

bowel motion during either labour or the ensuing 48 hours.<sup>1</sup> While this is true there is a very noticeable gestational age effect on the passing of meconium by a fetus during labour which gives some idea of the maturation of this aspect of gut function. Increasing gestational age is by far the strongest predictor of the presence of meconium in amniotic fluid at delivery with 10% of infants at 35 weeks' gestation, 17% at 40 weeks' gestation, and 25% at 42 weeks' gestation having passed meconium before delivery.<sup>2</sup> The presence of meconium in labour before 34 weeks' gestation is uncommon and infections such as listeria need to be excluded. The mechanism resulting in the passage of meconium is unknown but has been said to depend on hypoxia or acidosis,<sup>3</sup> with vasoconstriction of the fetal intestinal blood supply, hyperperistalsis, and anal sphincter relaxation.<sup>4</sup> However it seems highly likely that the passage of meconium is generally a normal physiological event reflecting increasing fetal maturity.

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- 1 Bisset WM. Development of intestinal motility. *Arch Dis Child* 1991;66:3-5.
- 2 Matthews TG, Stack JC, Unwin AR, Harrison RF. Is there a gestational age distribution in the passage of meconium. *Journal of Obstetrics and Gynaecology* 1983;4:23-4.
- 3 Walker J. Fetal anoxia. *Journal of Obstetrics and Gynaecology of the British Empire* 1954;61:162-80.
- 4 Saling E. *Fetal and neonatal hypoxia in relation to clinical obstetrical practice*. London: Arnold 1968:117.

#### Gastro-oesophageal reflux in apparent life threatening events

SIR,—Your editorial on surgery for gastro-oesophageal reflux stated that apparent life threatening events (ALTE or 'near miss' SIDS episodes) associated with confirmed gastro-oesophageal reflux was 'probably an absolute' indication for surgery.<sup>1</sup> While having some difficulty with the concept of probable absolutes the following points should be noted.

Gastro-oesophageal reflux is a common event in infants and the application of techniques such as continuous lower oesophageal pH measurement has resulted in its association with a variety of conditions from asthma, recurrent bronchitis, pneumonia, apnoeic periods in the newborn, cyanotic attacks, ALTE, rumination, torticollis, abnormal movements of the head and neck, and neuropsychiatric disorders.<sup>2</sup> However as Carré previously noted none of these disorders, with the exception of pulmonary infections, occurred with increased frequency in a 35 year prospective study of 710 children with partial thoracic stomach (hiatal hernia) and gastro-oesophageal incompetence.<sup>2</sup>

The presence of gastro-oesophageal reflux in infants who have suffered an ALTE does not establish a causal relationship. Before embarking on surgical treatment of a common, and usually harmless, condition it is imperative that a clear association be established between the two events—that is, it has to be shown that gastro-oesophageal reflux causes the ALTE in a particular child. Previous studies do not support surgical intervention with no clear causal relationship established and no benefit from fundoplication.<sup>3</sup>

Even if gastro-oesophageal reflux is shown to cause an ALTE in an infant we should perhaps ponder on why this infant reacts so differently from the majority of infants with gastro-oesophageal reflux.

At present the role of gastro-oesophageal reflux in ALTE is still unclear and needs to be firmly established before the various management options (including medical and conservative) can be prospectively evaluated.

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- 1 Kiely EM. Surgery for gastro-oesophageal reflux. *Arch Dis Child* 1990;65:1291-2.
- 2 Carré JI. Clinical significance of gastro-oesophageal reflux. *Arch Dis Child* 1984;59:911-2.
- 3 Rosen CL, Frost JD, Harrison GM. Infant apnoea: polygraphic studies and follow-up monitoring. *Pediatrics* 1983;71:731-6.

