Intraventricular Hemorrhage and Posthemorrhagic Ventricular Dilation: Current Approaches to Improve Outcomes

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Abstract

Intraventricular hemorrhage (IVH) and posthemorrhagic ventricular dilation (PHVD) are important complications of prematurity with short- and long-term implications for the patient and for nursing care. Several approaches have been shown to reduce the incidence of IVH and, more recently, mitigate the impact of IVH on long-term neurodevelopment. This article discusses the pathophysiology of IVH, with a focus on prevention strategies. Posthemorrhagic ventricular dilation is a common complication of severe IVH and has implications for neurodevelopmental sequelae. Both surgical and nonsurgical interventions for PHVD are described.

Keywords: IVH; neurology; PHVD; VP shunt

INTRAVENTRICULAR HEMORRHAGE (IVH), ALSO referred to as germinal matrix hemorrhage, is one of the most common and significant causes of brain injury in preterm infants.[1] The incidence of IVH of any grade in preterm infants is approximately 45 percent.[2] Despite improvements in care of the preterm infant, the incidence of severe IVH has not substantially declined in the last two decades, particularly in infants born between 22 and 25 weeks' gestational age (GA).[3] The long-term consequences of severe IVH are considerable and include increased mortality and morbidity, and poor neurodevelopmental outcomes.[4]

Severe IVH can lead to posthemorrhagic ventricular dilation (PHVD). The management and treatment of PHVD is important because early management may have a positive impact on neurodevelopmental outcome.[5] The purpose of this article is to review the pathophysiology of IVH and PHVD, provide medical and surgical strategies for optimal treatment, and discuss interventions for prevention of IVH. Nursing considerations for preterm patients with IVH and PHVD are provided as well as postoperative management implications.

VENTRICULAR SYSTEM PHYSIOLOGY

The ventricular system is comprised of the lateral ventricles (right and left), which drain into the third ventricle and subsequently

FIGURE 1 ■ Ventricular system anatomy.
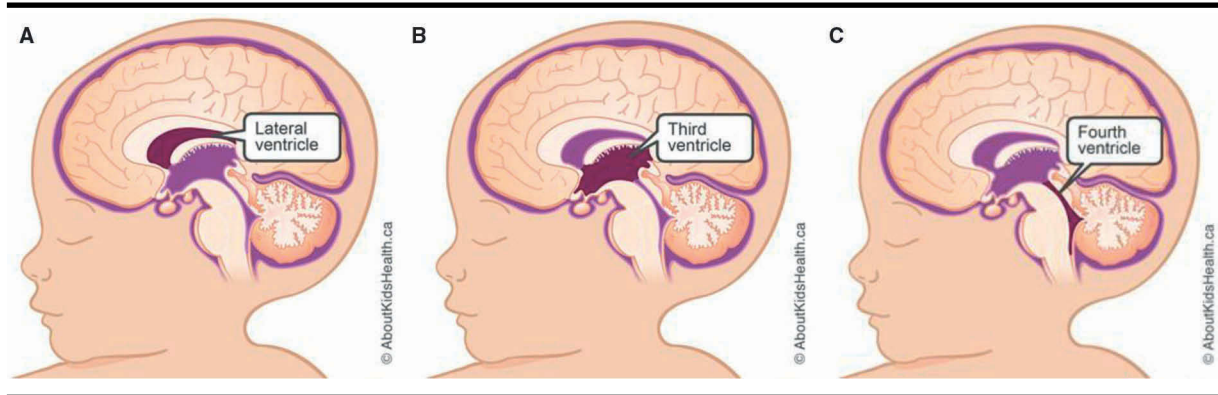
1A. Lateral ventricles house the choroid plexus where CSF is formed.
1B. The third ventricle drains through the aqueduct of Sylvius into the fourth ventricle.
1C. The fourth ventricle drains CSF to the spinal cord.
Abbreviation: CSF = cerebrospinal fluid.
Images used with permission the Hospital for Sick Children, www.aboutkidshealth.ca.

the fourth ventricle, proximal to the cerebellum (Figure 1). Cerebrospinal fluid (CSF) is formed in the choroid plexus and flows through the lateral ventricles to the third ventricle. The aqueduct of Sylvius separates the third and fourth ventricles. Obstruction can occur at any point along the ventricular system. A blockage which prevents the flow of CSF results in noncommunicating hydrocephalus. Communicating hydrocephalus occurs when CSF passes through the ventricles freely but is improperly absorbed by the cerebral and cerebellar subarachnoid processes.[6–8]

PATHOPHYSIOLOGY OF IVH

The risk of IVH is inversely related to the preterm infant's GA, with the most premature infants at highest risk. Although survival rates have improved over the last decade, the incidence of IVH remains high especially in very preterm infants.[8] The germinal matrix is the source of neuronal stem cells and over time, it matures and involutes. It is nearly nonexistent by 34–36 weeks' gestation, and therefore the risk of IVH is dramatically reduced by term.[8]

Preterm infants are at risk for IVH when there are fluctuations in cerebral blood flow. Lower systemic perfusion and CBF followed by increased perfusion and CBF precede the development of IVH.[9] These fluctuations are largely due to interventions such as intubation, ventilation and the use of high oxygen concentrations, during resuscitation in the delivery room. Fluctuations may also be caused physiological factors such as hypotension, patent ductus arteriosis and hypercarbia, post resuscitation.[8,9]

Although term infants rarely develop IVH, there are some conditions that may predispose them to ventricular bleeding, including coagulation abnormalities, collagen gene mutation, arteriovenous malformation, and sinovenous thrombosis.[10]

TIMING AND CLINICAL PRESENTATION

The most common time period for the development of IVH is within the first three days of life.[8] Fifty percent of IVH occurs on the first day, 25 percent on the second day, and 15 percent on the third day. According to Canadian Neonatal Network data, more than 50 percent of patients <25 weeks' gestation in Canada develop IVH (Figure 2).[11]

The majority of IVH is asymptomatic (silent) and only detected by routine ultrasound screening. Early clinical signs of IVH include irritability, anemia, or a sudden clinical deterioration when IVH is catastrophic. Less common later signs could include a full fontanel, rising head circumference, or seizures.[8]

INTRAVENTRICULAR HEMORRHAGE GRADING

Cranial ultrasound is the most reliable screening tool to detect IVH and to determine the severity of the bleeding.[12] Once identified, IVH is classified according to a grading scale developed by Papile in 1978[13] and later adapted by Volpe.[8] The most significant change to Papile's original grading system is in the description of a Grade IV IVH (Table 1).

Grade 1 IVH is bleeding that is restricted to the germinal matrix area. This classification may also be referred to as subependymal hemorrhage, because the bleeding occurs within the subependymal zone. Grade II IVH is described as a germinal matrix hemorrhage that extends into the ventricle but fills <50 percent of the lateral ventricles and does not cause ventricular dilation. The neurodevelopmental outcomes for infants with Grade I and II IVH are similar to those without IVH. Outcomes are specifically related to additional findings of white matter and cerebellar injury.[14]

Grade III and periventricular hemorrhagic infarction (PVHI) are more severe forms of IVH that can lead to PHVD and white matter injury with less favorable

