Neonatal Subgaleal Hemorrhage

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ABSTRACT

Subgaleal hemorrhages, although infrequent in the past, are becoming more common with the increased use of vacuum extraction. Bleeding into the large subgaleal space can quickly lead to hypovolemic shock, which can be fatal. Understanding of anatomy, pathophysiology, risk factors, differential diagnosis, and management will assist in early recognition and care of the infant with a subgaleal hemorrhage.

NEONATAL SUBGALEAL HEMORRHAGE IS AN infrequent but potentially fatal complication of childbirth, especially if accompanied by coagulation disorders. A subgaleal hemorrhage is an accumulation of blood within the loose connective tissue of the subgaleal space, which is located between the galea aponeurotica and the periosteum (Figure 1). Unlike a cephalohematoma, a subgaleal hemorrhage can be massive, leading to profound hypovolemic shock.[1,2] Although subgaleal hemorrhage has a low incidence rate, it is strongly associated with vacuum extraction devices, which have been increasingly used over the last decade.[3] Careful monitoring of infants following a difficult vacuum extraction or forceps delivery, along with early recognition of distinguishing features of a subgaleal hemorrhage, optimizes neonatal outcomes.

INCIDENCE

Several studies have documented the incidence of subgaleal hemorrhages and associated factors over the last 20 years. Table 1 summarizes the findings of some of these studies. Plauche evaluated the published reports of 123 cases of subgaleal hemorrhage. The documented incidence of subgaleal hemorrhage in this study was 0.4 in 1,000 spontaneous vaginal deliveries and 5.9 in 1,000 deliveries in centers that used vacuum extraction.[4] A number of other researchers reported similar results.[5–7] Gebremariam, however, has documented the incidence of subgaleal hemorrhage to be much higher: 3 per 1,000 live and term births and 19.7 per 1,000 vacuum extraction births.[8] More remarkable than the actual incidence is the six- to sevenfold increase in incidence when vacuum extraction is applied at delivery.

In a three-year study in Hong Kong, an infant born with vacuum-assisted extraction was 60 times more likely to develop a subgaleal hemorrhage than an infant born with other modes of delivery.[6] A five-year study from 1988 to 1993 revealed a 25-fold increase in subgaleal hemorrhages with vacuum extraction when compared to noninstrumented or unassisted birth through normal deliveries.[8] In 1998, the Food and Drug Administration (FDA) issued an advisory warning regarding the use of vacuum-assisted devices and stated that these devices may cause serious or fatal complications. The FDA also stated in the advisory that the law required hospitals and other facilities to report deaths, serious illnesses, and injuries associated with the use of medical devices.[9] Following the advisory, reporting of subgaleal hemorrhages increased 22-fold, suggesting a significant number of unreported adverse outcomes prior to the advisory.[3]

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TABLE 1 ■ Statistics Related to Subgaleal Hemorrhages[4–6,8,27,28]

Study (Study Period)	No. of Infants in Study	No. of Subgaleal Hemorrhages	Incidence per 1,000 Live Births	% Male	% Primi-paraous	% Vacuum Extraction	% >40 Weeks Gestation	% >3.4 kg	% Mortality	Infant's Race	% Coagu-lopathy	Location
Chadwick et al.[5] (1970–1993)	239,608	37	0.15	65	100	89	U	Mean 3.4 kg	5.4	89% Caucasian	19	Australia
Ahuja et al.[27] (1964–1969)	12,648	13	1.02	69	U	100	62	69	23	100% Caucasian	U	Scotland
Barrow & Peters[28] (1947–approx 1968)	23,500	18	0.8	83	44	11	U	72	22	67% African	U	Africa
Gebremariam[8] (1988–1993)	23,353	69	3	57	53	41	64	37	14	U	5.8	Ethiopia
Ng et al.[6] (1991–1993)	22,489	18	0.8	89	U	89	33	44	16.7	72% Chinese	U	Hong Kong
Plauche[4] (Approx 1963–1979)	U	123	0.4	U	U	49	U	U	22.8	U	29	U

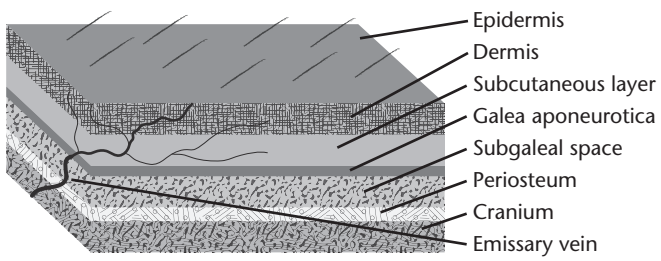
U = unreported.

ANATOMY

A review if the anatomy of the scalp helps in understanding subgaleal hemorrhages (see Figure 1). The scalp is composed of five layers: skin (epidermis and dermis), subcutaneous tissue, galea aponeurotica, subgaleal space, and periosteum (or pericranium). The skin contains hair follicles and sweat glands and is firmly bound to deeper tissues.[10] Below the skin is the subcutaneous layer, a dense network of connective tissue.[1] The galea aponeurotica, also referred to as the epicranial aponeurosis, is the third layer of tissue.[10] This dense, fibrous tissue covers the entire upper cranial vault from the occiput to the frontal and laterally to the temporal fascia.[2] Located below the galea aponeurotica and covering the same massive space is the subgaleal, or subaponeurotic, space.[1] This loose, fibroareolar tissue allows the scalp to slide on the periosteum.[10] Large, valveless emissary veins traverse the subgaleal space, which connects the dural sinuses inside the skull with the superficial veins of the scalp (see Figure 1).[11] The periosteum, the deepest layer of the scalp, strongly adheres to the surface of the cranium.[10]

FIGURE 1 ■ Cross-section of the scalp.

Note the location of an emissary vein in the large subgaleal space.



