ambulance service trust involved. By law the trust was liable for employees involved in accidents while on duty, but nevertheless the recourse to obtain compensation was through negotiation and delays.

As a result of our experiences we have undertaken a comprehensive review of our neonatal transport operations. Initial problems of incubator security have been temporarily addressed with ring bolts set into the floor of ambulances which allow secure anchorage with cargo straps. Incubators are loaded using portable ramps. In the long term, in cooperation with our ambulance service, we have redesigned our transport system with a reduced payload around a no lift platform that will be compatible with all types of stretcher. The ambulance service has also undertaken to modify incubator anchorages to accommodate newer stretcher types with transport incubators on board.

The standards of safety for neonatal transport incubators need to be reviewed. Attention needs to be given to reducing payload, and to ergonomic improvements for loading and securing incubators within ambulances, whether road vehicle or aircraft. The problems of different incubators, ambulances, and stretcher designs can be avoided by using a universal mating platform. Each unit needs to be aware of its responsibilities to its staff regarding liability in the event of an accident.

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Unfriendly incubators

Editor,—Fractures in infants of very low birth weight (<1500 g) not related to birth trauma are being recognised with increasing frequency.1 2 These fractures are usually associated with bone demineralisation, which is frequently present in very low birthweight infants,3 and almost two thirds of them involve the extremities.4 Fractures in these infants can occur as the result of ‘trauma’ during physiotherapy or other procedures such as placement of intravenous lines.4 5

We have observed four infants with birth weights under 1000 g who sustained traumatic fractures of the extremities (one arm in three cases, one leg in the other), and we believe that the contributing factor was accidental trauma of the extremities by or under the plastic tray on which the mattress of the incubator lies. The fractures, diagnosed radiologically between 4 and 6 weeks of age, were caused by signs of intercostal bruising. Two of these babies also had well documented bone demineralisation. Subsequent to these independent observations in Athens and Montreal, we conducted a survey of incubators used in intensive care units and found that in many (even in some of the latest models) there was sufficient gap between the plastic tray and the incubator wall for an arm or a leg of the baby to slip between the incubator’s wall and the plastic tray (figure). In many instances the nursing staff had tried to cover this gap with rolled sheets. In some incubators the tilting mechanism could not be securely locked at the desired tilted position.

Editor,—It is well known that hypoxic ischaemic encephalopathy (HIE) in neonates is associated with adverse neurological outcomes.1 2 The incidence of HIE can be a useful indicator of quality of intrapartum care provided to term infants,3 while the severity relates to subsequent neurological outcomes.2 The Doncaster perinatal service has received much adverse publicity suggesting that their incidence of HIE was excessive (“Trial of labour’, World in Action, Granada Television, 2 Nov 1992). We analysed data from the Trent Regional Neonatal Survey in order to establish if these criticisms were valid.

The Trent Regional Neonatal Survey collects data on all high risk babies admitted to perinatal units within the region. All babies greater than 35 weeks gestation admitted because of HIE were included in this analysis. HIE was diagnosed using modified criteria of Levene et al.2 Grade II HIE was defined as the infant having a history suggestive of asphyxia plus convulsions, while grade III was defined as grade II plus the need for respiratory support. Data was collected by two independent visiting observers. The last three years of complete data were analysed (1 April 1990–30 March 1993).

There were a total of 168 435 live births in 16 perinatal centres (five subregional and 11 smaller units) and 263 babies with HIE; 141 grade II and 122 grade III (table). The incidence of HIE for the region was 1·56 per 1000 live births (range 0·58–5·1 per 1000 live births). The Doncaster unit (number 6 in the table) had an incidence of 1·53 per 1000 live births— that is, similar to the region as a whole. It is clear that criticisms levelled at this perinatal unit were unfounded and based on anecdotal evidence rather than fact. Data relating to HIE is not routinely collected by most perinatal units, however, we believe such data provide a valuable method of auditing perinatal care. Only if the rate of HIE falls outside the ‘normal range’ is concern warranted.

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Excessive rates of asphyxia — accede or fact?

Editor,—It is well known that hypoxic ischaemic encephalopathy (HIE) in neonates is associated with adverse neurological outcomes.1 2 The incidence of HIE can be a useful indicator of quality of intrapartum care provided to term infants,3 while the severity relates to subsequent neurological outcomes.2 The Doncaster perinatal service has received much adverse publicity suggesting that their incidence of HIE was excessive (“Trial of labour’, World in Action, Granada Television, 2 Nov 1992). We analysed data from the Trent Regional Neonatal Survey in order to establish if these criticisms were valid.

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Diagnosis and management of non-immune hydrops in the newborn

Editor,—We read with interest the article by Stephenson et al on the diagnosis of non-immune hydrops in the newborn,1 and we would like to emphasize that a wide range of inborn errors of metabolism (IMD) have been reported to be associated with non-immune hydrops and in many cases a feasible pathogenetic mechanism can be hypothesised.2 As a cause of hydrops they are rare
but collectively they are significant and hence appropriate investigations should be considered. It is particularly important to consider IMD where more than one case of hydrops occurs in the same family or a twin pregnancy is a history of spontaneous abortion or unexplained deaths in close family members. We would like to describe our protocol for the investigation of fetal and neonatal hydrops for IMD, and the relevance of these investigations is considered when the more common causes of hydrops have been excluded.