Neuroimaging findings: GA <33 weeks
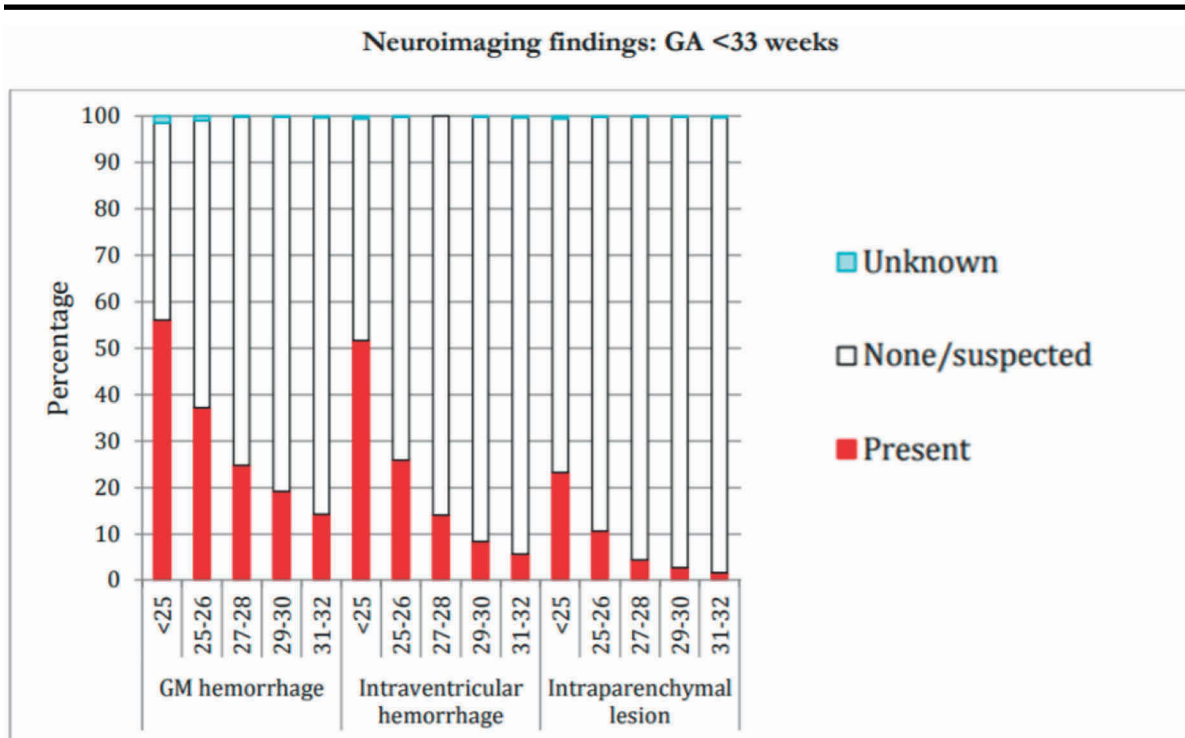
Note: Canadian Neonatal Network Data showing that the incidence of IVH is inversely proportional to lower gestational age groups.
Abbreviation: IVH = intraventricular hemorrhage.
Source: Shah PS, Yoon EW, Chan P and Members of the Annual Report Review Committee on behalf of the Canadian Neonatal Network Investigators. Canadian Neonatal Network Annual report 2017, page 33. Available at http://www.canadianneonatalnetwork.org/Portal/LinkClick.aspx?fileticket=XhPMIxFgc2M%3d&tabid=39. Accessed on Oct. 9, 2017. Published by the Canadian Neonatal Network, Toronto, Canada.

neurodevelopmental outcomes. In Grade III IVH, the hemorrhage extends to greater than 50 percent of the ventricle with associated dilation of the ventricle.[8]

Periventricular hemorrhagic infarction has been previously referred to as Grade IV IVH. Periventricular hemorrhagic infarction occurs when blood vessels in the area immediately surrounding the ventricles rupture as a result of pressure within the ventricles (Figure 3a). This bleeding causes damage to the white matter of the brain. As the blood is reabsorbed by the body, necrotic areas are left and cysts form in those areas (Figure 3b). These are classified as porencephalic cysts. They are formed rapidly following a PVHI and are contiguous with the ventricles, thus causing further ventricular dilation and increased CSF production, resulting in hydrocephalus. The evolution of porencephalic cysts which communicate with the ventricular system and/or the subarachnoid space, is different from periventricular leukomalacia, which occurs after periventricular white matter injury and develops into fluid-filled spaces.[15,16]

PREVENTION OF IVH

Several antenatal interventions as well as specific strategies at the time of delivery have been recommended to provide protection from IVH. Many centers have developed protocols for the care of preterm infants following delivery; some have proven to be effective in the improvement of long-term outcomes and the prevention of IVH, while others remain controversial.
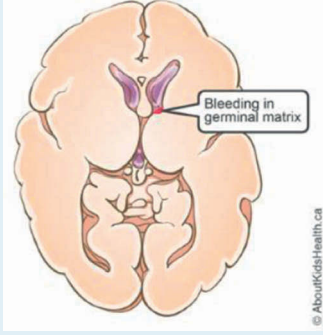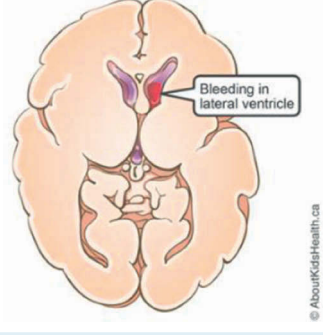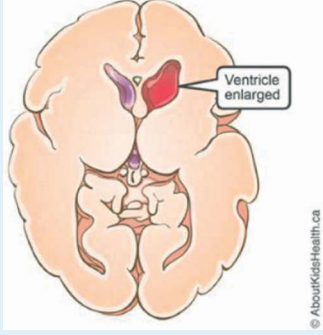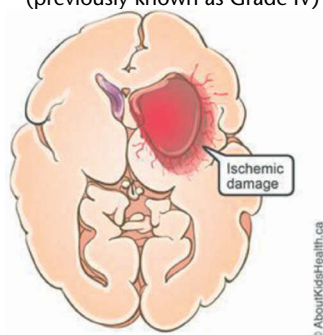
Prenatal Prevention

Glucocorticoids given to a mother prior to delivery lower the incidence of severe IVH, because of vasoconstriction in the fetal brain, which may provide neuroprotection; the protective effect is enhanced further when the course of steroids is completed at least 48 hours prior to delivery.[17,18] Magnesium sulfate given to the mother prior to delivery has not been shown to reduce the incidence of IVH but may be neuroprotective, because of anti-inflammatory effects.[18–20] The Society of Obstetricians and Gynaecologists of Canada recommends consideration of treatment with magnesium sulfate for mothers whose fetus is <34 weeks' gestation and who are expected to deliver within 24 hours. The American College of Obstetrics and Gynecology recommends treatment for mothers of fetuses <32 weeks' gestation.[20,21]

Delivery in a perinatal center is preferred because transport of the preterm infant from hospital to hospital may increase the risk of IVH.[22] Perinatal centers have experienced

TABLE 1 ■ IVH Grading and Description

Type of Hemorrhage	Description
Mild (Grade I)	Subependymal region and/or germinal matrix
Moderate (Grade II)	Bleed extends into the ventricles, filling 10%–40% of the ventricular system with little or no ventricular enlargement
Severe (Grade III)	Intraventricular bleed filling 50% or more of the ventricular system with or without ventricular dilation
Periventricular hemorrhagic infarction (previously known as Grade IV)	Extensive intraventricular hemorrhage with ventricular dilation and extension into the parenchyma of the brain with venous infarction

Abbreviation: IVH = intraventricular hemorrhage.
Images used with permission from the Hospital for Sick Children www.aboutkidshealth.ca.
Source: Inder TE, Perlman, JM, Volpe JJ. Preterm intraventricular hemorrhage/posthemorrhagic hydrocephalus. In: Volpe JL, Inder TE, Barra BT, et al., eds. *Volpe's Neurology of the Newborn.* 6th ed. Philadelphia, PA: Elsevier; 2018:637–695.

personnel who can provide timely, skilled interventions for these fragile patients, thus reducing the risk of IVH.[18,22]

Delayed cord clamping to enhance perfusion to the newborn's organs is another intervention that has been shown to reduce acute brain injury in preterm infants.[18] Delayed cord clamping of 30–60 seconds is recommended for all infants who do not need immediate resuscitation. In preterm patients requiring immediate interventions, optimal duration of cord clamping remains unclear.[18,23]

Umbilical cord milking is another potential intervention intended to reduce the risk of IVH. While thought to have a similar effect to delayed cord clamping, a recent study in extremely preterm infants suggests an increased risk of severe IVH and extreme caution should be used if considering this intervention.[24]

Prophylactic indomethacin is a treatment used to reduce the incidence of patent ductus arteriosus and IVH. A Cochrane review assessing the benefits of prophylactic indomethacin found a significant reduction in the incidence of IVH including severe IVH[25]; however, an earlier study showed no improvement in long-term neurodevelopmental outcomes.[26]

Clinical Practice Implications for Prevention of IVH

NICUs are increasing adopting preventive strategies with the intention of reducing the incidence of IVH.[27] While there is no single intervention to prevent IVH, combinations of measures (bundles) have been developed which have shown some promise in preventing IVH.[28] Postnatal interventions focus on avoiding fluctuations in CBF and promoting autonomic stability.[18] Nurses play an important role in managing interventions that will be beneficial to prevent IVH, particularly in the first 72 hours of life when preterm infants are most vulnerable. Most IVH "brain care bundles" focus on this critical time period. Key nursing interventions in brain bundles include: midline head position and elevating the head of the bed, minimal handling, reducing painful procedures, and hemodynamic and respiratory stability (Table 2).

