Birth Trauma

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Mark Tattersall, Devender Roberts,
and Leanne Bricker

Abstract

Birth trauma refers to the variety of injuries that can be sustained by the infant during the process of labour and delivery. It is a significant cause of neonatal morbidity and mortality. The process of birth involves a combination of mechanical forces acting upon the fetus that can produce tissue haemorrhage and disruption of physiological integrity. These factors may result from the method of delivery, route of delivery or fetal position and size. In addition, obstetric intervention may amplify the effects of these forces and cause or exacerbate birth trauma. Whilst one aim of the obstetrician is to prevent birth trauma by identifying fetuses at risk and making appropriate plans for delivery, most birth injuries are unavoidable and occur despite skilled obstetric and neonatal care. In any discussion of birth trauma it is important to realise that whilst caesarean delivery may be protective of some types of birth trauma, fetal injuries can be seen with caesarean section, even when this is performed as an elective procedure.

Keywords

Fetus • Birth injuries • Newborn • Newborn surgery

M. Tattersall, MA, BM, BCh, MRCOG (✉)
Department of Women's and Children Health,
University of Liverpool, Liverpool Women's
Hospital, Liverpool L8 7SS, UK
e-mail: mark.tattersall@doctors.org.uk

D. Roberts, MB, ChB, MRCOG
Liverpool Women's Hospital,
Crown Street, Liverpool L8 7SS, UK
e-mail: Devender.Roberts@lwh.nhs.uk

L. Bricker, MB, BCh, MRCOG
Corniche Hospital, Abu Dhabi, United Arab Emirates
e-mail: LeanneB@seha.ae

P.D. Losty et al. (eds.), *Rickham's Neonatal Surgery*, https://doi.org/10.1007/978-1-4471-4721-3_17

Birth trauma refers to the variety of injuries that can be sustained by the infant during the process of labour and delivery. It is a significant cause of neonatal morbidity and mortality. The process of birth involves a combination of mechanical forces acting upon the fetus that can produce tissue haemorrhage and disruption of physiological integrity. These factors may result from the method of delivery, route of delivery or fetal position and size [1]. In addition, obstetric intervention may amplify the effects of these forces and cause or exacerbate birth trauma. Whilst one aim of the obstetrician is to prevent birth trauma by identifying fetuses at risk and making appropriate plans for delivery, most birth injuries are unavoidable and occur despite skilled obstetric and neonatal care. In any discussion of birth trauma it is important to realise that whilst caesarean delivery may be protective of some types of birth trauma, fetal injuries can be seen with caesarean section, even when this is performed as an elective procedure [2].

Birth trauma has been estimated to occur in 3% of all live births and accounts for 2% of all neonatal mortality and 10% of all neonatal deaths in infants delivered at term [3]. Whilst the cause of much birth trauma is difficult to define and it can sometimes prove impossible to explain, a large number of risk factors have been identified for birth trauma and these can be divided into those pertaining to the mother and those relevant to the fetus, as shown in Table 17.1.

There are a wide variety of types of injury that can occur during the process of labour and birth and which will be discussed in this chapter. For ease, injuries have been broken down into the following types:

- Injuries to the Head
- Fractures
- Nerve injuries
- Anoxic injuries

Table 17.1 Risk factors for birth trauma [72]

Maternal	Fetal
Diabetes	Macrosomia
Obesity	Pregnancy prolonged beyond Term +18 days
Small pelvis	Abnormal presentation
Large pregnancy weight gain	Instrumental delivery
Short maternal stature	Perinatal depression
Induction of labour	Shoulder dystocia
Epidural usage	
History of macrosomic infant	

17.1 Head Injuries

The head is the most common part of the body injured during labour and delivery, which is due to the fact that this is usually the presenting part.

17.1.1 Superficial Injuries

Superficial head injuries can occur at all types of delivery and are relatively common. Bruising, abrasions and lacerations can occur at the time of spontaneous vaginal and instrumental deliveries. Rates of 0.1% of all deliveries [4], around 10% with ventouse deliveries and approximately 35% with forceps deliveries [5] have been quoted. Lacerations to the scalp, face, cheek or ear can occur at Caesarean section and it is good practice to specifically counsel mothers regarding this risk. It is estimated that the risk of such lacerations is approximately 1%, with the risk not necessarily being associated with any particular type of Caesarean section, the presentation, dilatation, presence of intact membranes or the experience of the surgeon [6–8]. If a full-thickness laceration is present, suturing is often required and consultation with a plastic surgeon is recommended to achieve a cosmetically satisfactory result, particularly for injuries to the face. The two most common types of injuries to the head are caput succedaneum and cephalohaematoma. Other head injuries include subgaleal haemorrhages, intracranial haemorrhages, nasal injuries and eye injuries. Figure 17.1 illustrates the location of the fluid collections in the major types of scalp injuries.

