Seminars in Fetal and Neonatal Medicine 24 (2019) 101047

Contents lists available at ScienceDirect

Seminars in Fetal and Neonatal Medicine

journal homepage: www.elsevier.com/locate/siny

Delivery room emergencies due to birth injuries

Tiffany McKee-Garrett

Baylor College of Medicine, Department of Pediatrics, Section of Neonatology, Houston, TX, USA

A B S T R A C T

Delivery room emergencies due to birth injuries are serious, usually unexpected, and can be distressing situations that necessitate immediate action to reduce neonatal morbidity and prevent neonatal mortality. Birth injuries requiring immediate, urgent care in the delivery room are uncommon, hence knowledge of obstetric risk factors and prenatal conditions linked to birth injury is an important first step in the management of affected neonates. Furthermore, immediate recognition of injury and quick action upon delivery is essential in order to achieve the best possible outcomes. This chapter briefly reviews the known risk factors associated with birth injury, and then discusses the identification and management of specific injuries that may require immediate treatment in the delivery room, or hasty management within hours after birth.

1. Introduction

Birth injury is described as an impairment of a newborn's body function or structure secondary to an adverse, often unexpected event that occurred during labor, delivery, or both. While most birth injuries are nonintentional, some injuries are the result of efforts to rapidly deliver an infant in the setting of maternal compromise or fetal distress. The majority of birth injuries are not life-threatening, and most do not require interventions in the delivery room. For purposes of this review, the more common birth injuries, such as fractures and lacerations, will not be discussed due their generally benign nature.

Fortunately, the overall incidence of birth injuries has declined due to enhancements in prenatal diagnosis and improvements in obstetrical care. The estimated incidence of birth injury is approximately 2% for single vaginal deliveries from the cephalic position, and approximately 1.1% for cesarean deliveries [1,2]. As these numbers indicate, mode of delivery and fetal position (cephalic versus breech) are some of the factors that influence the risk of birth injury. Moczygemba et al. estimated an overall birth trauma rate of approximately 2.6% when analyzing U.S. data from the Nationwide Inpatient Sample of over eight million newborns born between 2004 and 2005 [3]. Risk factors associated with birth injury have been well defined, enabling the obstetrician to recognize the potential for injury and adjust maternal care in order to avoid trauma to the neonate (e.g. elective cesarean delivery). Likewise, based on the obstetric and peripartum history, the neonatal team is able to anticipate certain scenarios in which emergent interventions in the delivery room may be necessary. However, it is important to note that despite best efforts to anticipate trauma to the neonate, significant birth injury can occur in the setting of a low risk gravida, uncomplicated pregnancy, and/or normal labor course.

This chapter focuses on the diagnosis and treatment of certain birth injuries that require intervention soon after delivery in order to decrease neonatal morbidity and, in some instances, prevent neonatal death. Fortunately, neonatal death attributed to birth injury is rare, with a reported incidence of less than 2% [4]. Management of these neonates may require urgent attention in the delivery room or, if the birthing facility has a neonatal intensive care unit (NICU) in close proximity to the delivery room, the neonate can be expeditiously transferred to the NICU for care. For purposes of this chapter, birth injuries with the following characteristics are deemed emergencies and require immediate management: 1) injuries associated with acute blood loss and shock, 2) injuries that may result in respiratory insufficiency or failure, and 3) injuries that carry a risk of neurological or organ impairment.

2. Risk factors

Known risks for neonatal birth injury have been attributed to maternal factors, fetal factors, and to the use of instrumentation during delivery. Likewise, a combination of these risk factors further increases the likelihood of birth injury. When significant risk factors for birth injury are present, a team of skilled providers should be assembled and present at delivery to provide immediate care to the newborn.

2.1. Maternal factors

Maternal obesity, defined as a body mass index (BMI) greater than 30 kg/m^2, is considered one of the most commonly occurring risk factors seen in obstetric practice and is associated with an increased risk of birth injury [5,6]. The etiology of this increased risk is likely due to a higher incidence of gestational diabetes, increased use of instrumentation (section 2.3), shoulder dystocia (section 2.4), and

E-mail address: tiffanym@bcm.edu.

https://doi.org/10.1016/j.siny.2019.101047

1744-165X/

increased incidence of large for gestational age (LGA) newborns in women who are obese. In analyzing outcomes from over 350,000 pregnancies, Jolly et al. reported that women with a BMI greater than 30 kg/m2 were more likely to deliver a newborn with excessive birth weight, have an instrumented delivery, and have a newborn that was admitted to the special care baby unit [7].

2.2. Fetal factors

Excessive fetal weight and abnormal presentation are associated with an increased risk of birth trauma. The term macrosomia is used to describe newborns with excessive birth weight and, although definitions vary in the literature, most experts define macrosomia as a birth weight of greater than 4000 to 4500 g. Studies show newborns with birth weights ≥4000–4500 g to be at increased risk for a variety of injuries related to birth trauma, including brachial plexus injury, phrenic nerve injury, fractures, soft tissue injuries, and abdominal injury. Nassar et al. reported a 7.7% risk of birth injury in a retrospective review of 231 newborns with birth weights > 4500 g [8]. When comparing newborns of birth weight ≥4000 g to newborns with birth weights of 3000–3999 g, Boutlet, et al., observed an increase risk of neonatal complications as the birth weight increased beyond 4000 g, with newborns of birth weight > 4500 g being at particular risk of morbidity and mortality [9]. Abnormal fetal lie, defined as fetal presentation other than cephalic, is also associated with an increased risk of birth trauma. Infants delivered via vaginal breech extraction are at particular risk for birth injury, and specific criteria have been established to minimize risk for women who desire a trial of vaginal breech delivery [10].

2.3. Use of instrumentation

Multiple publications have shown an increased incidence of birth injury with the use of forceps or vacuum assistance during delivery. Both forceps and vacuum delivery have an increased risk of birth injury when compared to non-operative vaginal delivery, or delivery by cesarean section, with or without labor [1,11]. Both forceps and vacuum extraction are recognized as acceptable for instrumented delivery [12], and each has been associated with specific types of birth injuries.

2.4. Shoulder dystocia

An obstetric emergency, shoulder dystocia is defined as a delivery that requires additional obstetric maneuvers to release the shoulders when gentle downward traction has failed to affect delivery [13]. Spong et al. generated an objective measure for shoulder dystocia, and proposed that a prolonged head-to-body delivery time of more than 60 s be included in the definition [14]. Shoulder dystocia is associated with both maternal and neonatal morbidity, and is often accompanied by other risk factors for birth injury, such as fetal macrosomia and instrumented delivery.

3. Resuscitation in the delivery room

Per international guidelines for neonatal resuscitation, every birth should be attended by at least one individual whose only responsibility is the care of the newborn. This individual is expected to be skilled in the initial steps of resuscitation: warm the neonate, clear secretions if copious and/or obstructing the airway, dry, stimulate, and initiate positive pressure ventilation if needed. Furthermore, every birthing facility should have additional personnel available who are skilled in all aspects of neonatal resuscitation, including endotracheal intubation, umbilical vein catheter placement, and the administration of medications, fluids, and blood products [15]. Volume expansion may be considered when blood loss is unknown or suspected based on exam findings such as pallor, weak pulse, poor perfusion, and if the infant's heart rate has not adequately responded to other resuscitative measures [16].

3.1. vol resuscitation

Volume resuscitation of the newborn in the delivery room is potentially life-saving and, fortunately, is a rare occurrence. In a large retrospective review of 37,972 liveborn infants, thirteen were treated with volume infusions in the delivery room and, of those thirteen, only three received volume for suspicion of hypovolemia [17]. Objective measures of newborn circulatory status in the delivery room are needed for assessing infants with suspected hypovolemic shock. Per the Neonatal Resuscitation Algorithm, assessment of heart rate is the mainstay by which to gauge the need for resuscitation, and to assess the newborn's response to resuscitative efforts [16]. Pulse oximetry during stabilization is traditionally used to monitor the newborn's heart rate however, electrocardiogram monitoring with the placement of three chest leads has been shown to provide more accurate heart rate values in a shorter period of time than pulse oximetry, and is less prone to movement artifact [18]. Non-invasive (cuff) blood pressures can be obtained during newborn stabilization, but these measurements have been shown to be inconsistent and may not be clinically useful as a means of assessing circulatory status. Invasive monitoring, such as placement of an arterial line, is not practical in the delivery room setting [19]. When birth history and the newborn's initial presentation suggest hypovolemic shock, volume resuscitation with whole blood or crystalloid solution (normal saline) should be urgently provided. Colloid infusions, such as albumin, are not advised as a treatment option during delivery room stabilizations [20]. Whole blood is preferred over normal saline because it provides volume, colloid, and oxygen carrying capacity, and urgent transfusion with uncrossmatched O-Rhesus-negative blood should be administered whenever available [20,21]. The recommended initial dose is 10–20 milliliters/kilogram, with repeat doses as needed based on the newborn's response.

