The data presented, in accordance with experimental results on the effects of temperature on breathing,3 suggest that raised respiratory rate may be partly attributable to increases in body temperature. We earlier reported that in children with cough or difficult breathing respiratory rate is a valid predictor of the presence of clinical or radiological pneumonia.2 The findings presented here do not challenge this, but they suggest that this relationship between fever and respiratory rate may account for some of the false positive diagnoses of pneumonia in children with cough or difficult breathing, fever and raised respiratory rate. This issue may be of particular importance in areas in which malaria is endemic, as it has been shown that there may be a substantial overlap of clinical presentation in children with malaria and pneumonia.2 The possible effect of this phenomenon on the specificity of raised respiratory rate as an indicator for pneumonia needs further investigation.

Effects of body temperature on respiratory rate in young children

Sr,.—Dr Simoes and colleagues have reported studies of variability in measurements of respiratory rate in young American children, but did not consider possible effects of body temperature on these measurements.1 Previous work suggested a weak association between body temperature and respiratory rate in young infants studied in Australia and Britain.2 Current World Health Organisation guidelines for the management of acute respiratory infections in children recommend that young children with cough or difficult breathing and raised respiratory rate should be treated for pneumonia irrespective of temperature.

In a community study of acute respiratory infections undertaken at the MRC Laboratories in the Gambia, weekly measurements of temperature and respiratory rate were made on a population including approximately 500 children under the age of 5 years, over a one year period. This study is described in detail elsewhere.3 Although these repeated observations are technically not independent, we consider that measurements of respiratory rate and temperature carried out not more than once weekly on a young child may reasonably be assumed to be independent. A total of 25 025 observations on 685 young children were made. In 70 instances abnormalities on chest radiography were found and these observations have been excluded from the following analysis. The relationships between temperature and respiratory rate for infants (5542 observations) and for children aged 1 to 4 years (19 413 observations), are shown in the figure. In both groups, mean respiratory rate shows a steady increase with increasing temperature of approximately 2.5 min °C over the temperature range shown. A similar analysis restricted to children with cough (2537 observations) showed a similar relationship (data not shown).

Consumer safety and child choking attacks

Sr,.—From time to time you publish letters which do not have any direct relevance to immediate past publications and it would be helpful if such letters indicated their origin. One such letter recently published gives no explanation as to why Drs Matthes, Sibert, and Levene were concerned about possible inhalation of foreign bodies from toys.4 Those paediatricians who help local authority consumer protection departments by assessing or commenting on the safety of toys will be aware that there has been a recent increase in the vigilance of trading standards officers regarding choking hazards to children because of a number of deaths. Dr Levene pointed out that the Child Accident Prevention Trust, which found little published evidence of any serious hazard from the inhalation of ingestion of hair plucked from toys.2 This report is being used by manufacturers to defend their products against legal action even though safer alternative materials are available.

Coroners’ records of accidental deaths

Sr,.—Dr Levene has demonstrated the potential of using coroners’ records as a source of data relevant to child accident prevention studies.1 In a similar retrospective study in this district using the coroner’s records we discovered 69 children aged under 15 years who had died as a result of or in association between the years 1980–9 inclusive. Road traffic accidents represented the commonest fatal accident with falls, drownings, and asphyxia accounting for the remainder. Head injury was the commonest reported cause of death. Most deaths occurred within 2 km of the child’s home while children were playing without supervision. We encountered an association between social class and incidence of accidental deaths with 10 times as many accidents occurring in classes IV and V than in I and II. There was, in addition, a clustering of cases in areas with high deprivation scores. This information was of great use to us in planning local child accident prevention strategy as it enabled us to target limited resources to areas where they were needed most. However, as in Dr Levene’s case, we were made aware of the limitations of using coroner’s records alone for this purpose. We discovered that inquiries relate to deaths occurring to children who died within the boundaries of our district only. During the period of our study we became aware that several local children had died while visiting other districts but this information would not
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’ inquisitions and child health records will yield basic data which might, 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, 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. End 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.


SIR:—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.3

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 propose 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.


Intraosseous infusion

SIR:—The intraosseous route for emergency infusions of fluids and drugs is under-utilised 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 one might 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 intraosseous 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 become bent and broken 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, that is to say 9 or 16, is usable in most situations for all ages of children.

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Familial asplenia

SIR,—We read the article on haemophilus septicaemia in congenital asplenia1 and the subsequent correspondence 2 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, 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 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 tomographic scan. Unfortunately the child 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

Coroner's records of accidental deaths.

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Arch Dis Child 1992 67: 664-665
doi: 10.1136/adc.67.5.664-b

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