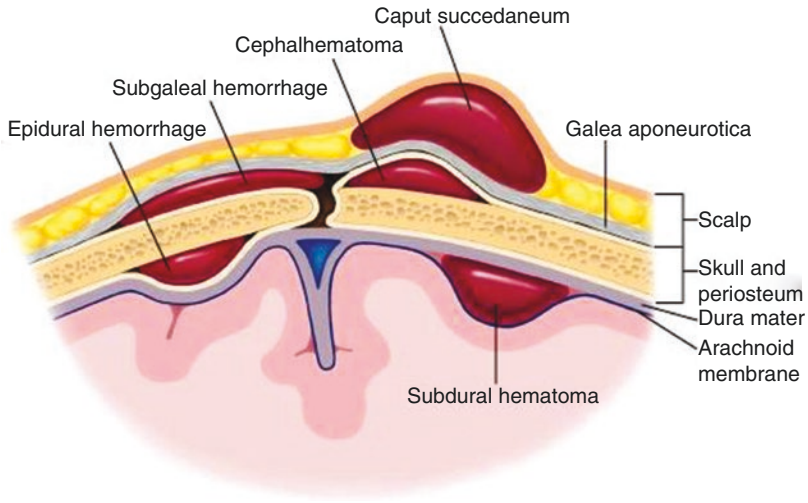

Fig. 17.1 Schematic diagram illustrating anatomical regions of the newborn skull and site(s) for potential birth injury(s)

17.1.2 Caput Succedaneum

A caput succedaneum is a subcutaneous, extra-periosteal collection of serosanguinous fluid which is associated with head moulding. This common lesion at birth is usually seen on the presenting portion of an infant's skull during vaginal birth and is often accompanied by bruising and petechiae. It is caused by the high pressure exerted on the fetal head during labour by the uterus and vaginal walls as the head passes through the birth canal. This prolonged pressure causes leakage of serosanguinous fluid from the subcutaneous tissues into the area between the scalp and the lining of the periosteum, causing oedema and bruising. Caput succedaneum is easily differentiated from a cephalohaematoma by the fact that the fluid accumulation crosses over the cranial sutures due to the collection being above the periosteum. Again in contrast to cephalohaematoma, a caput succedaneum is evident immediately after delivery and will subsequently decrease in size over time. A caput succedaneum has a soft, boggy feel and may display petechiae, purpura or ecchymosis. The swelling is generally 1–2 cm in depth and is most commonly seen in the midline with the circumference being highly variable in size and the margins usually being irregular in shape. In caput succedaneum, the collection of serous fluid characteristically shifts from side to side as the infant's head position is changed.

Risk factors for caput succedaneum include nulliparity, a prolonged second stage of labour, premature rupture of membranes and ventouse delivery, with the latter being a particular risk where the vacuum is applied for over 10 min, there is inappropriate cup placement or repetitive cup detachments. In the case of ventouse delivery, the 'artificial' caput succedaneum (commonly known as a 'chignon') is caused by a collection of interstitial fluid and microhaemorrhages occurring under the cup site and is helpful in keeping the vacuum cup more attached to the fetal scalp [9]. In rare cases of ventouse delivery, caput succedaneum can be associated with breakage of the skin of the scalp if the cup "pops off" the head and abrades the underlying skin. Although it is thought that pressure exerted on the fetal head during labour and delivery is the causative factor in the development of caput succedaneum, it is important to note that there are several cases reported in the literature where this injury has been described as being identified by ultrasound in the third trimester, suggesting that this injury does not always occur during labour and delivery. Caput succedaneum does not usually require any treatment, resolving spontaneously within the first few days after birth [10, 11].

Alopecia can occur in conjunction with caput succedaneum. This is usually in the form of a halo scalp ring, where hair loss develops as a result of tissue necrosis from prolonged pressure of the

scalp against the ring of the cervical os during the birth process. In most cases, the hair grows back over time, but scarring and permanent hair loss have been reported in association with caput succedaneum [12]. An important final point to note is that caput succedaneum can be confused with a subgaleal haemorrhage (discussed below), as this also crosses the suture lines. It is important that such a misdiagnosis is not made as this can have potentially catastrophic results due to the more serious nature of the latter diagnosis.

17.1.3 Cephalohaematoma

A cephalohaematoma is a collection of blood below the periosteum of the skull. This extracranial haemorrhage occurs when friction during the birth process causes the emissary and diploic veins between the periosteum and the skull to rupture. As the haematoma is subperiosteal, it does not extend across suture lines as the ligaments which attach the periosteum to the skull at the cranial suture lines contain the blood. A cephalohaematoma can thus usually be easily distinguished from a caput succedaneum or a subgaleeal haemorrhage, although where there is a caput succedaneum or scalp swelling overlying a cephalohaematoma, this can obscure its boundaries and make diagnosis more difficult. Figure 17.2 shows a MRI where the differences between a caput succedaneum and a cephalohaematoma can be clearly seen. A cephalohaematoma is not usually present at birth unless there is a history of prolonged head engagement and usually develops during the first 24 h of life due to the slow nature of subperiosteal bleeding. In contrast to caput succedaneum, there is not usually any discolouration of the overlying skin due to the accumulation being in a deeper, more vascular tissue plane. This also accounts for the increased blood content of cephalohaematomas, which continue to increase in size until pressure in the space builds and acts as a tamponade to stop further bleeding. Cephalohaematomas present as firm masses that may be either unilateral or bilateral covering one or more bones of the scalp. The lesion cannot usually be transillumi-