4. Cranial injuries

Birth injuries sustained to the head may involve intracranial structures and/or extracranial structures. Some of these injuries are associated with significant morbidity and mortality, hence it is imperative to recognize them early and initiate treatment as soon as possible.

4.1. Intracranial hemorrhages (ICH)¶

Intracranial hemorrhages are described according to location and, in order of incidence, include: 1) subdural, 2) subarachnoid, 3) epidural, and 4) intraventricular. Not surprisingly, the incidence of all types of intracranial hemorrhage is reported to be five to ten times higher for neonates delivered by forceps or vacuum extraction [1]. In general, birth injuries due to intracranial hemorrhage do not require urgent intervention in the delivery room setting. However, affected newborns may become symptomatic within hours of delivery, requiring early recognition and intervention to avoid significant morbidity. Subdural hemorrhage (SDH) refers to bleeding between the dura mater and the arachnoid layer of the brain caused by rupture of the bridging veins, and is usually tentorial or interhemispheric in location (Fig. 1). The reported incidence varies widely, as SDH has been reported to be a common finding upon imaging of the newborn brain shortly after delivery with incidences as high as 26% [22] and 46% [23], for infants born via instrumented and non-instrumented deliveries. Subarachnoid hemorrhage, the second most common type of neonatal intracranial hemorrhage, is caused by rupture of bridging veins in the subarachnoid space or small leptomeningeal vessels. Epidural hemorrhage (EDH) occurs between the dura and inner table of the skull and is attributed to injury to the middle meningeal artery. Typically located in the parietotemporal region, epidural hemorrhage is often accompanied by

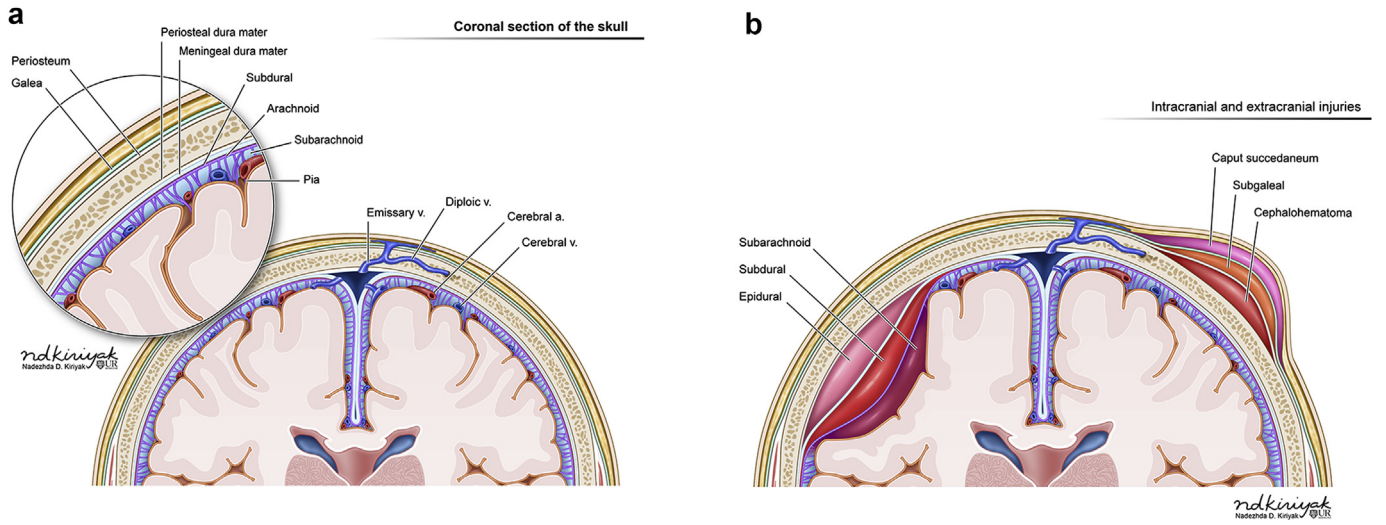

Fig. 1. A Coronal section of the skull. Used with permissions. B Intracranial and extracranial injuries. Used with permissions.

skull fracture which can enhance bleeding by shearing of the underlying blood vessels. Intraventricular hemorrhage (IVH) is most often associated with premature infants, with an increasing incidence as gestational age decreases. While uncommon, IVH has been reported in term infants, for which primary management rarely includes early intervention.

Management/Intervention: Newborns with ICH requiring urgent intervention usually exhibit symptoms within hours after delivery. However, because symptoms can be delayed, asymptomatic newborns with suspected ICH should be monitored closely after delivery in a nursery setting that allows frequent assessment of vital signs, serial physical exams, including serial measurement of frontal-occipital circumference (FOC), and ready access to diagnostic imaging. Symptoms associated with ICH include: apnea, seizures, respiratory depression, altered tone, decreased level of consciousness, and increased irritability [24]. These symptoms, particularly in the setting of vacuum or forceps delivery, require urgent imaging of the head. When performed by an experienced technician or radiologist, bedside ultrasound is a safe and rapid tool to assess the presence of intracranial hemorrhage, followed by definitive study with computed tomography (CT) or magnetic resonance imaging (MRI) [25]. While convenient and free of radiation exposure, images obtained via ultrasound are limited by the size of the fontanel, hence peripheral ICH can be missed, and epidural hemorrhage is rarely detected. CT has been described as the preferred method for rapid detection of ICH due to ease of access and shorter scan time [24]. To avoid significant morbidity, and mortality, neurosurgical intervention is sought in the setting of deteriorating neurological function with evidence of brain compression, or when symptoms and imaging are consistent with increased intracranial pressure. However, it is important to remember the flexibility of the newborn skull – owing to unfused sutures - and the presence of fontanels that serve as a “pop off” may compensate for the presence of increased intracranial pressure. For this reason, size or thickness of the bleed has also been used as a determinant for surgical intervention [26]. In an analysis by Pollina et al. of 41 newborns with cranial birth injuries, five infants (12%) required operative treatment, and two of the 41 infants died. Posterior fossa SDH, although uncommon, can be acutely life-threatening due to compression of the brainstem. In a retrospective analysis by Blauwblomme et al. of sixteen neonates with posterior fossa hematoma, nine neonates had evidence of brainstem dysfunction within the first 24 h of life, and eleven required surgical evacuation of the posterior fossa SDH [27]. EDH is more likely than SDH to present with signs of increased intracranial pressure in the first hours of life, likely due to the arterial etiology of the hemorrhage, particularly when accompanied by skull fracture. In his study of fifteen neonates with epidural hematomas, Heyman, et al. observed skull fractures in ten of the babies, with six of the ten fractures described as depressed. Seven neonates (46%) in this series were treated with craniotomy to evacuate the EDH, including all six babies with a depressed skull fracture [26].

4.2. *Extracranial injuries*¶

Extracranial injuries sustained as a result of birth trauma include: 1) caput succedaneum, 2) cephalohematoma, and 3) subgaleal hemorrhage (SGH). Of these, only SGH has been associated with significant morbidity and mortality. SGH is caused by traction on the scalp during delivery with subsequent shearing or severing of the emissary veins that lie in the space between the skull periosteum and epicranial aponeurosis (subgaleal space) [Fig. 1A and B]. SGH is associated with instrumented delivery, with vacuum assistance carrying a much higher risk of SGH than forceps. The incidence is estimated to be 4 per 10,000 non-instrumented deliveries, and as high as 64 per 10,000 vacuum-assisted deliveries [28,29]. Maternal nulliparity, Apgar score of less than 8 at five minutes, marks of vacuum cup over the sagittal suture, and marks of leading edge of the vacuum cup < 3 cm away from the anterior fontanel of the head have also been identified as risk specific factors for SGH [30]. Essentially a huge potential space, the subgaleal space extends from the orbital ridges anteriorly to the nape of the neck posteriorly and to the level of the ears laterally. Bleeding can be massive, allowing for sequestration of 20–40% of the newborn's blood volume, or more, into the subgaleal space [31]. The mortality rate for SGH is high due to the potential for hypovolemic shock and associated coagulopathy. Kilani and Wetmore report a mortality rate of 11.8% in their study of 34 newborns with SGH, all died from significant volume loss, coagulopathy, and shock [32]. In an analysis of data collected prospectively on 69 newborn with SGH, Gebremariam reported a 14% mortality rate [33]. A more recent analysis of 20 newborns diagnosed with SGH, eighteen of whom were delivered via vacuum assistance, reported a mortality rate of 15% [34]. Amar et al. reports the unusual presentation of severe extracranial cerebral compression in two newborns with massive SGH, in which one of the neonates did not survive [35].

