Transection of Spinal Cord

A Rare Obstetrical Complication of Cephalic Delivery

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Shulman, S. T., Madden, J. D., Shanklin, D. R., and Esterly, J. R. (1971). *Archives of Disease in Childhood*, 46, 291. **Transection of the spinal cord: a rare obstetrical complication of cephalic delivery.** A newborn infant, delivered following mid-forceps rotation, presented with apnoea, anaesthesia below the level of the mid-neck, and flaccid quadriplegia. At necropsy there was transection of the cord, and atlanto-occipital and atlantoaxial dislocations. Cord injury usually follows breech presentation, the lesion is in the lower cervical or upper thoracic segments, and results from excessive traction. By contrast, in the rare cases following cephalic delivery, the lesion is most often in the upper cervical cord and probably results from rotational forces.

A century ago, Parrot (1870) reported the clinical and pathological findings in a 3-day-old baby with cervical spinal cord damage evidenced by intact spinal reflexes and quadriplegia. Necropsy findings included meningeal lacerations and C_{6,7} cord transection. He related the lesions to the difficult breech delivery, during which '*un craquement très-fort*' had been heard from within the birth canal. Spinal cord injury had been noted previously in the nineteenth century. Kennedy (1836), Billard (1839), Weber (1851), and Little (1862) referred to spinal cord pathology, but only Billard (1839) considered such lesions a direct result of labour.

Newborn babies with spinal cord injuries surviving the neonatal period were first documented in the early 1900's (Handwerck, 1901; Beevor, 1902; Gött, 1909; Lawatschek, 1911; Burr, 1920). Survival into childhood was first reported in the 1920's (Kooy, 1920; Kohlbry, 1923; Valentin, 1924).

The classic articles on intrapartum spinal cord injury appeared between 1921 and 1927 (Crothers, 1922 and 1923a, b; Ford, 1925; Crothers and Putnam, 1927; Ford, Crothers and Putnam, 1927), revealing that the vast majority of cases followed

traumatic breech delivery. Crothers and Putnam (1927) reported 7 cases following cephalic delivery and 7 more from the literature (Jolly, 1895; Couvelaire, 1903; Stoltzenberg, 1911; Belfrage, 1923), and cited necropsy findings of spinal cord haemorrhage in 6 of 16 cephalic stillborn infants (Spencer, 1891). There have been only 3 subsequent reports of spinal cord injury with cephalic delivery, including but a single article in the English literature (Table I) (Föderl, 1931; Herzog, 1952; Towbin, 1964).

Case Report

A mature, 2500 g infant male was born to a gravida II, para I, 35-year-old black woman in the 39th week of gestation. The onset of labour was spontaneous but there was a history of rupture of the membranes two days before admission. Dilatation and effacement were uneventful. 50 mg pethidine was given two hours before delivery. Presentation was cephalic, in the occiput posterior position, and a mid-forceps rotation was performed under methoxyflurane anaesthesia. The second stage of labour was uneventful.

The Apgar score was 3 at one minute and 4 at five minutes. The heart rate was 120, but the infant was flaccid and failed to breathe spontaneously. With positive pressure oxygen, he became pink and maintained a normal cardiac rate, but made no respiratory effort. Over the next two hours there was no change in his condition despite intravenous nalorphine, levallorphan, and caffeine.