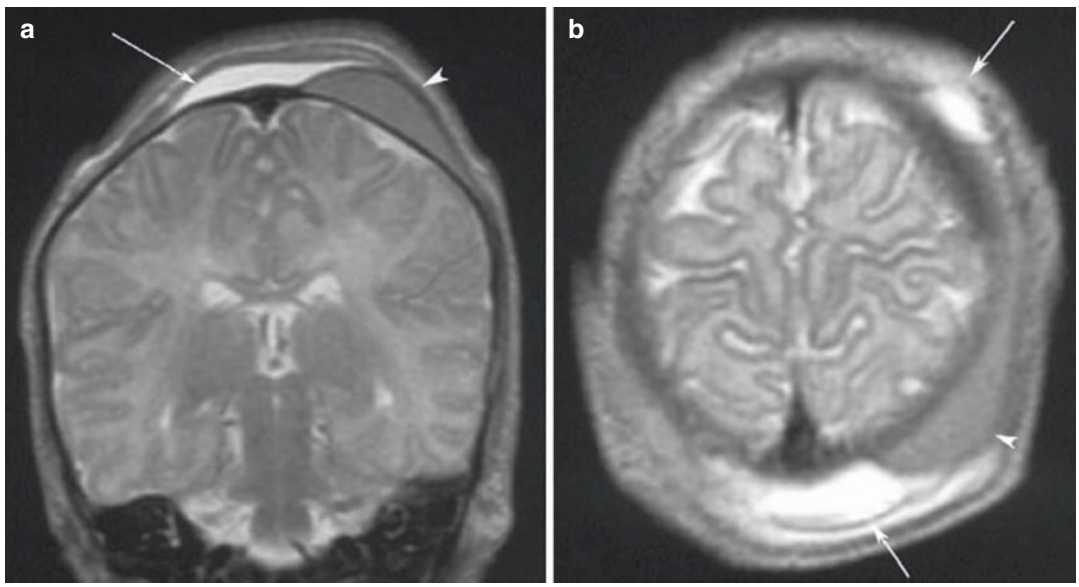

Fig. 17.2 Brain MRI. Coronal (**a**) and axial (**b**) images reveal the caput succedaneum (*arrow*) with crossing the suture line and the cephalhaematoma (*arrowhead*) without crossing the suture line. The low signal intensity of the cephalhaematoma suggests the subacute haematoma. From: Choi JW, Lee CH, Suh SI. Scalp swelling crossing the suture line on skull radiograph: is it always a sign of caput succedaneum? Pediatr Radiol (2006) 36: 364. doi:10.1007/s00247-006-0113-6

nated and are most commonly found in the parietal region, although it may occur over any of the bones of the skull. The right parietal bone is involved twice as commonly as the left and unilateral lesions are five-times more common than bilateral ones.

The incidence of cephalohaematomas has been estimated to be up to 2.5% of all live births [13–15]. The rate of cephalohaematoma is significantly increased with ventouse delivery (compared with normal vaginal delivery) [16, 17] and is also increased to a smaller extent by forceps delivery [18]. Other risk factors for cephalohaematomas include primiparity, macrosomic infants, prolonged labours and abnormal fetal positions such as occipito-transverse or occipito-posterior. Cephalohaematoma is twice as common in males than in females for reasons that are unknown. Although cephalohaematomas may be potentially more serious than caput succedaneum, they are usually benign if there is no underlying coagulation disorder and treatment is rarely indicated. The haematoma generally resolves spontaneously and completely by 3 months of age.

There are a number of important complications which may be associated with a cephalohaematoma. Linear skull fractures occur in approximately 5% of unilateral and 18% of bilateral cephalohaematomas. Routine radiography is not recommended, but should be undertaken if the cephalohaematoma is excessively large, there are neurological signs present or when a particularly difficult delivery has taken place. Such linear skull fractures rarely require treatment. Cephalohaematomas can become infected and when signs of sepsis are present and the focus of the infection cannot otherwise be explained, the cephalohaematoma should be suspected as the site of infection. There is an increased risk of infection if a scalp electrode has been used during labour, if a needle aspiration of the cephalohaematoma has previously been attempted or if there is already systemic infection existing in the infant. The most common organisms include E. coli and S. aureus. Needle aspiration is used to diagnose infection of a cephalohaematoma, but is only performed when other possible sources of infection have been excluded, as it may introduce organisms into a previously sterile area. Treatment of infected cephalohaematoma is usually with intravenous antibiotics for up to 2 weeks, with initial antibiotic selection made to cover both E. coli and S. aureus. When infection has been diagnosed following needle aspiration, antibiotics are usually adjusted according to sensitivity results and CT can be used to reveal any evidence of osteomyelitis, epidural abscess or subdural empyema, which may necessitate a longer course of intravenous antibiotics. In cases where there is no clinical improvement despite antibiotic treatment, surgical incision, drainage and evacuation may also be considered.

Anaemia may be a complication associated with larger cephalohaematomas and can occasionally require transfusion. Hyperbilirubinaemia is a common side effect of cephalohaematoma, which occurs when the red blood cells in a cephalohaematoma are destroyed, with an end metabolic product being bilirubin. Bilirubin levels should therefore be monitored whilst a significant cephalohaematoma resolves and phototherapy is effective is returning unconjugated bilirubin levels to normal.

