86]

Education and training is important for initial education and should be part of continuing professional development, as well.[86]

Policies and Guidelines

Providers who care for families that experience perinatal loss can become overwhelmed, especially if staff shortages exist.[87] It is essential that hospitals and health services develop policies and guidelines to help support staff, including access to counseling services and support networks.[87]

In the United Kingdom, statutory midwifery supervision provides a framework of professional support in which a named supervisor is readily available to listen, advise, and offer support when adverse clinical incidents occur.[91,92] This ensures that the provider is given time to reflect and learn from such events and enables the processing of emotions and experiences that arise and that, if not addressed, might persist.[93]

It is important for providers to be aware of their feelings of loss after a birth crisis. They are invested in a healthy woman and child, and can feel saddened by a catastrophic outcome. It is important not to become defensive or to prematurely admit to wrongdoing as a means of resolving personal grief. If providers have personally experienced a similar loss, it is at their discretion to reveal that information to the family. Although each situation is different, this might help to minimize or assuage the parent's grief.

Safety Bundle for Support After a Severe Maternal Event

The Council on Patient Safety in Women's Health Care and the Alliance for Innovation on Maternal Health have developed a patient safety bundle called *Support After a Severe Maternal Event* to address birth crises in a standardized and evidence-based manner. The bundle has four domains: readiness, recognition, response, and reporting/systems learning (https://safehealthcareforeverywoman.org/wp-content/uploads/2017/11/Support-after-Severe-Maternal-Event-Bundle.pdf), and serves as a guide for developing local standards of practice for managing birth crises (see pages 13 and 14).

US Racial Disparities in Stillbirth Rates

In the United States, non-Hispanic black and American Indian or Alaska Native women have a significantly higher stillbirth rate than white women. This is also true of prematurity and infant mortality rates. The stillbirth rate is 10.53 deaths per 1,000 live births and stillbirths for non-Hispanic black women and 6.22 for American Indian or Alaska Native women compared with 4.88 for non-Hispanic White women. The stillbirth rate for Hispanic women is 5.22 and for Asian or Pacific Islander is 4.68.[29]

The etiology of racial disparities is multifactorial. Higher rates of risk factors (eg, diabetes, hypertension) and implicit and explicit bias and racism appear to be factors. Some studies found higher stillbirth rates even among black women with higher education levels and adequate prenatal care.[29] More research is needed.

Birth Crisis in Global or Low-Resource Environments

Women in developing countries experience birth crises with greater frequency than those in developed countries. Of approximately 2.6 million annual stillbirths, 98% occur in low- or middle-income countries and 75% occur in sub-Saharan Africa and south Asia.[94] Social support is a key element of caring for women and their families. When the crisis occurs outside of the hospital setting, this support may come from family and the community. In the hospital setting, providers should be aware of cultural and religious morays and how they apply to the demonstration of empathy and sympathy. If necessary, translators should be used to avoid miscommunication and misunderstanding.

Summary

Birth crisis affects women and their families, providers, and medical staff. Management of these crises is important and challenging because of the emotional trauma, personal responsibility, and fear of litigation. In caring for women and families who experience birth crises, providers must seek to

provide mindful, compassionate, and empathetic care. Accusations and blame are never helpful and indeed may prevent or delay a positive resolution in the process of dealing with grief and loss.

Nursing Considerations: Birth Crisis

- Nurses are leaders who should set the tone in the room
- Advocate for mindful, compassionate and empathetic care for the entire family
- Validate parenthood and the grieving process
- Facilitate adequate time and interaction with the infant
- Dress the infant, wrap in warm blankets, and provide mementos
- Identify resources within your institution and community
- Implement a reflective process for providers involved in a birth crisis at your institution

COUNCIL ON PATIENT SAFETY IN WOMEN'S HEALTH CARE

safe health care for every woman

PATIENT SAFETY BUNDLE

Patient, Family, and Staff Support after a Severe Maternal Event

READINESS

Every unit

- Develop a unit-based protocol that includes resources for supporting patients, their families (including non-family support), and staff after a severe maternal event
- Establish a facility-based multidisciplinary response team that integrates clinical staff and mental health professionals
- Provide unit education on protocols and conduct unit-based drills (with post-drill debriefs) on patient, family, and staff support after a severe maternal event
- Develop a unit culture where patients, families, and staff are informed about potential risk factors and are encouraged to speak up when they feel concern for patient well-being and safety

