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 C6,7 cord transection. He related the lesions to the difficult breech delivery, during which 'un craquement trés-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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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 cephalo-haematoma 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 leukocytic 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 haematoxylin in the liver was compatible with mild inappronatus 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 paraspinous 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 mecha-
nism of injury is related to stretch injury. These
injuries can occur in any spinal cord region (Zell-
weger, 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
bruise 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 caudal lesions due to traction injury.

Fetal malposition, such as face or brow presenta-
tion 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

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 (C₃-C₅)
(Crothers and Putnam, 1927; Zellweger, 1945).
Because of the more caudal 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 sympa-
thetic 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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