Calcification can rarely be a complication of cephalohaematoma, which usually occurs when the lesion persists beyond 4 weeks of age. It can cause a misshapen head and such skull deformities can require treatment. Figure 17.3 shows a CT scan of a calcified haematoma. A persistent

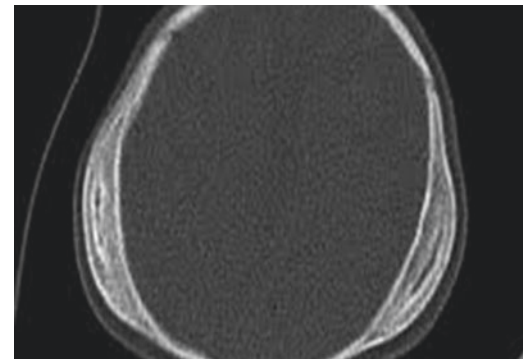

Fig. 17.3 Computed tomography scan showing calcified cephalohematoma. From: Kestle JRW. Tumors of the cranial vault in children. In Tonn J-C, Westphal M, Rutka, JT, eds. Oncology of CNS Tumors. Springer-Verlag: Berlin Heidelberg; 2010: 629–635

disfiguring calcified cephalohaematoma can require surgical treatment. The bony cap of a calcified cephalohaematoma is removed and the underlying material is debrided. A contouring surgical burr is used to reshape the skull and reform the correct anatomical form, with bony shavings used to fill in any depression left. Passive cranial moulding helmet therapy has also been used as an effective non-surgical treatment for calcified cephalohaematomas. It relies upon the malleability of the infant's head prior to 12 months of age and involves placing a moulded helmet on the infant's head for 18–20 h per day until the desired cranial shape is achieved. This non-surgical treatment is usually only successful in the case of partially calcified cephalohaematomas, as fully calcified cephalohaematomas do not usually respond as well.

17.1.4 Subgaleal Haemorrhage

Subgaleal haemorrhage is a potentially fatal lesion, which results from bleeding into the subaponeurotic space, a potential space between the epicranial aponeurosis of the scalp and the periosteum of bone. The epicranial aponeurosis is a sheet of fibrous tissue which covers the entire cranial vault from the orbital ridges anteriorly to the nuchal ridge posteriorly. Its lateral margins blend with the temporal fascia. The space formed is potentially a large one, estimated to be able to accommodate up to 360 ml of blood. The fact that such extensive blood loss is possible means that mortality rates from subgaleal haemorrhage have been estimated to be up to 22%. Diagnosis of subgaleal haemorrhage is generally made clinically, with a boggy swelling over the scalp generally being noted between 12 and 72 h after delivery, although in severe cases signs may be present at delivery. The swelling may be associated with bruising and periorbital or periauricular oedema. The infant usually has an irritable cry and signs of pain, especially when the head or scalp is manipulated. Severe cases are associated with cardiovascular collapse. Subgaleal haemaorrhage can be distinguished from cephalohaematoma in that the swelling crosses the suture lines. The presentation can often be insidious, which can cause delay in diagnosis and treatment, which contributes significantly to the high mortality. Subgaleal haemarrhage is most common after ventouse delivery [19, 20]. It is thought that use of a ventouse produces a shearing force to the scalp that tears the emissary veins and produces the haemorrhage, but it is important to note that subgaleal haemorrhage has also been observed in association with Caesarean section delivery. Subgaleal haemorrhage has been estimated to occur in less than 0.1% of all deliveries, but in up to 1% of ventouse deliveries [21].

The diagnosis of subgaleal haemorrhage is usually made by careful physical examination demonstrating a ballotable lesion which crosses the suture lines. It is important that treatment is not delayed whilst waiting for CT or MRI confirmation of the diagnosis. The priority is to stabilise the infant and radiologic examination can be safely deferred useless there are focal neurological signs indicating the potential for a subdural lesion. Serial head measurements can be used to monitor the evolution of a subgaleal haemorrhage, with it having been estimated that approximately 38 ml of blood have accumulated for each centimetre increase in head circumference. Signs that indicate increasing severity include severe anaemia, hypotension, weak pulses and tachycardia and once hypovolaemic shock has developed the infant's chances of survival are poor, due to decreased organ perfusion, myocardial damage and widespread cellular death. It is thus critical that a subgaleal haemorrhage is recognised early and treated with aggressive volume resuscitation in order to provide the best chances of full recovery. Treatment also requires careful monitoring of the infant's condition, regularly assessing vital signs, neurological status and clotting variables. If blood products are transfused, it is essential to monitor the haematocrit, coagulation studies and the platelet count until haemostasis is achieved.

17.1.5 Intracranial Haemorrhage

Intracranial haemorrhage, including subarachnoid haemorrhage and subdural haemorrhage should be considered after any difficult delivery

to prevent life-threatening complications. However, it is important to realise that the presence of such lesions in newborns is not always subsequent to birth trauma. For example, a series of six cases have been reported where intracranial haemorrhage occurred in the absence of birth trauma [22]. In three of these cases, there was a clotting factor deficiency, highlighting the role that such abnormalities play in intracranial haemorrhage. Intracranial haemorrhage is regarded as the most serious complication of neonatal alloimmune thrombocytopenia, a condition estimated to occur in 1 in 2000–5000 neonates [23].