From: Seery, G. (2002). Surgical anatomy of the scalp. *Dermatologic Surgery, 28,* 582. Reprinted by permission.

PATHOPHYSIOLOGY

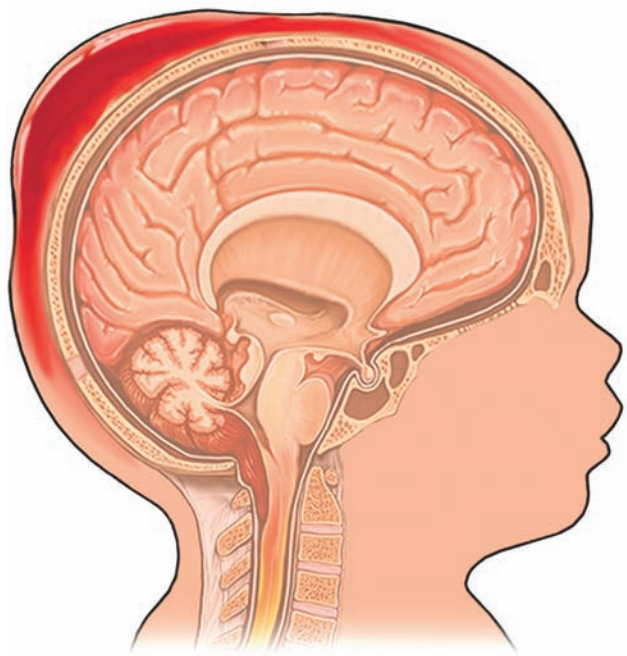
A neonatal subgaleal hemorrhage, also known as a subaponeurotic hemorrhage, develops as a collection of blood in the subgaleal space (Figure 2).[1] When shearing forces are applied to the scalp, large emissary veins in the subgaleal space sever or rupture, and blood accumulates (see Figure 1).[12] Because the galea aponeurotica covers the entire cranial vault, the subgaleal space creates a huge potential for hemorrhaging: From the orbits of the eyes to the nape of the neck and laterally to the temporal fascia, located above the ears. If the hemorrhage is massive, it can displace the ears anteriorly.[13] This space is not limited by sutures, so there are no anatomic barriers to prevent a massive hemorrhage.

The loose connective tissue of the subgaleal space has the potential to accommodate up to 260 ml of blood.[14] A neonate's total blood volume is approximately 80 ml/kg.[15] Robinson and Rossiter estimated that for every centimeter the head circumference exceeds normal limits approximately 40 ml of blood are lost to the subgaleal space.[16] Massive blood loss of this type could easily exceed the neonate's total blood volume and lead to profound, and many times lethal, hypovolemic shock. On postmortem examination, one infant who had died from a subgaleal hemorrhage was found to have a clot containing 50 ml of blood, and two others had clots containing 150 ml of blood.[6]

VACCUM EXTRACTION AND FORCEPS AS RISK FACTORS

Although subgaleal hemorrhages can occur spontaneously, most are associated with vacuum extraction or a combination

FIGURE 2 ■ Subgaleal hemorrhage, sagittal view.



From: Nucleus Medical Art. Retrieved May 1, 2006, from http://catalog.nucleusinc.com/generateexhibit.php?ID=14999&ExhibitKeywordsRaw=Birth+Injury+-+Head+Trauma+Due+to+Vacuum+Extraction+Delivery&TL=1793&A=2. Reprinted by permission.

FIGURE 3A ■ Optimum placement of vacuum extraction cup.

The center of the vacuum extraction cup is about 3 cm in front of the posterior fontanel, and the sagittal suture is midline under the cup. This is called the flexion point.

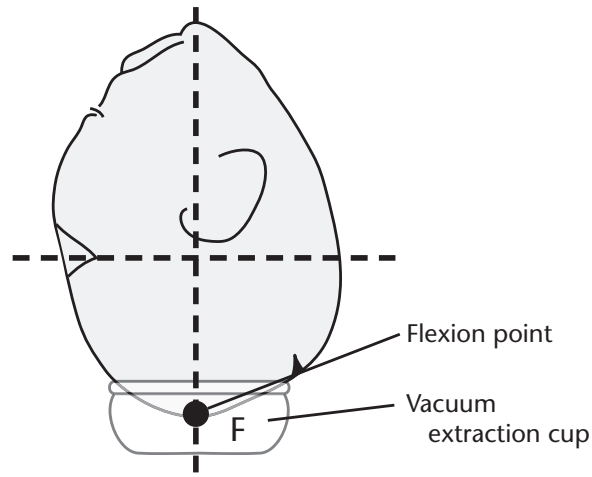
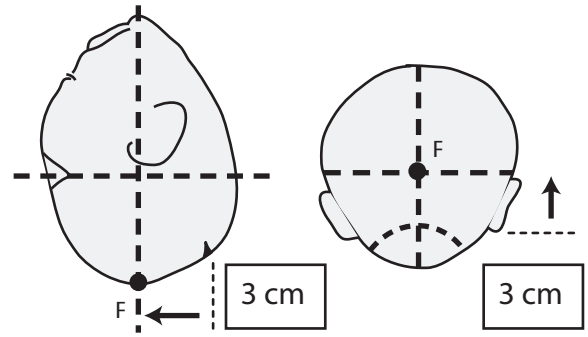


FIGURE 3B ■ Fetal head and direction of descent.