Management/Intervention: Early recognition and intervention is imperative to decreasing morbidity, preventing mortality, and improving long term outcomes. In a prospective observational study of 71 infants for which active screening for SGH, and early, aggressive treatment was initiated, the reported mortality rate was 2.8% [30], much lower than other published reports. SGH should be suspected in

Fig. 2. Newborn with severe subgaleal bleed subsequent to vacuum delivery. Scalp is boggy and fluctuant to palpation. Photo courtesy of Gerardo Cabrera-Meza MD.

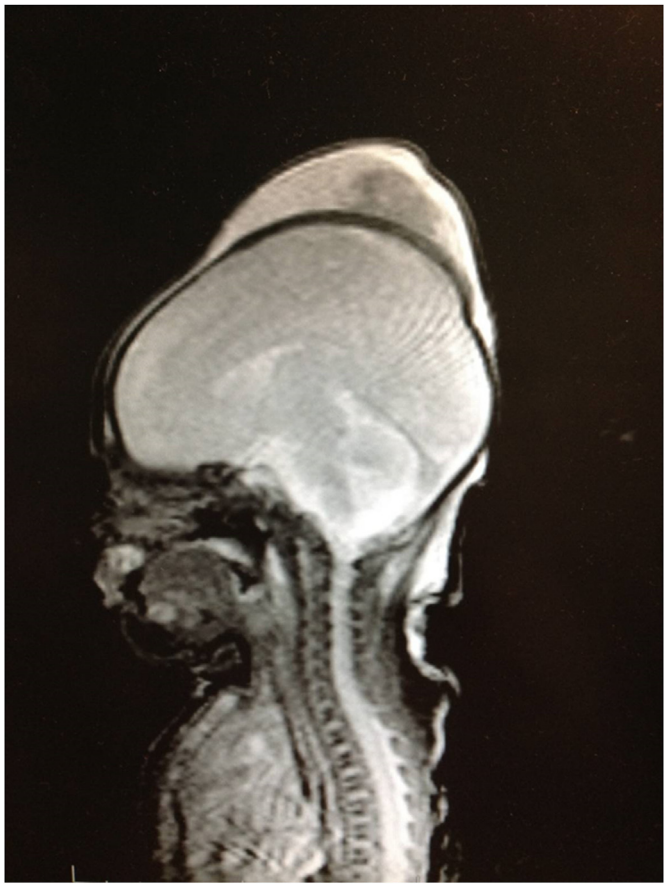

Fig. 3. Subgaleal bleed Image courtesy of Gerardo Cabrera-Meza MD.

any newborn presenting with fluctuant swelling of the head that crosses suture lines, particularly when delivery was complicated by the use of forceps or vacuum extractor (Figs. 2 and 3). While intervention in the delivery room is likely not needed, identification of newborns at risk for SGH should occur soon after delivery, and at risk infants should be admitted to a special care nursery for close monitoring. Monitoring of these infants must include: 1) continuous heart rate monitoring or frequent assessment of vital signs (every one to four hours), 2) serial measurements of FOC, as a one centimeter increase in FOC can

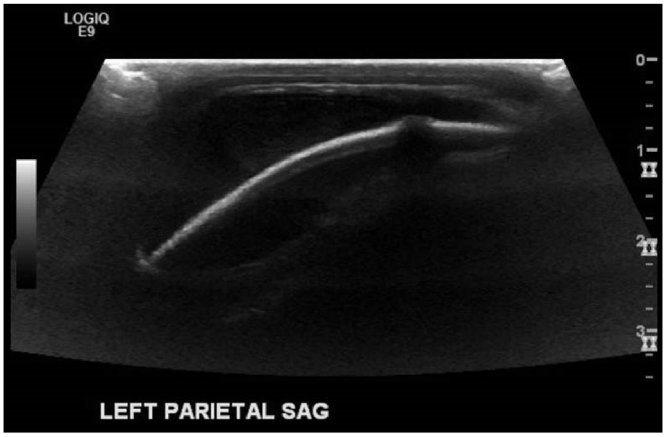

Fig. 4. Ultrasound view of subgaleal hemorrhage. Note fluid extending beyond the suture line. Courtesy of Amy Mehollin-Ray MD.

represent 30–40 mL of blood loss into the subgaleal space [36], and 3) serial hematocrit measurements. In the hands of an experienced operator, ultrasound is useful for rapid assessment of SGH at the bedside (Fig. 4). Head CT or MRI can provide a definitive diagnosis, but may not be practical in the acute setting, and treatment should not be delayed while awaiting CT or MRI if suspicion for SGH is high. Findings of decreasing hematocrit, increasing FOC, and/or tachycardia, are consistent with ongoing blood loss, for which central access via umbilical vessel catheterization should be obtained for the administration of volume expanders and monitoring of blood pressure. Volume expansion is provided with normal saline, whole blood, or packed red blood cells (PRBCs) [see section 3.1]. In a retrospective review of the use of emergency uncrossmatched blood transfusions during neonatal stabilization, Finn, et al. noted SGH as the indication for four of the thirty-nine newborns who received emergency transfusions [20]. Coagulation studies should be obtained due to the risk of consumptive coagulopathy with large bleeds, with transfusion of fresh frozen plasma, cryoprecipitate, and platelets as indicated. Case reports have shown administration of recombinant activated factor VII to be an effective therapy for infants with massive subgaleal hemorrhage [37,38].

5. Nerve palsies

Peripheral nerve palsies are a frequently described sequelae of birth injury. The three most often reported peripheral nerve injuries due to birth trauma are: 1) facial nerve palsy (incidence 0.1–0.7% of births [39]), 2) brachial plexus palsy (incidence 0.03–0.2% of births [40]), and 3) phrenic nerve palsy. Of these, phrenic nerve injury poses the highest risk of morbidity and mortality due to concomitant paralysis of the diaphragm. The incidence of phrenic nerve injury is reported to be 1:15,000 to 1:30,000 live births, with an estimated 10–15% mortality rate [41,42]. Affected newborns may require urgent intervention shortly after delivery due to significant respiratory distress.

5.1. Mechanism of injury

The phrenic nerve originates from the anterior rami of the cervical 3 through 5 (C3–C5) nerve roots and descends through the thorax to innervate the diaphragm. The phrenic nerve is the only source of motor innervation to the diaphragm, hence it plays a crucial role in breathing. The motor innervation activation allows the diaphragm to contract with inspiration, resulting in a flattened diaphragm and decreased intrapleural pressure, with relaxation and return to the dual dome shape during exhalation. Phrenic nerve injury due to birth trauma typically occurs when there is stretching or avulsion of the C3–C5 nerve roots due to extreme lateral flexion and traction of the neck at birth [43].

