SUBGALEAL HAEMORRHAGE IN THE NEWBORN: A CALL FOR EARLY DIAGNOSIS AND AGGRESSIVE MANAGEMENT

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Abstract: Subgaleal haemorrhage (SGH) is an important cause of preventable morbidity and mortality in the neonate. Its increased prevalence in recent years has coincided with the rise in the number of births assisted by vacuum extraction. Three deaths in Australia within the last 7 years have been the subject of two coronial inquests. Subsequent coronial reports have highlighted that neonatal death from SGH can be prevented if appropriate attention is paid to identification of risk factors, early diagnosis, close observation and aggressive treatment. To prevent unnecessary deaths, all involved in the care of the baby after birth need to be aware of the importance of prompt diagnosis, monitoring and early treatment of SGH.

Key words: delivery; infant; newborn; perinatal mortality; subgaleal haemorrhage; vacuum extraction.

Recent coronial inquests in Australia have drawn attention to the fact that subgaleal haemorrhage (SGH) in the newborn may be fatal but that deaths can be prevented.1 A coroner recommended that the Royal Australasian College of Physicians (RACP) impress upon their paediatric members the potential seriousness of SGH and that prompt, early diagnosis and treatment are required.1 This article arose from the response of the RACP (Paediatric Division) Policy and Advocacy Committee. It provides a broad review of the available evidence and focuses on the diagnosis and management of SGH in the newborn to prevent serious morbidity or mortality. All involved in perinatal care should be aware of the importance of prompt diagnosis and early treatment of neonatal SGH.

In Australia, state coronial inquests into three deaths from SGH identified factors contributing to mortality. These included initial delays in diagnosis, misdiagnosis, failure to recognise that an infant with an SGH may deteriorate quickly and delays in providing sufficiently aggressive management. Of the cases in question, one infant died at 7 h after a continuous SGH of 120–150 mL. Another infant who died 8 days after birth had ongoing transfusion requirements, increasing head size and progressive shock, resulting in multi-organ failure.1 A third infant died at 3 days from multi-organ failure, complicated by initial misdiagnosis of caput succedaneum and delays in intravenous resuscitation.2 The broader literature supports these factors as contributing to mortality and morbidity from SGH.3,4

An association between vacuum extraction and SGH is well described.3,4 Over the past three decades, increasing use of vacuum extraction to assist births has resulted in an increase in the prevalence of SGH in Australia and the developed world.5–10 Up to 5–10% of all births are estimated to be performed with vacuum,11 with higher rates in some centres. The United States Food and Drug Administration (FDA) issued a public health advisory in 1998, detailing a fivefold increase in the reported morbidity and mortality in the preceding 4 years from SGH after vacuum.12 In the following 6 months, 22 times more cases of vacuum-associated SGH were reported to the FDA.13 Health Canada issued a similar alert the following year after four deaths from vacuum-associated SGH, concluding that ‘All Health Care Professionals responsible for the post-natal care of infants whose delivery involved the use of Vacuum Assisted Delivery Devices must monitor the infant for signs of subgaleal haemorrhage’.14 The Royal Australian and New Zealand College of Obstetricians and Gynaecologists (RANZCOG) in 2009 issued a position statement with recommendations for appropriate vacuum extraction techniques to minimise the risk of SGH, patient selection and stratification of at-risk neonates for further observations.15 This document draws attention to the fact that ‘minimising the
morbidity and mortality of SGH following vacuum extraction requires a multifaceted approach, with the engagement of obstetricians, delivery suite and post-natal midwives, nursery and paediatric staff. However, deaths are continuing to occur despite available knowledge that early close monitoring, diagnosis and aggressive treatment can prevent and very significantly reduce mortality and morbidity from SGH.16

**Anatomy**

Naegele is cited as first describing SGH in 1819, by distinguishing true subperiosteal cephalohaematoma from false cephalohaematoma.17 Development of vacuum extraction by Malmström in the 1950s led to refinement of the nomenclature to the current term ‘subgaleal haemorrhage’,18 sometimes described in the literature as ‘subaponeurotic haemorrhage’.