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TABLE

Spinal Cord Injury Associated With Cephalic Delivery

Author	Year	Obstetric Factors	Clinical and Pathological Findings	Survival
Spencer	1892	Necropsy study of 16 cephalic stillbirths	Six with spinal cord haemorrhage	Stillborn
Jolly	1895	Face presentation rotated to vertex	Symmetrical arm paralysis felt to be secondary to cord injury	Alive at 'a few months'
Couvelaire	1903	Four cephalic cases with dystocia	Haemorrhage into medulla and upper cervical spinal cord in each	Neonatal deaths
Stoltzenberg	1911	One of 75 babies dying of 'asphyxia'	'Ruptured spinal column'	Neonatal death
Belfrage	1923	Spontaneous occiput posterior delivery, second of twins	Sensory and motor findings below T-2	Alive at 4½ yr
Crothers and Putnam	1927	Case 11: arrested partial delivery	Triplegia; probable avulsion of right brachial plexus with diffuse cord injury	Alive at 3 mth
		Case 15: difficult forceps delivery	Physiological transection in lower thoracic region	Alive at 3 yr
		Case 18: 'easy', spontaneous delivery	'Diffuse cord injury'	Alive at 8 yr
		Case 20: difficult forceps delivery	Right brachial plexus injury with diffuse cervical and upper thoracic injury	Alive at 1 yr
		Case 22: difficult forceps delivery	Right brachial palsy with diffuse cord damage	Alive at 18 mth
		Case 26: face presentation rotated to occiput posterior by high forceps	Mild 'diffuse cervical cord injury'; spastic legs	Alive at 10 mth
Föderl	1931	One of 21 infants with birth injuries	Probable avulsion of brachial plexi with haemorrhage into lower cervical cord	Alive at 2½ yr
Herzog	1952	Spontaneous delivery	High cervical luxation or subluxation with cord injury documented at necropsy	Died on 3rd dy
Towbin	1964	Two spontaneous deliveries	Scoliosis and spastic paraplegia; physiological lesion at T-10 level	Alive at 9 yr
Present case	1971	Occiput posterior with midforceps rotation	Haemorrhage into cord with epidural and subdural haemorrhage and avulsion of C-7 and C-8 spinal roots	Death in first 2 dy of life
			Transection at C ₁₋₂ with fracture dislocations of C ₁₋₂	Died at 12 hr

By the third hour, he was intubated and placed on a respirator. Though his extremities were flaccid, he was alert, moved his head and eyes, and had a suck reflex. A systolic heart murmur was heard. Priapism and withdrawal responses to painful stimuli in all limbs were noted at 6 hours of age, but he was otherwise unchanged and he did not move his extremities spontaneously. Absent sweating and no evidence of sensation (other than withdrawal responses to deep pain) were noted below the level of mid-neck. Priapism was persistent. X-rays of the cervical spine showed probable atlantoaxial and atlanto-occipital dislocations without obvious fractures.

Because of the radiological evidence of vertebral dislocations and clinical evidence of cervical spinal cord injury, the infant was placed in traction with chin straps. Within 15 minutes, however, he developed bradycardia which was irreversible despite removal from traction. He died shortly thereafter, 12 hours after birth.

At necropsy a cephalohaematoma was present over the right parietal region. The brain stem was surrounded by blood clots. The medulla and occipital lobes were also blood-stained, and the spinal fluid was grossly bloody. Discontinuity of the spinal cord and haemorrhage in the epidural and subarachnoid spaces were found at the level of C₁, 2. The atlas and axis were freely movable, and on subsequent dissection, a fracture of the odontoid process of the axis was demonstrated.

Histological sections of the proximal and distal

portions of the cord showed extravasated blood and a leucocytic infiltration between the nerve fibres. Necrosis and neuronal chromatolysis were found here and in the sections from the brain stem. Other findings included an acute chorioamnionitis, aspirated squames, and petechial haemorrhages and foci of polymorphonuclear cells in the lung. The degree of glycogen depletion and reduced haematopoiesis in the liver was compatible with mild intrapartum distress.

Discussion

Incidence. Severe spinal cord injury following cephalic delivery is decidedly uncommon. Mild injury appears to be frequent, though most neonatal necropsies do not routinely include an examination of the spinal cord. In a series of 600 fetal and neonatal necropsies with brain and cord examination, Towbin (1970) found evidence of significant spinal or brain stem injury in over 10%. The lesions include spinal epidural haemorrhage, meningeal laceration, and trauma to the nerve roots, arteries, ligaments, vertebral bodies, and even to the paraspinal musculature (Towbin, 1970; Schwartz, 1961).