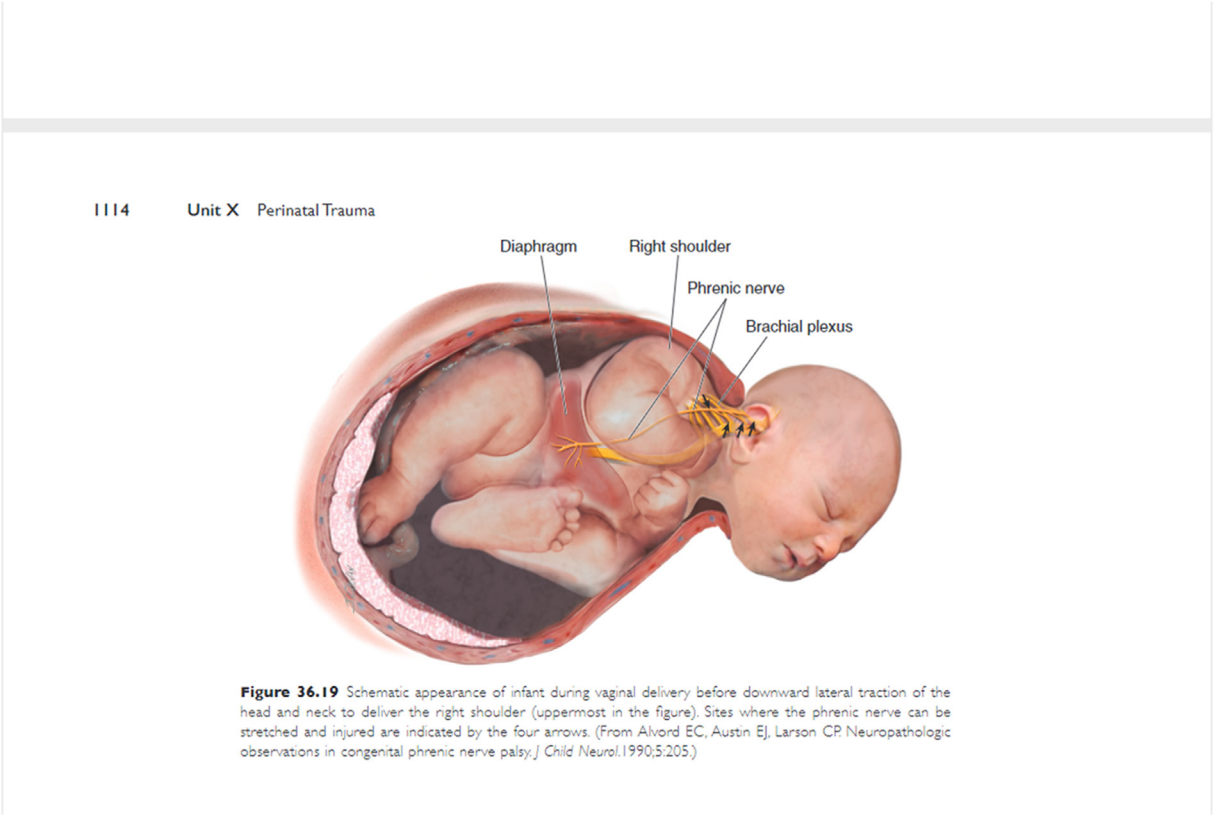

Fig. 5. Diagram illustrating mechanism of injury to brachial plexus and phrenic nerve due to downward lateral traction of head and neck during delivery From: Alvord EC, Austin EJ, Larson CP. Neuropathologic observations in congenital phrenic nerve palsy. J Child Neurol 1995; 5:205 Used with permissions from Elsevier [73].

Given the close proximity to the brachial plexus, a large majority of cases of neonatal diaphragmatic paralysis are associated with brachial plexus injury (Fig. 5). Approximately 80%–90% of newborns with diaphragmatic paralysis have an associated brachial plexus injury [42]. Conversely, a small percentage of newborns who present with a brachial plexus palsy will have concomitant phrenic nerve injury. A retrospective medical record review of 166 newborns with brachial plexus palsy reported a 2.4% incidence of concurrent phrenic nerve injury [44]. In another review of 191 babies with brachial plexus injury, Al-Qattan reported a 4.2% incidence of concurrent phrenic nerve palsy [45].

5.2. Risk factors

The risk factors for phrenic nerve injury are consistent with those described for all birth injuries and include fetal macrosomia, instrumented delivery, shoulder dystocia, and vaginal breech delivery (Fig. 6). Shoulder dystocia poses the greatest risk for brachial plexus injury, with Foad et al. reporting a 100 times greater risk of brachial plexus injury when delivery was complicated by shoulder dystocia [46]. The reported incidence of brachial plexus injury secondary to shoulder dystocia varies widely and ranges from 4% to 40% in the literature [47]. Vaginal breech delivery in particular has been associated with phrenic nerve palsy in several publications [48–50]. In a more recent analysis of 14 neonates with phrenic nerve injury, Stramrood, et al. noted that 57% of affected infants had a breech delivery [41].

Management/Treatment: Life-threatening respiratory compromise may result from phrenic nerve injury, requiring interventions to stabilize the infant in the delivery room setting. Phrenic nerve injury should be considered for newborns with acute respiratory distress at birth, particularly if the delivery was complicated by shoulder dystocia, brachial plexus injury is noted on exam, or if other risk factors exist for birth injury (macrosomia, use of instrumentation, etc). Low Apgar scores often accompany phrenic nerve injury [41], which is not unexpected given the association with difficult delivery and shoulder dystocia, and infants may require resuscitation at delivery. Phrenic nerve palsy can cause severe respiratory failure because of the newborn's relatively low reserve in respiratory function, relatively weak intercostal muscles, and high dependency on abdominal respiration [51]. Affected infants can exhibit paradoxical or see-saw breathing, tachypnea, and cyanosis soon after delivery, and symptoms will be more severe when paralysis is bilateral. Injury is bilateral in approximately 10% of cases, and unilateral in approximately 80% of cases, with the right side affected more often than the left [41,42]. Paralysis is ipsilateral thus, if accompanied by brachial plexus injury, careful examination of the newborn may reveal diminished breath sounds on the same side as the brachial plexus palsy. Chest radiograph (CXR) reveals elevation, or eventration, of the diaphragm on the affected side (Fig. 7), however, CXR may appear normal if the newborn is receiving positive pressure ventilation. Ultrasound, the preferred method of diagnosis because it does not utilize radiation, demonstrates lack of or paradoxical diaphragmatic movement and is helpful in confirming the diagnosis (Fig. 8). Initial treatment focuses on the acute management of respiratory symptoms, and may include supplemental oxygen and continuous positive airway pressure. Severely affected infants, typically those with bilateral paralysis, will require endotracheal intubation and mechanical ventilation, often in the delivery room setting. Plication of the diaphragm is recommended for infants who are unable to wean from respiratory support, or for infants with persistent respiratory distress leading to failure to thrive. Timing of plication varies in published case reports [41,48,51,52], although, in the majority of cases, plication is successful and infants are able to be weaned from respiratory support shortly thereafter.

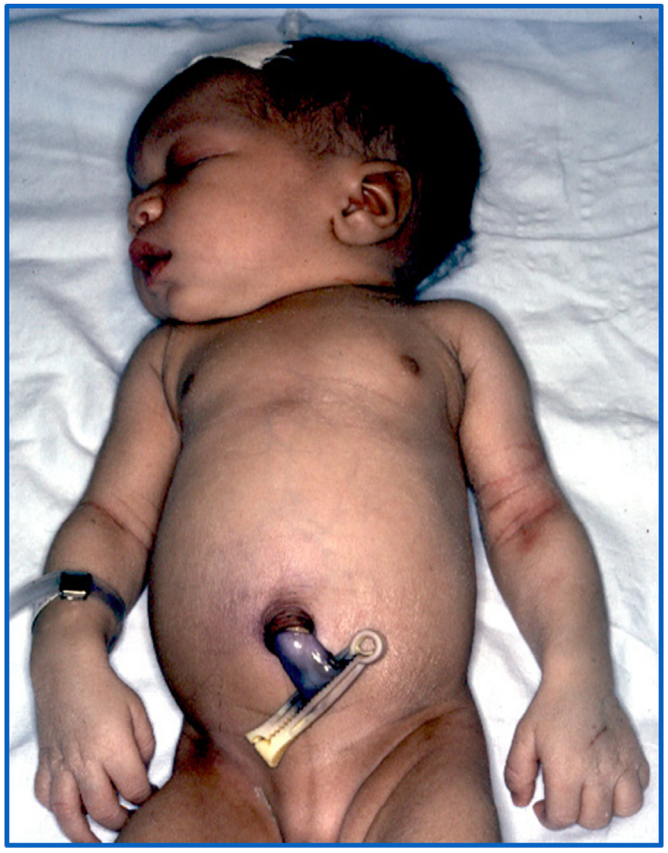

Fig. 6. Newborn with flaccid upper extremities due to bilateral brachial plexus injury. Photo courtesy of Gerardo Cabrera-Meza MD.

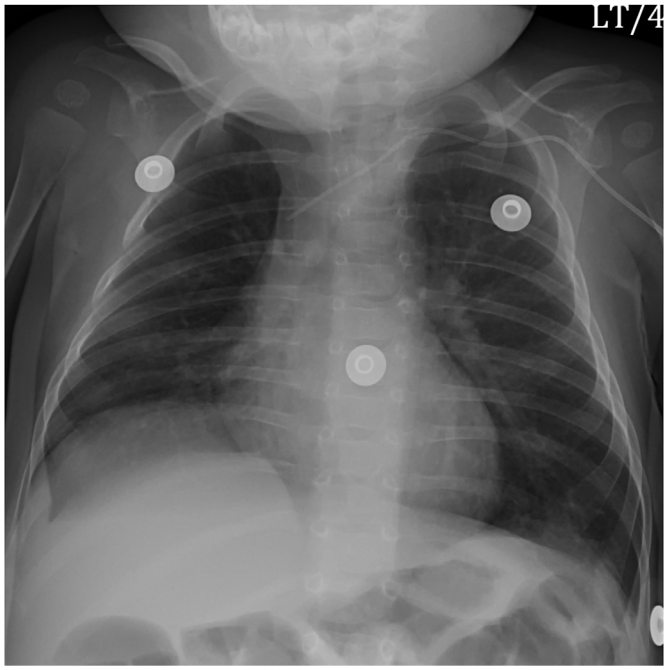

Fig. 7. Paralysis of right diaphragm Photo courtesy of: Amy Mehollin-Ray MD.

