NEONATAL NERVE PALSIES:
A CONTEMPORARY OBSTETRIC PERSPECTIVE

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Background:
Birth trauma and its often incorrect inference of iatrogenic causation has led to unfortunate implications for the affected child, the parents, the obstetrician and the midwife due to unwarranted medico-legal attention in our current litigious society.
A more discerning evaluation of neonatal nerve palsies following labour and delivery has led to a better understanding of their aetiology with potentially more appropriate outcomes for all parties involved.
Keywords: birth trauma, iatrogenic, medico-legal, aetiology

INTRODUCTION

The birth process is a blend of compression, contractions, torques and traction. These forces may have deleterious effects if there is associated fetal malpresentation, immaturity or congenital anomalies. Despite historical observational argument that clinician applied traction is the primary mechanism of congenital neuropathy associated with vaginal delivery, there is increasing contemporary evidence that alternative mechanisms such as in-utero mal-adaptation, endogenous compression from the bony pelvis and maternal expulsive efforts may be more significant in the aetiology of obstetric nerve palsies.[1,2]

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Brachial Plexus Palsy (BPP) is the commonest congenital nerve palsy and is the subject of great controversy with regard to aetiology, prediction, prevention and clinical management.[3]

The incidence of BPP is difficult to obtain as there are significant ascertainment and observational biases with a tendency to under reporting particularly as the majority of cases are either transient or temporary with just 10% of reported cases resulting in permanent BPP (approximately 1 in 15-20,000 births).[4,5] Sadly despite our accumulating knowledge of endogenous factors in the aetiology of BPP and its inconstant relationship to physician applied traction along with its poor prospective predictive characteristics and the inadequate preventive measures available, neonatal BPP is still the single most common cause for litigation related to shoulder dystocia.[6,7]

As a consequence BPP receives disproportionate attention by risk managers but it must be remembered that almost any peripheral nerve can be affected similarly and is just as deserving of our discerning examination and hence this review of peripheral nerve palsies, cranial nerve palsies and cervical cord injury of the neonate.

GENERAL COMMENTS

The incidence of peripheral nerve palsies in neonates is unknown with the body of knowledge being restricted to case reports and small cohort studies.

Within these case series the aetiology and mechanisms of the neurological defect whilst varied are often grouped together as 'birth trauma' resulting in misleading conclusions with regard to diagnosis, treatment, prognosis and prevention. The mechanism of the neurological deficit will vary depending on the location and the course of the peripheral nerve.

Lower limb palsies are extremely rare in cephalic vaginal deliveries whilst "traumatic/birth injury" causes are more commonly associated with both footling and frank breech deliveries which in turn may be related to the position in labour rather than the manoeuvres to effect delivery.

Fetal size and resilience are additional risk factors for peripheral nerve injury with preterm and low birth weight babies being at higher risk not only for nerve injury but also for musculoskeletal injury.

This contrasts with BPP where infants with macrosomia (relative or actual) may be at higher risk of neurological injury.[8,9]

Amniotic bands, compression bands, severe prolonged oligohydramnios and structural uterine abnormalities have all been associated with peripheral nerve palsy.[10,11]

The mechanism of injury relates to external nerve compression usually at the level where the affected nerve courses superficially in close proximity to the bone.

Distal limb hypoplasia can occasionally co-exist.[12]

External compression can affect upper limb nerves resulting in diagnostic confusion with BPP. As with BPP similar compression forces may also occur in utero during labour with fetal parts compressed against the maternal bony pelvis by the previously described significant endogenous uterine forces resulting in peripheral neuro-praxia or nerve palsy.

Similarly associated congenital anomalies of the fetus of a genetic or syndromal origin may exert compressive or structural mass effects resulting in peripheral nerve palsy.

Although trauma and compression account for the larger proportion of peripheral nerve palsies in neonates, other significant factors include viral infection,[13] ischaemia and demyelinating disease which can all occur in-utero with nerve palsy being present at birth.[14]

A clear cause for neonatal peripheral nerve palsy and other palsies is often never identified.

INDIVIDUAL PERIPHERAL NERVE PALSIES

Sciatic nerve

The sciatic nerve receives branches from the L4-S3 nerve roots and exits the pelvis through the greater sciatic foramen.

Compression injury typically occurs at this level while traction injury can occur anywhere along the entire course of the sciatic nerve.

Palsy can result in a variety of lower limb problems ranging from posterior thigh paralysis to anterior tibial weakness and foot drop depending on the site of the injury.

