deformity'. Since 1960 the aetiology of congenital sternomastoid torticollis (CST) has been studied during observation of over 20,000 newborn infants. It proved possible to diagnose the condition at birth, though the torticollis was frequently 'latent' at this time because of neck shortness. Its presence was confirmed by histological examination in the case of 5 infants dying within 1-3 hours of delivery. Interstitial fibrosis, present at birth, was observed to increase during the postnatal period leading to hardening of the muscle or to frank 'tumour' formation. The incidence of CST, 0.3% at birth, subsequently diminished as the majority of muscles recovered; in the remainder torticollis became more apparent as the neck lengthened during early childhood. CST was noted after nontraumatic delivery, including caesarean section in 3 cases. It was always unilateral. Characteristically the infant lay with head flexed laterally onto one shoulder. The ear on this side, the affected one, was usually upturned and the jaw tilted away; often plagiocephaly was present. In a number of cases it was possible to show the similarity between the moulded postnatal appearance and 'position of comfort', and the radiologically determined prenatal posture.

During a study of 6756 consecutive deliveries, CST was observed in association with plagiocephaly ($P < 0.001$), facial deformities ($P < 0.05$), ipsilateral mandibular asymmetry ($P < 0.001$), congenital postural scoliosis ($P < 0.001$), CDH ($P = 0.12$), and congenital deformities of the feet ($P < 0.001$). A similar pattern of maternal pregnancy characteristics was observed as was encountered in association with these other congenital postural deformities. Thus, 53% of infants with CST were firstborn (expected 36%), and 20% presented by the breech (expected 5%). Maternal oligohydramnios was a frequent accompaniment and CST was noted to occur in association with bilateral renal anomalies causing fetal anuria.

These observations strongly support experimental work which suggests that CST is due to ischaemic fibrosis and shortening of the muscle secondary to venous occlusion which may be caused by persistent lateral flexion and rotation of the neck before birth. Trauma to the shortened muscle during delivery may be responsible for secondary damage to the muscle in some cases.

J. A. S. DICKSON, C. T. LEWIS, and V. A. J. SWAIN. Queen Elizabeth Hospital for Children, London. 'Milk bolus obstruction in the neonate'. In the years 1964 to 1972, 17 cases of milk bolus obstruction in neonates were treated at this hospital. The condition which was first seen in 1964 now accounts for 6% of the neonatal intestinal obstructions. The diagnosis should be suspected in a male infant (13 out of 14) who after progressing normally and passing changing stools becomes obstructed around the 5th day (range—1st to 10th). All were on a prepared cow's milk feed and 11 out of 14 a full-cream milk feed. 7 out of 14 of the babies passed blood by the rectum. On the plain x-ray there were small gut fluid levels and replacement of normal gas pattern in the right iliac fossa by an inspissated 'faecal' mass sometimes containing bubbles of gas or with

a ground glass appearance. Most had gas in the rectum. The first 14 babies were operated on, the last 3 were treated with Gastrografin orally or by enema. All the babies survived and, except the severely premature or abnormal, progressed normally. The cause appears to be inability of the babies to absorb the solid content of the feed which may also have been overconcentrated.

A. G. MCPHERSON. Southmead Hospital, Bristol. 'Neonatal peritonitis'. Neonatal peritonitis remains a dangerous condition. Factors complicating diagnosis and management include its low incidence, the diversity of aetiological factors found, and the severity of underlying or associated disease. 28 cases presenting since 1957 at Southmead General Hospital were reviewed.

Thirteen aetiological factors relating to mother or baby were found. 13 infants were premature, 10 had severe respiratory problems at birth. Maternal hydramnios was present in 7. 6 babies had had exchange transfusion.

The commonest findings were gangrenous volvulus and cystic fibrosis. Combined factors were often present. The usual presentation is with bilious vomiting, abdominal distension, and failure to pass normal meconium. 4 cases had distended abdomens at birth. Plain abdominal x-rays are of great diagnostic value. Pneumoperitoneum is diagnostic but absent from early films. Treatment is surgical. Close liaison with the paediatrician who sees the baby first is essential. In assessing results it is pointed out that many of these babies are small and/or suffering from serious conditions such as haemolytic disease, cystic fibrosis, or gangrenous volvulus. 8 cases were considered unfit for surgery. Of 20 submitted to surgery, 12 survived, mostly in the later part of the series.

J. A. DODGE. University Hospital of Wales, Cardiff. 'Maternal factor in infantile hypertrophic pyloric stenosis'. In a study of 526 patients with infantile hypertrophic pyloric stenosis in the Belfast area, it became evident that a purely genetic aetiology was unlikely. Environmental factors such as birth rank, social class, seasonal variation, and type of feeding all appeared to influence the incidence of the disorder. Furthermore, though the infants deviated significantly from the general population in respect of their ABO blood groups, with a deficiency of group A, their mothers showed an even greater divergence from the control distribution. This suggests that the intrauterine environment may predispose to the development of infantile hypertrophic pyloric stenosis, perhaps by transplacental passage of a teratogen.

Experimental work in dogs succeeded in reproducing hypertrophic pyloric stenosis in pups born to mothers who were treated with pentagastrin during pregnancy. This animal model may be relevant to the disorder in the human and indicates a mechanism by which maternal variations can produce pathology in the infant.

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