6. Spinal cord

Although quite rare and usually catastrophic, spinal cord injuries due to birth trauma require urgent management in the delivery room setting. Injury to the upper cervical spinal cord is more common than injury to the lower cervical and thoracic spinal cord. The true incidence of birth-related spinal cord injury is difficult to determine, with the majority of incidence-specific data being published in the 1990s. In a review of 15 cases occurring over a span of thirteen years, the incidence of high cervical spinal cord injury was estimated at 0.15 per 10,000 live births [53], while an earlier publication by Rehan and Seshia reported an incidence of 0.34 per 10,000 (1 in 29,000) live births complicated by serious spinal cord injury [54].

6.1. Mechanism of injury and risk factors

Spinal cord injury due to birth trauma is the result of excessive traction or rotational forces on the spine during difficult delivery. The neonate in particular is susceptible to spinal cord injury due to lax ligaments, weak muscles, and incomplete mineralization of the vertebrae, which can allow the spine to stretch further than the spinal cord [55]. Injuries described in the literature include hematomyelia (intramedullary spinal cord hemorrhage), extraspinal hematoma, disruption or transection of the spinal cord, and malalignment or dislocation of the spine [56,57]. Similar to other birth injuries, vaginal breech delivery and instrumented (forceps) delivery are the most commonly cited risk factors for birth-related spinal cord injuries. Upper cervical spine lesions are typically associated with vertex delivery, while lower cervical and thoracic spine injuries (cervicothoracic) are often associated with breech delivery. Forceps rotation during vertex delivery – specifically, rotation of 90° or more from occipitoposterior or occipitotransverse position – has been shown to be a major risk factor for high cervical spine injury (above C4), as demonstrated in a review of 15 cases of upper spinal cord injury by Metigolgou et al. in which the delivery of 14 of the 15 affected neonates involved forceps rotation [53]. Vaginal breech delivery assumes an increased likelihood of lower cervical and thoracic spine injury due to application of excessive pulling forces to achieve delivery of an entrapped head (Figs. 9 and 10). In a retrospective review of 22 neonates with spinal cord injury, all fourteen patients with upper cervical spinal cord injury had vertex presentations, and the six patients with cervicothoracic spinal cord injury all had a breech presentation [58].

Management/Treatment: Depending on the level of spinal cord insult, affected neonates may present at birth with hypotonia, flaccid tetraplegia or flaccid paraplegia, respiratory distress, or apnea. Other birth related injuries may also be evident, such as brachial plexus palsies, or fractures. When risk factors and initial presentation raise the possibility of spinal cord injury, the newborn's head, neck and spine must be immobilized as soon as possible after delivery. Personnel skilled in neonatal resuscitation and endotracheal intubation should be available at delivery, as neonates with lesions above C4 will likely have apnea in the delivery room [53] (Fig. 11). Ultrasound should be urgently obtained at bedside to assist with the diagnosis. In addition to easy access and fast results, ultrasound is the preferred imaging modality due to incomplete ossification of the posterior vertebral arch which allows for visualization of intra- and extra spinal lesions, as well as spinal disruption and malalignment [54,57]. Vertebral fractures or spinal dislocations may accompany the injury, hence frontal and lateral radiographs of the spine should be obtained to identify neonates with these injuries, as some may be amendable to surgical therapy [25,42,56]. Magnetic resonance imaging (MRI) should be obtained early if the nature of the lesion is not clear, and to differentiate between edema, ischemia, or hemorrhage [25]; however, a normal early MRI must be cautiously interpreted as it does not necessarily rule-out spinal cord injury [54,58]. MRI done after the acute phase of injury can be useful to predict long term prognosis. Newborns with upper cervical spinal cord injury have been shown to have worse outcomes than those with cervicothoracic and lower spinal cord injury [56,58]. MacKinnon et al. noted a 50% mortality rate in their analysis of 22 cases of birth-related spinal cord injury, with significant morbidity (ventilator dependency, paraplegia) in the majority of survivors. Age of first

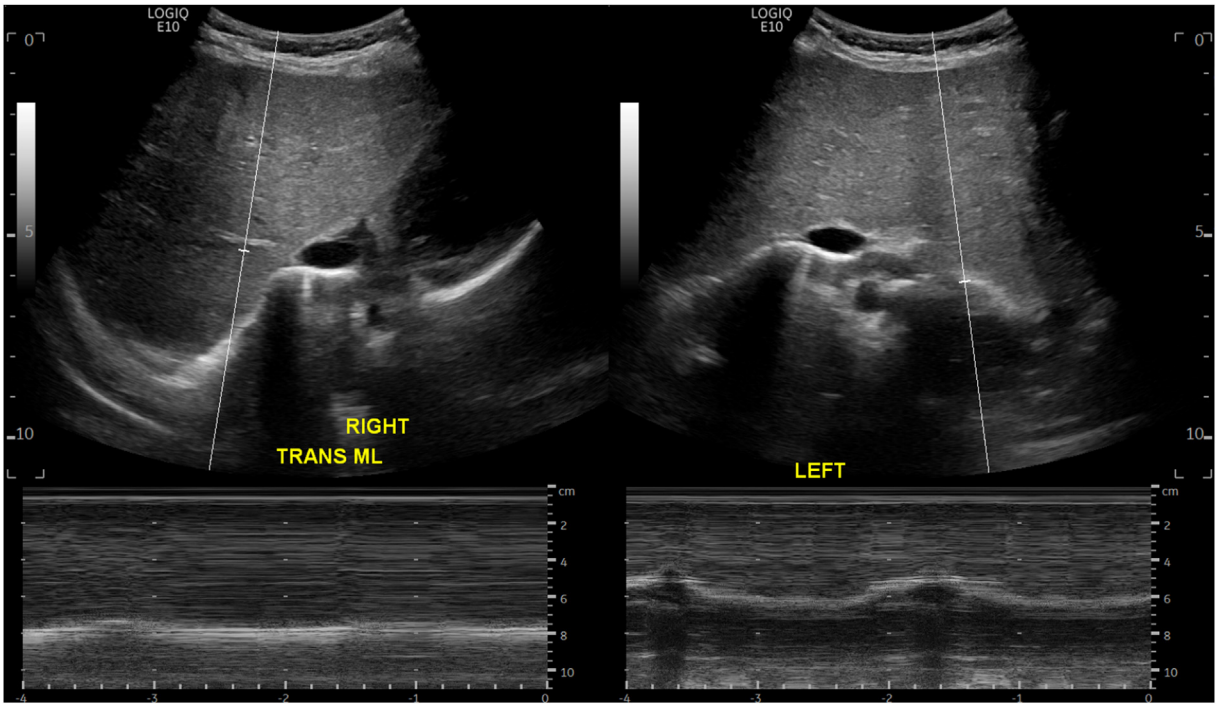

Fig. 8. US images for patient in Fig. 7. Term infant with hypotonia, poor feeding, respiratory concerns. R diaphragm persistently elevated on CXRs, M-mode US shows paralysis of R diaphragm, normal motion of L Courtesy: Amy Mehollin-Ray MD.

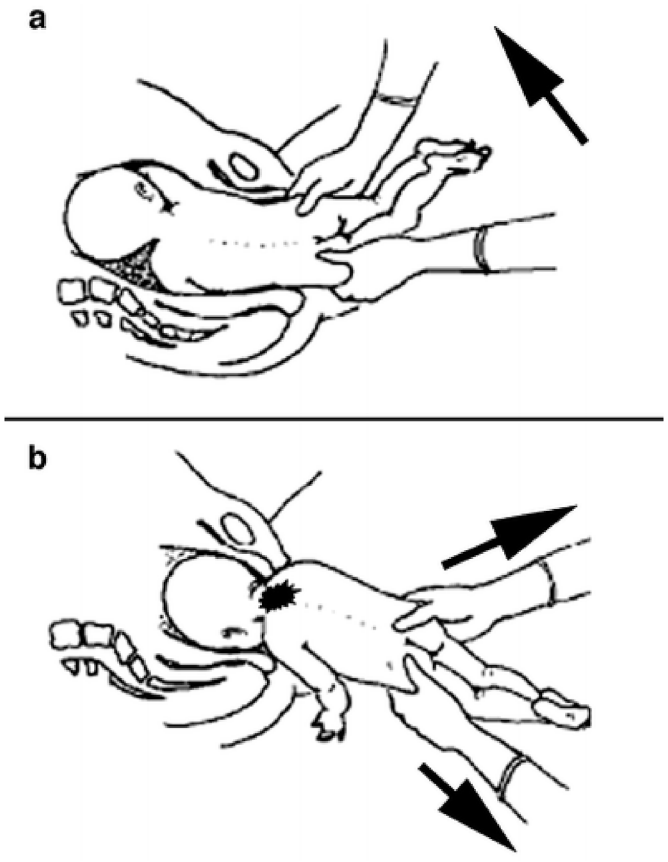

Fig. 9. Mechanism of spinal cord injury during vaginal breech extraction with fetal head entrapment with excessive traction maneuvers. From: Vialle R, Piétin-Vialle C, Vinchon M, Dauger S, Ilharreborde B, Glorion C. Birth-related spinal cord injuries: a multicentric review of nine cases. Childs Nerv Syst 2008; 24(1):79–85. Used with permissions.

spontaneous breath and rate of recovery of motor function in the first three months were noted to be outcome predictors [58].