Subarachnoid haemorrhage is the most common intracranial lesion detected after birth. Where it is secondary to birth trauma, it commonly occurs in conjunction with another cranial lesion, such as caput succedaneum, cephalohaematoma or a skull fracture. A subarachnoid haemorrhage occurs when bridging veins tear or vessels rupture and bleed into the subarchnoid space. It is a relatively benign lesion and rarely has complications, being often discovered as an incidental finding on CT scan. When symptomatic, the most common presentation is with transient seizure activity on day 2 or 3 of life in an otherwise healthy infant. If signs of apnoea, lethargy or abnormal neurology are present, imaging should be undertaken to confirm the diagnosis and exclude other pathology. In rare cases, massive haemorrhage can develop leading to progressive hypovolaemic shock and death. Severe cases can require craniotomy and haematoma evacuation and aggressive management of coagulopathies. Hydrocephalus is a rare complication, which develops due to adhesions or meningeal inflammation blocking the flow of cerebrospinal fluid.

A subdural haemorrhage occurs when tears in the falx, tentorium or bridging cortical veins bleed between the dura and arachnoid. It is thought that excessive vertical moulding of the cranium in a difficult delivery provides excessive stretch on these vessels. Subdural haemorrhages can vary in severity from small asymptomatic lesions which may present as an incidental finding on imaging to a massive haemorrhage associated with deteriorating neurological function and signs of brain stem compression. Severe haemorrhages tend to present between 12 and 72 h after birth with abnormal neurological signs that may include seizures, apnoea, hypotonia, cyanotic episodes, hemiparesis, decreased movements and unequal or sluggish pupils. A full fontanelle or a rapidly increasing head circumference can be indicative of raised intracranial pressure and should therefore prompt urgent review of management. The majority of subdural haemorrhages occur over the cerebral hemispheres or within the posterior fossa. Haemorrhages in the posterior fossa are clinically important as they can cause brainstem compression, which may present with symptoms of abnormal pupil reactivity, changes in vital signs, severe, progressive neurological deterioration or even coma. Subdural haemorrhage associated with tentorial tears can cause particularly severe haemorrhage and are generally seen in situations where there is rapid forcing of the head through the birth canal, for example in precipitate labour or vaginal breech delivery.

Subdural haemorrhages are best diagnosed by the use of CT or MRI, although cranial ultrasound may detect a very large bleed. The treatment of subdural haemorrhages depends upon the location and extent of the haemorrhage, as well as its progression. Small haemorrhages may be successfully managed conservatively with careful continuing assessment of vital signs, haematocrit, platelet count and perfusion status [24]. Transfusion of packed red cells, platelets and clotting factors should take place as indicated. Anticonvulsant treatment should be added if seizure activity is present. If there is seizure activity of abnormal or focal neurological findings, consultation should taken place with a neurologist and neurosurgeon. Surgery is needed the minority of cases [24–26] and is considered if there are signs of brainstem compression or deterioration in neurological status. Smaller lesions may be treated using Burr holes or subdural drains in order to decrease brain compression from accumulated blood, but severe lesions may require craniotomy and haematoma removal. The prognosis for subdural haemorrhage depends upon the extent and severity of the bleeding. The prognosis is generally good if only a small haemorrhage is present and the infant experiences only transient seizure activity. However, developmental delay

or cerebral palsy have been described in severe cases. Although short-term outcome appears favourable in the majority of cases, long-term outcome remains unclear and careful follow-up is required [26].

17.1.6 Nasal Injuries

Nasal injuries of some form are estimated to occur in between 0.5% and 1% of live births [4]. The injury usually involves dislocation of the cartilaginous portion of the septum from the vomerine groove and columella and present with deviation of the tip of the nose to one side with leaning of the columella and flattening of the nasal aperture and frequently bruising. These injuries are usually benign and rarely result in long-term complications, but as newborn infants are obligate nose breathers, severe septal deviation can compromise breathing. Ideally, nasal injuries should be detected and treated early, ideally within the first 3 days of life. Manual manipulation of the nose into proper alignment is usually performed by an Ear Nose and Throat specialist and involves, steadying the head, grasping the dorsum of the nose and lifting a nasal elevator to push the anterior end of the septum into the septal groove and columella.

17.1.7 Eye Injuries

Birth trauma to the eye is relatively common. The commonest type of injury are benign conjunctival haemorrhages, which are frequently observed after normal vaginal delivery [10]. These appear as a bright red patch or crescentic band located on one side of the iris or completely surrounding it and are due to the rupture of small capillaries in the conjunctivae. These dilated blood vessels are thought to be damaged due to increased venous pressure in the head and neck produced by compression of the fetal thorax and/or abdomen by uterine contractions during labour and delivery. The presence of a nuchal cord may also contribute [27]. These conjunctival haemorrhages require no treatment and resolve within 7–10 days with no risk of permanent damage [28].