F = Flexion point

From: Vacca, A. (2004). Vacuum-assisted delivery: Improving patient outcomes and protecting yourself against litigation. *OBG Management* (Suppl), S2. Reprinted by permission.

of vacuum and forceps (see Table 1). The risk of infant injury is greatest when multiple methods—such as vacuum extraction followed by forceps—are employed in a delivery.[17]

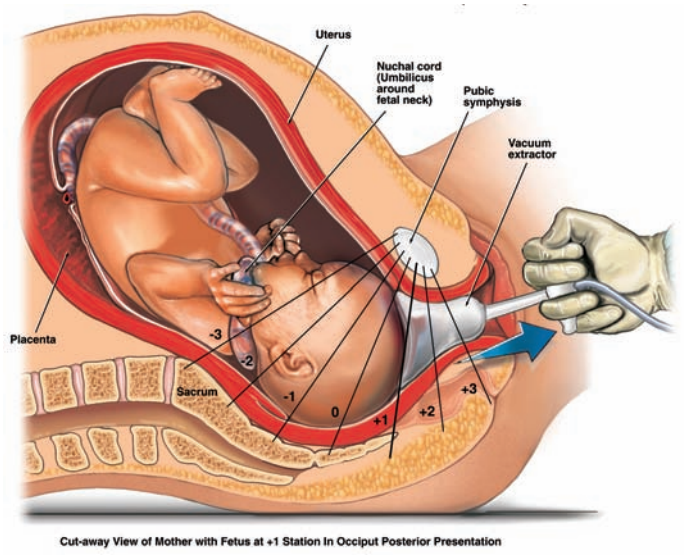
A vacuum extraction cup exerts traction directly on the fetal scalp in an attempt to assist the mother in the birth. The vacuum cup must be applied on the infant's head so that the least amount of force needed is used to maintain the traction to extract the infant. The vacuum device is positioned correctly when the posterior fontanel is about 3 cm from the center of the cup and the sagittal suture is midline under the cup.[18] This is called the flexion point. (Figure 3A and 3B). Regardless of the head's position, the clinician must be able to locate the flexion point for correct positioning of the vacuum device (Figure 4).[19] Incorrect placement of the vacuum device contributes significantly to the development of a subgaleal hemorrhage.[5]

Multiple "pop-offs" (dislodgment of the suction cup), applications exceeding ten minutes, increased number of pulls, and incorrect manipulation of the vacuum-assisted device also contribute to the development of a subgaleal hemorrhage.[19] Steady, smooth pulls with rotation that occurs naturally as a part of normal labor are safer than jerking, rocking, or rotational pulls.[19,20] Incorrect traction may result in descent of only the scalp and not of the infant's entire head. When the scalp alone descends, the galea is pulled away from the skull, tearing the emissary veins and causing subsequent bleeding into the subgaleal space.[20]

The use of vacuum-assisted birth devices has grown significantly in the U.S.[3] In 1980, forceps were used at a rate of 17.7 times in 100 vaginal births, whereas vacuum extraction was used less than 1 time in 100 vaginal births. By 2000, forceps were used only 4 times in 100 vaginal births, but vacuum extraction was up to 8.4 times in 100 vaginal births.[21]

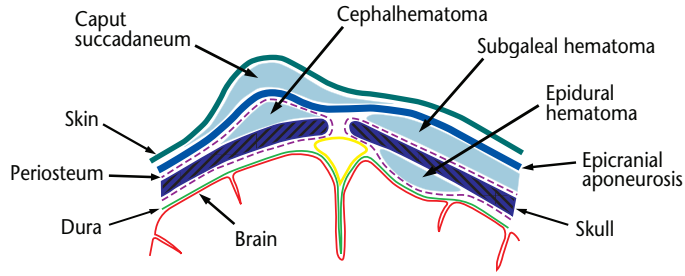
In the 4 years from 1994 to 1998, the FDA received reports of 12 deaths and nine serious injuries resulting from vacuum-assisted delivery, or approximately five events per year.[3] Although these events occurred in an extremely small percentage of births, the FDA was concerned because of a fivefold increase over the preceding 11 years. In May 1998, the agency issued a health advisory addressing the need for caution when using vacuum-assisted devices. The advisory was to make health care professionals who use these devices aware of the life-threatening complications associated with

FIGURE 4 ■ Vacuum extractor on flexion point.



From: Nucleus Medical Art. Vacuum extraction with fatal head injury—Medical Illustration. Retrieved May 1, 2006, from http://catalog.nucleusinc.com/enlargeexhibit.php?ID=1699. Reprinted by permission.

FIGURE 5 ■ Hemorrhages of the scalp.



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them. Also of concern was the fact that health care professionals responsible for caring for infants were not being alerted when a vacuum device had been used and therefore did not monitor for signs and symptoms of a subgaleal hemorrhage.[9] In 1999, after experiencing a similar association between vacuum-assisted devices and subgaleal hemorrhages, the Health Protection Branch of Health Canada issued its own warning.[22]

OTHER RISK FACTORS

Risk factors for a subgaleal hemorrhage also include pregnancies complicated by cephalopelvic disproportion, maternal exhaustion, prolonged second stage of labor, prematurity, postmaturity (>40 weeks), large, heavy infants, nonreassuring fetal status and fetal distress, and birth asphyxia. Maternal exhaustion can occur during prolonged and difficult labor. Friction against the maternal pelvic bone may also be increased when labor is prolonged or difficult. This friction places the infant at increased risk for developing a subgaleal hemorrhage. Furthermore, any labor that is arduous and prolonged can result in hypoxia.[20] Longer second-stage labor and longer vacuum procedures may provide time for the accumulation of more interstitial scalp fluid, thereby leaving tissues more vulnerable to injury.[7] Ng and associates demonstrated that a prolonged second stage of labor was an obstetric indication that placed an infant at risk. They documented that the mother had experienced a prolonged second stage of labor in 50 percent of infants with a subgaleal hemorrhage.[6] Subgaleal hemorrhages in the premature infant may be secondary to bleeding abnormalities associated with prematurity.[20] In addition, a large, heavy infant places increased force against the maternal pelvic bone, potentially increases the risk of vascular stretching and/or laceration, and thus increases the risk for subgaleal hemorrhage.[3]

