SCIATIC PALSY AFTER UMBILICAL CATHETERISATION: BRIEF REPORT

M. C. LYNCH

During 1949 and 1950, 38 cases of neonatal sciatic palsy after injection of umbilical vessels were described (Mills 1949; Hudson, McCandless and O'Malley 1950; McFarland 1950). Typically, paralysis was accompanied by ipsilateral discoloration of the buttock skin which progressed to scarring. McFarland (1950) proposed arterial thrombosis as the aetiology. The cause was thought to be mistaken one of the umbilical arteries for the vein, and injecting a thrombogenic analptic drug into it. In a shocked neonate, retrograde arterial flow may carry a bolus injection into the inferior gluteal artery via the hypogastric and internal iliac arteries. The inferior gluteal artery supplies the upper part of the sciatic nerve and the overlying area of skin on the buttock.

The administration of analptic agents via the umbilical route declined markedly after these reports. A further and similar case was described by Shaw in 1960, but no other examples appear in the literature. A recent case is described in this report.

Case report. A boy was delivered by breech presentation at 34 weeks' gestation. Fetal distress merited intubation, ventilation and the insertion of an umbilical “venous” catheter. The following drugs were administered via the catheter in the first hour of life: 4 ml 4.2% bicarbonate solution, 30 ml plasma, 10 ml 10% dextrose solution, 1 mg dexamethasone, 30 mg phenobarbitone and 30 mg benzylpenicillin. Forty-five minutes after injection, “bruising” of the right buttock, calf and heel became apparent. The child was noted not to move his right leg.

Over the ensuing weeks, the cutaneous discoloration faded, leaving an area of slough, and later of dense scar tissue, in the right buttock. A right sciatic nerve palsy was diagnosed. At 11 months of age the scarred area was explored; the two trunks of the sciatic nerve appeared normal and were free of scar. Since then the motor palsy has shown no recovery; calcaneal tendon lengthening and tibialis posterior transfer to the dorsum of the foot have proved necessary to correct equinovarus deformity. Sensory anaesthesia remains complete in the distribution of the lateral popliteal nerve at three and a half years of age.

Discussion. The administration of thrombogenic analptic agents by umbilical injection in the distressed neonate has been largely replaced by improved attention to the airway and intubation. However, the umbilical vessels are still utilised for injection or catheterisation for the administration of fluids and drugs. It is as difficult today as it ever was to distinguish the umbilical vein from either of the two arteries in a neonate with peripheral circulatory collapse. The apparent disappearance of neonatal sciatic palsy from mistaken injections may result more from alteration in the nature of the drugs and fluids administered by this route than from increased accuracy in identification of the umbilical vein. In the case reported here, it would appear that the 10% dextrose solution was the preparation most likely to have induced the thrombosis.

This thrombogenic solution is commonly used in clinical practice, and it is therefore difficult to comprehend why the condition does not occur more frequently. The current popular texts of neonatology do not mention the condition as a complication of umbilical injection or catheterisation. By reporting this case, it is hoped that any other recent cases may be highlighted. If additional cases are reported, the poor overall prognosis for recovery in this condition would merit a serious review of the practice of umbilical injection in the newborn.

REFERENCES