The scalp comprises five layers: skin; dense connective tissue; the tough fibrous layer of the galea aponeurotica, also known as the epicranial aponeurosis; loose connective tissue permitting movement of the galea; and the dense periosteum tightly encasing each cranial bone and their diploic veins. The subgaleal space exists immediately superior to the periosteum, and inferior to the tough fibrous sheath of the galea as it extends uninterrupted across the cranial vault from the frontalis muscle to the posterior nuchal lines and laterally to the temporalis muscle. Many fine emissary veins traverse the loose connective tissue of the subgaleal space, providing drainage of the superficial veins of the scalp into the intradural venous sinuses.

Application of external force to the scalp, such as encountered at birth or particularly by the tractional and rotational forces with the use of vacuum extraction, can result in rupture of veins and haemorrhage into different layers of the scalp.19 Most significantly, SGH may result from rupture of emissary veins resulting in haemorrhage, without anatomical tamponade, into the subgaleal space. A 1-cm increase in the depth of the subgaleal space may contain approximately 40 mL to 260 mL of blood.18,20 Because the circulating blood volume of the neonate is about 90 mL/kg body weight21 in a 3-kg baby, a serious 20% reduction in circulating blood volume occurs with a haemorrhage of only 54 mL.

**Diagnosis**

The clinical picture of SGH is variable and progressive and necessitates repeated assessment.1,22 Initial clinical presentation ranges from no signs at birth with a slow insidious onset3 to rapidly progressive; mean time to diagnosis is 1 to 6 h after birth.16,23 SGH severity can be classified using criteria relative to degree of head circumference expansion, the type of supportive treatment required to correct hypovolaemic shock and hyperbilirubinaemia24 (Table 1). The mild, moderate (development of hypovolaemic shock) and severe (associated with coagulopathy) grading system is useful but does not assist in predicting the potential for progression of mild SGH to severe SGH.

Approximately 6% of SGH cases are asymptomatic, 15–20% are mild, 40–50% are moderate and 25–33% are severe.25,26 Classical clinical signs of SGH may be absent. Presentation is with a diffuse, boggy (like pitting oedema), gravity-dependent scalp swelling that obscures the sutures initially and across time organises to a tense haematoma.27 An increase in head swelling and circumference is common, and accurate measurement of head circumference is essential. In large haemorrhages, eyelids may swell and ears may be displaced inferiorly because of the mass effect (Fig. 1).

SGH should be distinguished from other common causes of a head lump in the newborn, as these rarely produce...
hypovolaemia or mortality. Caput succedaneum or chignon is characterised as a localised, serosanguinous, transudative collection potentially with ecchymoses, located above the galea and occurs in normal birth or at the site of suction cup application. An early SGH may appear clinically similar to a caput succedaneum, which often diffuses quickly to a minor swelling of the scalp and is either static or resolves. With progression, an SGH may appear as an expanding bogginess, increasing in depth with extent and time. Haemorrhage beneath the periosteum, caused by ruptured diploic veins, results in a localised, tense swelling coursing the extracranial extent of the cranial bone and does not cross a suture line. This condition termed cephalohaematoma or subperiosteal haematoma is managed conservatively (Fig. 2).