Neonatal sciatic nerve palsy may occur after prolonged hip flexion associated with breech presentation[15] or following traction associated with breech extraction.[16]

A review of neonatal sciatic nerve palsy by Ramos-Fernandez et al (1998)[17] found that the majority of cases had no apparent cause other than a small number of babies following intra-gluteal intramuscular injection post delivery

Peroneal nerve

The peroneal nerve is derived from L4-S2 nerve roots as part of the sciatic nerve as it divides to form the tibial and common peroneal nerves at the level of the popliteal fossa. The common peroneal nerve then courses around the head of the fibula which subjects it to the risk of compressive or traumatic injury.

Peroneal nerve palsy results in foot drop and diminished sensation on the dorsum of the foot. Congenital peroneal nerve has also been reported in association with breech presentation and delivery, compression bands and concomitant talipes equino-varus.[18,19]

Recovery of peroneal nerve palsy post delivery is variable and dependent on the length of time of nerve compression which is unpredictable.

Phrenic nerve

Phrenic nerve palsy occurs in approximately 1:10,000 live births.[20]

The phrenic nerve arises from C3-C5 nerve roots and innervates the ipsi-lateral half of the diaphragm.

Loss of function results in eventration of the diaphragm characterised by elevation of the hemi-diaphragm.

More than 85% of cases are right sided.[21]

Bilateral phrenic nerve palsy has been reported and is usually a result of demyelination or as part of a genetic/syndromal condition.

Congenital phrenic nerve palsy is frequently (70-80%) associated with BPP which might imply a traction related injury if conventional thought is applied[22] but as with contemporary thought now applied to BPP, an antenatal or intra-partum aetiology is more likely as congenital phrenic nerve palsy has been reported after uncomplicated caesarean section.[23]

Initial management consists of ventilatory support awaiting spontaneous resolution usually within a few days.

Surgical plication of the diaphragm may be necessary for those neonates who fail to improve spontaneously with a good outcome expected in 86% of cases with unilateral affectation.[22]

Bilateral affectation has a worse prognosis with a 50% neonatal mortality rate.[21]

A review by Stamrood et al[22] examined 14 cases of neonatal phrenic nerve palsy and found that 8 infants were vaginal breech deliveries, 5 infants were vaginal cephalic deliveries and 1 infant was delivered by caesarean section. Of the 5 infants having cephalic vaginal births only 1 was associated with shoulder dystocia whilst 3 babies were macrosomic.

As with BPP, the authors concluded that intra-uterine mechanical factors were responsible for the phrenic nerve palsy identified post delivery.

Radial, Ulnar and Median Nerve

Distal upper limb nerve palsies have been reported but are extremely rare. Nerve conduction studies serve to differentiate isolated upper limb radial, ulnar and median nerve palsies from those associated with BPP.

Isolated radial nerve palsy (with good shoulder function and intact flexion of the elbow) has been identified following trauma, intra-uterine compression, fat necrosis in the radial groove (seen in 68% cases), fractures and occasionally after sphygmo-manometry.[24,25]

A retrospective study by Alsubhi et al[6] of 25 neonates with isolated radial nerve palsy demonstrated that 18 infants (75%) had a complete recovery within a range of one week to six months after birth.

Congenital hypoplasia of the distal ulnar nerve resulting in nerve palsy has also been reported.[27]

CRANIAL NERVE PALSIES

Any discussion of obstetric neonatal nerve palsies would not be complete without a review of the 12 cranial nerves all of which can have aberrant function at birth.

The most common prolonged congenital cranial nerve palsies occur with the oculomotor (CN III), trochlear (CN IV) and abducens (CN VI) nerves controlling eye movement.

Neonates/infants may present with ocular muscle paresis, strabismus, pupillary dilatation and reduced visual acuity.

A review by Ng et al (2005)[13] found that 33% of cases were of congenital origin, 28% were traumatic origin, 22% were of neoplastic origin, 11% were of vascular origin and 6% were of infectious aetiology.

The recurrent laryngeal nerve (a branch of the vagus nerve – CN XI) can be affected at birth and is thought to result from fetal positioning in-utero where the head is rotated and flexed laterally.

This same mechanism may occur during labour and delivery resulting in unilateral congenital recurrent laryngeal nerve palsy.[28]

Clinically the neonate may present with a hoarse cry, respiratory stridor or swallowing difficulties. Occasionally bilateral recurrent laryngeal nerve palsy may occur which is usually secondary to hypoxia or brain stem haemorrhage resulting in severe respiratory distress and/or asphyxia.

Spontaneous resolution of unilateral recurrent laryngeal palsy may take 6 – 12 months with supportive gavage feeding and interim tracheostomy often being necessary.

Facial nerve (CNVII) palsy is the most common cranial nerve palsy at birth but most are transient and usually resolve within a few days after delivery.