From the dead fetus a skin biopsy can be taken for IMD and this should be done as soon as possible. The cultured cells can be assayed for β-galactosidase (GM1 gangliosidosis, Morquio’s disease type B, galactosialidosis), β-glucuronidase (Sly’s disease), β-glucosidase (Gaucher’s disease), α-neuraminidase (mucolipidosis I, N-acetylgalactosamine-6-sulfate sulphatase (Morquio’s disease type A), sialic acid (sialic acidaemia) and cholestero esterification (Niemann-Pick C). DNA from the cultured cells can be tested for expanded trinucleotide repeat sequence found in myotonic dystrophy.

In cases where hydrops has been diagnosed antenatally amniotic fluid can be tested for I-cell disease or there is a family history of ichthyosis disorders. Other assays can be undertaken on cultured amniotic fluid cells or chorionic villus tissue (direct or cultured). In the live neonate investigations which can be performed on blood include neuraminidase, plasma lysosomal enzymes, plasma carnitine, iron, and ferritin (neonatal haemochromatosis). A skin biopsy may be preferable if a large number of lysosomal enzyme assays are to be assayed. A random urine sample can be investigated for mucopolysaccharide excretion and oligosaccharide chromatography. Liver tissue may be of value for liver iron quantitation and possibly also for enzyme assays. If liver is to be taken, it should be frozen immediately and stored below −20°C.

While not all these tests will be indicated on every case of hydrops it is important to consider informed consent for investigations as a cause of non-immune hydrops and to collect appropriate specimens for investigations where indicated. Hence discussion with a specialist centre for inherited metabolic disorders is important.

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Do maternal β-sympathomimetics influence the development of retinopathy in the premature infant?

EDITOR.—Recent studies suggest that a number of factors other than the injudicious delivery of oxygen contribute to the development of retinopathy in the premature infant.1 2 One hundred and fifty infants on our neonatal unit who required oxygen treatment for more than 60 days were reviewed retrospectively in order to determine the range of factors contributing to this pathology.

All infants requiring oxygen for more than two weeks were examined at two weekly intervals by an ophthalmic surgeon (ES). Fourteen cases of retinopathy greater than stage 3 were identified. These patients were matched for gestation (+1 week), birth weight (±50 g), sex, and ethnic group with infants who had oxygen requirements for more than 60 days, but did not have retinopathy. The two sets of cases showed no significant differences in their Apgar scores, requirement for exchange transfusions, or the incidence of clinically significant patent ductus arteriosus.

Mothers of eight of the retinopathy group had received infusions of β-sympathomimetics (seven ritodrine, one salbutamol) to arrest premature labour: only one of the non-retinopathy group received ritodrine (p<0.008). This observation may be of physiological importance, as animal experiments have suggested that the retinal circulation is strongly influenced by these pharmacological agents acting jointly through the sympathetic nervous system and endothelial factors.3 β-Sympathomimetics are known to induce transient ischaemic changes in the myocardium of the newborn if given over long periods by intravenous infusion.4 Maternal ritodrine has been associated with reduced blood pressure in the newborn.5 6 In the premature infant in whom antenatal retinal perfusion is influenced by β-sympathomimetics, the retina may be at increased risk of retinal ischaemia and predispose to retinopathy. This observation merits review in a larger series of infants. Further, it casts doubt as to the safety of β-sympathomimetics which are widely used in the therapy of premature labour with little evidence to support their efficacy.

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Immunisation and C reactive protein in infants on neonatal intensive care units

EDITOR.—C reactive protein estimation is a well known indirect method of detecting bacterial infection in neonates.1 Premature infants on our neonatal unit receive their primary course of immunisation for diptheria, pertussis, tetanus and Haemophilus influenzae as per the immunisation schedule at two, three, and four months postnataally.2 Plasma C reactive protein estimation is performed as part of a weekly or daily infection surveillance. Serial estimations are continued in infants with an increased concentration (normal <4 mg/l). We noticed, as an incidental finding, that values increased within 24 hours of immunisation.

During a period of three months (November 1993 to January 1994) 12 babies had C reactive protein estimations before and after immunisation. The median value preimmunisation was 0.8 mg/l (range 0.4–2.6) and postimmunisation the median value was 0.8 mg/l (range 0.8–4.0). The median duration of increased C reactive protein was two days. During the initial period of the study one of the babies had a full infection screen based on the raised C reactive protein and clinical findings (systemically unwell postimmunisation).

Plasma C reactive protein is known to increase after immunisation with diphtheria, pertussis, and tetanus vaccines in malnourished children after nutritional recovery.3 To the best of our knowledge it has not been reported in premature infants who have been immunised. It is important to realise that immunisation leads to an increase in the circulating reactive protein concentrations so that unnecessary investigations may be avoided.

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Diagnosis and management of non-immune hydrops in the newborn

EDITOR.—The article by Stephenson and colleagues concerning the diagnosis and management of non-immune fetal hydrops was both informative and timely. The number of reported associations was particularly thorough.1 I would like to make a minor point regarding the inclusion of asplenia syndrome among the cardiovascular associations. Asplenia syndrome (right atrial isomerism) is characterised by complex cardiac defects including primitive atrioventricular septal defects and abnormal atrioventricular connections, many of which are listed in Stephenson’s table 1. Nevertheless, it is far more common to see polysplenia syndrome (left atrial isomerism) in association with fetal hydrops.2 3 Left atrial isomerism is well associated with primitive atrioventricular block, as well as other structural cardiac lesions.3 Complete heart block can occur, rarely, in right atrial isomerism, as in one case in Schmidt’s study.