Midline and Elevated Head Positioning. Routine care of preterm infants includes repositioning. However, during the first 72 hours of life, when IVH is most prevalent, maintaining the head in a neutral position may avoid variation in CBF that may lead to IVH. Studies have suggested that turning the head 90 degrees to the side can alter CBF, causing a decrease in jugular blood flow.[29,30] While some NICUs have recommended midline positioning as part of neuroprotective strategies for preterm infants,[31,32] a 2017 systematic review concluded that midline positioning was neither beneficial or detrimental in reducing IVH.[33] Additional research is required to answer this question. However, midline positioning is a simple strategy that can be accomplished using assistive products such as gel pillows. With this intervention, extremely premature infants should not be positioned prone but can be positioned supine or side-lying while maintaining a neutral midline position.

FIGURE 3 ■ Periventricular hemorrhagic infarction and subsequent porencephalic cyst formation.
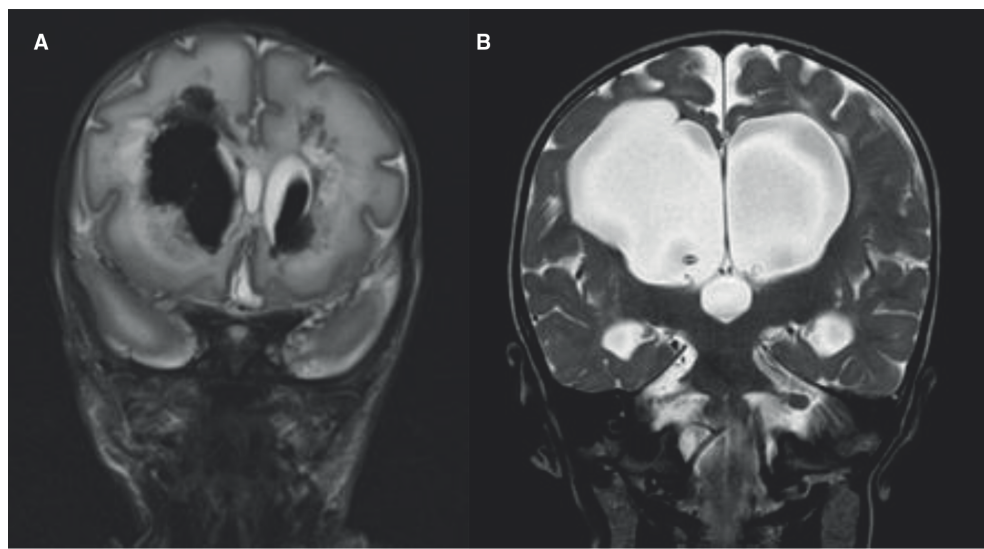
3A. MRI image of right-sided periventricular hemorrhagic infarction in a preterm infant. Note the significant dilation of the right ventricle and the fan-shaped appearance of the infarction.

3B. MRI image of the same patient with porencephalic cyst development by term equivalent age.

Abbreviation: MRI = magnetic resonance imaging.
Courtesy of the Hospital for Sick Children, Toronto, Ontario.

TABLE 2 ■ Clinical Practice Strategies for Neuroprotection in the First 72 Hours of Life

- Midline head position
- Elevating the head of the bed
- Minimal handling/gentle care approach
- Minimizing painful procedures
- Maintaining cerebral autoregulation
- Avoid hypotension/hypoperfusion
- Avoid hypercapnia and hypocapnia

Source: Ryan M, Lacaze-Masmonteil T, Mohammad K. Neuroprotection from acute brain injury in preterm infants. *Paediatr Child Health.* 2019;24(4):276–282.

Elevated head positioning, defined as the head of the bed elevated 30 degrees, is an intervention done simultaneously with midline head positioning.[33] The rationale for this strategy is that it reduces cerebral venous pressure and improves oxygenation.[29,30,34] Nurses should avoid lowering the head of the bed for routine procedures and diagnostic tests such as x-rays. Two studies examining the combination of these two strategies continued for 96 hours, found a reduction in the progression to PVHI in extremely low birth weight infants <1,000 grams, but not an overall reduction in the incidence of IVH.[33,35] Maintaining a midline, neutral head position with the head of the bed at 30 degrees for the first 72–96 hours, is an important strategiy that may prevent progression of IVH and provide a visual cue for minimal handling and care with positioning

Minimal Handling/Pain Management. In preterm infants, minimal handling can promote autonomic stability. Minimal handling refers to a gentle approach in caring for extremely premature infants, minimizing care and procedures. While not directly shown to reduce IVH, indirect activities that reduce stress and promote neurodevelopment may be beneficial during the first 72-hour period and beyond. Minimizing stimulation and routine procedures can reduce fluctuations in blood flow.[36] Painful procedures can also affect brain development. In preterm infants, painful experiences can lead to reduced white matter and subcortical gray matter.[37] Therefore, minimizing painful procedures, such as heel stick, venous blood draws, routine tracheal suctioning, lumbar puncture, and other procedures may be neuroprotective and reduce harm. The insertion of an umbilical arterial catheter that can be used to draw blood for lab tests, avoid painful heel sticks, and monitor BP is recommended in infants <26 weeks' GA.[38]

Even seemingly innocuous handling may be detrimental. For example, a study of 47 infants born at <35 weeks' gestation and weighing <2,000 grams demonstrated that diaper changes resulted in increased distress and pain scores.[39] The use of a nest for postural support during diaper changes was found to reduce signs of stress and pain.[39] Gentle diaper changes that avoid lifting by the ankles and only slightly raising the buttocks can reduce the stress of the procedure.

MAINTAINING CEREBRAL AUTOREGULATION

Preterm infants have impaired autoregulation, meaning CBF is not regulated and can fluctuate with alterations in systemic BP.[8,9] Prevention of hypotension and hypoperfusion is important in reducing fluctuations in CBF. While hypotension is not well defined, a mean arterial pressure of less than

the infant's GA is generally accepted; however, a mean BP of <30 mmHg has been associated with a higher risk of severe IVH.[40] Measures of hypoperfusion such as pulses, color, and capillary refill time should be assessed, and findings considered when determining if treatment for hypotension is required. The management of hypotension with inotropes is not recommended in the preterm infant as it is associated with brain injury.[41] Fluid boluses, a common treatment for hypotension, should be used with caution, and are not recommended for routine use in the preterm infant. It is unclear if there is a benefit in the management of hypotension.[42]

Respiratory management should include avoiding extreme hypercapnia and hypocapnia. In a retrospective review of 840 neonates weighing 401–1,250 grams at birth with a mean gestational age of 26 weeks, those with severe IVH had higher maximum CO_2 levels (median CO_2 level of 72 mmHg), and lower minimum CO_2 levels (median CO_2 level of 34 mmHg) as well as fluctuations in CO_2 levels.[43] Critical lab work, such as arterial blood gases, provide confirmation of clinical changes and provide guidance for perfusion and ventilation strategies in a timely manner. Nurses and respiratory therapists can help to regulate fluctuations in CBF by maintaining adequate ventilation and monitoring CO_2 to prevent hypo- and hypercarbia and to promptly recognize acute respiratory changes.