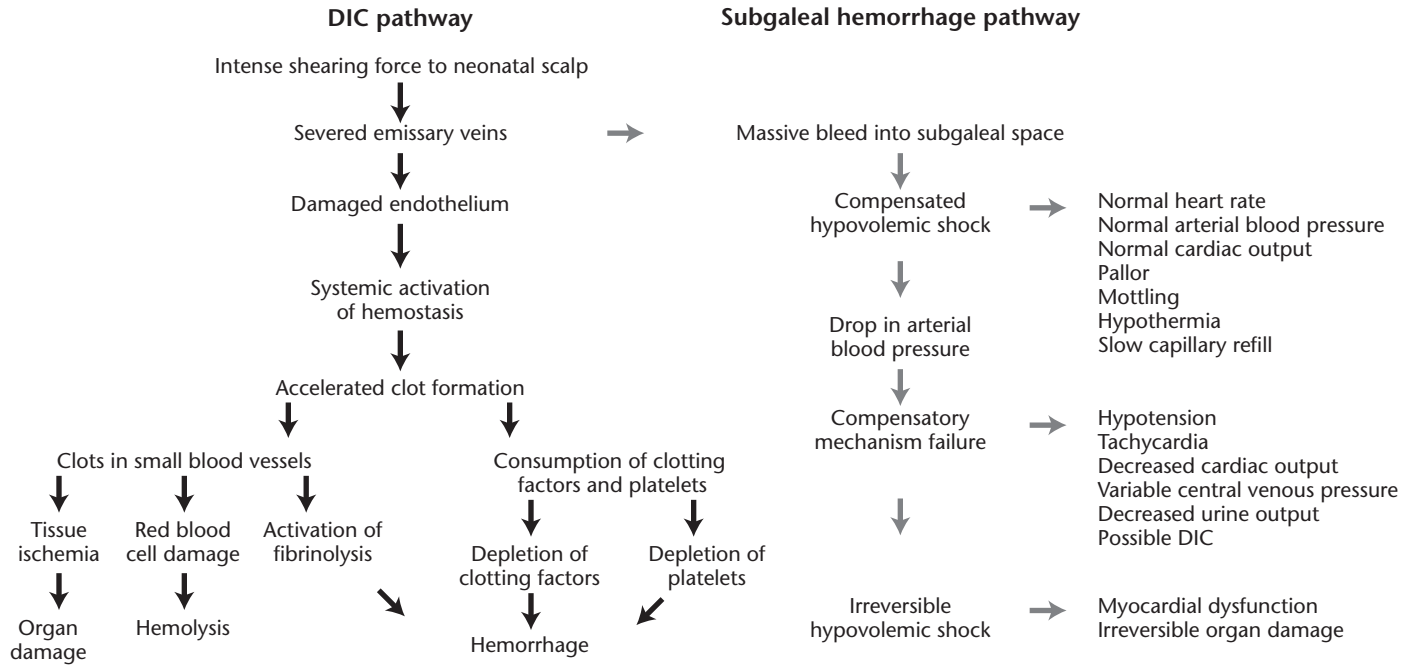
Fetal distress and birth asphyxia have also shown strong correlation to subgaleal hemorrhaging.[6] Nonreassuring fetal status, fetal distress, and birth asphyxia may be due to an underlying abnormal labor.[3,20] Of the 18 cases Ng and associates reviewed, 9 involved fetal bradycardia.[6] Infants of primiparous women are also at risk for developing subgaleal hemorrhage because of the increased resistance of heavy perineal muscles stretching the scalp and causing emissary veins to tear. Primiparous women also have an increased incidence of operative delivery requiring forceps, vacuum extraction, and/or cesarean section.[23]

Neonatal coagulopathy may play a significant role in subgaleal hemorrhages, but its association as a cause is controversial.[2] Researchers are uncertain as to whether the coagulopathy problem exists before the subgaleal hemorrhage or afterward.[12] Vitamin K, factor VIII (hemophilia A), and factor IX (hemophilia B) deficiencies have all been associated with subgaleal hemorrhages.[24] Male infants are also at higher risk than females, possibly because of their increased incidence of bleeding disorders.[25]

CLINICAL FEATURES

The clinical features of a subgaleal hemorrhage make this condition difficult to distinguish from other birth-related scalp injuries (Figure 5). Infants delivered by vacuum extraction often develop an artificial caput succedaneum, an edematous fluid collection in the scalp that typically resolves without complications 12 to 18 hours after birth but leaves a normal caput succedaneum with a circular area of ecchymosis.[26] In some cases, caput succedaneum, cephalohematoma, and subgaleal hemorrhage coexist.[24] If the hemorrhage is severe, the infant develops clinical features rapidly. Most subgaleal hemorrhages develop gradually, however, over several hours up to a few days. They do not present with clinical features until extensive blood loss has occurred.[2] Ahuja and colleagues evaluated 13 cases of subgaleal hemorrhage, which

FIGURE 6 ■ Pathophysiology of subgaleal hemorrhage and DIC.



Adapted from: Rimar, J. (1988). Recognizing shock syndromes in infants and children. *MCN. The American Journal of Maternal Child Nursing, 13,* 32–37; and Emery, M. (1992). Disseminated intravascular coagulation in the neonate. *Neonatal Network, 11(8), 7*.

presented 30 minutes to 30 hours after birth, with a mean onset of 9 hours.[27]

An increasing head circumference is a uniform characteristic of this condition.[28] As the hemorrhage fills the subgaleal space, it exerts pressure on the brain tissue, causing neurologic disturbances and possible seizures.[1,2] Hematocrit values decline as blood is lost from the circulatory system into the subgaleal space. Ecchymoses can appear around the eyes and ears, and hyperbilirubinemia may develop.[2]

The hallmark of this condition is the presence of a fluctuating mass that straddles cranial sutures, fontanels, or both.[13] In some cases, the swelling is difficult to distinguish from scalp edema.[2] The fluctuating mass, sometimes referred to as a wave, is important for distinguishing the subgaleal hemorrhage from other scalp injuries.[11]

