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Neonatal Sciatic Palsy

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References to the occurrence of sciatic palsy in the neonatal period are infrequent and several authors have described the condition as a very rare one. However, Mills in 1949 and Hudson *et al.* in 1950 published relatively large series of cases numbering eight and 20 respectively. The latter series included 11 cases previously collected by Fahrni (1950). Apparently independently, they arrived at the interesting hypothesis that the injection of analeptic drugs into the umbilical cord for purposes of resuscitation had resulted in damage to the sciatic nerve. Later, in 1950, McFarland, in an editorial comment on Fahrni's paper which had been submitted two years previously, again stressed the common factor of umbilical cord injections in all 11 cases. The possible danger of such injections was then widely accepted.

Despite the continued use of umbilical cord injections in many centres, no further papers on this subject appeared until 1960, when Shaw described three cases, including twin boys. Many orthopaedic surgeons and paediatricians of wide experience have never encountered a single case (Seddon, 1960), suggesting that the condition occurs regionally and sporadically. Consequently it is believed that the occurrence of five European cases in three years in an area with a total European population of 50,000 persons merits reporting.

CASE REPORTS

Case 1.—C.A., a full-term infant, was born on 17th October, 1956, and weighed 5 lb. 14 oz. Because of disproportion, delivery was accomplished by mid-forceps under general anaesthetic. At birth the infant was pale and apnoeic. Nikethamide was injected into the

umbilical cord and improvement was then rapid. Soon after birth the right lower limb was noted to be immobile and held flexed at the hip; subsequently there was persistent weakness of the right leg and foot. The infant was referred to us at age 18 months because of weakness of the leg and foot drop. The right calf was wasted and the Achilles tendon tight; the foot was atrophic and showed equinovarus deformity; the right ankle reflex and plantar response were absent; there was loss of sensation over the first sacral dermatome and saddle area. At age 3½ years elongation of right tendon Achilles was performed. Considerable weakness of dorsiflexion of the foot persists and the child has to wear a caliper for walking. There was a tendency to trophic ulceration because of poor sensory recovery in the sole and plantar aspects of the toes.

Case 2.—B.A., a male infant weighing 6 lb. 3 oz., was born at full term on 8th April, 1959, by elective caesarian section. As he was limp and slow to cry, an injection of nikethamide was given into the umbilical cord. On the third day profound weakness of the entire left lower limb was noted for the first time; there was associated loss of sensation in the sciatic distribution. Later an abscess developed in the right buttock in relation to a pilonidal sinus. However, there were no apparent bruising, mottling or vascular changes in the buttock or lower limb. At three weeks the infant could move the left knee, but not the foot, and knee reflexes were equal. At age nine months he could stand on the foot, which was slightly smaller than the right; minimal weakness of dorsiflexion of the foot was the only abnormality noted. He was not seen thereafter.

Case 3.—P. van S., a male infant, was born by lower segment caesarian section, performed for cephalo-pelvic disproportion after labour had been in progress 15 hours. The infant was apnoeic after delivery, but is said to have cried actively after two minutes following an injection of 1.5 ml. nikethamide into the cord. Birth weight was 7 lb. 15 oz. After 24 hours the right foot was found to be limp and immobile. Clinically, the foot and toes on that side were

flaccid and the ankle reflex was absent; both knee jerks were present and equal; complete anaesthesia for pinprick was evidenced from the knee downwards; there was no difference in temperature between the two lower limbs and all the pulses were palpable. At age 10 weeks he was thriving, but there was some loss of bulk in the right calf and flexion contracture of the right big toe; movements of the ankle were present, but rather weak. He was not seen again.

Case 4.—M. McG. was delivered by forceps under general anaesthesia on 27th May, 1959, following artificial rupture of membranes and prolapse of an arm. She was slow to breathe and cry. Nikethamide 1 ml. was injected into the cord and this dose was repeated after five minutes; thereafter the infant improved. On discharge from hospital after one week, the mother noticed that the left foot was smaller than the right and that pressure over the lower part of the back and buttocks seemed to cause pain. Clinically, there was already equinovarus deformity of the foot due to paralysis of evertors and dorsiflexors and "hammer" deformity of the big toe; the left ankle jerk was absent and sensation was impaired below the knee. At 20 months profound weakness persisted and lengthening of the tendon Achilles was performed. Following this, she could walk only with a below-knee caliper and significant disability persists. Sensation is now apparently normal.

Case 5.—B.K., a female infant, was born to a mother with severe diabetes, who had had a normal child by caesarian section two years previously. In this second pregnancy she had required an average of 80 units of insulin daily to control her glycosuria. When eight weeks pregnant she had a brisk haemorrhage and then two smaller bleeds later in pregnancy. Delivery was by caesarian section at 37 weeks. At operation it was noted that liquor was extremely scanty, that the presentation was breech with extended legs and sacrum to the left, and that the placenta was situated normally in the upper segment. A few moments after delivery, when the child was crying normally, a crescentic area of bluish discolouration was noted over the right buttock. This was considered to be a congenital naevus. It was also noted that the infant was not moving the right leg. The discolouration faded slowly over the first week, but paresis of the leg persisted. The child was referred to us at age six months, when the only

abnormalities noted were in the right lower limb. At rest the knee lay in extension and the foot in firm equino-varus; active movements below the knee were restricted to inversion of the foot and flexion of the foot and toes. Hamstring contraction was judged as being less powerful than on the left side. Though no sensory changes had been noted previously, marked hyperalgesia was evident over the sole and dorsum of the foot. Thereafter spontaneous improvement was rapid. At age nine months active eversion and dorsiflexion of the foot occurred both spontaneously and in response to appropriate stimuli, and there were no residual sensory disturbances. The foot was slightly smaller than the normal one.