POSTHEMORRHAGIC VENTRICULAR DILATION

Posthemorrhagic ventricular dilation occurs when infants with severe IVH, typically Grade III or more, develop ventricular dilation that causes pressure on the surrounding tissue leading to damage to the white matter. PHVD occurs in 30–50 percent of infants with Grade III or IV IVH.[5,8] In 30–40 percent of infants PHVD will resolve without intervention while approximately 60 percent will require intervention.[5,44] The speed at which PHVD develops depends on the severity of the initial IVH. Some infants develop PHVD in the days following IVH, while in others it may take weeks to develop.[8] In obstructive PHVD (noncommunicating) a clot following the hemorrhage may cause obstruction at the level of the aqueduct, disrupting CSF flow and causing a more subacute hydrocephalus. Posthemorrhagic ventricular dilation caused by an acute injury is the result of both the development of particulate clots that may impair CSF flow and impaired reabsorption of CSF.[8]

Effective treatment of PHVD may mitigate secondary brain injury, damage to the white matter of the brain, and improve long-term neurodevelopmental outcomes.[5] However, there is significant variability in the timing and approach to intervention. Some centers utilize an early intervention strategy incorporating initial lumbar puncture then surgical insertion of a ventricular access devise (VAD), such as an Ommaya reservoir if needed, to mitigate progression of PHVD and to preserve the white matter. Ventriculoperitoneal (VP) shunts are utilized only in those who have progressive PHVD after early intervention.[44] Other centers utilize a later intervention approach. The intervention is based only on symptomatology

and not on ultrasound findings or head circumference.[44] Symptoms of PHVD requiring urgent intervention because of increased intracranial pressure include fullness of the fontanels, rapid increase in head circumference, vomiting, bradycardia, or sunsetting appearance of the eyes.[6]

Diagnosis of PHVD

Serial cranial ultrasounds should be completed for surveillance of patients with progressive ventricular dilation. Surveillance is usually recommended for patients who are <32 weeks' gestation, because these infants are at the highest risk of developing PHVD.[5] Measurements of the ventricular size using ventricular index (VI) and anterior horn width (AHW) are common practice (Figure 4).[45,46] The VI is the distance between the falx and the lateral wall of the anterior horn in the coronal plane at the level of the third ventricle. The AHW is the width of the distance from the medial walls of the lateral ventricles at the widest points. Measurements are then plotted on a standardized scale, developed by Levene and Davies (Figure 5).[45,46] When patients meet a threshold where the VI, as described by Levene in 1981, enters the severely dilated zone with measurements >p97 + 2sd + 4mm and AHW, as described by Davies in 2000, reaches >10 mm, then intervention should be considered.[45,46]

With use of Doppler ultrasound, resistive index (RI) may be included in the assessment of the need for ventricular

FIGURE 4 ■ Measurement of anterior horn width and ventricular index.
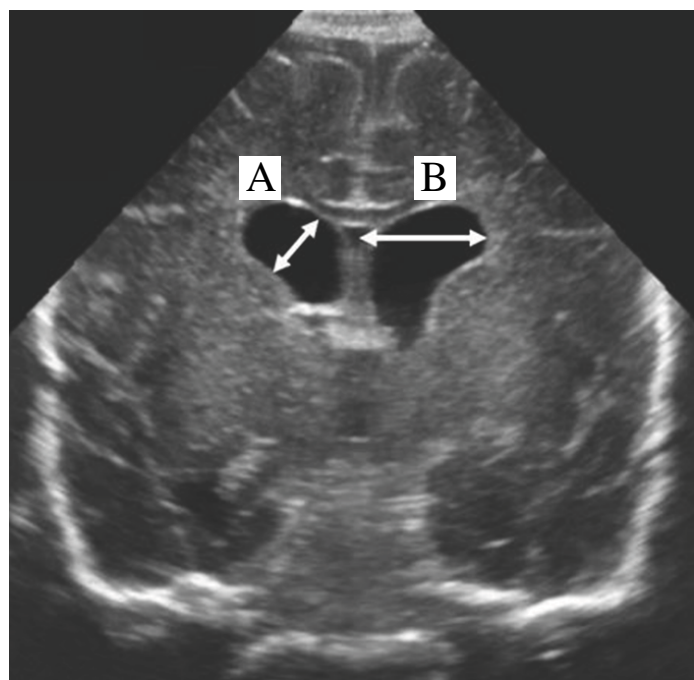
*Note:* Measurement A on the left, indicates the anterior horn width as the the largest diagonal width between the wall of the frontal horns of the lateral ventricles. Measurement B on the right indicates the ventricular index measured on the coronal plane at the level of the third ventricle, as the width from the midline to the most lateral wall of the ventricle.
Courtesy of the Hospital for Sick Children, Toronto, Ontario.

drainage. Resistive index is a calculation of the systolic and diastolic velocities measured in the cerebral arteries. Normal RI values are 0.65–0.85. High RI (>0.85) may indicate low blood flow velocity and high resistance in patients with PHVD suggesting a need for ventricular drainage.[12]

Management of PHVD

In an attempt to protect the vulnerable white matter from further injury several approaches to reducing the size of the ventricles have been developed. Both nonsurgical and surgical approaches have a role.

FIGURE 5 ■ Graph showing measurements of Levene and Davies.
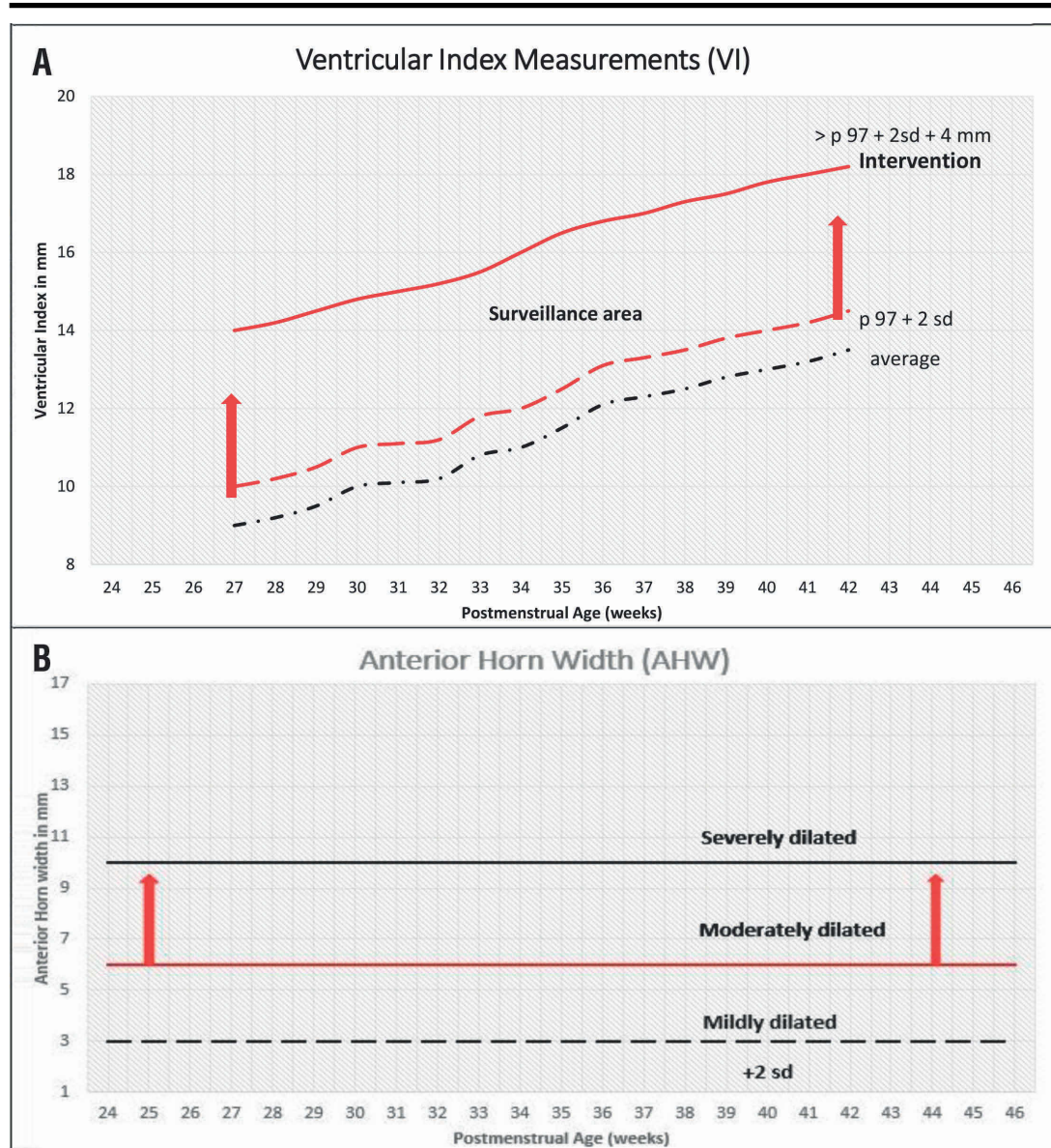
5A. Ventricular Index (VI) measured by ultrasound is plotted on the Levene Index where the black dashed line shows the average VI at each gestational age and the red dashed line indicates p97%ile +2 sd, where surveillance for PHVD should begin and the solid red line indicating p97%ile + 2 sd + 4 mm, where intervention is indicated.