The incidence is of the order of 2.1 per 1,000 live births.

The extra-temporal course of the facial nerve places it at risk of compression by the sacral promontory and the ischial spines during labour and at delivery from obstetric forceps.[29, 30]

Facial nerve palsy can have a variable clinical presentation depending on the level of the lesion. Upper facial nerve palsy may result in the loss of voluntary eyelid closure and facial droop while lower facial nerve palsy will resulting mouth and lip droop with unilateral loss of facial expression. The differential diagnosis includes nuclear agenesis (Mobius syndrome), congenital absence of facial muscles, Poland's syndrome, Goldenhaar syndrome, Craniofacial syndrome and intra-cranial haemorrhage.[31]

Laing et al (1996)[32] performed a retrospective case control study of 61 children with established facial nerve palsy and measured the odds ratio of recognised risk factors for birth injury in each pregnancy of maternal primiparity, increased birth weight and forceps delivery. A number of 13.2% of the women were delivered by forceps compared to 10.2% in the general unaffected obstetric population (OR 1.34; 95% CI 0.61-2.07), 39.6% were born to primiparous women compared to a national rate of 36.7% (OR 1.13; 95% CI 0.65-1.96) and only 18.9% of babies weighed more than 3.5 Kg at birth (OR 0.37; 95% CI 0.19-0.74). They concluded that there was no association between the development of permanent congenital facial nerve palsy and recognised risk factors for birth injury suggesting an intra-uterine rather than a traumatic aetiology for established facial nerve palsy.

Traumatic facial nerve palsy in neonates is associated with a good prognosis while non-traumatic facial nerve palsies often result in a poor functional outcome.

Palsies of other cranial nerves are extremely rare and usually do not have a direct obstetric relationship.

Case reports of diminished neurological function involving the olfactory, optic, hypoglossal, trigeminal and vestibular cochlear nerves exist but more commonly are seen in association with other congenital syndromes.[33]

CERVICAL SPINAL CORD INJURY

Any contemporary review of neonatal nerve palsy should include the rare occurrence of cervical spinal cord injury at birth.

Injury may result from excessive traction or rotation forces on the cervical spine during labour and delivery.

Although both are rare, traction injury is more common during labour and delivery of breech presentation where the lower cervical and upper thoracic spine is more likely to be affected while torsion injury is more common in vertex presentation, labour and delivery where the upper and mid cervical spine is more likely to be affected. However cervical cord involvement can be associated with hyperextension of the fetal neck (so called "flying" or "star gazing" fetus) where even elective caesarean section may not offer protection against cervical cord injury.[34]

Additionally cervical spinal cord anomalies may be present including glio-neuronal heterotopias, which will either present or predispose to cervical cord injury in the neonate.[35]

Ever since the signature report by Pridmore et al (1974)[36]: "Spinal Cord Injury Of The Fetus During Delivery With Kielland's Forceps" the continued use of rotational forceps in obstetric practice has been questioned. However detailed examination of this case and of a number of cohort studies including that of Menticoglou et al (1995)[37] has identified a number of confounding factors which may have played a role in cervical cord injury and have hitherto been ignored. These factors include prolonged labour, prolonged rupture of the membranes with chorio-amnionitis and inappropriate pharmacological augmentation of labour.

Similar observations and confounding factors have also been found in neonates undergoing vacuum delivery resulting in cervical spinal cord injury.

Neonates with cervical cord injury may present with spinal shock with flaccidity below the level of the injury initially, with spasticity developing within days or sometimes weeks after birth.

There may be delay in the diagnosis of less severe injuries with consequent delay in the institution of appropriate management.

The use of MRI may help to delineate the cervical cord injury in addition to excluding any pre-existing pathology and surgically treatable lesions including congenital tumours or haematomas which may be exerting pressure on the cervical spinal cord.[38,39]

CONCLUSIONS

A more discerning contemporary appraisal of the clinical evidence surrounding neonatal obstetric nerve palsies has led to the more sensible conclusion that they are not the invariable consequence of clinician applied traction but are very likely a combination of endogenous and exogenous factors which in the main cannot be predicted ante-natally or intra-partum thus making prevention unlikely.

Armed with this knowledge the obstetrician and the paediatrician can provide clarification of the sequence of events of neonatal nerve palsy to the parents and later to the affected individual, all of whom may have to live through or with a permanent disability.

This insight should modify unnecessary or inappropriate medico-legal activity afforded to neonatal nerve palsies which in turn will allow all parties to concentrate more appropriately on the management of any long term disability and to direct their energies and finance into ensuring that the affected individuals receive the required sympathetic and continued management that tragically appears to be under resourced in most communities.

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