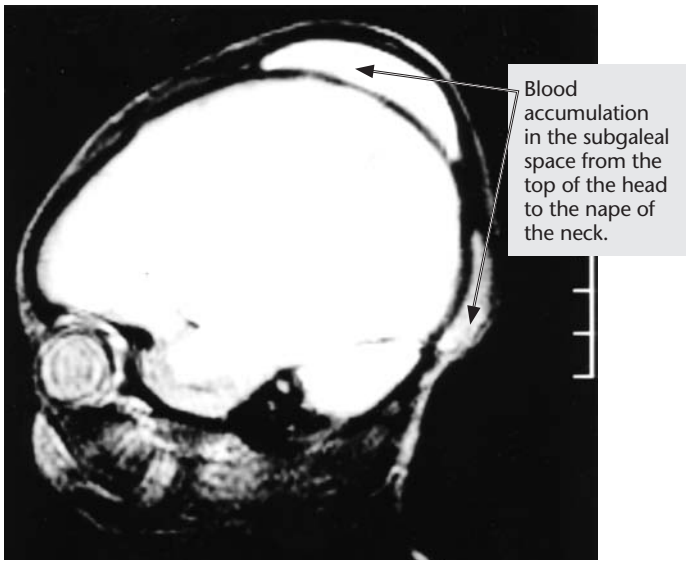
Initially, the infant may present with what appears to be a caput succedaneum, especially common in vacuum-assisted deliveries. The scalp may feel boggy or tight.[13] The swelling can shift when the head is repositioned and indent with palpation.[2] The condition may also present without the hallmark mass to distinguish it, however.[6] Pallor and hypotonicity, sometimes accompanied by increased heart and respiratory rates, may be the only early signs the infant displays.[2] Over time, discoloration of the scalp and eyelids begins to appear, signaling the collection of blood deep beneath the aponeurotic layer.[5]

Hypovolemic Shock

As blood is lost into the subgaleal space, signs of hypovolemic shock appear. A loss of 20 to 40 percent of an infant's circulating blood volume will result in acute shock.[2] In a 3 kg infant, 20 to 40 percent blood loss would equal approximately 50 to 100 ml. As established earlier, the subgaleal space is capable of holding 260 ml.[14] Profound shock and loss of the infant's blood volume can occur.

Initially, symptoms of shock may be nonspecific. Compensatory mechanisms cause vasoconstriction to maintain blood supply to vital organs (Figure 6).[29] During this compensatory stage, the infant may present with normal heart rate and blood pressure accompanied by pallor, mottling of the skin, hypothermia, lethargy, and capillary refill greater than three seconds.[29,30] With inadequate tissue perfusion, metabolic acidosis can occur. As the hemorrhage continues, the compensatory mechanisms fail, and the hallmark signs of hypovolemic shock begin to appear: hypotension, tachycardia, tachypnea, cyanosis, and oliguria.[29,31] Accumulation of acid products from anaerobic metabolism can cause increasing metabolic acidosis. If blood loss continues, the infant enters the uncompensated stage of hypovolemic shock, and arterial blood pressure falls, hastening a phase that is irreversible and fatal.[29,31] For unknown reasons, vital signs may be normal for a short period of time in this phase even though death is inevitable.[13] Disseminated intravascular coagulation (DIC) may also develop during this phase.

FIGURE 7 ■ Magnetic resonance imaging of head demonstrating blood in the subgaleal space.



From: Florentino-Pineda, I., Ezhuthachan, S. G., Sineni, L. G., & Kumar, S. P. (1994). Subgaleal hemorrhage in the newborn infant associated with silicone elastomer vacuum extractor. *Journal of Perinatology, 14,* 97. Reprinted by permission.

DISSEMINATED INTRAVASCULAR COAGULATION

In a subgaleal hemorrhage, damage to the endothelium triggers the hemostatic system to begin the coagulation cascade to stop bleeding. Although the hemostatic system may respond appropriately at the onset of a subgaleal hemorrhage, massive blood loss, leading to the characteristic hypovolemic shock, soon causes widespread activation of coagulation. When normal counterbalancing regulatory mechanisms are overwhelmed by the widespread coagulation response, DIC develops and exacerbates the subgaleal hemorrhage.[32]

Clinical manifestations of DIC vary and may be unpredictable in the neonate.[33] Because DIC is a diffuse, systemic disorder, bleeding or oozing of blood occurs at multiple sites. Bleeding can also occur in the gastrointestinal, genitourinary, pulmonary, and central nervous systems. Petechia, echymosis, and purpura may be visible in infants with DIC. Microclots in the capillaries may cause fingers, toes, nose, and ears to appear cyanotic, and clots obstructing blood flow to organ systems may cause organ dysfunction.[33] Shock is a common underlying disorder in DIC.[34]

DIAGNOSIS

A baseline complete blood count is important for diagnosing a subgaleal hemorrhage. During an acute hemorrhage, however, the hemoglobin level may not accurately reflect the severity of the hemorrhage until hemodilution and reconstitution of the plasma volume occur.[24] Because no single lab test is sensitive or specific enough to enable a diagnosis of DIC, the whole clinical picture must be taken into account. The most common combination of lab tests to diagnose DIC is platelet count, prothrombin time (PT), partial thromboplastin time (PTT), measurement of one or two clotting factors and inhibitors (such as antithrombin), and a test for fibrin degradation products.[35] Abnormalities indicative of DIC include a prolonged PT and PTT, a reduced fibrinogen level, thrombocytopenia, and increased D-dimers or fibrin/fibrinogen degradation products.[36]

Factor levels and other clotting studies must be obtained to rule out other primary bleeding disorders, such as hemophilia A, hemophilia B, and vitamin K deficiency, also known as hemorrhagic disease of the newborn. A PTT measures the vitamin K–dependent clotting factors VII, IX, and X and prothrombin. Factor VIII is deficient in hemophilia A. Factor B, also referred to as Christmas factor, is deficient in hemophilia B. The outcomes of these tests guide effective treatment strategies of the infant with a subgaleal hemorrhage.