5B. Anterior Horn Width (AHW) is measured by ultrasound and potted on the Davies Graph, where the black dashed line indicates the minimum dilation of the ventricles, the red line indicates moderately dilated ventricles and the black solid line indicates severely dilated ventricles.

Note: Measurements of ventricular dilation, from Levene (1981) and Davies (2000).

With permission from Levene, MI. Measurement of the growth of the lateral ventricles in preterm infants with real-time ultrasound. *Arch Dis Child*. 1981;56(12):900–904 and Davies MW, Swaminathan M, Chuang SL, et al. Reference ranges for the linear dimensions of the intracranial ventricles in preterm neonates. *Arch Dis Child Fetal neonatal Ed*, 2000; 82:F218–F223.

Nonsurgical Management. Studies have suggested that the initial nonsurgical management strategy include performing serial lumber punctures (LPs), to relieve pressure from the CSF and remove debris from the ventricles.[5,44] The goal of lumbar punctures is to remove 10 mL/kg of CSF on two to three consecutive days.[6] This procedure is normally done at the bedside under sterile conditions. Ultrasound surveillance continues during this stage of care and the measurements of VI and AHW continue to guide care. If the VI and AHW are reduced as a result of LPs, weekly ultrasound surveillance should continue until the infant is 32 weeks' GA and the ventricles are stable. If ventricular dilation persists despite serial LPs, surgical intervention is considered.[5,44]

Surgical Management. There are several options for neurosurgical management of progressive PHVD. Depending on the size and general clinical status of the patient, it may be preferable to begin with a temporizing procedure before determining the need to proceed to a more definitive surgery. This is both because the definitive treatments may not be feasible in such small patients, and because there is a subset of patients that may not go on to require permanent CSF diversion after a period of initial treatment.[4]

Management strategy of PVHD must carefully balance the risks of treatment for this vulnerable group of infants with the intended benefit.[5,47] Although there is a growing body of research showing the neurodevelopmental benefits of earlier intervention, there is not yet conclusive evidence to support one strategy over the other, leading to practice variations existing across institutions.[48,49]

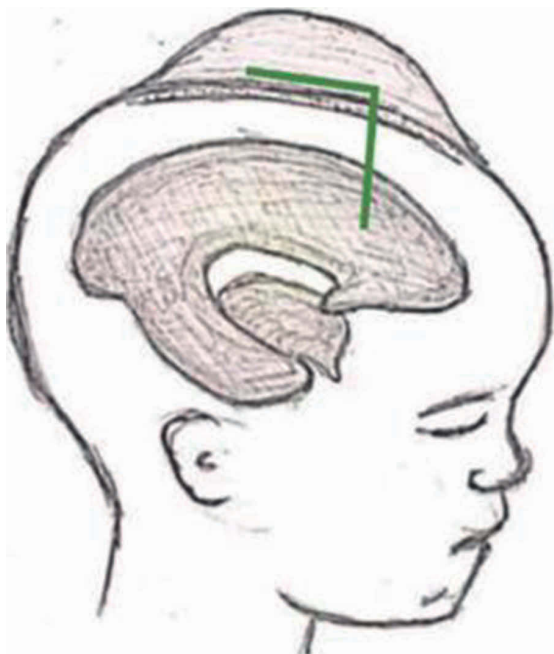**FIGURE 6** ■ Ventriculosubgaleal shunt.

Image courtesy of Stephanie Bernardo. Used with permission.

Temporizing Neurosurgical Procedures. Temporary options for the surgical management of PHVD include the placement of a ventriculosubgaleal (VSG) shunt, shown in Figure 6. A catheter is surgically placed with the proximal tip in the lateral ventricle of the brain and the distal end in a pocket in the subgaleal space, thus allowing for continuous diversion of CSF. The reported benefits of this method include decompression of the ventricular system without the need for frequent percutaneous tapping. Another benefit to the VSG shunt is the ability to maintain a closed system which can also minimize the risk of electrolyte abnormalities as it allows CSF, which contains sodium, to be reabsorbed rather than removed.[49] This method has fallen out of favor in some institutions because of complications that include the lack of control over rate of decompression, hemorrhage at the site of insertion, CSF leak, and infection.[48]

A second option is surgical placement of an external ventricular drain (EVD). This involves insertion of a catheter into the ventricular system which is tunneled under the skin and exits the scalp where it is attached to an external collection chamber. Similar to the VSG shunt, the EVD eliminates the need for percutaneous tapping.[48] A benefit of the EVD is the theoretical ability to control the rate of CSF removal by adjusting the level of the drainage chamber, though this is not always straightforward to maintain. Significant risks with EVDs in infants include infection, overdrainage, and electrolyte abnormalities related to removal of CSF.[50] There are also positioning and nursing care difficulties associated with these drains because they require frequent leveling and monitoring to ensure desired drainage parameters are maintained. Because of these risks, EVD insertion is generally reserved for specific clinical situations, such as severe meningitis, but is not standard for routine management of PHVD.[49]

The other commonly performed temporizing procedure for PHVD management is the placement of a ventricular access device (VAD).[49] This involves the surgical placement of a catheter with the proximal end in the ventricular system and the distal end attached to a silicone dome which is implanted in a subgaleal pocket. The VAD allows for serial CSF aspiration through the dome via percutaneous tap. Because this is a closed system it allows easy access to aspirate CSF when clinically indicated but does not drain spontaneously, thereby allowing full provider control over the volume removed and the frequency. Due to the need for serial taps there is the risk of infection, though this has been shown to be quite low when proper sterile technique is used.[51] There is also pain associated with tapping the VAD, although this is generally very well tolerated with nonpharmacological pain management strategies. The tapping procedure, similar to a venipuncture, involves inserting a small butterfly needle (25 gauge) directly into the VAD through the epidermis (Figure 7). Sucrose, swaddling, and containment, as well as a pacifier for nonnutritive sucking are recommended for needle puncture procedures and are well tolerated for VAD tapping.[52]

FIGURE 7 ■ Accessing a ventricular access device.
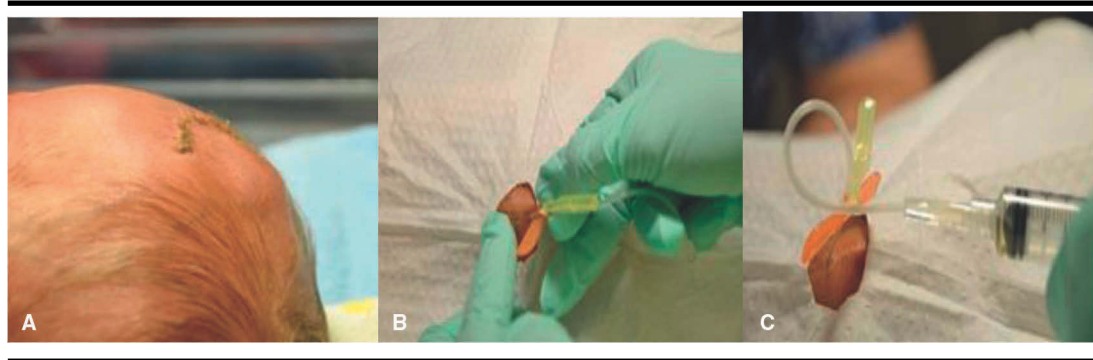
7a. Note newly inserted VAD under the skin with c-shaped incision.
7b. Insertion of butterfly needle into VAD under sterile conditions.
7c. Slow removal of CSF.

