for Sick Children, Glasgow, during the last 5 years the condition has been seen most commonly in association with hypertonic dehydration. During this period 14 infants have been diagnosed as having renal venous thrombosis and in 6 of these the lesion was thought to be bilateral. All 14 infants were aged less than 6 months and in each the biochemical findings of hypertonic dehydration were present. In all infants renal enlargement and haematuria were detected and in 8 cases the renal enlargement was confirmed radiologically. 13 of the 14 infants studied had demonstrable thrombocytopenia and in 4 of these detailed serial coagulation studies were carried out. All infants received intravenous fluid therapy and 5 were treated with peritoneal dialysis. In 6 infants continuous intravenous heparin was administered during the acute stage of the illness. 7 infants are alive with normal renal function. One is alive but with impaired renal function bilaterally and one has required nephrectomy for systemic hypertension. 4 infants died during the acute stage of the illness and a further child died 15 months after the acute illness of unrelated disease, his kidneys being normal histologically at necropsy.

Combined Sessions with British Association of Paediatric Surgeons

J. WAGGET. Hospital for Sick Children, Newcastle. 'Parenteral feeding: surgical indications and techniques'. Intravenous feeding is indicated in any condition where feeding by the gastrointestinal tract is contraindicated for more than a few days, but where return to normal function can eventually be expected. Surgical cases include many of the congenital abnormalities, and any surgery of the gastrointestinal tract, complicated by sepsis, ileus, or fistulae formation which prevents normal feeding. In the past many such patients had increased morbidity and mortality, simply because of the added factor of starvation.

Our team effort in Newcastle is mainly directed towards newborn surgical patients, but our regimen can also be used in older children. It has been designed so that it can be given either by central or peripheral vein and it is flexible enough to allow for replacement of large gastrointestinal fluid, and electrolyte losses, where necessary. We use fat as one of the principal calorie sources, a crystalline amino acid solution, and a third solution which supplies dextrose plus extra electrolytes and vitamins. These three main solutions are infused by pump by a series of 'Y' connections. The metabolic problems associated with this unphysiological method of preventing starvation have been lessened since we have had a more balanced regimen whose constituents have been delivered to the patient steadily throughout the 24-hour period. The technique of preparing infusion set-up and its delivery to the patient were outlined.

M. PANTERBRICK. Newcastle. 'Parenteral feeding: metabolic changes'. This paper reported the personal experience with newborn surgical patients fed intravenously using a regimen which included fat, carbohydrate, and crystalline amino acids. The regimen is designed so that it can be given either by peripheral vein or into the central venous system. Emphasis was given to phosphate requirements and amino acid patterns during infusion and their relation to acid-base status.

J. T. HARRIES. Institute of Child Health and The Hospital for Sick Children, Great Ormond Street, London. 'Parenteral feeding—complications'. Intravenous feeding represents an important advance in the management of certain conditions, but the serious complications limit the more widespread use of this form of treatment at present. Cumulative data suggest that complication rates may be as high as 60–70%. The following complications have been reported.

(*1*) *Septicaemia.* The commonest infecting organisms are *Candida albicans*, *Staphylococcus aureus*, and *albus*, and other Gram-negative bacteria. Risk of septicaemia is increased when using I.V. catheters, particularly if placed in central veins, and all peripheral veins should be utilized before resorting to central ones. A strict aseptic technique is probably the most important single factor in prevention. (*2*) *Metabolic acidosis.* Severe lactic acidosis may follow fructose infusions in hepatic disease or anoxic states, particularly if infusate contains ethanol (e.g. Aminosol-fructose-ethanol). High titratable acidity of some amino acid infusates may also induce acidosis. Dehydration, anoxia, and electrolyte imbalance must be corrected before initiating I.V. feeding; controlled infusion rates and frequent biochemical monitoring are essential during I.V. feeding. (*3*) *Hypophosphataemia*, despite phosphate supplementation, may lead to haemolytic anaemia, weakness, and seizures. Phosphate consumption during anabolic phase may contribute to pathophysiology of hypophosphataemia. (*4*) *Phlebitis and venous obstruction* secondary to hypertonicity and H^+ ion concentration of infusates, and/or infection. Frequency may be reduced by inclusion of isotonic fat emulsion in regimen. (*5*) *Catheter dislodgement and extravasation of fluid.* Correct initial catheter placement and subsequent careful handling important; extravasation of hypertonic solutions may cause tissue necrosis and secondary infection. (*6*) *Hypoglycaemia* with severe hypothermia may follow abrupt termination of I.V. feeding. (*7*) *Fluid retention* with cardiac failure may accompany infusions of amino acid solutions due to high sodium concentration. (*8*) *Dehydration* secondary to osmotic diuresis particularly likely to occur during introduction of hypertonic infusates (e.g. osmolality of Aminosol-fructose-ethanol and Vamin = 1975 and 1275 mOsm/kg, respectively). (*9*) *Hyperuricaemia* due to increased hepatic synthesis or uric acid may accompany fructose infusions. (*10*) *Hypocalcaemia, hypokalaemia, hepatic dysfunction, and thrombocytopenia* may also complicate I.V. feeding.

With increasing experience of I.V. feeding additional complications will undoubtedly become recognized.

P. M. DUNN. Southmead Hospital, Bristol. 'Congenital sternomastoid torticollis: an intrauterine postural

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 ($P < 0{\cdot}001$), facial deformities ($P < 0{\cdot}05$), ipsilateral mandibular asymmetry ($P < 0{\cdot}001$), congenital postural scoliosis ($P < 0{\cdot}001$), CDH ($P = 0{\cdot}12$), and congenital deformities of the feet ($P < 0{\cdot}001$). 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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