Children who lost weight, regardless of therapeutic regimen, showed a reduced growth velocity ($90.1 \pm 22.2\%$) compared to those who gained weight ($99.6 \pm 24.5\%$) but the difference was not statistically significant and did not correlate with the amount of weight lost. Children who received diet plus fenfluramine showed a mean growth velocity of only $82.3 \pm 23.6\%$ compared to $99.5 \pm 23.2\%$ for diet alone and $99.3 \pm 26.4\%$ for diet plus chlorphentermine or diethyl propion. The difference was only significant at the 10% level.

In view of the doubtful value and possible dangers of anorectic drugs in childhood, careful monitoring of the growth of obese children treated with these agents is indicated.

British Association of Paediatric Nephrology

C. CHANTLER, J. S. CAMERON, R. H. R. WHITE, and C. S. OGG. Guy's Hospital, London and Birmingham Children's Hospital. 'Long-term stability of remission in the nephrotic syndrome after treatment with cyclophosphamide'. 57 children with the nephrotic syndrome and minimal changes on renal biopsy, who had relapsed repeatedly and showed corticosteroid-induced toxicity, were treated with cyclophosphamide from 1966 to 1969. At that time an initial dose of 5 mg/kg per day was used, and leucopenia maintained for an average of 12 weeks. All these children have now been followed for more than 4 years since the end of their treatment, some for 7 years.

At present, 18 children still remain in remission, 35 have relapsed, and 4 are dead, 2 in relapse. One child died of measles pneumonia shortly after treatment with cyclophosphamide, and another died of cerebral tumour in 1973. One other child developed Hodgkin's disease also in 1973, aged 19.

The rate of relapse has been exponential over the first 5 years after treatment with a half-time of 3 years. This compares favourably with the near 100% relapse rate in similar children treated with corticosteroid withdrawal alone, but it is no better over the first 2 years than cyclophosphamide treatment at 3 mg/kg per day for 8 weeks. There was no suggestion that remissions were more stable in those treated for longer periods, nor did stability of remission relate to age or the duration of disease before treatment. These data permit a better assessment of benefit versus toxicity in the treatment of relapsing nephrotic children.

S. R. MEADOW. Department of Paediatrics and Child Health, Leeds. 'Poststreptococcal nephritis—a rare disease?'. (To be published in full in the *Archives*.)

M. J. DILLON and JENNIFER RYNESS. The Hospital for Sick Children and Institute of Child Health, London. 'Plasma renin activity and aldosterone concentration in children'. The central role of the renin angiotensin aldosterone system in the control of salt balance and blood pressure is well established. However, only limited data are available in children because of the large volumes of blood hitherto required for the estimation of plasma renin activity and aldosterone concentration.

For this reason, semimicro methods for the measurement of plasma renin activity (PRA) by radioimmunoassay (angiotensin I generation rate) and plasma aldosterone concentration (PAldo) by radioimmunoassay have been developed utilizing 0.5 ml and 1.0 ml plasma, respectively.

It was found that in healthy children on free diets the PRA varied inversely with age. In infants the mean value was 1392 pgAI/ml per hr with a progressive decrease through childhood to the mean adult value of 87 pgAI/ml per hr. There was some evidence of a negative correlation between PRA and sodium turn-over, estimated from the urinary sodium/creatinine ratio. The mean value for PAldo in children over the age of 1 year was 6.2 ng/100 ml, but in infants was 20.2 ng/100 ml.

PRA was in the range from 1000–200 pgAI/ml per hr in several hypertensive children, without PAldo necessarily being above the upper limit of normal. On the other hand, in children with salt-losing states PRA was much greater, usually over 10,000 pgAI/ml per hr, and in the majority of these children PAldo was over 30 ng/100 ml.

S. MELLER. Queen Mary's Hospital for Children, Carshalton. 'Significance of bacteriuria in Cardiff schoolgirls'. Over a 2-year period all infant and junior schools in the City of Cardiff were visited by a mobile bacteriuria screening laboratory. 11,939 girls aged 5 to 11 (89% of the total population) were screened, at an estimated cost of 75 pence per child. Bacteriuria was confirmed in 207 girls, a prevalence of 1.7%. Full clinical and radiological data were obtained in 180 children who are participating in a randomized controlled trial of treatment.

Although some urinary symptoms were common, notably incomplete bladder control and offensive urine, few children had a history suggestive of serious infection past or present. As a group, their general health was good and they were of normal stature. However radiological evidence of pyelonephritis was found in 26% and vesicoureteric reflux in 34%. 74 children have completed the first year of follow-up. In the control group, 24% had a spontaneous bacteriological remission without treatment. In the treatment group, 19% had a recurrence within 6 months and 54% within 12 months of a successful short course of appropriate antibiotic: 11% continued to have bacteriuria despite repeated courses of treatment.

Although screening for bacteriuria in this age group identifies one child with pyelonephritis out of every 260 girls examined, it has yet to be shown that a treatment programme can influence the natural history of the condition.

Anna MURPHY. Royal Hospital for Sick Children, Glasgow. 'Renal venous thrombosis in hypertonic dehydration'. Renal venous thrombosis is a well-recognized clinical entity of early infancy. The typical case is characterized by renal enlargement, haematuria, uraemia, and thrombocytopenia. In the Royal Hospital

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