The classic signs of early shock associated with symptomatic SGH are tachycardia, reduced spontaneous activity, pallor, poor peripheral refill and mild respiratory distress.28 SGH should be excluded in all cases of shock, or as contributing to other causes of shock such as sepsis, hypoxic ischaemic encephalopathy and haemorrhage at other sites. Transiently compensated shock may result in stable haemodynamics, which is falsely reassuring. Undue reliance should not be placed upon a clinical picture of haemodynamic stability alone, a fact highlighted by the coronial inquest.1

Poor end organ tissue perfusion results in low urine output, hypotonia, lethargy, cyanosis and seizures. Early anaerobic metabolism from tissue ischaemia can be identified biochemically by a rising lactate or worsening base deficit.28 Deranged liver function tests may indicate ischaemic hepatopathy, and elevated creatinine may indicate developing renal failure, acute tubular necrosis and cortical necrosis, all of which portend poorer outcomes.29,30 Poor cerebral perfusion may result in cerebral oedema, brain injury and seizures.28

A fall in haemoglobin (Hb) is a late sign in severe haemorrhage.21 Anaemia may not develop in the early stages of massive haemorrhage because of insufficient time for fluid shift to result in haemodilution. Persisting or large volume haemorrhage may progress to disseminated intravascular coagulopathy (DIC).28 Early biochemical signs of coagulopathy such as prolonged activated partial thromboplastin time, international normalised ratio (INR) and a decreasing fibrinogen should be sought in moderate and severe cases. Jaundice and hyperbilirubinaemia may result from breakdown of the extravasated Hb and typically occur during the resolution of the established subgaleal haematoma.

Management may be complicated by other coexisting injuries. Hypoxic ischaemic encephalopathy occurs in 62–72% of SGH.24 Brain trauma resulting in cerebral oedema and/or intracranial haemorrhage occurs in 33–40%.24,28 Less common associations include subdural haematoma,30 dural tear with herniation,32 superior sagittal sinus rupture,31 pseudomeningocele14,15 and encephalocele,16 and subconjunctival and retinal haemorrhage. Elevated intracranial pressure (ICP) from the SGH mass effect is reported.23 Skull fractures may be associated.16,19,24

**Risk Factors**

Primiparity accounts for 95% of SGH cases.15 Labour history risk factors include vacuum delivery (odds ratio = 7.17), failed vacuum, repeated or prolonged vacuum,25,26 difficult vacuum characterised by extraction over more than three contractions,20 20-min extraction time or more than two cups of detachments,13 placement of vacuum cup near the anterior fontanel sagittal suture or with marks at less than 3 cm away from the anterior fontanel,16 prolonged second stage (>120 min),37 prolonged rupture of membranes (>12 h), malposition of the fetal head, forceps delivery particularly where this is a high or mid-cavity forceps delivery, fetal distress, macrosomia, prematurity, low birthweight, male sex 2:1 to 8:1, low Apgar scores, need for resuscitation at birth and cord blood acidosis,28,31,37

**Incidence**

Reported incidences of SGH in retrospective studies are 0.1 to 0.6 per 1000 spontaneous vaginal deliveries and 3 to 7.6 per 1000 vacuum extractions2,12,18,22,26,28,31,37 Higher incidences of 41.4 per 1000 and 210 per 1000 vacuum extractions are reported in prospective surveillance studies.16,38,39 Sixty to 89% of cases of SGH are related to vacuum extraction.24,31,38 SGH can also occur post-caesarean section.30

**Management**

The possibility of SGH should always be considered after a vacuum delivery, especially if delivery has been difficult and the infant requires resuscitation. A focused clinical history can identify the neonate’s risk factors for SGH where early aggressive treatment is critical. Prompt initial assessment by a neonatologist, paediatrician or other experienced staff member with careful examination of scalp and repeat review at 1 and 4 h is recommended for the at-risk neonate. Transfer to an intensive care unit or special care nursery is advised for close observation. If the nursery provides low-dependency care, consider transfer to a high-dependency area and seek specialist paediatric advice.