7. Visceral injury

Injuries to intra-abdominal organs are a rare consequence of birth trauma and can be acutely life threatening due to the risk of organ rupture and severe hemorrhage. These injuries may initially go unnoticed due to lack of visible signs of trauma. Injuries to the liver, spleen, and adrenals have been reported in the literature, with hepatic injury occurring more often than injury to other organs. The incidence of hepatic injury due to birth trauma is unknown, with estimations in the literature based on series from several years ago where subcapsular liver hematoma or hemoperitoneum were noted at the time of autopsy [59,60]. Adrenal hemorrhage is less common than hepatic injury, but occurs more often than splenic injury. Published studies in which several thousand newborns were screened with abdominal ultrasound in the first few days after birth report an incidence of adrenal hemorrhage of 1.9–2.8 per 1000 deliveries [61,62]. Splenic injury is rare in the newborn and precise incidence is unknown, with less than fifty cases reported in the world's literature as of the year 2000 [63].

7.1. Mechanism of injury and risk factors

Several risk factors for birth-related injury to intra-abdominal organs have been identified. As with the previously described birth traumas, macrosomia, breech delivery, “difficult delivery”, and use of instrumentation have been associated with injury to abdominal organs [64]. Compression of the thorax during delivery can cause direct injury to the liver in the area where the costal margin of the rib cage comes in contact with the anterior hepatic surface; compression on the chest can also force the liver downward and cause tearing at the ligamentous peritoneal insertion [65]. Resuscitation at delivery with chest compressions has also been identified as a risk factor for liver injury due to the liver's superficial location adjacent to the heart [64]. The liver is not fully protected by the rib cage in neonates, with a normal anatomic location of up to three centimeters below the right costal margin. These

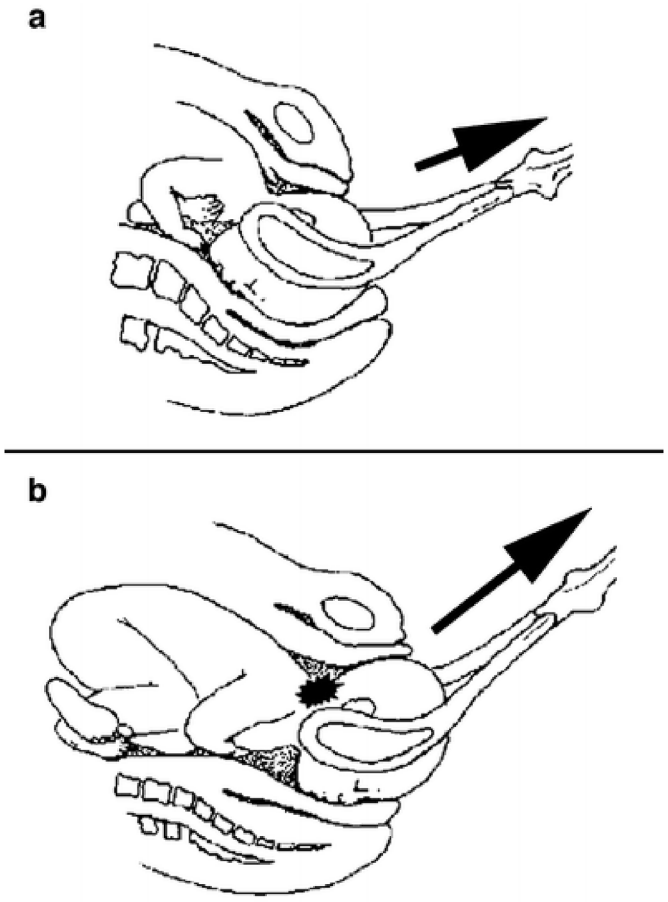

Fig. 10. Mechanism of spinal cord injury forceps assisted vaginal delivery demonstrating possible stretch injury due to excessive traction forces from the forceps. From: Vialle R, Piétin-Vialle C, Vinchon M, Dauger S, Ilharreborde B, Glorion C. Birth-related spinal cord injuries: a multicentric review of nine cases. Childs Nerv Syst 2008; 24(1):79–85. Used with permissions.

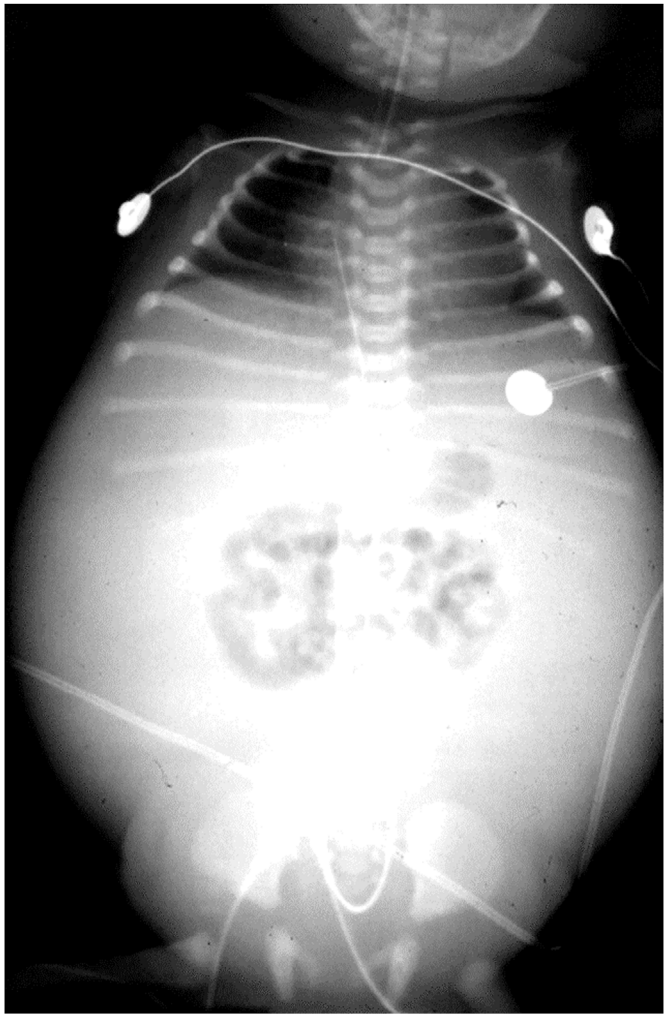

Fig. 11. X-ray demonstrating spinal cord transection at level above C4. Newborn is intubated. Photo courtesy of: Gerardo Cabrera-Meza MD.

factors, in addition to a friable consistency, contribute to the susceptibility of the liver to injury, particularly in premature infants [60]. Similar mechanisms have been proposed for birth-related injury to the spleen. However, unlike the liver, the anatomic locale of the spleen in the left upper quadrant of the abdomen, between the fundus of the stomach and the diaphragm, offers protection against injury during the birth process [66]. The adrenal glands are relatively large and highly vascularized in the neonatal period, features that contribute to the susceptibility of the gland to trauma during delivery [67]. Risk factors most often associated with neonatal adrenal hemorrhage are traumatic vaginal delivery and macrosomia, with the right side more often injured than the left, likely due to compression of the gland between the liver and the spine [68]. In a retrospective study of over 26,000 newborns screened with abdominal ultrasound on day two of age, macrosomia (birth weight $>$ 4 kg) was noted in 21% of the newborns with adrenal hemorrhage [62]. Unilateral adrenal gland injury is more common than bilateral, with bilateral hemorrhage occurring in 5–15% of cases [69].

