it, the improvement in the survival rates from 1976 to 1983 is not so striking (Fig. 1). Indeed the overall survival of babies weighing 1251 to 1500 g from 1976 to 1983 (68.5%) would not have been significantly greater than it was from 1970 to 1975 (60-0%). The contribution of mechanical ventilation to the survival rates of babies is summarised in Fig. 2.

We agree with Barson et al that good quality neonatal intensive care influences obstetrical management and leads to elective preterm delivery of high risk fetuses, thereby reducing the risk of stillbirth from asphyxia. Indeed, we submitted that idea as written and oral evidence to the House of Commons Social Services Committee in 1980.3 There are great pitfalls in using information from perinatal necropsy surveys as a basis for auditing neonatal care. Audit requires a multidisciplinary approach including the assessment of neurodevelopmental handicap in surviving babies. We recently drew attention to the improving prognosis of low birthweight babies after the introduction of intensive care at this hospital (British Paediatric Association Annual Meeting, 1984). We conclude that neonatal intensive care has improved the survival of immature babies with established respiratory distress or recurrent apnoea irrespective of any beneficial influence it might have had on babies asphyxiated at birth. Those who might be tempted to direct their resources solely towards resuscitation of asphyxiated babies while relegating continuing neonatal care must be warned.

References

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Food allergy and infantile colitis

Sir,

We read with interest the important paper by Jenkins et al1 on the role of food allergy as a major cause of infantile colitis. The authors state 'we have shown that food allergic colitis . . . accounts for most, if not all, children who present in the first year of life suffering from colitis.' This has not been our experience at St Bartholomew’s Hospital and the Queen Elizabeth Hospital for Children.

Table Details of 11 children aged under 1 year presenting with colitis and response to elimination diet

<table>
<thead>
<tr>
<th>Case No</th>
<th>Diagnosis</th>
<th>Age at onset of symptoms</th>
<th>Responded to elimination diet</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Indeterminate colitis and subacute colitis</td>
<td>1 wk</td>
<td>No</td>
</tr>
<tr>
<td>2</td>
<td>Indeterminate colitis</td>
<td>3 wk</td>
<td>No</td>
</tr>
<tr>
<td>3</td>
<td>Food allergic colitis</td>
<td>5 wk</td>
<td>Yes</td>
</tr>
<tr>
<td>4</td>
<td>Food allergic colitis</td>
<td>6 wk</td>
<td>Yes</td>
</tr>
<tr>
<td>5</td>
<td>Indeterminate colitis</td>
<td>10 wk</td>
<td>No</td>
</tr>
<tr>
<td>6</td>
<td>Food allergic colitis</td>
<td>3 mths</td>
<td>Yes</td>
</tr>
<tr>
<td>7</td>
<td>Indeterminate colitis</td>
<td>4 mths</td>
<td>No</td>
</tr>
<tr>
<td>8</td>
<td>Behcet’s colitis and subacute colitis</td>
<td>4 mths</td>
<td>No</td>
</tr>
<tr>
<td>9</td>
<td>Behcet’s colitis and subacute colitis</td>
<td>5 mths</td>
<td>No</td>
</tr>
<tr>
<td>10</td>
<td>Indeterminate colitis</td>
<td>6 mths</td>
<td>No</td>
</tr>
<tr>
<td>11</td>
<td>Food allergic colitis</td>
<td>7 mths</td>
<td>Yes</td>
</tr>
</tbody>
</table>
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 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 Behçet'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


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Hypoplastic optic nerves and pituitary dysfunction

Sir,

In the paper by Stanhope et al1 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 al5 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


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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' and tetanus' 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 paralisning agent had caused joint contractures in the infant. Experimental animal data4 showed that early in chick embryogenesis,