RECOGNITION

Every patient, family, and staff member

- Perform timely assessment of emotional and mental health status of patients, their families, and staff during and after a severe maternal event
- Build capacity among staff to recognize signs of acute stress disorder in patients, their families, and staff after a severe maternal event

RESPONSE

Every severe maternal event

- Provide timely and effective interventions to patients, their families, and staff during and after a severe maternal event
- Communicate a woman's condition with the patient and her family, when appropriate, after a severe maternal event
- Offer support and resources to patients, their families, and staff after a severe maternal event

October 2015

COUNCIL ON PATIENT SAFETY
IN WOMEN'S HEALTH CARE
safe health care for every woman

PATIENT SAFETY BUNDLE

Patient, Family, and Staff Support after a Severe Maternal Event

REPORTING/SYSTEMS LEARNING

Every unit

- Establish a culture of huddles for high-risk patients and post-event debriefs to identify successes and opportunities for improvement
- Conduct a multidisciplinary review of severe maternal morbidity events for systems issues, to include patient perspectives where feasible
- Monitor outcomes and process metrics in perinatal quality improvement (QI) committee

Standardization of health care processes and reduced variation has been shown to improve outcomes and quality of care. The Council on Patient Safety in Women's Health Care disseminates patient safety bundles to help facilitate the standardization process. This bundle reflects emerging clinical, scientific, and patient safety advances as of the date issued and is subject to change. The information should not be construed as dictating an exclusive course of treatment or procedure to be followed. Although the components of a particular bundle may be adapted to local resources, standardization within an institution is strongly encouraged.

The Council on Patient Safety in Women's Health Care is a broad consortium of organizations across the spectrum of women's health for the promotion of safe health care for every woman.

October 2015

Reprinted from Council on Patient Safety in Women's Health Care. AIM-Supported Patient Safety Bundles. Patient, Family, and Staff Support after a Severe Maternal Event. Available at https://safehealthcareforeverywoman.org.