*Abbreviation:* CSF = cerebrospinal fluid.
Courtesy of the Hospital for Sick Children, Toronto, Ontario.

Regardless of which method is used, there should be ongoing reassessment of the need for, and tolerance of, temporary CSF diversion.[48] This can be done through a combination of serial cranial ultrasounds to determine ventricular size, CSF cell counts, and assessment for increased intracranial pressure by monitoring apnea and bradycardic events, measuring head circumference, and assessing fontanels.[53]

Long-Term Neurosurgical Management. Following a period of temporization, it must be determined if the infant is dependent on CSF diversion. If so, a more definitive hydrocephalus treatment is required. It is theorized that early removal of debris due to IVH may prevent permanent impairment of normal CSF absorption thereby reducing the need for long-term treatment in a subset of infants.[5] Published outcomes show that approximately 60 percent of infants treated with a temporizing procedure will go on to require long-term CSF diversion; the remainder will have no further progression of their ventricular enlargement.[48,54] If need for long-term CSF management has been shown, considerations for the timing of treatment include the infant's size, GA, comorbidities, and general stability for surgery.[55] It is generally feasible to convert to a long-term management strategy as the infant approaches term age and a weight of at least 2 kg.

The mainstay of permanent hydrocephalus treatment is insertion of a VP shunt which diverts the excess CSF from the ventricles to the peritoneal cavity where it can be absorbed.[55] This involves surgically implanting a catheter with the proximal end in the ventricular system of the brain that is connected to a valve underneath the skin which controls the flow of CSF. The valve is then connected to another catheter with the distal end in the peritoneal cavity. Enough tubing is placed on initial insertion to accommodate growth to full adult height. The tubing is tunneled entirely under the skin through incisions on the head and abdomen. It has generally been shown that shunting can be considered after a minimum weight of 2 kg has been attained because the risk of infection and hardware complications is higher in smaller infants.[56]

FIGURE 8 ■ Ventriculoperitoneal shunt.
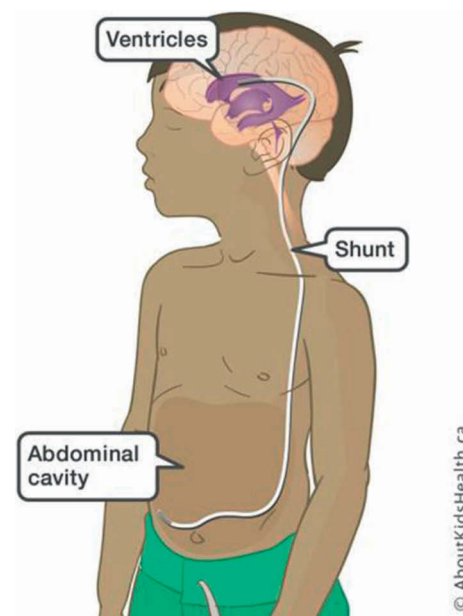
Note: The path of VP shunt from the ventricular system to the abdominal cavity.
Abbreviation: VP = ventriculoperitoneal.
Courtesy of the Hospital for Sick Children, Toronto, Ontario.

In patients who are not able to accept a catheter into the peritoneal cavity because of abdominal comorbidities such as severe necrotizing enterocolitis (NEC) there is the possibility of a ventriculoatrial (VA) shunt with the distal catheter in the atrium of the heart. VA shunts are associated with a higher rate of complications that include migration, cardiac arrhythmias, thrombus formation, and nephritis.[47,57] In older children, ventriculopleural (VPL) shunts are possible as well. This involves placement of the distal catheter in the pleural space. This technique is not tolerated well in children under

TABLE 3 ■ Surgical Nursing Implications

- Surgical incisions in the infant are at risk of infection, breakdown, and poor healing because of skin fragility and other factors.
- It is important to visualize and assess incisions regularly and ensure that they are clean.
- Minimize friction, pressure, and tension on incisions.
- Early identification of any leaking, wound breakdown, or swelling is key.

seven years of age because of a smaller pleural surface and therefore a significant risk of developing a pleural effusion and respiratory distress.[57]

Although CSF shunting is the preferable treatment, it is not without risks and complications. Despite the introduction of a strict protocol across most neurosurgical centers, there remains a 5–10 percent risk of shunt infection.[58] The risk of infection is almost exclusively within the first six months after a shunt operation though rarely infection can occur in a delayed fashion.[54] There are also the lifetime risks of symptomatic shunt malfunction because of obstruction, migration, and fractured tubing.[49] Children with shunts are more likely to have multiple hospitalizations and neurosurgical procedures over the course of their lives with 40–50 percent of children requiring at least one shunt revision by the age of two.[57]

There is an alternative surgical management option for hydrocephalus in the form of endoscopic third ventriculostomy (ETV) with or without choroid plexus cauterization (CPC). This involves an endoscopic technique to make an internal alternate pathway for CSF flow by creating a stoma in the third ventricle to divert fluid out of the ventricular system into the basal cisterns. Choroid plexus cauterization is sometimes added to ETV surgery to improve the efficacy of this procedure by cauterizing as much as possible of the choroid plexus, where CSF is produced, thus decreasing the overall circulating volume of CSF. Although this treatment is useful in some forms of hydrocephalus, at this time it remains an unfavorable treatment option in young infants with post-IVH hydrocephalus because of high failure rates. Approximately 80 percent of those treated with ETV go on to require a shunt.[59] ETV may be considered in patients that require CSF diversion at an older age. Postoperative nursing care focuses on close monitoring of the incision site and prevention of infection (Table 3).

NEURODEVELOPMENTAL OUTCOMES FOLLOWING IVH AND PHVD

The neurodevelopmental outcome for infants with IVH and PHVD depends on the size and location of the hemorrhage, the location of the parenchymal injury, and the toxic effects of blood products on brain development.[8] Cerebral palsy is a likely outcome for infants with PVHI who have experienced white matter injury. Infants with Grade III IVH have a 50 percent chance of neurodevelopmental sequelae and those with PHVI have a 75 percent risk.[8] Several approaches to reducing the size of the ventricles have been developed, in an attempt to protect the vulnerable white matter from

further injury. A recent study has suggested that the early intervention approach to PHVD improves both cognitive and motor outcomes at 18 months of age.[5] This approach needs to be evaluated further to ensure it has longer-term impact for improvement in outcomes at school age.[5]

CONCLUSION

Intraventricular hemorrhage and PHVD are significant complications of prematurity. Despite ongoing attempts to reduce the incidence and severity of IVH, there has been little impact on this global problem. In recent years, the focus has shifted to bundles of preventive measures and early intervention to mitigate complications of PHVD as a result of severe IVH.