In the at-risk but asymptomatic neonate, RANZCOG recommend that cord pH, lactate, haematocrit and platelet count be
taken at birth, as well as basic observations hourly for 2 h and second hourly for 6 h. Other guidelines suggest 15 minutely blood pressure (BP) measurements for 4 h15,41 or 15 minutely BP measurements for 1 h, then hourly for 4 h, and then forth hourly thereafter.15 BP monitoring alone should be not relied upon, as hypotension is a late sign of severe SGH. Close vital sign observations should include hourly head circumference measurements and monitoring for signs of hypoperfusion, such as tachycardia, reduced spontaneous activity, pallor, poor peripheral refill and mild respiratory distress. The most successful monitoring regime with the lowest published mortality performed formal structured assessments at 1, 6 and 24 h after birth, with close monitoring of all at-risk neonates; all SGH were transferred to the neonatal intensive care unit (NICU) with cardiorespiratory and BP monitoring.16

In cases of shock or a fall in Hb, consider early aggressive circulatory support with normal saline and packed red blood cells.42 Inotropes, vasopressors, and multiple red cell transfusions may be required.43 A rising lactate, developing or persisting acidemia, hypotension, coagulopathy or anaemia should prompt significant escalation of therapy, including intubation and ventilation (Fig. 3).

Consider repeating Hb and lactate measurements 2–4 h after the initial assessment. Biochemical indicators of progressing

**SGH Management Flow Chart**

- **Vacuum or forceps delivery**
- **Take a cord pH**
  - Examine at 1 and 4 h post-delivery
- **No signs of SGH**
  - **Signs of SGH:**
    - Vague generalised expanding scalp bogginess, crossing suture lines
  - **Admit** NICU, high-dependency unit, monitor heart rate, respirations, oxygen saturation, blood pressure. Take blood for FBC, lactate, coagulation profile
- **No signs of shock**
  - Continue monitoring for another 24 h
  - **Signs of shock:**
    - Tachycardia, respiratory distress, oxygen requirement, pallor, poor capillary refill
      - Normal saline bolus ± packed red blood cells transfusion
      - Consider intubation and ventilation ± inotropes
      - For coagulopathy, consider fresh frozen plasma, platelets, cryoprecipitate or recombinant activated Factor VII
- **Routine care**

Fig. 3 SGH management flow chart. FBC, full blood count; NICU, neonatal intensive care unit; SGH, subgaleal haemorrhage.
shock and impending collapse are a falling Hb, a rising lactate and a prolonged INR. The study with the lowest reported mortality applied treatment triggers for transfusion at Hb 140 g/L or coagulopathy correction at INR 1.5.16 Such aggressive treatment triggers take into consideration that haemorrhage may be ongoing, despite stable clinical signs. Correction of acidosis is important to reduce worsening of coagulopathy and cardiac output. Aggressive correction and regular monitoring of coagulopathy are vital with support from potentially large volumes of fresh frozen plasma, platelets and cryoprecipitate.

Up to 81% of neonates with SGH may develop coagulopathy.16 Correction of coagulopathy may assist in arresting haemorrhage or the development of DIC.

Successful source control of massive diffuse haemorrhage is reported with recombinant activated factor VII.26,44,45 Recombinant activated factor VII promotes clot formation and stability by binding to platelets, initiating a thrombin burst independent of tissue factor (VIII and IX) and reducing fibrinolysis.44 Combination with tranexamic acid (120 mcg/kg bolus) has been described as successful.44

Once the infant’s condition is stabilised, medical imaging is recommended to confirm diagnosis and to identify confounding morbidities;46 however, imaging should never delay or interfere with treatment. Reports of head bandaging suggest it should not be used. In one report, two-head bandaged neonates died28 and in another there was no benefit.26 Bandaging may increase the SGH mass effect and elevate ICP, particularly in a setting of diastasis of cranial sutures, associated head injury or cerebral oedema. Relief of symptomatic mass effect with neurosurgical evacuation with Jackson-Pratt drain has been reported.23

After organisation of the SGH, jaundice and hyperbilirubinaemia ensue in approximately 60% of infants.16 Phototherapy and exchange transfusions may be required.47 The haematoma may also become a nidus for infection.47 Additional organ support may be required for complications of ischaemic injury, with success reported with peritoneal dialysis.48

