Indeed, three children presenting with colitis in the first year of life had such severe, chronic inflammatory bowel disease that subtotal colectomy was eventually required. Indeed, we have emphasised[2] that infantile colitis is a heterogeneous syndrome with a variety of causes that can only be identified by means of colonoscopy and multiple mucosal biopsy as well as response to dietary manipulation. Over the past four years, 11 children (see Table) have presented at these two hospitals with colitis (chronic bloody diarrhoea) whose onset was under the age of 1 year. Only four proved to have food allergic colitis. Five had an indeterminate colitis which could not be categorised as ulcerative colitis or Crohn's disease. Two had features of Behcet's colitis.[3]

In our experience chronic, inflammatory bowel disease rather than food allergic colitis is the major cause of infantile colitis presenting under the age of 1 year.

References

[1] Jenkins, HR, Pincott JR, Soothill JF, Milla PJ, Harries JT. Food allergy: the major cause of infantile colitis. *Arch Dis Child* 1984;**59**:326–9.
[2] Chong SKF, Walker-Smith JA, Blackshaw AJ, Morson BC. Colitis in early infancy and childhood: a prospective study. *Gut* 1983;**5**:A462–63.
[3] Chong SKF, Walker-Smith JA, Wright V, Nishigami T, Raafat F, Morson BC. Intestinal Behcet's disease. *Paediatric Research* 1983;**46**:427.

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Hypoplastic optic nerves and pituitary dysfunction

Sir,

In the report by Stanhope *et al*[1] we were interested to read that one additional patient presented with hypoglycaemia and conjugated hyperbilirubinaemia. The latter was also the presentation in one of our patients and we feel that this is a genuine association which needs emphasis.

This boy, the first child of healthy, unrelated Caucasian parents, was born at 38 weeks' gestation and weighed 2·5 kg. Two cyanotic episodes in the first day were associated with hypoglycaemia (blood glucose concentration 0·8 mmol/l), polycythaemia (haemoglobin concentration 20 g/dl) and hypothermia (33°C). Suspected bacterial infection was treated, but was not confirmed by cultures. Hypoglycaemia did not recur. His serum unconjugated bilirubin concentration rose to a maximum of 336 μmol/l at 5 days and was treated with phototherapy. Subsequently, hepatosplenomegaly, conjugated hyperbilirubinaemia (maximum concentration 35 mmol/l) and raised serum γ glutamyltranspeptidase (maximum 555 IU/l) occurred and resolved during the first 12 weeks of life. No infective or metabolic cause of cholestasis was identified. During investigation nystagmus was noted and ophthalmoscopy showed gross optic nerve hypoplasia. On cerebral ultrasound examination he had symmetrically dilated lateral ventricles and absence of the septum pellucidum. At 15 months of age he shows normal growth but remains severely visually handicapped.

Previous reports have drawn attention to jaundice in infants with septo-optic dysplasia. Two of 15 patients in one series[2] had 'giant cell hepatitis', while prolonged jaundice with or without raised serum transaminases was noted in one case report[3] and two of four children in another series.[4] Johnsen *et al*[2] suggested that the presence of hepatitis implied a viral aetiology of septo-optic dysplasia. It is perhaps more likely that either hyposecretion of thyroxine or cortisol, or both produce cholestasis, though in our patient endocrine dysfunction has not been documented.[5]

References

[1] Stanhope R, Preece MA, Brook CGD. Hypoplastic optic nerves and pituitary dysfunction. *Arch Dis Child* 1984;**59**:111–4.
[2] Johnsen MD, Richardson JR, Krohel GB. Neurologic concomitants of optic nerve hypoplasia. *Neurology* 1976;**26**:391.
[3] Harris RJ, Hass L. Septo-optic dysplasia with growth hormone deficiency (De Morsier syndrome). *Arch Dis Child* 1972;**47**:973–6.
[4] Patel H, Tze WJ, Crichton JU, McCormick AQ, Robinson GC, Dolman CL. Optic nerve hypoplasia with hypopituitarism. *Am J Dis Child* 1975;**129**:175–80.
[5] Copeland KC, Franks RC, Ramamurthy R. Neonatal hyperbilirubinemia and hypoglycaemia in congenital hypopituitarism. *Clin Pediatr (Phila)* 1981;**20**:523–6.

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Pancuronium bromide induced joint contractures in the newborn

Sir,

The paper by Sinha and Levene,[1] suggesting that pancuronium (a drug used frequently in the neonatal intensive care unit) is associated with a 30% incidence of 'severe joint contractures' is indeed disturbing. These data raise questions regarding the risk:benefit ratio of pancuronium treatment.

After careful review of the data presented and the published reports cited, I have several criticisms. Regarding the latter, Sinha states 'in humans, maternal paralysis for status epilepticus[2] and tetanus[3] have been associated with joint contractures in the neonate', but in one cited case no contractures were noted.[2] Furthermore, in the second case,[3] after a prolonged period of muscle paralysis (10 days) early in pregnancy, in a critically ill patient with multiple medical problems who had received several other medications, it was suggested that the paralysing agent had caused joint contractures in the infant. Experimental animal data[4] showed that early in chick embryogenesis,