Cranial ultrasound does not offer optimal views of the brain when scalp swelling is massive, as with a subgaleal hemorrhage. Computed tomography (CT) or magnetic resonance imaging (MRI) provides better images when massive swelling is involved.[37] Figure 7 shows an MRI demonstrating blood in the subgaleal space. A diagnosis can be made based on clinical conditions, but a CT or MRI is recommended to confirm the diagnosis.[13]

Differential Diagnoses

Two other scalp injuries seen in the newborn are caput succedaneum and cephalohematoma (see Figure 5). These scalp injuries differ from a subgaleal hemorrhage in several ways. Table 2 lists the three injuries and defining characteristics of each.

A caput succedaneum contains a collection of serosanguineous fluid in the subcutaneous tissue of the fetal scalp that subsides soon after delivery.[2] It is located where the vacuum cup attaches to the scalp or overlying the presenting part of the scalp in a vertex delivery. Caputs differ from subgaleal hemorrhages in that they have distinct borders that do not enlarge after delivery, and they are not fluctuant.[13]

A cephalohematoma is a collection of blood beneath the periosteum of a cranial bone.[2] Because it is located beneath the periosteum, it is unable to cross suture lines and is therefore a self-limiting hemorrhage.[1] It is usually unilateral and firm and is often located over the parietal bone. A cephalohematoma increases in size over a 24-hour period. The self-limiting size and firm rather than fluctuant consistency distinguish it from a subgaleal hemorrhage.[4]

Complicating diagnosis is the occurrence of a caput and/or cephalohematoma in addition to a subgaleal hemorrhage.[24] Often a subgaleal hemorrhage is diagnosed retrospectively 12 to 24 hours after delivery, when a caput begins to take on other characteristics, such as diffuse borders and increasing size.[6] Diagnostic and lab tests may aid in the diagnosis of a suspected subgaleal hemorrhage.

TABLE 2 ■ Birth Injuries of the Head

Injury	Incidence	Sutures and Scalp	Birth History	Associated Symptoms and Treatment	Prognosis
Cephalo-hematoma	4–25 in 1,000 deliveries	Does not cross suture lines Firm, fluctuant mass Usually unilateral Usually located over parietal bone No discoloration of scalp Distinct borders Increase in size at 12–24 hours	Forceps delivery Prolonged or difficult delivery More frequent in males More frequent in primiparas	Usually no other symptoms Skull fracture Severe blood loss (rare) Intercranial hemorrhage (rare) Infection (rare) Phototherapy for hyperbilirubinemia	Resolves in 2 weeks to 6 months
Caput succedaneum	Frequent	Crosses suture lines Soft, pitting, superficial edema over presenting part in vertex delivery Ecchymosis, petechia, or purpura over presenting part Maximum size at birth	Vertex delivery Vacuum extraction	Usually no other symptoms Resolves in several days Blood loss minimal	Excellent
Subgaleal hemorrhage	4 in 10,000 deliveries 64 in 10,000 vacuum extraction deliveries	Crosses suture lines Diffuse swelling progressively spreading from base of neck to orbits to ears Possible periorbital swelling (may not appear initially) Possible displacement of ears anteriorly Possible crepitus	Vacuum extraction Difficult or traumatic delivery including combined vacuum and forceps	Falling hematocrit Hypotonia Pallor Hypovolemic shock Seizures Skull fracture(s)	25% mortality rate

MANAGEMENT

Despite all efforts to prevent adverse consequences, subgaleal hemorrhages are difficult to treat and proved to be fatal approximately 16 percent of the time in the six studies evaluated in Table 1. Managing a neonate with a subgaleal hemorrhage includes being aware of the factors that place newborns at risk for this severe type of hemorrhage and being able to identify signs and symptoms early. The newborn nursery or NICU nurse should know the complete birth history of the neonate to whom care is provided. A vacuum-assisted delivery should alert the nurse to monitor for signs of a subgaleal hemorrhage, such as a fluctuant mass that straddles cranial sutures, fontanels, or both.[13] Any delivery that includes multiple attempts and/or the use of forceps in combination with a vacuum device requires the bedside nurse to monitor the neonate vigilantly for the first 48 hours.

Health care professionals must not be lulled into complacency by a neonate who is breathing unassisted on room air and/or whose initial lab work is normal. Signs and symptoms of subgaleal hemorrhage do not always present immediately; the infant may compensate for small amounts of acute bleeding.[5] If a subgaleal hemorrhage is suspected, therapy must include replacing blood volume, treating for shock to maintain adequate organ perfusion, treating neurologic disturbances if present, and managing coagulation disorders to stop bleeding.

Treatment of the Infant

Restoring circulating blood volume is crucial in treating an infant with a subgaleal hemorrhage. Blood products should be administered as quickly as possible to replace volume and correct the anemia caused by a falling hematocrit. A rough calculation for replacing blood lost into the subgaleal space is approximately 40 ml of blood for every 1 cm increase in frontal occipital circumference (FOC).[16]

Amar and colleagues present a case in which an infant with a subgaleal hemorrhage underwent surgery to evacuate blood from the subgaleal space after the FOC had increased by 9 cm. During the surgery, 150 ml of blood were evacuated, and 200 ml of blood were subsequently diverted through a Jackson-Pratt drain.[1] This correlates closely with the calculation of 40 ml of blood replacement for every 1 cm of increased FOC.