References

1. Heazell AEP, Siassakos D, Blencowe H, et al; Lancet Ending Preventable Stillbirths Series study group. Lancet Ending Preventable Stillbirths investigator group. Stillbirths: economic and psychosocial consequences. *Lancet*. 2016;387(10018):604-616.
2. Gold KJ. Navigating care after a baby dies: a systematic review of parent experiences with health providers. *J Perinatol*. 2007;27(4):230-237.
3. Farrow VA, Goldenberg RL, Fretts R, Schulkin J. Psychological impact of stillbirths on obstetricians. *J Matern Fetal Neonatal Med*. 2013;26(8):748-752.
4. Beck CT, LoGiudice J, Gable RK. A mixed-methods study of secondary traumatic stress in certified nurse-midwives: shaken belief in the birth process. *J Midwifery Womens Health*. 2015;60(1):16-23.
5. Sanghavi DM. What makes for a compassionate patient-caregiver relationship? *Jt Comm J Qual Patient Saf*. 2006;32(5):283-292.
6. Youngson R. *Time to care: How to love your patients and your job*. Raglan, NZ: Rebelheart Publishers; 2012.
7. Koopmans L, Wilson T, Cacciatore J, Flenady V. Support for mothers, fathers and families after perinatal death. *Cochrane Database Syst Rev*. 2013;(6): CD000452.
8. Kavanaugh K, Trier D, Korzec M. Social support following perinatal loss. *J Fam Nurs*. 2004;10(1):70-92.
9. Royal College of Obstetics and Gynaecology. *Late Intrauterine Fetal Death and Stillbirth: Greentop Guideline 55*. London: Royal College of Obstetics and Gynaecology; 2010.
10. Royal College of Obstetricians and Gynaecologists. Information for you - When your baby dies before birth. An information leaflet for parents. 2012. Available at https://www.rcog.org.uk/globalassets/documents/patients/patient-information-leaflets/pregnancy/pi-when-your-baby-dies-before-birth.pdf.
11. Lalor JG, Begley CM, Devane D. Exploring painful experiences: impact of emotional narratives on members of a qualitative research team. *J Adv Nurs*. 2006;56(6): 607-616.
12. Nordlund E, Börjesson A, Cacciatore J, et al. When a baby dies: motherhood, psychosocial care and negative affect. *Br J Midwifery*. 2012;20(11):780-784.
13. Drotar D, Baskiewicz A, Irvin N, et al. The adaptation of parents to the birth of an infant with a congenital malformation: a hypothetical model. *Pediatrics*. 1975;56(5): 710-717.
14. Bainbridge L. Not quite perfect! Diagnosis of a minor congenital abnormality during examination of the newborn. *Infant.* 2009;5(1):28-31.
15. Surkan PJ, Rådestad I, Cnattingius S, et al. Events after stillbirth in relation to maternal depressive symptoms: a brief report. *Birth*. 2008;35(2):153-157.
16. Black BP, Fields WS. Contexts of reproductive loss in lesbian couples. *MCN Am J Matern Child Nurs*. 2014; 39(3):157-162, quiz 163-164.
17. Avelin P, Erlandsson K, Hildingsson I. Rådestad I. Swedish parents' experiences of parenthood and the need for support to siblings when a baby is stillborn. *Birth*. 2011;38(2):150-158.
18. Ostler T. Grief and coping in early childhood: the role of communication in the mourning process. *Zero Three*. 2010;31(1):29-37. https://eric.ed.gov/?id=EJ926598.
19. Roose RE, Blanford CR. Perinatal grief and support spans the generations: parents' and grandparents' evaluations of an intergenerational perinatal bereavement program. *J Perinat Neonatal Nurs*. 2011;25(1):77-85.
20. Kempson D, Murdock V. Memory keepers: a narrative study on siblings never known. *Death Stud*. 2010;34(8): 738-756.
21. Bennett M, Chichester M. *Forgotten voices: How perinatal loss affects grandparents.* Los Angeles, CA: The Preliminary program for Association of Women's Health, Obstetric and Neonatal Nurses; 2008.
22. O'Leary J, Warland J, Parker L. Bereaved parents' perception of the grandparents' reactions to perinatal loss and the pregnancy that follows. *J Fam Nurs*. 2011;17(3): 330-356.
23. Van Dinter MC, Graves L. Managing adverse birth outcomes: helping parents and families cope. *Am Fam Physician*. 2012;85(9):900-904.
24. Gamble J, Creedy D, Moyle W, et al. Effectiveness of a counseling intervention after a traumatic childbirth: a randomized controlled trial. *Birth*. 2005;32(1):11-19.
25. National Center for Health Statics. Fetal Deaths. 2018. Available at https://www.cdc.gov/nchs/nvss/fetal_death.htm.
26. Hoyert DL, Gregory EC. Cause of fetal death: data from the fetal death report, 2014. *Natl Vital Stat Rep*. 2016; 65(7):1-25.
27. MacDorman MF, Gregory EC. Fetal and perinatal mortality: United States, 2013. *Natl Vital Stat Rep*. 2015; 64(8):1-24.
28. ACOG Committee Opinion no. 748: the importance of vital records and statistics for the obstetrician-gynecologist. *Obstet Gynecol*. 2018;132(2):e78-e81.
29. American College of Obstetricians and Gynecologists; Society for Maternal-Fetal Medicine. Management of Stillbirth: Obstetric Care Consensus No, 10. *Obstet Gynecol*. 2020;135(3):e110-e132.
30. Centers for Disease Control and Prevention. What is stillbirth? 2019. Available at https://www.cdc.gov/ncbddd/stillbirth/facts.html.
31. World Health Organization. Stillbirth. 2019. Available at https://www.who.int/maternal_child_adolescent/epidemiology/stillbirth/en/.
32. National Health Service. Definition of stillbirth. 2013. Available at http://www.nhs.uk/conditions/Stillbirth/Pages/Definition.aspx.
33. Australian Institute of Health and Welfare. Definition of stillbirth. 2013. Available at http://meteor.aihw.gov.au/content/index.phtml/itemId/327266.
34. Lawn JE, Yakoob MY, Haws RA, et al. 3.2 million stillbirths: epidemiology and overview of the evidence review. *BMC Pregnancy Childbirth*. 2009;9(Suppl 1):S2.