REFERENCES

1. Ramenghi LA. Germinal matrix-intraventricular haemorrhage: Still a very important brain lesion in premature infant. *J Maternal Fetal Neonatal Med.* 2015;28:2259–2260. https://doi.org/10.3109/14767058.2013.1031952
2. Kadri H, Mawla A, Kazah J. The incidence, timing, and predisposing factors of germinal matrix and intraventricular hemorrhage (GMH/IVH) in preterm neonates. *Child's Nervous System.* 2006;22(9):1086–1090. https://doi.org/10.1007/s00381-006-0050-6
3. Stoll BJ, Hanson NI, Bell EF, et al. Trends in care practices, morbidity and mortality of extremely preterm neonates 1993–2012. *JAMA.* 2015;314:1039–1051. https://doi.org/10.1001/jama.2015.10244
4. Mukerji A, Shah V, Shah PS. Periventricular/intraventricular hemorrhage and neurodevelopmental outcomes: A meta-analysis. *Pediatrics.* 2015;136(6):1132–1143. https://doi.org/10.1542/peds.2015-0944
5. Leijser L, Miller SP, van Wezel-Meijler G, et al. Posthemorrhagic ventricular dilatation in preterm infants: When best to intervene? *Neurology.* 2018;90(8):e698–e706. https://doi.org/10.1212/WNL.0000000000004984
6. Gomella TL. Neurologic diseases. In: Gomella TL, ed. *Neonatology: Management, Procedures, On-Call Problems, Diseases and Drugs.* 5th ed. New York, NY: McGraw Hill; 2014:488–511.
7. Limperopolos C. Imaging the fetal brain. In: Shevell MI, Miller SP, eds. *Acquired Brain Injury in, the Fetus and Newborn.* London, UK: Mac Keith Press; 2012:18–28.
8. Inder TE, Perlman, JM, Volpe JJ. Preterm intraventricular hemorrhage/posthemorrhagic hydrocephalus. In: Volpe JL, Inder TE, Barra BT, et al., eds. *Volpe's Neurology of the Newborn.* 6th ed. Philadelphia, PA: Elsevier; 2018:637–695.
9. Noori, S, McCoy, M, Anderson, MP, Ramji, F, Seri, I. Changes in cardiac function and cerebral blood flow in relation to peri/intraventricular hemorrhage in extremely preterm infants. *J. Peds.* 2014;164(2):264–270. https://doi.org/10.1016/j.jpeds.2013.09.045
10. Inder TE, Perlman JM, Volpe, JJ. Intracranial hemorrhage: Subdural, subarachnoid, intraventricular (term infant), miscellaneous. In: Volpe JL, Inder TE, Barra BT, et al., eds. *Volpe's Neurology of the Newborn.* 6th ed. Philadelphia, PA: Elsevier; 2018:593–622.
11. Shah PS, Yoon EW, Chan P, Members of the Annual Report Review Committee on behalf of the Canadian Neonatal Network Investigators. Canadian neonatal network annual report 2017, Page 33. http://www.canadianneonatalnetwork.org/Portal/LinkClick.aspx?fileticket=XhPMIxFgc2M%3d&tabid=39. Accessed on Oct. 9, 2017. Published by the Canadian Neonatal Network, Toronto, Canada. Accessed on Oct. 9, 2019.
12. Meijler G, Steggerda SJ. Neonatal cranial ultrasonography: Advantages and aims. In: *Neonatal Cranial Ultrasonography.* 3rd ed. Switzerland: Springer; 2019;3–5. https://doi.org/10.1007/978-3-319-77815-0_1

N E O N A T A L   N E T W O R K

13. Papile LA, Burstein J, Burstein R, Koffler H. Incidence and evaluation of subependymal and intraventricular hemorrhage: A study of infants with birth weights less than 1500 grams. *J Pediatr*. 1978;92(4):529–534. https://doi.org/10.1016/S0022-3476(78)80282-0

14. Limperopolos C, du Plessis AJ, Volpe, JJ. Cerebellar hemorrhage. In: Volpe JL, Inder TE, Barra BT, et al., eds. *Volpe's Neurology of the Newborn*. 6th ed. Philadelphia, PA: Elsevier; 2018:623–636.

15. Neil JJ, Volpe JJ. Encephalopathy of prematurity: Clinical-neurological features, diagnosis, imaging, prognosis, therapy. In: Volpe JL, Inder TE, Barra BT, et al., eds. *Volpe's Neurology of the Newborn*. 6th ed. Philadelphia, PA: Elsevier; 2018;425–457.

16. El-Feky M, Ho MI. Porencephaly. https://radiopaedia.org/articles/porencephaly, Accessed January 15, 2020.

17. Norman M, Piedvache A, Berch K, et al. Effective prenatal intensive care in Europe (EPICE) research group. Association of short antenatal corticosteroid administration-to-birth intervals with survival and morbidity among very preterm infants. Results from EPICE cohort. *JAMA Pediatr*. 2017;171(7):678–686. https://doi.org/10.1001/jamapediatrics.2017.0602

18. Ryan M, Lacaze-Masmonteil T, Mohammad K. Neuroprotection from acute brain injury in preterm infants. *Paediatr Child Health*. 2019;24(4):276–282. https://doi.org/10.1093/pch/pxz056

19. Doyle LW, Crowther CA, Middleton P, Marret S, Rouse D. Magnesium sulphate for women at risk of preterm birth for neuroprotection of the fetus. *Cochrane Database Syst Rev*. 2009(1):CD004661. https://doi.org/10.1002/14651858.CD004661.pub3

20. American College of Obstetricians and Gynecologists Committee on Obstetric Practice, Society for Maternal-Fetal Medicine. ACOG committee opinion No. 455. Magnesium sulfate before anticipated preterm birth for neuroprotection. *Obstet Gynecol*. 2010; 115:669. Reaffirmed 2018. https://doi.org/10.1097/AOG.0b013e3181d4ffa5

21. Magee L, De Silva D, Sawchuck D, Synnes A, von Dadelszen P. Magnesium Sulfate For Fetal Neuroprotection Consensus Committee; Maternal Fetal Medicine Committee. SOGC Best Practice Guidelines. Magnesium for neuroprotection. *J Obstet Gynaecol Can*. 2019;41(4):505–522. https://doi.org/10.1016/j.jogc.2018.09.018

22. Mohamed MA, Aly H. Transport of premature infants is associated with increased risk for intraventricular haemorrhage. *Arch Dis Child Fetal Neonatal Ed*. 2010;95(6):F403–F407. https://doi.org/10.1136/adc.2010.183236

23. Rabe H, Gyte GML, 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*. 2019(9):CD003248. https://doi.org/10.1002/14651858.CD003248.pub4

24. Katheria A, Reister F, Essers J, et al. Association of umbilical cord milking vs delayed umbilical cord clamping with death or severe intraventricular hemorrhage among preterm infants. *JAMA* 2019;332(19):1877–1886. https://doi.org/10.1001/jama.2019.16004

25. Fowlie PW, Davis PG, McGuire W. Prophylactic intravenous indomethacin for preventing mortality and morbidity in preterm infants. *Cochrane Database Syst Rev*. 2010;(7):CD000174. https://doi.org/10.1002/14651858.CD000174.pub2

26. Schmidt B, Davis P, Moddemann D, et al.; Trial of Indomethacin Prophylaxis in Preterms Investigators. Long-term effects of indomethacin prophylaxis in extremely low-birth-weight infants. *N Engl J Med*. 2001;344(26):1966–1972. https://doi.org/10.1056/NEJM200106283442602

27. Hendley SC, Passarella M, Lorch SA, Lee HC. Survey of preterm neuro-centric care practices in California neonatal intensive care units. *J Perinatol*. 2019;39(2):256–262. https://doi.org/10.1038/s41372-018-0283-8

28. Schmid MB, Reister F, Mayer B, Hopfner RJ, Fuchs H, Hummler H. Prospective risk factor monotiling reduces intracranial hemorrhage rates in preterm infants. *Dtsch Arztebl Int*. 2013;110(29–30):489–496. https://doi.org/10.3238/arztebl.2013.0489

29. Goldberg RN, Joshi A, Moscoso P, Castillo T. The effect of head position on intracranial pressure in the neonate. *Crit Care Med*. 1983;11(6):428–430. https://doi.org/10.1097/00003246-198306000-00006

30. Cowen F, Thoresen M. Changes in superior sagittal sinus blood velocities due to postural alterations and pressure on the head of the newborn infant. *Pediatr*. 1985;75:1085.