The infant who has developed hypovolemic shock may require inotropic support during restoration of circulating blood volume. A decreased heart rate, improved capillary refill, improved color, increased arterial pressure, and improved urine output indicate effective inotropic support. Metabolic acidosis is common in hypovolemic shock and DIC. This results from inadequate tissue perfusion and accumulation of acid products from anaerobic metabolism.[13] Sodium bicarbonate may be needed to correct the metabolic acidosis. Fluid replacement may also aid the infant in metabolizing excess acids.[38]

Intra- and extracranial compression from subgaleal hemorrhages can result in neurologic deterioration of the infant and may require hyperventilation to counteract cerebral edema.[1] Hyperventilation has the potential to lower intracranial pressure via reflex vasoconstriction.[39] A CT scan of the head aids the clinician in evaluating the severity of the hemorrhage and the possible brain compression secondary to it. In cases involving increased intracranial pressure, a neurosurgical consultation is obtained so that the hemorrhage can be evaluated. Although pressure bandages were at one time suggested as therapy, they are not recommended in situations of cerebral edema and are no longer indicated in general.[1] Phenobarbital can be administered for seizure, or suspected seizure activity.

Treating an infant with a coagulation disorder is complicated. Early detection and prompt medical intervention are important to stop the bleeding and thus to reduce morbidity and mortality. The primary goal in treating the infant who has developed DIC is to correct the underlying cause of the subgaleal hemorrhage. Additional therapies include transfusions with different blood products: Packed red blood cells are administered to correct anemia, fresh frozen plasma (FFP) to replace clotting factors, cryoprecipitate to provide a higher concentration of factor VIII and fibrinogen per unit volume than FFP, and platelet concentrate to correct ongoing bleeding and thrombocytopenia and to maintain platelet counts between 20,000 and 50,000/mm^3.[33,36,40] Exchange transfusions may be necessary in severe cases of DIC and to avoid volume overload.[36] Thrombosis can be as problematic as the bleeding aspects of DIC. Although heparin therapy is used to treat DIC in adults, it is controversial and usually not recommended as routine treatment for DIC in the neonate.[36]

Specific strategies for monitoring the neonate at risk for a subgaleal hemorrhage include the following:

- Checking head circumference on admission and following up, with documentation, every 8 hours for 48 hours
- Periodically assessing and documenting findings of the scalp for diffuse (spreading) swelling covering any portion of the cranial vault from the bridge of the nose to the base of the neck and from ear to ear, location of fluid in relation to cranial sutures and fontanels and ability of fluid to shift dependently, pitting of scalp, crepitus, and ecchymosis
- Checking for fluid fluctuance of the scalp, which becomes tense, and for pitting
- Watching for bluish-black bruising of the cranial vault, especially in the frontal and suboccipital areas
- Educating parents regarding hallmark signs for any infant delivered by vacuum extraction and discharged less than 48 hours after birth
- Assessing for signs of hypovolemia: lethargy, hypotonicity, pallor, increased heart rate, increased respiratory rate, and decreased urine output; if present, frequently monitoring blood pressure and checking blood gases to monitor respiratory status
- Watching for falling hematocrit
- Monitoring for signs of diffuse bleeding, which may indicate a coagulopathy
- Monitoring for development of oozing from needle stick sites and umbilicus
- Watching for neurologic disturbances: irritability, lethargy, weak suck, hypotonia, seizures, bulging fontanel, tachypnea

Family-Centered Care

Families anticipate and prepare for the arrival of a new baby for many months. Few are prepared for the birth of a critically ill infant. Caring for the family of the critically ill infant, an important aspect of nursing, requires a great deal of skill. The nurse must be able to appropriately interpret behaviors and emotions that surround the birth of the critically ill infant and help these families adjust to the crisis situation.

Family-centered neonatal care, based on open and honest communication among parents and health care professionals, is standard in many NICUs. Because of the high morbidity and mortality rates associated with subgaleal hemorrhages, parents must have the same facts and interpretation of those facts as health care professionals. This information must be complete, specific, detailed, and meaningful.[41]

SUMMARY

Although the possibility of developing a subgaleal hemorrhage is small, vacuum extraction—even when performed within recommended guidelines—is an important risk factor associated with the condition. Controversy exists in regard to the safety of vacuum extraction devices. Birth histories that include vacuum extraction should alert the bedside nurse to monitor the neonate for signs and symptoms of a subgaleal hemorrhage. Early recognition and treatment are vital to reduce morbidity and mortality. The nurse caring for the infant with a subgaleal hemorrhage who has developed DIC must utilize knowledge of both conditions and think critically to assist the infant and the family.

REFERENCES

1. Amar, A. P., Aryan, H. E., Meltzer, H.S., & Levy, M. L. (2003). Neonatal subgaleal hematoma causing brain compression: Report of two cases and review of the literature. *Neurosurgery Online, 52,* 1470–1481.
2. Uchil, D., & Arulkumaran, S. (2003). Neonatal subgaleal hemorrhage and its relationship to delivery by vacuum extraction. *Obstetrical and Gynecological Survey, 58,* 687–693.
3. Ross, M. G., Fresquez, M., & El-Haddad, M. A. (2000). Impact of FDA advisory on reported vacuum assisted delivery and morbidity. *The Journal of Maternal-Fetal and Neonatal Medicine, 9,* 321–326.
4. Plauche, W. (1980). A complication of instrumental delivery. *Journal of the American Medical Association, 244,* 1597–1598.
5. Chadwick, L. M., Pemberton, P. J., & Kurinczuk, J. J. (1996). Neonatal subgaleal haematoma: Associated risk factors, complications and outcome. *Journal of Paedatric Health, 32,* 228–232.