More serious eye damage is rare, but damage to the cornea and haemorrhage into the orbit of the eye have been described [29]. Corneal damage can occur due to an inappropriately placed forceps blade slipping over the orbital wall and exerting pressure on the cornea. Such an injury may permanently impair vision. Orbital haemorrhage can occur during difficult delivery and presents as either unilateral or bilateral proptosis. Treatment with antibiotic ointment is essential to prevent infection and patching usually results in complete recovery.

17.2 Fractures

17.2.1 Skull Fractures

Skull fracture should be suspected in any case of cephalohaematoma or subarachnoid haemorrhage. Between 10 and 25% of cephalohaematomas are associated with a skull fracture. Skull X-ray is indicated when physical assessment suggests a fracture or there are abnormal neurological signs [11]. Skull fractures can occur with any type of delivery, including Caesarean section, but are most commonly associated with instrumental delivery [30]. Neurosurgical consultation is highly recommended in the case of any fracture. Skull fractures can be linear or depressed. Linear skull fractures are usually asymptomatic and heal without intervention, although a repeat X-ray should be performed at 6 weeks of age to exclude a leptomeningeal cyst, an uncommon complication that results from extrusion of the leptomeninges into the fracture site and can interfere with the normal healing process. A depressed skull fracture is seen as either an indentation of the skull or a “ping-pong” type defect. Surgical intervention is usually needed to elevate the skull and prevent brain compression, as when such fractures are treated early they have an excellent prognosis in the absence of underlying brain injury.

17.2.2 Clavicular Fracture

Clavicular fractures are the most common type of fracture that may occur as a result of birth trauma.

In some studies, they have been reported to be the most common type of birth trauma. Rates of between 0.2% and 5% have been reported [3, 31–37]. This large variability in observed incidence probably results from differences in the way in which infants were screened for clavicular fracture as many fractures are asymptomatic or present late and also whether the study was prospective or retrospective. Although a displaced fractured clavicle may be easy to diagnose clinically, a non-displaced fracture may often only be apparent if the neonate is subjected to X-rays or multiple physical examinations as some fractures may not be apparent until 1 week after birth.

Although some clavicular fractures may occur either intentionally or non-intentionally during attempts to relieve shoulder dystocia, the majority occur following vaginal deliveries not reported as being complicated by difficulty delivering the shoulders [33, 35]. Although clavicular fractures are also associated with higher birth weights [3, 33], nearly 80% of newborns with clavicular fractures weigh less than 4000 grams [36]. This means that in the majority of cases, the risk of clavicular fracture cannot be appreciated prior to delivery. Isolated clavicular fractures usually heal without sequelae, although they can be associated with brachial plexus, phrenic and right recurrent laryngeal nerve injuries and so these must be excluded if a clavicular fracture is diagnosed in a newborn.

17.2.3 Long Bone Fractures

Fractures of the long bones due to birth trauma are uncommon, since the force required to break such bones is much higher [38]. For example, femoral fractures have been estimated to occur once in every 10,000 deliveries [38, 39].

17.3 Nerve Injuries

17.3.1 Brachial Plexus Injury

Brachial plexus injuries at birth are frequently attributed to the use of excessive lateral traction on the neck during a difficult delivery of the shoulders in a vertex presentation or the delivery of the aftercoming head in a vaginal breech delivery and it is also thought that direct compression of the plexus may occur with the use of forceps. Early studies implicated vaginal breech deliveries as the cause of the majority of brachial plexus injuries. For example, in 1973, 72% of neonates with this type of injury were estimated to have been delivered in this way [40] and the risk of brachial plexus injury in vaginal breech delivery has been estimated to be 17 times that of a vertex presentation [32]. However, with changes in the management of breech presentation the association of brachial plexus injury with shoulder dystocia has become more important. Other risk factors for brachial plexus injury include primiparity, prolonged labour, heavier birth weight and forceps delivery [3, 16, 41, 42]. The incidence of brachial plexus injury has been estimated by a variety of studies to be between 0.4 and 0.9 per 1000 live births [40, 41, 43–47], although a single study suggested a rate as high as 2.6 per 1000 live births [3].

Upper brachial plexus injuries are the most common type seen following delivery. These palsies were first described clinically by Smellie in 1764 in his midwifery text and in 1874 Erb localised the lesion to the junction of 5th and 6th cervical roots (now known as Erb's point) and also credited Duchenne with the original clinical dissection 2 years previously. It is noteworthy that Erb recognised that the cause of the palsy was excessive lateral traction of the neck during delivery and condemned the use of the 'Prague manoeuvre' common at the time, in which strong traction was placed on the shoulder to deliver the aftercoming head in breech presentations. Erb-Duchenne palsies comprise 90% of all brachial plexus injuries and cause paralysis of the deltoid, infrascapular and flexor muscles of the forearm. The presentation is thus with a flaccid upper arm, internal shoulder rotation, elbow extension, forearm pronation, wrist and finger flexion (the "waiter's tip" deformity) and an asymetric Moro reflex. The arm falls limply to the side, but the grasp remains intact. Figure 17.4 shows the characteristic appearance of a child with an Erb-Duchenne palsy. The Moro reflex is present in cases of shoulder girdle fracture and so can differentiate such pseudopalsies from brachial