35. Frøen JF, Friberg IK, Lawn JE, et al; Lancet Ending Preventable Stillbirths Series study group. Stillbirths: progress and unfinished business. *Lancet*. 2016;387(10018): 574-586.
36. World Health Organization. *Definitions and indicators in Family Planning Maternal & Child Health and Reproductive Health*. 2001, Geneva: WHO Press.
37. Kochanek KD, Murphy SL, Xu JQ, Arias E. Deaths: final data for 2017. *Natl Vital Stat Rep*. 2019;68(9):1-77.
38. Korteweg FJ, Bouman K, Erwich JJ, et al. Cytogenetic analysis after evaluation of 750 fetal deaths: proposal for diagnostic workup. *Obstet Gynecol*. 2008;111(4): 865-874.
39. Rosenstein MG, Cheng YW, Snowden JM, et al. Risk of stillbirth and infant death stratified by gestational age. *Obstet Gynecol*. 2012;120(1):76-82.
40. American College of Obstetricians and Gynecologists' Committee on Practice Bulletins—Obstetrics, Society for Maternal-Fetal Medicine. ACOG Practice Bulletin no. 204: fetal growth restriction. *Obstet Gynecol*. 2019; 133(2):e97-e109.
41. Lawn JE, Blencowe H, Waiswa P, et al; Lancet Ending Preventable Stillbirths Series study group. Lancet Stillbirth Epidemiology investigator group. Stillbirths: rates, risk factors, and acceleration towards 2030. *Lancet*. 2016;387(10018):587-603.
42. Gardosi J, Giddings S, Buller S, et al. Preventing stillbirths through improved antenatal recognition of pregnancies at risk due to fetal growth restriction. *Public Health*. 2014;128(8):698-702.
43. The Stillbirth Collaborative Research Network Writing Group. Causes of death among stillbirths. *JAMA*. 2011; 306(22):2459-2468.
44. Burden C, Bradley S, Storey C, et al. From grief, guilt pain and stigma to hope and pride - a systematic review and meta-analysis of mixed-method research of the psychosocial impact of stillbirth. *BMC Pregnancy Childbirth*. 2016;16(1):9.
45. Peters MD, Lisy K, Riitano D, et al. Caring for families experiencing stillbirth: Evidence-based guidance for maternity care providers. *Women Birth*. 2015;28(4): 272-278.
46. Ellis A, Chebsey C, Storey C, et al. Systematic review to understand and improve care after stillbirth: a review of parents' and healthcare professionals' experiences. *BMC Pregnancy Childbirth*. 2016;16:16.
47. Serwint J, Rutherford L. Sharing bad news with parents. *Contemp Pediatr*. 2000;17(3):45-66.
48. Trulsson O. Rådestad I. The silent child—mothers' experiences before, during, and after stillbirth. *Birth*. 2004; 31(3):189-195.
49. Chakhtoura NA, Reddy UM. Management of stillbirth delivery. *Semin Perinatol*. 2015;39(6):501-504.
50. Burgoine GA, Van Kirk SD, Romm J, et al. Comparison of perinatal grief after dilation and evacuation or labor induction in second trimester terminations for fetal anomalies. *Am J Obstet Gynecol*. 2005;192(6): 1928-1932.