31. Al-Salam Z, Al-Alaiyan S, Alallah J, et al. The golden hour approach: Practical guidelines of the Saudi neonatology society on managing very low birth weight infants in the first hour of life. *J Clin Neonatol*. 2016;5:222–229. https://doi.org/10.4103/2249-4847.194178

32. Carteaux P, Cohen H, Check H, et al. Evaluation and development of potentially better practices for the prevention of brain hemorrhage and ischemic brain injury in very low birth weight infants. *Pediatrics*. 2003;111(4): e489–e496.

33. Romantsik O, Grazia-Calevo M, Brushettini, M. Head midline position for preventing the occurrence or extension of germinal matrix-intraventricular hemorrhage in preterm infants. *Cochrane Database Syst Rev*. 2017(7):CD 012362. https://doi.org/10.1002/14651858.CD012362.pub2

34. Limperopoulous C, Gauvreau K, O'Leary H, et al. Cerebral hemodynamic changes during intensive care of preterm infants. *Pediatr*. 2008;122(5):e1006–e1013. https://doi.org/10.1542/peds.2008-0768

35. Kochan M, Leonardi B, Firestine A, et al. Elevated midline head positioning of extremely low birth weight infants: Effects on the cardiopulmonary function and the incidence of periventricular-intraventricular hemorrhage. *J. Perinatol*. 2019;39:56–62. https://doi.org/10.1038/s41372-018-0261-1

36. Ballabh P. Pathogenesis and prevention of intraventricular hemorrhage. *Clin Perinatol*. 2014;41(1):47–67. https://doi.org/10.1016/j.clp.2013.09.007

37. Brummette S, Grunau R, Chau V, et al. Procedural pain and brain development in premature newborns. *Ann Neurol*. 2012;71(3):385–396. https://doi.org/10.1002/ana.22267

38. Shahid S, Dutta S, Symington A, Shivananda S. Standardizing umbilical catheter usage in preterm infants. *Pediatrics*. 2014;133(6):e1742-e1752. https://doi.org/10.1542/peds.2013-1373

39. Comaru F, Miura E. Postural support improves distress and pain during diaper change in preterm. *J Perinatol*. 2009;29:504–507. https://doi.org/10.1038/jp.2009.13

40. St Peter D, Gandy C, Hoffman SB. Hypotension and adverse outcomes in prematurity: Comparing definitions. *Neonatology*. 2017;111(3):228–233. https://doi.org/10.1159/000452616

41. Abdul Aziz AN, Thomas S, Murthy P, et al. Early inotropes use is associated with higher risk of death and/or severe brain injury in extremely preterm infants. *J Matern Fetal Neonatal Med*. 2019:1–8. https://doi.org/10.1080/14767058.2018.1560408

42. Osborne D, Evans NJ. Early volume expansion for prevention of morbidity and mortality in very preterm infants. *Cochrane Database Syst Rev*. 2009;3:1–69.

43. Fabres J, Phillips V, Howard G, et al. Both extremes of arterial carbon dioxide and the magnitude of fluctuation in arterial carbon dioxide pressure are associated with severe intraventricular hemorrhage. *Pediatrics*. 2007;119(2):299–305. https://doi.org/10.1542/peds.2006-2434

44. De Vries LS, Liem KD, van Dijk K, et al. Early versus late treatment of posthaemorrhagic ventricular dilatation: Results of a retrospective study from five neonatal intensive care units in The Netherlands. *Acta Paediatrica*. 2002;91(2):212–217. https://doi.org/10.1111/j.1651-2227.2002.tb01697.x

45. Levene, MI. Measurement of the growth of the lateral ventricles in preterm infants with real-time ultrasound. *Arch Dis Child.* 1981;56(12):900–904. https://doi.org/10.1136/adc.56.12.900

46. Davies MW, Swaminathan M, Chuang SL, et al. Reference ranges for the linear dimensions of the intracranial ventricles in preterm neonates. *Arch Dis Child Fetal Neonatal Ed.* 2000;82:F218–F223. https://doi.org/10.1136/fn.82.3.F218

47. Gilard V, Chadie A, Ferracci FX, et al. Post hemorrhagic hydrocephalus and neurodevelopmental outcomes in a context of neonatal intraventricular hemorrhage: An institutional experience in 122 preterm children. *BMC Pediatr.* 2018;18(1):288. https://doi.org/10.1186/s12887-018-1249-x

48. Badhiwala JH, Hong CJ, Nassiri F, Hong BY, Riva-Cambrin J, Kulkarni AV. Treatment of posthemorrhagic ventricular dilation in preterm infants: A systematic review and meta-analysis of outcomes and complications. *J Neurosurg Pediatr.* 2015;16(5):545–555. https://doi.org/10.3171/2015.3.PEDS14630

49. Sandoval PV, Rosales PH, Hernandez DGQ, Naranjo EAC, Navarro VC. Intraventricular hemorrhage and posthemorrhagic hydrocephalus in preterm infants: Diagnosis, classification, and treatment options. *Childs Nerv Syst.* 2019;35(6):917–927. https://doi.org/10.1007/s00381-019-04127-x

50. Limbrick DD, Mather A, Johnston JM, et al. Neurosurgical treatment of progressive posthemorrhagic ventricular dilation in preterm infants: A 10-year single-institution study clinical article. *J Neurosurg Pediatr.* 2010;6(3):224–230. https://doi.org/10.3171/2010.5.PEDS1010

51. Kormanik K, Praca J, Garton HJL, Sarkar S. Repeated tapping of ventricular reservoir in preterm infants with post-hemorrhagic ventricular dilatation does not increase the risk of reservoir infection. *J Perinatol.* 2010;30(3):218–221. https://doi.org/10.1038/jp.2009.154

52. McNair C, Campbell M, Johnston C, Taddio A. Nonpharmacological management of pain during common needle puncture procedures in infants. In: Jain L, Hamrick SM, Ing C, eds. *Anesthesia, Sedation and Pain Control. Clinics in Perinatology*, 2019:46.

53. Ferguson-Clarke L, Williams C. Neurological examination in infancy and childhood. *Paediatr Nurs.* 1998;10(4):29–35. https://doi.org/10.7748/paed.10.4.29.s27

54. Wellons JC, Shannon CN, Kulkarni AV, et al. A multicenter retrospective comparison of conversion from temporary to permanent cerebrospinal fluid diversion in very low birth weight infants with posthemorrhagic hydrocephalus clinical article. *J Neurosurg Pediatr.* 2009;4(1):50–55. https://doi.org/10.3171/2009.2.PEDS08400

55. Mazzola CA, Choudhri AF, Auguste KI, et al. Pediatric hydrocephalus: Systematic literature review and evidence-based guidelines. Part 2: Management of posthemorrhagic hydrocephalus in premature infants. *J Neurosurg Pediatr.* 2014;14:S8–S23. https://doi.org/10.3171/2014.7.PEDS14322

56. Ellenbogen JR, Waqar M, Pettorini B. Management of post-hemorrhagic hydrocephalus in infants. *J Clin Neurosci.* 2016;31(1);30–34. https://doi.org/10.1016/j.jocn.2016.02.026

57. Hanak BW, Bonow RH, Harris CA, Browd SR. Cerebrospinal fluid shunting complications in children. *Pediatr Neurosurg.* 2017;52(6):381–400. https://doi.org/10.1159/000452840

58. Kestle JRW, Riva-Cambrin J, Wellons JC, et al. A standardized protocol to reduce cerebrospinal fluid shunt infection: The hydrocephalus clinical research network quality improvement initiative. *J Neurosurg Pediatr.* 2011;8(1):22–29. https://doi.org/10.3171/2011.4.PEDS10551

59. Kulkarni AV, Riva-Cambrin J, Rozzelle CI, et al. Endoscopic third ventriculostomy and choroid plexus cauterization in infant hydrocephalus: A prospective study by the Hydrocephalus Clinical Research Network. *J Neurosurg Pediatr.* 2018;21(3):214–223. https://doi.org/10.3171/2017.8.PEDS17217

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