Fig. 17.4 An 8-month-old boy with Erb's palsy at birth had no shoulder abduction or elbow flexion against gravity. From: Shigematsu K, Yajima H, Kobata Y, Kawamura K, Maegawa N, Takakura Y. Oberlin partial ulnar nerve transfer for restoration in obstetric brachial plexus palsy of a newborn: case report. J Brachial Plex Peripher Nerve Inj. 2006 Sep 29;1:3

plexus palsy. The Klumpke paralysis, described in 1885, involves the 5th, 6th and 7th cervical roots. Clincially, this differs from the Erb-Duchenne paralysis in that the grasp reflex is also absent. Horner's syndrome (ptosis, miosis and anhidrosis) can also be present if sympathetic fibres from the 1st thoracic root are also involved.

Before 1970, the literature suggested a poor prognosis for brachial plexus injuries with full recovery only occurring in the minority of patients [43, 48]. However, more recent reports have suggested full recovery occurs spontaneously in 80–95% of cases [40, 45, 46, 49]. It is now generally accepted that initial management of brachial plexus injuries should be with physiotherapy and dynamic splints and surgery should be delayed for at least 3 months and reserved to cases resistant to conservative measures [50].

It has been proposed that performing elective Caesarean section for fetuses estimated to be relatively macrosomic might be effective in reducing the incidence of shoulder dystocia and hence brachial plexus injuries. However such an approach is limited by the fact that ultrasound estimation of fetal weight is relatively inaccurate and that about half of brachial plexus injuries occur in the absence of shoulder dystocia [51–53] and calculations have suggested that more than 1000 Caesarean sections and their associated expense and morbidity would be required to prevent a single permanent brachial plexus injury, using any possible threshold to perform Caesarean section [54, 55].

17.3.2 Phrenic Nerve Injury

When a phrenic nerve injury occurs as a result of birth trauma, the majority occur in conjunction with a brachial plexus injury [4]. Given that the phrenic nerve innervates the diaphragm and this is the major effector of spontaneous respiration in the newborn, the injury should be suspected when there is evidence of respiratory distress following a difficult labour or delivery, particularly if there is evidence of a brachial plexus injury. Although respiratory distress usually presents on the first day of life, this may be delayed until up to 1 month of age [4]. The infant's breathing is usually laboured and thoracic, and auscultation reveals decreased breath sounds on the affected side. The diagnosis is confirmed by chest X-ray, which will show elevation of the affected hemidiaphragm, and fluoroscopy which reveals paradoxical "see-saw" movement of the affected side. It is important to realise that radiography can sometimes provide a false negative result, particularly if the patient is receiving ventilatory support. Recovery is usually spontaneous, but pulmonary infection can be a serious complication and so prophylactic antibiotics may be helpful. An affected infant should be placed on the affected side and respiratory support provided as necessary. Feeding by nasogastric tube may be utilised to save the infant energy to utilise for breathing. Diaphragmatic pacing can be used and surgical plication of the diaphragm is occasionally necessary.

17.3.3 Facial Nerve Injury

Facial nerve palsy acquired due to birth trauma needs to be distinguished from developmental facial paralysis and is estimated to occur in less than 1% of live births [4, 56]. Factors predictive of the injury include forceps delivery, a prolonged second stage of labour, increased birth weight and primiparity [3], although over 90% of facial nerve palsies due to birth trauma occur in infants delivered with the use of forceps [32, 56]. In a forceps

delivery, the injury is thought to be caused by pressure from the forceps blade on the stylomastoid foramen or compression of the bone overlying the vertical segment of the facial canal. When an injury occurs following normal delivery, it is believed that the pressure is caused by the sacral promontory. The prognosis for these injuries is good, with spontaneous recovery usual in a timeframe ranging from hours to weeks. Electromyography (EMG) is recommended, together with recording of the auditory brainstem response to exclude auditory nerve involvement. Although some experts recommend surgical intervention as early as 1–3 months of age, most reports favour observation for up to 1 year or up to 2 years if there have been improved EMG findings.

17.3.4 Laryngeal Nerve Injury

The major problem caused by injury to the laryngeal nerve is vocal cord paralysis, due to dysfunction of the motor supply to the larynx. However, it has been estimated that only 19% of vocal cord paralysis in newborns is attributable to birth trauma [4]. The injury occurs when the laryngeal nerves are overstretched during delivery and presentation is usually immediately after delivery. The presenting signs depend upon whether the vocal cord paralysis is unilateral or bilateral. 60% of cases involve bilateral paralysis and these cases tend to present with a high-pitched cry, inspiratory stridor and potentially a compromised airway. The remaining cases involve unilateral paralysis and tend to have more mild stridor and a hoarse or breathy cry. In either group, there may also be dysphonia, dysphagia or aspiration. Laryngeal nerve injury has been reported in all types of delivery.