Management/Treatment: Solid organ injury may occur in two phases, 1) initial subcapsular hemorrhage follow by, 2) rupture of the hematoma with resultant hemoperitoneum. Neonates with solid organ hemorrhage that remains contained within the capsule may have a delayed presentation, with symptoms of anemia (tachycardia, tachypnea, poor feeding) emerging several days after the injury. Conversely, neonates with solid organ rupture present with acute decompensation, sudden pallor, and the classic triad of shock, anemia, and bluish discoloration of the abdomen [55]. Rupture of the liver occurs far more commonly than splenic rupture, although both have been well described in the literature and both constitute a life-threatening emergency for the neonate [64,66,70,71]. Depending on the severity of the injury and rate of bleeding, neonates may present in the delivery room or within hours of delivery, as the hematoma tends to increase up to 4–5 cm prior to rupture into the peritoneal cavity [64]. Abdominal ultrasound at the bedside is a rapid diagnostic tool and should allow differentiation of blood from other forms of ascites [66]. Computed tomography (CT) can be obtained to confirm the source of bleeding, if the neonate is stabilized sufficiently for transport to the CT scanner. In the event of subcapsular rupture, rapid diagnosis and immediate treatment are crucial to reducing morbidity and mortality. Treatment includes aggressive management of hypovolemic shock, and clotting factor replacement as indicated [70]. Volume resuscitation with blood is preferable, although an isotonic crystalloid solution such as normal saline can be used while awaiting the arrival of blood [see section 3.1]. Persistent coagulopathy is treated with fresh frozen plasma, cryoprecipitate, or platelet transfusion if warranted. Conservative management with correction of coagulopathies, blood transfusion, and serial/intensive monitoring is preferred to surgical intervention, although some infants will require laparotomy if bleeding continues despite these efforts [55,63,70]. Splenectomy, previously the standard of care for neonates with splenic rupture, is to be avoided when possible due the increased risk of postsplenectomy sepsis. Successful nonsurgical management of splenic rupture has been reported [63,72]. Nonsurgical management of adrenal hemorrhage is the standard, with spontaneous resolution reported in the majority of cases

[69]. Although adrenal insufficiency as a sequelae of hemorrhage is rare, it has been reported in infants who sustained bilateral injury. In an analysis of 74 neonates with adrenal hemorrhage, Gyurkovits, et al. noted adrenal insufficiency in one infant who had sustained bilateral hemorrhage [62]. Likewise, Zessis et al. report a case of bilateral hemorrhage with subsequent adrenal insufficiency requiring persistent therapy in a newborn with macrosomia and traumatic vaginal delivery [68].

8. Conclusion

Birth injuries with the potential for significant morbidity and mortality must be identified as soon as possible after delivery, and treatment should follow within minutes to hours after birth to ensure the best possible outcome for the neonate. A medical team trained in newborn resuscitation, including persons with advanced resuscitation skills including endotracheal intubation and umbilical vessel catheterization, should be available for every delivery for which there are risk factors for birth trauma.

Declaration of competing interest

The author has no conflicts of interest.

Practice points

- Risk factors for birth trauma are: instrumented delivery, vaginal breech extraction, fetal macrosomia, maternal obesity, and shoulder dystocia.
- Life-threatening birth injuries are rare, require treatment within minutes to hours after delivery, and include: cranial hemorrhage, diaphragm paralysis, spinal cord injury, and visceral injuries.
- Whole blood is the preferred treatment for acute hemorrhage in the neonate, and O-Rhesus-negative, uncrossmatched blood can be given in the acute setting.
- Bedside ultrasound can be useful for rapid evaluation of birth injuries, however MRI or CT may be needed to confirm the diagnosis.
- Intra-abdominal bleeding should be considered for neonates who present with shock, pallor, anemia, and abdominal distension.

References

[1] Demissie K, Rhoads GG, Smulian JC, et al. Operative vaginal delivery and neonatal and infant adverse outcomes: population based retrospective analysis. BMJ 2004;329:24–9.
[2] Alexander JM, Leveno KJ, Hauth J, et al. Fetal injury associated with cesarean delivery. Obstet Gynecol 2006;108:885–90.
[3] Moczygemba CK, Paramsothy P, Meikle S, et al. Route of delivery and neonatal birth trauma. Am J Obstet Gynecol 2010;202. 361.e1.
[4] Reichard R. Birth injury of the cranium and central nervous system. Brain Pathol 2008;18:565–70.
[5] Fitzsimons KJ, Modder J, Greer IA. Obesity in pregnancy: risks and management. Obstet Med 2009;2(2):52–62.
[6] Cedergren M. Maternal morbid obesity and the risk of adverse pregnancy outcome. Obstet Gynecol 2004;103(2):219–24.
[7] Jolly MC, Sebire NJ, Harris JP, Regan L, Robinson S. Risk factors for macrosomia and its clinical consequences: a study of 350,311 pregnancies. Eur J Obstet Gynecol Reprod Biol 2003;111(1):9–14.
[8] Nassar AH, Usta IM, Khalil AM, Melhem ZI, Nakad TI, Abu Musa AA. Fetal macrosomia (> or =4500 g): perinatal outcome of 231 cases according to the mode of delivery. J Perinatol 2003 Mar;23(2):136–41.
[9] Boulet SL, Alexander GR, Salihu HM, Pass M. Macrosomic births in the United States: determinants, outcomes, and proposed grades of risk. Am J Obstet Gynecol 2003 May;188(5):1372–8.
[10] ACOG Committee Opinion Number 745. Mode of term singleton breech delivery. Committee on Obstetric Practice. Obstet Gynecol 2018;132(2).
[11] Towner D, Castro MA, Eby-Wilkens E, Gilbert WM. Effect of mode of delivery in nulliparous women on neonatal intracranial injury. N Engl J Med 1999;341:1709–14.
[12] ACOG Practice Bulletin No. 154. Summary: operative vaginal delivery. Obstet Gynecol 2015;126(5):1118.
[13] Hansen A, Chauhan SP. Shoulder dystocia: definitions and incidence. Semin Perinatol 2014;38(4):184–8.
[14] Spong CY, Beall M, Rodrigues D, Ross MG. An objective definition of shoulder dystocia: prolonged head-to-body delivery intervals and/or the use of ancillary obstetric maneuvers. Obstet Gynecol 1995 Sep;86(3):433–6.
[15] Perlman JM, Wyllie J, Kattwinkel J, et al. Neonatal Resuscitation: 2010 International consensus on cardiopulmonary resuscitation and emergency cardiovascular care science with treatment recommendations. Pediatrics 2010;126(5):e1319–44.
[16] Wyckoff MH, Aziz K, Escobedo MB, et al. Part 13: neonatal Resuscitation 2015 American Heart Association guidelines update for cardiopulmonary resuscitation and emergency cardiovascular care. Circulation 2015;132:S543–60.
[17] Wyckoff MH, Perlman JM, Laptook AR. Use of volume expansion during delivery room resuscitation in near-term and term infants. Pediatrics 2005;115(4):950–5.
[18] Katheria A, Rich W, Finer N. Electrocardiogram provides a continuous heart rate faster than oximetry during neonatal resuscitation. Pediatrics 2012 Nov;130(5):e1177–81.
[19] Finn D, Roehr CC, Ryan CA, Dempsey EM. Optimising Intravenous volume resuscitation of the newborn in the delivery room: practical considerations and gaps in knowledge. Neonatology 2017;112:163–71.
[20] Finn D, Dorrian A, Sheehy J, Dempsey EM, Ryan CA. Emergency uncross-matched blood transfusions in a tertiary neonatal unit. Acta Paediatr 2017 Feb;106(2):218–22.
[21] Wyllie J, Perlman J, Kattwinkel J, et al. Part 7: neonatal resuscitation 2015 international consensus on cardiopulmonary resuscitation and emergency cardiovascular care science with treatment recommendations. Resuscitation 2015;95. e169–201.
[22] Looney CB, Smith JK, Merck LH, et al. Intracranial hemorrhage in asymptomatic neonates: prevalence on MR images and relationship to obstetric and neonatal risk factors. Radiology 2007;242(2):535–41.
[23] Rooks VJ, Eaton JP, Ruess L, Petermann GW, Keck-Wherley J, Pedersen RC. Prevalence and evolution of intracranial hemorrhage in asymptomatic term infants. AJNR Am J Neuroradiol 2008;29(6):1082–9.
[24] Pollina J, Dias MS, Li V, Kachurek D, Arbesman M. Cranial birth injuries in term newborn infants. Pediatr Neurosurg 2001;35(3):113–9.
[25] Chaturvedi A, Chaturvedi A, Stanescu AL, Blickman JG, Meyers SP. Mechanical birth-related trauma to the neonate: an imaging perspective. Insights Imaging 2018;9(1):103–18.
[26] Heyman R, Heckly A, Magagi J, Pladys P, Hamlat A. Intracranial epidural hematoma in newborn infants: clinical study of 15 cases. Neurosurgery 2005;57(5):924–9.
[27] Blauwblomme T, Garnett M, Vergnaud E, et al. The management of birth-related posterior fossa hematomas in neonates. Neurosurgery 2013;72(5):755–62.
[28] Plauche WC. Subgaleal hematoma. A complication of instrumental delivery. J Am Med Assoc 1980;244:1597–8.
[29] Ng PC, Siu YK, Lewindon PJ. Subaponeurotic haemorrhage in the 1990s: a 3-year surveillance. Acta Paediatr 1995;84:1065–9.
[30] Boo NY, Foong KW, Mahdy ZA, Yong SC, Jaafar R. Risk factors associated with subaponeurotic haemorrhage in full-term infants exposed to vacuum extraction. BJOG 2005;112(11):1516–21.
[31] Uchil D, Arulkumaran S. Neonatal subgaleal hemorrhage and its relationship to delivery by vacuum extraction. Obstet Gynecol Surv 2003;58(10):687–93.
[32] Kilani RA, Wetmore J. Neonatal subgaleal hematoma: presentation and outcome–radiological findings and factors associated with mortality. Am J Perinatol 2006;23(1):41–8.
[33] Gebremariam A. Subgaleal haemorrhage: risk factors and neurological and developmental outcome in survivors. Ann Trop Paediatr 1999;19(1):45–50.
[34] Lee SJ, Kim JK, Kim SJ. The clinical characteristics and prognosis of subgaleal hemorrhage in newborn. Korean J Pediatr 2018;61(12):387–91.
[35] Amar AP, Aryan HE, Meltzer HS, Levy ML. Neonatal subgaleal hematoma causing brain compression: report of two cases and review of the literature. Neurosurgery 2003;52(6):1470–4.
[36] Robinson RJ, Rossiter MA. Massive subaponeurotic haemorrhage in babies of African origin. Arch Dis Child 1968;43(232):684–7.
[37] Strauss T, Kenet G, Schushan-Eisen I, Mazkereth R, Kuint J. Rescue recombinant activated factor VII for neonatal subgaleal hemorrhage. Isr Med Assoc J 2009;11(10):639–40.
[38] Hünseler C, Kribs A, Eifinger F, Roth B. Recombinant activated factor seven in acute life-threatening bleeding in neonates: report on three cases and review of literature. J Perinatol 2006;26(11):706–13.
[39] Hughes CA, Harley EH, Milmoe G, Bala R, Martorella A. Birth trauma in the head and neck. Arch Otolaryngol Head Neck Surg 1999;125(2):193–9.
[40] Chauhan SP, Rose CH, Gherman RB, Magann EF, Holland MW, Morrison JC. Brachial plexus injury: a 23-year experience from a tertiary center. Am J Obstet Gynecol 2005;192(6):1795–800.
[41] Stramrood CA, Blok CA, van der Zee DC, Gerards LJ. Neonatal phrenic nerve injury due to traumatic delivery. J Perinat Med 2009;37(3):293–6.
[42] Volpe JJ. Chapter 36 - injuries of extracranial, cranial, intracranial, spinal cord, and peripheral nervous system structures. In: Volpe JJ, Inder TE, Darras BT, editors. Volpe's neurology of the newborn. sixth ed.Elsevier; 2018. p. 1093–123.
[43] Alvord EC, Austin EJ, Larson CP. Neuropathologic observations in congenital phrenic nerve palsy. J Child Neurol 1990;5(3):205–9.
[44] Bowerson M, Nelson VS, Yang LJ. Diaphragmatic paralysis associated with neonatal brachial plexus palsy. Pediatr Neurol 2010;42(3):234–6.