Mechanism of injury. The spinal cord is firmly anchored by the cauda equina and the

brachial plexi but is only loosely attached to the dura in the thoracolumbar region. The neonatal vertebral column is poorly ossified and less rigid than in the older infant (Stern and Rand, 1959), and the cord is relatively less elastic than its encasement, documented by direct observations on decapitation of mature stillborn infants by linear tension (Duncan, 1874).

In breech presentations it is clear that the mechanism of injury is related to *stretch injury*. These injuries can occur in any spinal cord region (Zellweger, 1945), though lower cervical and/or upper thoracic lesions are most common (Ford, 1925; Schwartz, 1961; Stern and Rand, 1959; Leventhal, 1960). Because of the frequency of vertebral displacement, however, Potter (1961) concluded that crush injury might also play a role in cord damage after breech delivery.

The mechanism of injury during cephalic delivery is less obvious. Difficult or prolonged labour is often noted, and occasionally (as in the present patient) there is a history of rotational manipulation; in these infants it is clear that injury resulted from excessive *torsion* rather than traction. With forceps extraction, stretch forces may also play a role. The site of cord injury in cephalic presentation is nearly always cephalad to the brachial attachments, most compatible with the hypothesis of torsion injury. This is in contrast to the more caudad lesions due to traction injury.

Fetal malposition, such as face or brow presentation or *in utero* opisthotonos or 'flying fetus', may render the fetus vulnerable to unphysiological force (Knowlton, 1938; Melody, 1948; Taylor, 1948). Likewise, congenital vertebral anomalies may predispose to injury (Föderl, 1931; Zellweger, 1945). Indeed, x-rays in infants with suspected cord injury may be of more value in ruling out congenital spinal anomalies than in diagnosing vertebral fracture and/or dislocation because of the sparsely ossified spinal column and the difficulty in positioning ill infants (Crothers, 1959; Stern and Rand, 1959; Ford, 1966).

Clinical features. Most liveborn infants with intrapartum cord injuries die in the early neonatal period from respiratory problems or associated brainstem injury. As in the present case, apnoea may be the dominant symptom in patients with lesions cephalad to the phrenic nuclei (C_3 – C_5) (Crothers and Putnam, 1927; Zellweger, 1945). Because of the more caudad lesions produced, apnoea is rarely present after breech cord injury.

Several clinical syndromes appear in surviving infants (Crothers, 1959): (a) unilateral or bilateral

brachial palsies and/or cranial nerve deficits, (b) permanent flaccidity and anaesthesia below the level of physiological (and/or anatomical) cord transection, and (c) initial flaccidity and anaesthesia (spinal shock) but with the subsequent development of lively spinal reflexes (Head and Riddoch, 1917). In addition, there may be less severe lesions with partial physiological cord transection and varying amounts of destruction of ascending or descending tracts. The return of reflex activity after a period of spinal shock depends upon the absence of significant damage in the distal cord segment. In the present patient, spinal shock was not found; it has been suggested that children have an earlier and more active return of reflex activity (Ford, 1925).

The lesions in these cord injuries extend over many vertebral segments (Crothers and Putnam, 1927; Stern and Rand, 1959), and the diagnosis in the neonate requires the presence of non-selective, non-progressive neurological signs, most often with a history of a difficult, usually breech, delivery (Crothers and Putnam, 1927; Leventhal, 1960).

Though priapism may be present in any patient with spinal cord lesion (most commonly in those with cervical lesions or with transection, Austin, 1961), this sign has not been previously reported in cord injury in the newborn. The mechanism of priapism in these injuries may be related to sympathetic vasopressor interruption, leading to penial engorgement.

Conclusions

Serious and even lethal spinal cord lesions can and do occasionally result from obstetrical injury to the fetus. In breech deliveries, injury to the cord may result from excessive longitudinal traction. With cephalic presentation, spinal cord lesions are considerably less common, and the mechanism is often unclear. The case presented here is probably unique in that cord transection most likely resulted from the shearing forces associated with a forceps rotation of the head of 180 degrees. Since many infants with less severe injuries survive the neonatal period, the prompt recognition of spinal cord lesions is essential.

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