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Birth Injuries Resulting in Neurologic Insult

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ABSTRACT

Neurological insult caused by nerve injury due to birth trauma is, unfortunately, a relatively common event. The two most common injuries are facial nerve injury and brachial plexus injury (BPI). This article will discuss the following aspects of birth trauma-related neurological/nerve injury: incidence, epidemiology, pathophysiology, assessment, treatment, and outcomes. Implications for practice are also described.

Neurological insult caused by nerve injury due to birth trauma is, unfortunately, a relatively common event. The two most common injuries are facial nerve injury and brachial plexus injury (BPI). This article will discuss the following aspects of birth trauma-related neurological/nerve injury: incidence, epidemiology, pathophysiology, assessment, treatment, and outcomes.

Facial Nerve Injury

Facial nerve injury can cause paralysis (or palsy) resulting in weakness of the facial muscles, which can impact feeding/nursing in newborns, and facial expression, speech, and social interaction in children or adults.

Incidence

Facial nerve palsy is reported to occur at rate of 0.8 to 7.5 cases per 1000 births, with 78–90% associated with birth trauma. Although facial nerve injury is associated with the use of forceps (8.8 cases per 1000 births), a third of cases occur in spontaneous vaginal delivery without instrumentation.[1,2] Other risk factors may include prolonged labor, and/or a birth weight greater than 3500 grams.[3]

Epidemiology

Facial nerve palsy was described over two centuries ago. Sir Charles Bell is credited with discovering that the facial nerve is responsible for facial muscle movement in 1821, and idiopathic facial palsy is named in his honor.[3] Forceps trauma as an etiology for facial nerve paralysis was recognized and studied by Landouzy in 1839. It occurs equally in males and females.[1]

Pathophysiology

The facial nerve is the seventh cranial nerve and has both motor and sensory components; it innervates the facial muscles and stapedius muscle in the middle ear, as well as portions of taste sensation, and control of the salivary and lacrimal (tear) glands. It is particularly at risk of trauma because of its extra-cranial exposure, exiting the skull via the stylomastoid foramen, and then running between the mastoid and styloid processes.

Facial nerve birth trauma may be due to pressure on the stylomastoid foramen, or by compression of the bone that lies over the facial canal by the maternal sacral promontory and/or ischial spines, or the posterior blade of the forceps.[1,3] The pressure induces swelling, which interferes with nerve conduction, causing denervation and paralysis. Since the facial nerve is a peripheral nerve, the injured side will show the effect of the trauma.

The differential diagnosis of facial nerve paralysis includes congenital absence of the facial muscles, neonatal asymmetric crying facies (a syndrome of abnormal facial muscle and/or nerve development), Mobius syndrome (a complex congenital anomaly involving multiple cranial nerves and often associated with limb anomalies), Poland's syndrome (a rare congenital anomaly characterized by unilateral chest wall hypoplasia and hand abnormalities), and Goldenhar syndrome also known as oculo-auriculo-vertebral (OAV) (a rare congenital defect characterized by incomplete development of the ear, nose, soft palate, lip, and mandible). Bilateral facial palsy may resemble the facies associated with congenital myotonic dystrophy.[4]

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Assessment

Physical examination may reveal bruising from the forceps. Serial examinations of the face while the newborn is crying will reveal facial asymmetry on the affected side, which manifests as sagging of the eyebrow, inability to close the eye, lack of the usual nasolabial fold, and pulling of the mouth away from the affected side, hampering mouth closure and effective nursing latch.

Imaging is generally not needed unless birth trauma, such as skull fracture, is also suspected. The House–Brackmann facial nerve grading scale uses a range of I–VI to rate the findings of the physical examination as normal, mild, moderate to severe (total paralysis).[4]

Treatment

Facial nerve palsy from birth trauma almost always completely resolves without intervention. Because the newborn may be unable to close the affected side's eye, eye care to prevent corneal abrasion is important. This can be accomplished with lubricating ophthalmic drops. Feeding issues due to latch/suck may be another complication of facial paralysis. Lactation support may be needed for breast pumping and feeding support until muscle movement returns.

Outcomes

Fortunately, greater than ninety percent of traumatic birth injuries to the facial nerve resolve spontaneously without long-term sequelae. The time to recovery ranges from 3 days to 4 months, with a mean recovery period of 24 days, and median recovery time of 17.5 days.[1,5]

Brachial Plexus Injury

Brachial plexus injury (BPI), also referred to as birth brachial plexus injury (BBPI), or obstetric brachial plexus injury (OBPI), can result in neonatal brachial plexus palsy (NBPP), also called brachial plexus birth palsy (BPBP) or birth brachial plexus palsy (BBPP). These terms refer to traumatic injury of the bundle of nerves (the brachial plexus at C5-T1) that innervate the muscles of the shoulder, arm, elbow, wrist, hand, fingers, and diaphragm (via the phrenic nerve). There is a range to the extent of the trauma, and therefore to the injury, treatment, and outcome.