6. Ng, P. C., Siu, Y. K., & Lewindon, P. J. (1995). Subaponeurotic haemorrhage in the 1990s: A 3-year surveillance. *Acta Paediatrica, 84,* 1065–1069.
7. Teng, F., & Sayre, J. (1997). Vacuum extraction: Does duration predict scalp injury? *Obstetrics and Gynaecology, 89,* 281–285.
8. Gebremariam, A. (1999). Subgaleal haemorrhage: Risk factors and neurological and developmental outcomes in survivors. *Annals of Tropical Paediatrics, 19,* 45–50.
9. Food and Drug Administration. (1998). FDA public health advisory: Need for caution when using vacuum assisted delivery devices. Retrieved July 18, 2005, from http://www.fda.gov/cdrh/fetal598.html.
10. Seery, G. (2002). Surgical anatomy of the scalp. *Dermatologic Surgery, 28,* 581–587.
11. Davis, D. (2001). Neonatal subgaleal hemorrhage: Diagnosis and management. *Canadian Medical Association Journal, 164,* 1452–1454.
12. Plauche, W. (1979). Fetal cranial trauma due to vacuum extraction. *Obstetrics and Gynecology, 53,* 750–757.
13. Cavlovich, F. (1995). Subgaleal haemorrhage in the neonate. *Journal of Obstetric, Gynecologic, and Neonatal Nursing, 25,* 397–404.
14. Eliacher, E., Bret, A. J., Bardiaux, M., Tassy, R., Phealpin, J., & Schneider, M. (1963). Hematome sous-cutane cranien du nouveau-ne. *Societe Francaise de Pediatrie, 20,* 1105–1111.
15. Moloney-Harmon, P. (1991). Initial assessment and stabilization of the critically injured child. *Critical Care Nursing Clinics of North America, 3,* 399–409.
16. Robinson, R., & Rossiter, M. (1968). Massive subaponeurotic haemorrhage in babies of African origin. *Archives of Disease in Childhood, 43,* 684–687.
17. Benedetti, T. J. (1999). Birth injury and method of delivery. *The New England Journal of Medicine, 341,* 1758–1759.
18. Putta, L. V., & Spencer, J. P. (2000). Assisted vaginal delivery using the vacuum extractor. *American Family Physican, 62,* 1316–1320.
19. Vacca, A. (2004). Vacuum-assisted delivery: Improving patient outcomes and protecting yourself against litigation. *OBG Management* (Suppl), S1–S7.
20. Bird, G. (1982). The use of the vacuum extractor. *Clinical Obstetrics and Gynecology, 9,* 641–661.
21. Kozak, L. (2002). US trends in obstetric procedures, 1990–2000. *Birth, 29,* 157–161.
22. Health Protection Branch, Health Canada. (1999). The use of vacuum assisted delivery devices and fetal subgaleal haemorrhage. Retrieved September 19, 2005, from http://www.hc-sc.gc.ca/dhp-mps/medeff/advisories-avis/prof/1999/alert-110_nth-ah_e.html.
23. Towner, D., Castro, M. A., Eby-Wilkens, E., & Gilbert, W. M. (1999). Effect of mode of delivery in nulliparous women on neonatal intracranial injury. *The New England Journal of Medicine, 314,* 1709–1714.
24. Florentino-Pineda, I., Ezhuthachan, S. G., Sineni, L. G., & Kumar, S. P. (1994). Subgaleal hemorrhage in the newborn infant associated with silicone elastomer vacuum extractor. *Journal of Perinatology, 14,* 95–100.
25. Hall, S. (1992). Simultaneous occurrence of intracranial and subgaleal hemorrhages complicating vacuum extraction delivery. *Journal of Perinatology, 12,* 185–187.
26. McQuivey, R. (2004). Vacuum-assisted delivery: A review. *The Journal of Maternal-Fetal & Neonatal Medicine, 16,* 171–179.
27. Ahuja, G. L., Willoughby, M. L., Kerr, M. M., & Hutchison, J. H. (1969). Massive subaponeurotic haemorrhage in infants born by vacuum extraction. *British Medical Journal, 3,* 743–745.
28. Barrow, E., & Peters, R. L. (1968). Exsanguinating haemorrhage into the scalp in newborn infants. *South African Medical Journal, 42,* 265–267.
29. Kelley, D. (2005). Hypovolemic shock: An overview. *Critical Care Nursing Quarterly, 28,* 2–19.
30. Diehl-Oplinger, L., & Kaminski, M. (2004). Choosing the right fluid to counter hypovolemic shock. *Nursing, 34,* 52–54.
31. Rimar, J. (1988). Recognizing shock syndromes in infants and children. *MCN. The American Journal of Maternal Child Nursing, 13,* 32–37.
32. LaPointe, L., & Von Rueden, K. (2002). Coagulopathies in trauma patients. *AACN Clinical Issues, 13,* 192–203.
33. Dressler, D. (2004). Coping with a coagulation crisis. *Nursing, 34,* 58–62.
34. Emery, M. (1992). Disseminated intravascular coagulation in the neonate. *Neonatal Network, 11*(8), 5–14.
35. Levi, M. (2004). Current understanding of disseminated intravascular coagulation. *British Journal of Haematology, 124,* 567–576.
36. Williams, M. D., Chalmers, E. A., Gibson, B. E., Haemostasis and Thrombosis Task Force, & British Committee for Standards in Haematology. (2002). The investigation and management of neonatal haemostasis and thrombosis. *British Journal of Haematology, 119,* 295–309.
37. King, S., & Boothroyd, A. (1998). Cranial trauma following birth in term infants. *The British Journal of Radiology, 71,* 233–238.
38. Fraser Askin, D. (1997). Interpretation of neonatal blood gases, Part II: Disorders of acid base balance. *Neonatal Network, 16*(6), 23–29.
39. Stocchetti, N., Maas, A., Chieregato, A., & van der Plas, A. (2005). Hyperventilation in head injury: A review. *Chest, 127,* 1812–1827.
40. Kuehl, J. (1997). Neonatal disseminated intravascular coagulation. *The Journal of Perinatal & Neonatal Nursing, 11,* 69–77.
41. Harrison, H. (1993). The principles for family-centered neonatal care. *Pediatrics, 92,* 643–650.

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