51. Boyle A, Preslar JP, Hogue CJ, et al. Route of delivery in women with stillbirth: results from the Stillbirth Collaborative Research Network. *Obstet Gynecol*. 2017;129(4): 693-698.
52. Gómez Ponce de León R, Wing D, Fiala C. Misoprostol for intrauterine fetal death. *Int J Gynaecol Obstet*. 2007; 99(Suppl 2):S190-S193.
53. Gawron LM, Kiley JW. Labor induction outcomes in third-trimester stillbirths. *Int J Gynaecol Obstet*. 2013; 123(3):203-206.
54. Goldenberg RL, Culhane JF, Iams JD, Romero R. Epidemiology and causes of preterm birth. *Lancet*. 2008; 371(9606):75-84.
55. Rådestad I, Surkan PJ, Steineck G, et al. Long-term outcomes for mothers who have or have not held their stillborn baby. *Midwifery*. 2009;25(4):422-429.
56. Kingdon C, Givens JL, O'Donnell E, Turner M. Seeing and Holding Baby: systematic review of clinical management and parental outcomes after stillbirth. *Birth*. 2015;42(3):206-218.
57. Mills TA, Ricklesford C, Cooke A, et al. Parents' experiences and expectations of care in pregnancy after stillbirth or neonatal death: a metasynthesis. *BJOG*. 2014; 121(8):943-950.
58. Page JM, Christiansen-Lindquist L, Thorsten V, et al. Diagnostic tests for evaluation of stillbirth: results from the Stillbirth Collaborative Research Network. *Obstet Gynecol*. 2017;129(4):699-706.
59. Silver RM. Optimal "work-up" of stillbirth: evidence! *Am J Obstet Gynecol*. 2012;206(1):1-2.
60. Man J, Hutchinson JC, Heazell AE, et al. Stillbirth and intrauterine fetal death: factors affecting determination of cause of death at autopsy. *Ultrasound Obstet Gynecol*. 2016;48(5):566-573.
61. Heazell AE, McLaughlin MJ, Schmidt EB, et al. A difficult conversation? The views and experiences of parents and professionals on the consent process for perinatal postmortem after stillbirth. *BJOG*. 2012;119(8): 987-997.
62. Leduc L. No. 394 - stillbirth investigation. *J Obstet Gynaecol Can*. 2020;42(1):92-99.
63. Perinatal Society of Australia and New Zealand. Clinical practice guideline for perinatal mortality. 2009. Available at https://sanda.psanz.com.au/assets/Uploads/Full-Version-PSANZ-Guidelines-2012.pdf.
64. Perinatal Society of Australia and New Zealand, Centre of Research Excellence Stillbirth. Clinical practice guideline for the care of women with decreased fetal movements for women with a singleton pregnancy from 28 weeks' gestation. 2019. Available at https://www.acn.edu.au/wp-content/uploads/element-3-decreased-fetal-movements-clinical-practice-guideline.pdf.
65. Lamont K, Scott NW, Jones GT, Bhattacharya S. Risk of recurrent stillbirth: systematic review and meta-analysis. *BMJ*. 2015;350(350):h3080.
66. Chescheir N, Menard MK. Scheduled deliveries: avoiding iatrogenic prematurity. *Am J Perinatol*. 2012;29(1): 27-34.