[45] Al-Qattan MM, Clarke HM, Curtis CG. The prognostic value of concurrent phrenic nerve palsy in newborn children with Erb's palsy. J Hand Surg Br 1998;23(2):225.
[46] Foad SL, Mehlman CT, Ying J. The epidemiology of neonatal brachial plexus palsy in the United States. J Bone Joint Surg Am 2008;90(6):1258–64.
[47] Dajani NK, Magann EF. Complications of shoulder dystocia. Semin Perinatol 2014;38(4):201–4.
[48] de Vries TS, Koens BL, Vos A. Surgical treatment of diaphragmatic eventration caused by phrenic nerve injury in the newborn. J Pediatr Surg 1998;33(4):602–5.
[49] Zifko U, Hartmann M, Girsch W, et al. Diaphragmatic paresis in newborns due to phrenic nerve injury. Neuropediatrics 1995;26(5):281–4.
[50] Commare MC, Kurstjens SP, Barois A. Diaphragmatic paralysis in children: a review of 11 cases. Pediatr Pulmonol 1994;18(3):187–93.
[51] Shiohama T, Fujii K, Hayashi M, et al. Phrenic nerve palsy associated with birth trauma–case reports and a literature review. Brain Dev 2013;35(4):363–6.
[52] Garge SS, Passi GR, Ghanekar D. Early thoracoscopic plication of diaphragm in a newborn with brachial plexus palsy and concurrent phrenic nerve palsy. J Indian Assoc Pediatr Surg 2017;22(3):165–7.
[53] Menticoglou SM, Perlman M, Manning FA. High cervical spinal cord injury in neonates delivered with forceps: report of 15 cases. Obstet Gynecol 1995;86(4 Pt 1):589–94.
[54] Rehan VK, Seshia MM. Spinal cord birth injury–diagnostic difficulties. Arch Dis Child 1993;69:92–4.
[55] Uhing MR. Management of birth injuries. Pediatr Clin N Am 2004;51(4):1169–86.
[56] Vialle R, Piétin-Vialle C, Vinchon M, Dauger S, Ilharreborde B, Glorion C. Birth-related spinal cord injuries: a multicentric review of nine cases. Childs Nerv Syst 2008;24(1):79–85.
[57] Simon L, Perreaux F, Devictor D, Millotte B, Husson B, Waguet J. Clinical and radiological diagnosis of spinal cord birth injury. Arch Dis Child Fetal Neonatal Ed 1999;81(3):F235.
[58] MacKinnon JA, Perlman M, Kirpalani H, Rehan V, Sauve R, Kovacs L. Spinal cord injury at birth: diagnostic and prognostic data in twenty-two patients. J Pediatr 1993;122(3):431–7.
[59] Charif P. Subcapsular hemorrhage of the liver in the newborn. An inquiry into its causes. Clin Pediatr (Phila) 1964;3:428–31.
[60] French CE, Waldstein G. Subcapsular hemorrhage of the liver in the newborn. Pediatrics 1982;69(2):204–8.
[61] Felc Z. Ultrasound in screening for neonatal adrenal hemorrhage. Am J Perinatol 1995;12(5):363–6.
[62] Gyurkovits Z, Á Maróti, Rénes L, Németh G, Pál A, Orvos H. Adrenal haemorrhage in term neonates: a retrospective study from the period 2001-2013. J Matern Fetal Neonatal Med 2015;28(17):2062–5.
[63] Bickler S, Ramachandran V, Gittes GK, Alonso M, Snyder CL. Nonoperative management of newborn splenic injury: a case report. J Pediatr Surg 2000;35(3):500–1.
[64] Share JC, Pursley D, Teele RL. Unsuspected hepatic injury in the neonate-diagnosis by ultrasonography. Pediatr Radiol 1990;20(5):320–2.
[65] Gruenwald P. Rupture of liver and spleen in the newborn infant. J Pediatr 1948;33(2):195–201.
[66] Lewis L, Sanoj KM, Poojari G, Kamath SP. Neonate subcapsular splenic hematoma. Indian J Pediatr 2008;75(9):950–2.
[67] Akin MA, Coban D, Doganay S, Durak Z, Kurtoglu S. Intrahepatic and adrenal hemorrhage as a rare cause of neonatal anemia. J Perinat Med 2011;39(3):353–4.
[68] Zessis NR, Nicholas JL, Stone SI. Severe bilateral adrenal hemorrhages in a newborn complicated by persistent adrenal insufficiency. Endocrinol Diabetes Metab Case Rep 2018;2018:17–0165.
[69] Abdu AT, Kriss VM, Bada HS, Reynolds EW. Adrenal hemorrhage in a newborn. Am J Perinatol 2009;26(8):553–7.
[70] Vachharajani A, Paes B. Spontaneous rupture of the liver presenting as scrotal bruising. Am J Perinatol 2001;18(3):147–9.
[71] Osifo OD, Numbere PP, Ovueni EM. Intra-abdominal solid visceral birth injury to neonates following normal spontaneous vaginal delivery. Am J Case Rep 2011;12:19–22.
[72] Hui CM, Tsui KY. Splenic rupture in a newborn. J Pediatr Surg 2002;37(4):E3.
[73] Alvord EC, Austin EJ, Larson CP. Neuropathologic observations in congenital phrenic nerve palsy. J Child Neurol 1990;5:205. Copyright Elsevier: 1995.