under normal circumstances be recorded by our local coroner’s office. In our efforts to resolve the difficulty, we encountered a more complete but less detailed source of data. In those districts where child health surveillance and school health records are computerised, a comprehensive list is kept of children who have died in order that grieving parents are not inadvertently sent invitations to attend child health surveillance appointments. The list is usually complete and includes local residents who have died outside of the district boundaries. The information contained in these lists usually just extends to the child’s name, address, and cause of death with no further details. However, we found these records of use in supplementing data supplied by the coroner’s records.

We would concur with Dr Levene in her plea for a comprehensive prospectively compiled childhood accident mortality database. Coroners’ inquests and child health surveillance will yield basic data which, perhaps, be usefully supplemented by confidential inquiries into the circumstances of individual accidents. Collation of locally relevant data is a key element in child accident prevention which should be a priority for every health authority.

Dr Rushforth and colleagues comment:
We note Dr Jones’ suggestion of a possible route of a percutaneous central venous catheter, of the diploic veins, to lie in the middle meningeal vein to explain a subdural collection. However, the diploic veins are absent at birth and do not develop until around 2 years of age.1 The infant in the case report was still less than 37 weeks’ corrected gestational age at the time of the incident.2

It may be possible to suppose passage of a catheter via an emissary vein to lie in the sigmoid sinus. However, this route is tortuous and would not be supported by the appearance on the original radiograph.

We agree that if a catheter will not advance, it is probably lodged in a small vein and should be withdrawn. Free flow of blood back through the catheter would support its tip being in a large vein, as the case in our report.2 However, the ideal position for central venous catheters should be the right atrium where risk of retrograde flow is less.

Subdural fat effusion complicating parenteral nutrition

Str.,—Rushforth et al recently reported an unusual complication of parenteral nutrition via a Silastic catheter.1 They stated the catheter was probably in the external jugular vein presumably based on a plan of film of the head and neck.

It seems to me that this assumption is unfounded, principally because the catheter would not advance past 9 cm. In my experience this is caused by the catheter wedging in a small vessel. It would thus seem more likely that the catheter had found its way via the deep temporal vein into the anterior diploic vein with free communication to meningeal veins.2 Rupture of these veins would result in a subdural collection.

In my opinion the message of the paper of Rushforth et al should be that if a Silastic catheter will not advance, it is probably wedged in a small vein. The catheter should therefore be withdrawn 1 or 2 cm before readvancing. If this fails the tip should be left one centimetre proximal to the site of obstruction and thus presumably in a larger vein.

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Health for all children

Str.,—The importance of early diagnosis of biliary atresia has been stressed repeatedly by hepatologists and is now acknowledged by those responsible for child health surveillance programmes.1 However, it does seem premature to suggest that we should change the age at which the infant’s first formal postnatal review is performed,2 particularly as the revised programme of child health surveillance recommends that this review should be combined with the first immunisation at the age of 8 weeks.

Each district in the UK will produce one new case of biliary atresia every four or five years. We need to know what strategies would be best to achieve early detection of such a rare condition. Formal screening may be less effective than professional education combined with easy rapid access to the appropriate tests and the expertise to interpret them. The paediatric hepatology team at King’s College Hospital is uniquely well placed to determine what work load would result, and what unforeseen problems might arise, if the various strategies they propose were to be implemented. Our experience with neonatal screening for hearing loss has taught us how much effort and commitment are required to make such programmes work effectively, even when run by an enthusiast.

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Intraosseous infusion

Str.,—The intraosseous route for emergency infusions of fluids and drugs is underutilised in this country and Drs Ryder, Munro, and Doull do well to remind us of its simplicity and efficacy when vascular access is difficult and speed essential.1 However, when discussing the various sites for intraosseous infusions I believe they include the sternum. This site is too hazardous to recommend in my opinion. The upper tibial shaft is safe and favoured as the intraosseous infusion site of first choice by most emergency physicians. The lower end of the femur or humerus are other useful alternatives.

Specially designed intraosseus needles are available and have a shelf life of approximately five years. Their advantage over using hollow shafted needles (for example a large butterfly or needle of large bore intravenous cannula) is that they are less likely to be dislodged by body particles and can more easily be anchored in situ. Their cost is not prohibitive (about £10–£15 each) and it would seem appropriate for all emergency trolleys or boxes to include such needles. One size, age 16, is usable in most situations for all ages of children.

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Familial asplenia

Str.,—We read the article on haemophilia septicaemia in congenital asplenia1 and the subsequent correspondence2 with great interest. We would like to describe our experience, which illustrates the importance of familial asplenia and also a potential disadvantage of not performing a necropsy.

A 21 month old girl, the second child of non-consanguineous white Australian parents, was presented to a peripheral hospital with a short history of fever and delirium. She was profoundly shocked with widespread purpura and ecchymoses. A presumptive diagnosis of meningococcemia was made, resuscitation with artificial ventilation, antibiotics and plasma was commenced, and transfer to this hospital was requested. On arrival here she was moribund and died within minutes. Blood cultures were sterile. Necropsy was requested but declined by the distraught parents.

The parents subsequently had a third child, a boy, who presented at the age of 4 months with fever and a rapid onset of shock and purpura. He was found to have purpura fulminans, with confluent ecchymoses of his distal limbs, ears and nose, and with purpura on his lips. Cultures of blood and cerebrospinal fluid grew Streptococcus pneumoniae. Blood films showed numerous erythrocytes containing Howell-Jolly bodies. An ultrasound scan revealed no spleen and asplenia was confirmed by tomoscopy scan. The child died 1 month later, while the scan was normal. His sister’s original blood film was retrieved from the peripheral hospital and the red cells were also found to contain many Howell-Jolly bodies. The oldest child is well, has a normal peripheral blood film, and has a spleen on ultrasound.

The patient required intensive resuscitation with artificial ventilation, colloid, blood, and
Subdural fat effusion complicating parenteral nutrition.

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