**Mortality**

The association between fatal SGH and vacuum extraction has been well described.49 Early studies reported a very high mortality of 17–25%18,24,31,37 for neonates with SGH admitted to NICU; this has improved to 5–14% in more recent studies.22,26,28 The lowest reported mortality is 2.8%, from a population where 21% of all vacuum-delivered neonates were diagnosed with SGH; these neonates underwent repeat assessment and close monitoring for progression.16 Mortality from SGH can be improved with increased surveillance and early aggressive management in the NICU.16

**Longer Term Outcomes**

Longer term outcomes in neonates who survive the acute SGH are generally good, with reported adverse outcomes ranging from normal28,42 to adverse outcomes of 17%, 22,24,33 A single report of a higher rate of disability of 33%28 presumably reflects a more severe grade of SGH resulting in cerebral injury (Figs 4,5). Neurological sequelae include seizures, neurodevelopmental delay and cerebral palsy. Poorer neurodevelopmental outcomes are more common with severe SGH.28

Fig. 4 Sagittal magnetic resonance scan. The large arrow indicates subgaleal haemorrhage; the small arrows indicate signal intensity changes indicative of haemorrhagic infarction. This image is from Cheong et al.27 Reprinted here with permission. All rights reserved.

Fig. 5 Coronal magnetic resonance scan. The large arrow indicates subgaleal haemorrhage. A clear loss of grey/white matter differentiation can be observed, suggesting widespread cellular injury. This image is from Cheong et al.27 Reprinted here with permission. All rights reserved.
Conclusion

SGH is an important cause of preventable morbidity and mortality in the neonate. Identification of risk factors, early diagnosis, close observation and aggressive treatment and support can prevent and very significantly reduce mortality and morbidity from SGH. All involved in perinatal care should be aware of the importance of prompt diagnosis and early treatment of neonatal SGH.

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References

Subgaleal haemorrhage in the newborn

What baseline tests will you order?

a. FBC, lactate and check to see if a cord pH was measured
b. None. Only close monitoring is required at this stage
c. Coagulation profile to exclude a predisposing coagulopathy, which caused the SGH
d. Blood culture and start IV antibiotics
e. Head ultrasound to define the extent of haemorrhage

Answer: a

b. Incorrect. Baseline Hb should be measured in case baby’s condition deteriorates rapidly.
c. Incorrect. Consider coagulopathy if baby has signs of early shock and condition is deteriorating.
d. Incorrect. There are no other clinical signs of sepsis at this stage.
e. Incorrect. Imaging is only recommended once baby’s condition is stabilised.

3 A baby who is being monitored for SGH is noted at 5 h of age to have developed tachycardia, poor peripheral perfusion and mild respiratory distress. Blood pressure remains stable and head circumference has not increased. Hb at birth was 168. As you take a blood culture and start antibiotics, you also perform a blood gas that shows the following: pH 7.2, CO2 35, Hb 135, lactate 8.

What will you do next?

a. Continue to observe as blood pressure and head circumference are stable
b. Take more blood for a coagulation profile
c. Give FFP to prevent progression into disseminated intravascular coagulation and seriously consider intubation and ventilation
d. Resuscitate with an intravenous normal saline fluid bolus and organise a cross-match for blood transfusion and seriously consider intubation and ventilation
e. Resuscitate with intravenous activated VII and seriously consider intubation and ventilation

Answer: d

a. Incorrect. Hypotension is a late sign of shock; early signs of shock require aggressive early intervention.
b. Incorrect. Intervention is more important at this point.
c. Incorrect. Correction of hypovolaemia is the first step in management of shock.
d. Correct. The baby has evidence of shock from hypovolaemia secondary to SGH and requires immediate intervention.
e. Incorrect. Activated factor VII may be considered to control massive diffuse haemorrhage.