South African Medical Journal
Suid-Afrikaanse Tydskrif vir Geneeskunde
P.O. Box 643, Cape Town Posbus 643, Kaapstad

Cape Town, 11 June 1955 Vol. 29 No. 24 Kaapstad, 11 Junie 1955
Weekly 2s. 6d. Weekliks 2s. 6d.

SCIATIC NERVE PALSY IN NEWBORN INFANTS

REPORT OF A CASE

A. PENN, M.B., CH.B. (RAND)
Surgical Registrar
and
W. T. ROSS, F.R.C.S. (Eng.)
Orthopaedic Surgeon, Germiston Hospital

Baby B. was born in the Germiston Hospital on 15 June 1954. His mother, a multipara aged 33 years, had been admitted on 9 June with severe pre-eclamptic toxaemia in the 34th week of her pregnancy. After labour had been induced surgically, a placenta praevia was detected and a lower-segment Caeserean section performed. The baby was easily delivered, without traction on the legs, but showed signs of asphyxia, and was resuscitated with oxygen and an injection of 1 c.c. of nikethamide (coramine) into the umbilical cord. His weight was 4 lb.

The following day it was noticed that the baby did not move the right lower limb, and that there was induration and oedema of the right buttock. It was verified that no injection had been given into this buttock. Further examination disclosed complete paralysis of the anterior and posterior tibial muscles, the peronaei, and the flexors and extensors of the toes; and marked weakness of the calf muscles. The plantar reflex was absent and the skin of the sole and dorsum of the foot appeared to be anaesthetic. The circulation of the limb seemed normal. A diagnosis was made of sciatic-nerve palsy, probably due to thrombosis of the inferior gluteal artery. A small plaster splint was applied to control the drop-foot deformity. Within 7 days, the induration of the buttock had disappeared but, 8 months later, there has been little recovery in the nerve palsy. The right leg is wasted and is slightly shorter than the other. A plaster night-splint is still worn, and has prevented the development of any equinus deformity. It is hoped that some further recovery will still take place.

Paralysis of the sciatic nerve in newborn infants does not appear to have been described in the literature until as recently as 1949, when Mills[1] published his findings in 8 cases born in the Birmingham Maternity Hospital between December 1946 and October 1947. In each of these infants Mills found that the paralysis was unilateral and accompanied by circulatory changes in the affected limb, mainly in the region of the skin of the buttock, varying from oedema and induration to actual sloughing and ulceration. These babies had all been born in a state of white asphyxia and had all been given at least one injection of nikethamide into the umbilical cord. Labour in each case had been prolonged and difficult, 6 being vertex presentations and 3 delivered by Caesarean section. Seven of these babies had oedema of the buttock, which in 3 cases went on to ulceration. One had ulceration of the skin overlying the front of the tibia. Two of the babies died; of the survivors, 5 showed complete recovery of the nerve lesion, but one still had a complete drop-foot at the age of 2 years.

Mills advanced the hypothesis that the palsy and the induration of the buttock were in each case due to thrombosis of the inferior gluteal (sciatic) artery, which supplies the sciatic nerve and the adjoining skin in the gluteal region. Before birth this artery is the largest branch of the hypogastric (internal iliac) artery and constitutes the main arterial blood supply for the lower limb. After birth, this function is taken over by the external iliac artery. Mills pointed out that if the drug were injected into either of the two umbilical arteries in the umbilical cord, it might be forced into the inferior gluteal artery and its branches, and possibly cause thrombosis, with subsequent ischaemia of the sciatic nerve and the overlying gluteal skin. The flow of blood in the single umbilical vein is, of course, towards the heart. He concluded, in the absence of other evidence, that the lesions in these 8 cases were due to injection of nikethamide into an umbilical artery.

Many doctors, even those with very wide experience, have never seen this syndrome of neonatal sciatic palsy; and most have not even heard of it. It seems incredible that such an obvious clinical finding should have passed unrecognized unless, as Mills suggested, it was indeed a new neonatal syndrome. Bates and Page[2] (1949)

reported a similar case in which nikethamide had been used as an analeptic. Hudson, McCandless and O'Malley[3] (1950) recorded 20 similar cases, which had occurred in two hospitals in Liverpool between 1945 and 1948. The drug injected into the umbilical cord in the Liverpool series of cases was 'cycliton', a synthetic compound having a chemical formula resembling that of nikethamide (coramine). These workers stated that no ill effects had been noticed after the use of lobeline, which is not a synthetic compound. In general, they agreed with the theory of Mills that the lesions were consistent with the injection of a drug into an umbilical artery, with noxious effects on the inferior gluteal artery and the tissues supplied by it.

Fahrni,[4] working in Liverpool, studied 11 of the cases subsequently described by Hudson and his co-workers, and in 1948 submitted his findings for publication in the British Journal of Bone and Joint Surgery, advancing the theory that the lesions might be caused by pressure of a foetal hand on the sciatic nerve and buttock during labour. Fahrni's paper was not published until 1950 and in the meantime further investigations were carried out in the University of Liverpool as to the exact nature of the cause of this syndrome, which tended to support the theory that the cause of the lesions in the sciatic nerve and gluteal skin was the intra-umbilical injection of a drug, and not local pressure, although exact experimental proof is still lacking. In the 20 Liverpool cases there were 17 vertex presentations, 2 breech presentations, and 1 Caesarean section. Six cases showed sloughing of the gluteal skin, and the majority of the 20 cases at the end of 2 years still showed evidence of sciatic-nerve weakness, such as drop-foot, wasting and shortening of the limb.

DISCUSSION

All that the baby seen by us in Germiston seems to have in common with the babies described in England is that he showed asphyxia at birth, had a drug injected into the umbilical cord, and has a palsy of the sciatic nerve. Therefore, it seems to us, in the absence of any contrary evidence, that the hypothesis put forward by Mills adequately explains the causation of the syndrome of neonatal sciatic palsy accompanied by circulatory changes in the affected leg.

The condition seems to be rare and it is obvious that not every infant who has an intra-umbilical injection will get sciatic palsy. As there are two umbilical arteries and only one umbilical vein, it is probable that many injections have been made into an umbilical artery without ill effects, and without doing any good! Not infrequently one sees slightly older children with wasting and slight shortening of one limb, due to no obvious cause. Usually the diagnosis is made, with no real evidence to support it, of 'missed polio'. Do these cases perhaps fall into the category of 'missed neonatal sciatic palsy of mild degree'?

Whether intravenous or other injections of stimulants are successful in resuscitating asphyxiated babies, is debatable. It seems that intra-umbilical injections carry a certain risk and, if they must be done, every effort should be made to inject the drug into the umbilical vein only, since the recovery of sciatic-nerve lesions in infants is poor.

SUMMARY

1. A case of neonatal sciatic palsy in an infant is described.

2. A theory as to the causation of the lesion is discussed.

3. Injection of drugs into the umbilical cord seems to be dangerous.

REFERENCES

1. Mills, W. G. (1948): Brit. Med. J., **2,** 956 and (1949): *Ibid.*, **2,** 464.
2. Bates, C. and Page, A. P. M. (1949): *Ibid.*, **2,** 756.
3. Hudson, F. P., McCandless, A. and O'Malley, A. (1950): *Ibid.*, **1,** 223.
4. Fahrni, W. H. (1950): Bone Jt. Surg., **32B,** 1, 1950.