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 emphasised² 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.

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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 series² had 'giant cell hepatitis', while prolonged jaundice with or without raised serum transaminases was noted in one case report³ 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

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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 epilepticus² and tetanus³ 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 data⁴ showed that early in chick embryogenesis,

prolonged administration of a paralysing agent consistently resulted in limited deformities at the time of hatching.

The data cited above are used as supportive evidence for implicating pancuronium as a cause of joint contractures in one infant who received pancuronium for six hours and in a second infant who had been paralysed for 24 hours. The second infant had been noted to have joint contractures before the start of treatment. It would not be unreasonable to expect that prolonged use of muscle paralysis (as in the third case) could be associated with the development of contractures.

I am also confused as to exactly how the aminoglycoside or the phenobarbitone treatment increased the risk of contractures. If the authors are indeed worried about prolonging the action of pancuronium, neostigmine (and atropine) has been used for years to reverse paralysis.

In those infants requiring prolonged paralysis, physiotherapy may be justified to prevent contractures. The data presented, however, should not limit the use of muscle paralysis where appropriately indicated.

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A prospective study of chlamydial, mycoplasmal, and viral infections in a neonatal intensive care unit

Sir,

There is still much to be learned about the microbiology of the newborn, and we were glad to see the article by Drs Rudd and Carrington.¹ Our interest has been in respiratory syncytial virus infections in childhood, and in 1969 we studied an outbreak of neonatal infection in a maternity hospital in Newcastle upon Tyne.² Knowing how severe illness from this virus can be in later infancy, we were surprised how mild it was in the neonate; no more than a 'cold'.³

Virus isolation is not an end but a beginning. We need to ask what is happening to the host in this encounter, what may happen in the future, and whether there is any connection between the two.

The mildness of neonatal respiratory syncytial virus infections can be deceptive; it may cause sudden unexplained ('cot') death:⁴ this is uncommon, but suggests the

need for discreet oversight through the danger period. Apart from this, is mild neonatal infection unimportant? If it is not, how is it related to the severe epidemic 'bronchiolitis' and 'pneumonia' which may follow in the next six months;^{3 5} and to the excess of recurrent wheezing illness which occurs in the infected in the next five years? Have the authors uncovered any studies of neonatal respiratory syncytial virus infection followed into later childhood which can answer this?

Our attempt to find hard evidence has been prevented by inability to complete hospital experience with the full picture in the community from which the severe hospital illness is drawn. Without all the facts we can only speculate. Our hypothesis, which has not been disproved, is that mild first infection sensitises and leads to an allergic reaction expressed as 'bronchiolitis' which may go on from this to recurrent wheezing illness in the early years of childhood.^{6 7} At the same time our minds are not closed to the possibility that later severity reflects structural damage. Whichever interpretation is correct, mild respiratory syncytial virus infections in the newborn may set in train a pattern of severe and recurrent illness.

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Drs Rudd and Carrington comment:

It seems that not all neonates,⁴ and infants⁸ infected with respiratory syncytial virus develop severe illness. Factors which contribute to the severity of infection include preterm birth⁴ and the use of mechanical ventilation,⁹ and it may be that pre-existing bronchopulmonary dysplasia as well as transplacentally acquired immunity are important.

The relation between respiratory syncytial virus infection in the neonatal period and wheezing illness in childhood is of great interest, and there is an urgent need for prospective studies in this field. It would, however, be misleading to study respiratory syncytial virus in isolation. The symptoms produced by rhinovirus are indistinguishable from respiratory syncytial virus infection in the neonate.⁹ *Chlamydia trachomatis*, which produces pneumonitis during the first few months of life, may go unrecognised and untreated. *Ureaplasma urealyticum* did not seem to cause illness in our study, although this organism has been isolated from infants with pneumonitis. Indeed, since publication of this work we have found that two babies with pneumonia from whom only *Mycoplasma hominis* was isolated also produced an immune response to this organism. Thus, it may be that the wheezing illnesses of childhood result from the early sensitisation to a number of different microorganisms.

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