67. Spong CY, Mercer BM, D'alton M, et al. Timing of indicated late-preterm and early-term birth. *Obstet Gynecol*. 2011;118(2 Pt 1):323-333.
68. Farrant BM, Stanley FJ, Hardelid P, Shepherd CC. Stillbirth and neonatal death rates across time: the influence of pregnancy terminations and birth defects in a Western Australian population-based cohort study. *BMC Pregnancy Childbirth*. 2016;16:112.
69. Miller DA, Rabello YA, Paul RH. The modified biophysical profile: antepartum testing in the 1990s. *Am J Obstet Gynecol*. 1996;174(3):812-817.
70. Hofmeyr GJ, Novikova N. Management of reported decreased fetal movements for improving pregnancy outcomes. *Cochrane Database Syst Rev*. 2012;(4): CD009148.
71. Borgatta L, Kapp N; Society of Family Planning. Clinical guidelines. Labor induction abortion in the second trimester. *Contraception*. 2011;84(1):4-18.
72. Dodd JM, Crowther CA. Misoprostol for induction of labour to terminate pregnancy in the second or third trimester for women with a fetal anomaly or after intrauterine fetal death. *Cochrane Database Syst Rev*. 2010; 4(4):CD004901.
73. Hassall R, Rose J, McDonald J. Parenting stress in mothers of children with an intellectual disability: the effects of parental cognitions in relation to child characteristics and family support. *J Intellect Disabil Res*. 2005;49(Pt 6):405-418.
74. Sanders J, Morgan S. Family stress and adjustment as perceived by parents of children with autism or Down syndrome: implications for intervention. *Child Fam Behav Ther*. 1997;19(4):15-32.
75. Mbugua MN, Kuria MW, Ndetei DM. The prevalence of depression among family caregivers of children with intellectual disability in a rural setting in Kenya. *Int J Family Med*. 2011;2011:534513.
76. Mak WW, Ho SM. Caregiving perceptions of Chinese mothers of children with intellectual disability in Hong Kong. *J Appl Res Intellect Disabil*. 2007;20(2):145-156.
77. Floyd F, Gallagher E. Parental stress, care demands, and use of support services for school-age children with disabilities and behavior problems. *Fam Relat*. 1997;46(4):359-371.
78. American College of Obstetricians and Gynecologists Committee on Patient Safety and Quality Improvement. Committee Opinion no. 590: preparing for clinical emergencies in obstetrics and gynecology. *Obstet Gynecol*. 2014;123(3):722-725.
79. Salas E, Gregory ME, King HB. Team training can enhance patient safety—the data, the challenge ahead. *Jt Comm J Qual Patient Saf*. 2011;37(8):339-340.
80. Pettker CM, Thung SF, Norwitz ER, et al. Impact of a comprehensive patient safety strategy on obstetric adverse events. *Am J Obstet Gynecol*. 2009;200(5):492. e1-492.e8.
81. Centers for Disease Control and Disease Prevention. Pregnancy Mortality Surveillance System. Available at https://www.cdc.gov/reproductivehealth/maternalinfanthealth/pregnancy-mortality-surveillance-system.htm.
82. Kildea S, Pollock WE, Barclay L. Making pregnancy safer in Australia: the importance of maternal death review. *Aust N Z J Obstet Gynaecol*. 2008;48(2):130-136.
83. United Nations Population Fund, World Health Organization, United Nations Children's Fund, World Bank Group, the United Nations Population Division. Trends in maternal mortality: 2000 to 2017: estimates by WHO, UNICEF, UNFPA, World Bank Group and the United Nations Population Division. 2019. Available at https://www.unfpa.org/featured-publication/trends-maternal-mortality-2000-2017.
84. The World Bank. Maternal mortality ration (modeled estimate, per 100,000 live births) — Ethiopia, Eritrea. Available at https://data.worldbank.org/indicator/SH.STA.MMRT?locations=ET-ER.
85. Mander R. The midwife's ultimate paradox: a UK-based study of the death of a mother. *Midwifery*. 2001;17(4): 248-258.
86. Nallen K. Midwives' needs in relation to the provision of bereavement support to parents affected by perinatal death. Part one. *MIDIRS Midwifery Digest*. 2006;16(4): 537-542.
87. Modiba LM. Experiences and perceptions of midwives and doctors when caring for mothers with pregnancy loss in a Gauteng hospital. *Health SA Gesondheid*. 2008;13(4):29-40.
88. McCool W, Guidera M, Stenson M, Dauphinee L. The pain that binds us: midwives' experiences of loss and adverse outcomes around the world. *Health Care Women Int*. 2009;30(11):1003-1013.
89. Fenwick J, Jennings B, Downie J, et al. Providing perinatal loss care: satisfying and dissatisfying aspects for midwives. *Women Birth*. 2007;20(4):153-160.
90. Alghamdi R, Jarrett P. Experiences of student midwives in the care of women with perinatal loss: a qualitative descriptive study. *Br J Midwifery*. 2016;24(10):715-722.
91. Nursing and Midwifery Council. Supervision, support and safety NMC quality assurance of LSAs 2010-2011. 2011. Available at www.nmc-uk.org/publications/midwifery-supervision/.
92. Nursing and Midwifery Council. Supporting nurses and midwives through lifelong learning. 2002. Available at https://www.epilepsy.org.uk/sites/epilepsy/files/professionals/Nurse%20portfolio%20docs/11.%20NMC%20-%20Supporting%20Nurses%20and%20Midwives%20through%20lifelong%20learning.pdf.
93. Deery R. An action-research study exploring midwives' support needs and the affect of group clinical supervision. *Midwifery*. 2005;21(2):161-176.
94. Frøen JF, Lawn JE, Heazell AER, et al. Ending preventable stillbirths: an executive summary for the Lancet's series. 2016. Available at http://www.thelancet.com/pb/assets/raw/Lancet/stories/series/stillbirths2016-execsumm.pdf.

CPD20011965

American Academy of Family Physicians
11400 Tomahawk Creek Parkway
Leawood, Kansas 66211-2680
(800) 274-2237, ext. 7506 | www.aafp.org/also