At the time of diagnosis it may not be possible to assess and predict the severity of the brachial plexus injury or the outcome or long-term morbidity of the palsy. Recovery from this injury, which can cause loss of sensation, and/or paralysis with passive range of motion greater than active, ranges from spontaneous complete healing, to partial improvement, to permanent residual sequelae such as paralysis, disability and limb deformity.

In a report by Thatte and Mehta (2011),[6] BPI's occurrence is described as right-sided sixty percent of the time, left-sided thirty-seven percent of the time, and bilateral in three percent of cases.

Brachial plexus injury may be concomitant with other injuries such as fractures of the clavicle and/or humerus, low cord pH (<7.2), hypoxic ischemic encephalopathy, and need for NICU admission.

Incidence

The incidence of BPI increases in direct proportion to gestational age and birth weight. Other factors that may influence the underlying rate of injury include parity, mode of delivery, length of the second stage of labor, use of oxytocin, instrumented/operative vaginal delivery, and maternal diabetes. Even with these known variables, BPI/NBPP is not always predictable. Rates of injury range from 0.3–3.8 per 1000 live births, and are approximately distributed by birth types as follows: 0.3 of 1000 cesarean deliveries, 1 of 1000 vaginal births, and 4 of 1000 operative/instrumented vaginal births.[7] In a hallmark study by Langer, Berkus, Huff, and Samueloff (1991)[8] it was reported that the incidence of shoulder dystocia was 15% to 42% in infants of diabetic mothers with birth weight more than 4000 and 4500 grams. Related to shoulder dystocia, the rate of BPI has been reported to range from 4–40% to 26–64% of cases.[9,10] Permanent NBPP occurs in approximately 1 in 10,000 births (as many as 20 to 30% may have residual neurological sequelae), and is a reason for litigation in approximately 1 in 45,000 births.[11,12]

Epidemiology

The first reported description of brachial plexus palsy following a protracted labor and difficult delivery of the newborn was in 1768, and is attributed to a British obstetrician named William Smellie. Since then, Iffy, Varadi, and Papp (2015)[13] have described that the rates of shoulder dystocia in eleven countries on four continents have remained stable, similar to those in the United States during the middle of the last century. However, in recent times, the U.S. shoulder dystocia occurrence rate has risen to a level four times more frequent than elsewhere in the world, and continues to rise steadily. This is in spite of improvements in obstetrical care in terms of management of postmaturity, diabetes, protracted labor, difficult instrumented extractions, and use of high dose pitocin in labor. The authors go on to comment on the association of rapid timing of delivery (less than two minutes) in the majority of cases of shoulder dystocia with birth injury or death.

The results of a retrospective case–control study of 144 cases of brachial plexus injury suggested that very few factors contributing to brachial plexus injury are modifiable, making it an unpredictable and probably unavoidable event.[14] Why is it that the rates of brachial plexus injury have not improved over time, even with the increase in cesarean delivery, and despite shoulder dystocia drills and training with birth simulators?

The reason for this lack of decline in the incidence of this form of birth injury is not completely understood. While the rates of shoulder dystocia in the United States have risen over time, and many cases of BPI occur in conjunction with shoulder dystocia, the majority of shoulder dystocia events have no brachial plexus sequelae.[10,13] This suggests that other antecedents account for the remaining cases. Historically, brachial plexus injury was thought to result from intervention of a birth attendant — the use of excessive downward traction following delivery of the head, causing stretch injury to the nerve bundle, but this does not account for all cases. It has been postulated that the pathogenesis of BPI might include intrauterine maladaptation such as the lack of the expected rotation of the shoulders into an oblique position, or the posterior shoulder impacting against the sacral promontory in an early stage of labor for an unknown reason.[15,16]

Pathophysiology

The brachial plexus is like a tree with branches — a network of nerve fibers consisting of nerve roots that connect to the spinal cord (tree trunk) and the peripheral nerves (tree branches) that innervate the shoulder on down the arm to the hand. Brachial plexus injury can be caused by either upward or downward traction on the shoulder girdle. Some of the nerve roots are more susceptible to trauma from traction than others, and may avulse, or tear from the spinal cord. Damage may be caused by stretch or compression of the nerve fibers resulting in lack of nerve impulse conduction (neurapraxia), or the trauma may cause rupture of the axon and myelin sheath with the connective tissue preserved (axonotmesis), or in the most severe cases, tearing or avulsion of the nerves from the nerve root (neurotmesis) may occur.[6,17] There are several types of palsies — partial to complete muscle paralysis that can occur with nerve injury. These range from lack of hand movement, to lack of movement of the entire arm and shoulder, depending on which nerves are injured. Injury to the nerves at C8 to T1 (lower nerve trunk) can cause paralysis of only the hand, and is known as Dejerine-Klumpke or Klumpke's palsy. If the injury involves C5 to C7 (middle trunk) this may result in loss of wrist extension, with the