DISCUSSION

Of the five cases mentioned, the first four infants have in common a history of delivery under general anaesthetic, of varying degrees of asphyxia and of an immediate injection of nikethamide into the umbilical cord. In these essential respects these cases are similar to the two large series mentioned (Mills, 1949, and Hudson *et al.*, 1950) and also to cases reported by Bates and Page (1949) and by Shaw (1960).

It is believed that complications result from inadvertent injection of the drug into an umbilical artery, the usually prominent vein being collapsed and inconspicuous in a shocked and apnoeic infant. The ease with which this error can occur was illustrated in an infant who died recently in an asphyxiated state following delivery. In an attempt at resuscitation nikethamide had been injected into the cord just before death. On dissection of the cord one of the arteries was noted to have been punctured and there was considerable peri-arterial extravasation of blood.

It is postulated that the drug causes vasoconstriction in the umbilical artery into which it is injected and in the proximal vessels, particularly the hypogastric and inferior gluteal arteries. In almost all reported cases the sciatic paralysis has been accompanied by ischaemic changes in the ipsilateral limb and buttock, varying in degree from gangrene of the lower limb, perforation of the colon and ulceration of the buttock to minor swelling and bruising in the gluteal region. The possible local mechanisms of action have been discussed extensively by Hudson *et al.* However, there appear to be many variables which determine the nature and degree of any ensuing damage. These include:

- (1) Precise nature of the drug used.
- (2) The total amount injected.
- (3) The proportion thereof which enters an artery.
- (4) The force of injection and amount of "milking" of the cord used.
- (5) The severity of asphyxia.

In consequence, it is not surprising that the severity of the ischaemic manifestations varies greatly from case to case and from one report to another. A notable feature of the four cases described here has been the complete absence of overt evidence of vascular changes in the buttock or leg of the affected side; superficially the pathology might appear to have been entirely a neurotoxic effect. Another significant factor was that in the four cases the cord injections were given by the same consultant anaesthetist. As in the reports of Mills (1949), Bates and Page (1949) and Shaw (1960), nikethamide was the substance used for resuscitation in the four cases described here. Another synthetic analeptic with a rather similar chemical structure, Cycliton, was incriminated in the 20 cases described by Hudson *et al.* (1950).

In the reported series there has been considerable variation, not only in the initial clinical picture, but also in the residual disability. Two fatal cases have been recorded. Of the four cases mentioned here, two remain with serious disability and two were showing rapid improvement when last examined. It is of interest that of the two cases with residual disability, one (C.A.) has a considerable sensory deficit, while the other (M. McG.) has fully recovered sensation.

The fifth case reported differs from the others in several respects: though delivered by caesarian section, the child cried lustily on delivery; no cord injection was given; she showed bluish discolouration of the buttock immediately after extraction; paralysis was evident only in the lateral popliteal division of the sciatic nerve (calf muscle contraction and sensation in the sole of the foot were never lost). It is believed that this is one of the rare cases where traction damage to the lateral popliteal division of the sciatic nerve occurs in a breech presentation with extended legs. The mechanism has been described by Craig and Clark (1958). As is the usual event in such traction lesions, recovery was almost complete at age one year. It is assumed that the buttock discolouration was the result of additional local pressure *in utero*.

Mention should be made of a third cause of sciatic paralysis encountered in the neonatal period—that due to injections given directly into or near to the sciatic nerve in the buttock. The vulnerability of the nerve in infants and consequent danger of buttock injections are so well known as to require no elaboration. Nevertheless, in the same three-year period we have encountered three such cases in older infants

CONCLUSIONS

Five cases of neonatal sciatic palsy are described. In four cases this followed injection of nikethamide into the cord for neonatal asphyxia. The fifth case is presented as an example of the rare traction injury to the lateral popliteal division occurring in breech presentations.

The precise mechanism whereby cord injections of certain analeptic substances produce these unfortunate results is not yet fully elucidated.

As the sciatic paralysis caused by inadvertent injection into an umbilical artery not infrequently produces severe permanent disability, and as two fatal cases are recorded in the literature, the method must be condemned. It is of interest to report that since wide publicity has been given locally to the hazards, no further cases of sciatic paralysis in the newborn have been encountered.

Occasionally, however, urgency provides the necessity for such injections. In this event it has been suggested recently by Holmes (1961) that the cord be compressed proximally to make the vein more prominent and thereby avoid any possibility of intra-arterial injection.

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