#### Nutrition and bronchopulmonary dysplasia

SIR,—We read the article by Wilson *et al* on nutrition and bronchopulmonary dysplasia<sup>1</sup> with interest and should like to report some of our own data for comparison. Between January 1980 and December 1989, 4389 infants under 72 hours of age have been admitted to the neonatal intensive care unit at Liverpool Maternity Hospital. Two hundred and forty two of these developed bronchopulmonary dysplasia, which was defined as being present if an infant had received ventilatory support for over 24 hours in the first 72 hours of life and had a persistent requirement for supplementary oxygen together with an abnormal chest radiograph on day 28. We performed a multiple logistic regression analysis of these data with bronchopulmonary dysplasia as the dependent variable and sex, birth weight, gestation, survival to discharge, and year of birth as independent variables. This showed an inverse correlation between the occurrence of bronchopulmonary dysplasia and birth weight which was independent of gestational age (odds ratio for a rise of 250 g in birth weight above 500 g=0.83; 95% confidence interval=0.77 to 0.90).

We have also investigated in detail the growth of nine infants with severe bronchopulmonary dysplasia who have recently been discharged from hospital receiving supplementary oxygen. Of the six that had been born at more than 28 weeks' gestation only two were below the 3rd centile for weight at birth, three were between the 10th and 50th, and one was above the 90th. At term all nine infants were below the 3rd centile for weight and at discharge from hospital eight were still below the 3rd centile.

The above findings are in agreement with those of Wilson *et al* and lend strong support to the hypothesis that infants who develop bronchopulmonary dysplasia fail to achieve optimal growth postnatally as well as antenatally.

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- 1 Wilson DC, McClure G, Halliday HL, Reid M McC, Dodge JA. Nutrition and bronchopulmonary dysplasia. *Arch Dis Child* 1991;66:37-8.

#### Continued need for pneumococcal prophylaxis after splenectomy

SIR,—In a recent issue of this journal Drs Murdoch and Dos Anjos reported two children who died of overwhelming pneumococcal infection, five and eight years after splenectomy.<sup>1</sup> They might have included in their report a patient at Guy's Hospital who died last summer 30 years after splenectomy performed at the age of 2 years at the Evelina Children's Hospital.

The girl was admitted to the Evelina Children's Hospital in 1959 when I was paediatric registrar. She had idiopathic hypoglycaemia—at that time labelled McQuarrie's syndrome. Heavy doses of steroid controlled her symptoms but rendered her grossly cushingoid. A single insulin estimation (30 ml blood from a femoral vein that was incubated on a rat's diaphragm) showed high circulating insulin concentrations and a subtotal pancreatectomy was carried out. To achieve this it was necessary to remove her spleen. The operation cured her hypoglycaemia and did not cause diabetes mellitus. The child thrived well physically and intellectually.

Some 20 years later, when I met a case of nesidioblastosis, I asked for the original blocks of her pancreas to be brought out, appropriately stained, and confirmed the diagnosis that she also had nesidioblastosis. At a meeting on hypoglycaemia at Guy's Hospital I presented the case history and the 'patient' attended; she was healthy and holding down a good job. The fact of splenectomy was noted but since she was now in her early 20s and very well, no prophylactic penicillin was suggested.

She married, bore two children successfully, and never had any obvious problem with infection. On the summer of her death she became unwell with a high fever and joint pains in the knee. She was seen in Guy's Hospital casualty department where the abdominal scar was attributed to pancreatectomy, and the casualty officer was unaware of the splenectomy. Radiographs were taken and tests were initiated but she was not admitted. In the small hours of that night she was brought in dead to King's College Hospital. Necropsy showed overwhelming infection with *Streptococcus pneumoniae*.

This tragic story, together with other cases encountered in my 30 years as a paediatrician, leads me to endorse Drs Murdoch and Dos Anjos' recommendations on prophylaxis, though they would not have saved the woman described here. (Pneumovax II was not available in 1959 and the infection occurred long after she had passed 'increased epidemiological risk of pneumococcal infection such as school, university, or the armed forces'.) Therefore, prophylactic penicillin should be continued for life. As this will inevitably lead to problems with compliance, an alternative is that the patient should carry a card and always have a large dose of amoxicillin capsules to be taken at once in any sudden feverish systemic illness, and seek medical advice promptly. I would also add that wherever the circumstances allow, Pneumovax II should be given before splenectomy.

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- 1 Murdoch IA, Dos Anjos R. Continued need for pneumococcal prophylaxis after splenectomy. *Arch Dis Child* 1990;65:1268-9.