Direct laryngoscopy allows the otolaryngologist to directly observe paralysis of the vocal cords. Investigation usually also involves a modified barium swallow and the early involvement of a speech therapist may help optimise feeding [4]. Management of unilateral vocal cord paralysis is usually conservative with monitoring for any possible aspiration and treatment of any gastro-esophageal reflux. Bilateral vocal cord paralysis more commonly requires treatment with tracheostomy, with the mean time before decannulation being over 4 years. Further surgical management is usually reserved for cases where permanent paralysis seems likely. Arytenopexy has been used to lateralise the laryngeal structures and increase the tracheal opening, with the aim of allowing for decannulation. Alternatively, a muscle-pedicle reinnervation procedure has been described with 50% of patients successfully decannulated with no further loss of voice. Unfortunately, it has been reported that this procedure can result in a further loss of voice and so it has been suggested that procedures such as this should be delayed until the child is old enough to take part in the decision process [4].

17.4 Anoxic Injuries

Cerebral palsy is a term used to describe a group of motor impairment syndromes through to occur secondary to disorders of early brain development. Encephalopathy is the precursor of cerebral palsy and has clinical signs which include altered states of arousal (i.e. hyperalterness with or without seizures or unresponsiveness), abnormalities of muscle tone and strength, focal neurological deficits or delayed developmental milestones). Although encephalophathy is often described as ‘hypoxic-ischaemic encephalopathy (HIE)’, such clinical signs can reflect multiple mechanisms of brain injury over any time course with the underlying pathological processes including genetic, developmental, metabolic-toxic, infectious, traumatic and neoplastic-infiltrative processes [57]. Maternal infection and chorioamnionitis are associated with an increased risk of subsequent cerebral palsy [58, 59], which together with the findings of raised inflammatory cytokines in infants with HIE [60] and in those who subsequently develop cerebral palsy [61], provides evidence that inflammation also plays a significant role in the development of these brain injuries. There are a number of different mechanisms by which hypoxia and subsequent ischaemia can lead to neuronal or glial cell loss and brain damage. Brain ischaemia is associated with a fall in both extracellular and intracellular pH. Whilst mild acidosis is protective, severe aci-

dosis promotes free radical production and cytotoxic oedema. The brain is particularly vulnerable to oxidative stress injury due to its high metabolic rate and the fact that it has minimal antioxidant activity. In addition changes in pH and ionic flux induced by hypoxia lead to depolarisation of the cellular membrane and increased intracellular calcium levels, which activate multiple enyzymatic pathways that release further oxygen free radicals [57].

Despite obstetric and neonatal interventions in developed countries being successful in detecting and reducing intrapartum morbidity, particularly from intrapartum hypoxia, a number of longitudinal studies have shown that despite a decrease in perinatal mortality, the incidence of cerebral palsy has remained relatively unchanged [62, 63]. Such data suggested that the proportion of cases of cerebral palsy which are due to birth asphyxia, and which therefore could potentially be prevented due to improved intrapartum care, is much less than previously thought. Such a view has been confirmed by studies which have examined cases of cerebral palsy and come to the conclusion than intrapartum asphyxia is unlikely to be a cause in any more than 10% of all cases of cerebral palsy [64, 65]. All of this evidence points to events in the antepartum period as be causative of the majority of cases of cerebral palsy, a finding which is most important not only in relation to litigation, but also with respect to the possible causes and prevention of cerebral palsy.

Neuroimaging can be helpful to assess both severity and the timing of causation in encephalopathic infants. Cranial ultrasound can be used to detect abnormalities within the thalami and basal ganglia, as well as in the periventricular parenchymal or intraventricular regions. Cystic lesions have been shown to develop over 14 days [66] and so their detection within days after birth suggests an antenatal timing of causation. However, magnetic resonance imaging (MRI) is the most sensitive technique for imaging the neonatal brain. The gestational age at birth is predictive of the pattern of hypoxic-ischaemic injury with hypoperfusion resulting in a periventricular border zone of white matter injury in the premature, but injury to the subcortical white matter and parasagittal cortex in term neonates [67]. It has been determined that diffusion based-MRI values are usually normal on the first day after injury, decrease between 2 and 4-days after injury and return to normal after approximately 7 days [66]. Such knowledge can be useful in timing the causation of an encephalopathy. Together with electroencephalography, MRI can also be useful to predict prognosis in neonatal encephalopathy, with the presence of cortical and basal ganglia abnormalities being predictive of a poor outcome [68, 69]. Traditional care for neonatal encephalopathy has been supportive, but recent trials have shown benefits from the use of selective brain cooling to improve outcomes and it is likely that there is a short therapeutic window of up to 6 h during which interventions such as this may be effective, so early identification of the neonate with such brain injuries may become increasingly important in the future [70, 71].

Conclusion

It is clear that there are a wide variety of different injuries that can be sustained during the birth process. The majority of these will be minor and of no long-term consequence to the infant or their family, but there some of these injuries will result in long-term impairment or disability. Although some infants can be highlighted to be at increased risk of specific injuries due to factors relating to the mother, the fetus or the mechanism of delivery, it is clear that the majority of birth trauma is difficult to predict and thus avoid, despite skilled obstetric and neonatal care. Although caesarean delivery is often portrayed as the means by which all birth trauma could be avoided, it is important that medical staff remind patients that such a means of delivery is not free from risk and indeed can be associated with significant fetal trauma, as well as maternal morbidity.

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