LETTERS TO THE EDITOR

Providing medical care for children

SIR,—David Hull has produced a masterly overview of the problems facing the medical services for children in this country.¹ Throughout his article he clearly promotes the principles of partnership with parents and cooperation between professionals involved in the care of children which underpin the Children Act 1989. We are therefore concerned that his comments concerning the concept of 'children in need' as found in the Children Act might lead to some misunderstanding among paediatricians. The act states 'a child shall be taken to be in need if: (a) he is unlikely to achieve or maintain . . . a reasonable standard of health or development without the provision for him of services by a local authority, (b) his health or development is likely to be significantly impaired . . . without such services OR (not and) (c) he is disabled'.

Thus all disabled children are by definition children in need within the terms of the act, whatever the ability of their parents and entitled to the services provided under part III and schedule 2. All will be eligible for inclusion on the register of disabled children that the local authority will be obliged to set up.

Assessment of all children in need will turn on the child's health, development or disability, and need for services and not on a professional judgment of their parents' abilities, although in many cases they may be a factor. One of the many challenges of the Children Act will be to avoid stigmatisation of families with a child in need who use local authority services. Paediatricians and other child care professionals, if they are effectively to help local authorities identify children in need and encourage the use of services, must have a clear understanding of the concepts within the Children Act. This understanding will also empower professionals in partnership with parents to campaign for the resources local authorities will need if they are to fulfil successfully their obligations under the act to children in need.

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1 Hull D. Providing medical care for children. *Arch Dis Child* 1991;66:643-6.

Life threatening 'epilepsy'

SIR,—In an earlier issue of the journal, Drs Brown and Godman reported a 5 year old girl with spells of unconsciousness which later proved to be caused by ventricular tachycardia and fibrillation.¹ Confusion in diagnosis

occurred at first, leading to the presumption of epilepsy as the cause of the 'spells'. This misdiagnosis was based on several medical history items: (i) possible fetal distress preceding the child's birth, (ii) questionable delayed motor development and hypotonia, and (iii) a family history of a cousin with spells caused by ketotic hypoglycaemia. These historical factors suggested that the patient's spells were likely epileptiform. Later complete cardiological evaluation proved that the loss of consciousness was not epileptiform, but rather cardiogenic in origin.

I would like to illustrate further the diagnostic difficulty which can be encountered with long Q-T syndrome in paediatric patients. I cared for an adolescent girl with spells. When she came to see me she had been tentatively diagnosed as having epilepsy and was already receiving phenytoin for that diagnosis. Her spells disappeared during phenytoin treatment. For a variety of reasons I chose to wean and then discontinue the phenytoin. After this had been completed the girl began having more spells of loss of consciousness. At that point I obtained an electrocardiogram (ECG) which demonstrated a long Q-T interval. Holter monitoring confirmed ventricular ectopy, consistent with the diagnosis of long Q-T syndrome.

Presumably phenytoin, a class Ib anti-arrhythmic agent and an antiepileptic agent, was therapeutic for the patient's problems, despite the fact that the diagnosis under consideration was in error.

The vast number of spells in the paediatric age group are probably benign and due to either breath holding in infancy or situational causes of syncope or epilepsy. Despite this, the relatively rare child with a cardiac conduction defect accounting for her spells certainly exists and her spells are certainly life threatening. Their diagnosis often depends on the consultant first seeing them. If the child described above had first seen a paediatric cardiologist, I am quite sure her long Q-T syndrome would have been promptly diagnosed. On the other hand, as a paediatric neurologist first seeing her, I reasonably first considered a diagnosis of seizure disorder.

I do not agree with Garson who in effect states that all new presumed epilepsy patients should have an ECG because of the risk of the spell being of cardiac origin.² In fact, I do not think that every new presumed epilepsy patient needs an ECG. Patients presenting with spells are best benefited by the physician thoughtfully considering the broad diagnostic differential of the common paediatric complaint of spells, whether they be due to neurological, cardiological, psychiatric, or metabolic causes.

Neither the patient of Brown and Godman patient nor my patient was harmed by delayed diagnosis. I wish only to add yet one more confounding factor which may possibly be encountered in the care of children with spells.

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1 Brown DC, Godman MJ. Life threatening 'epilepsy'. *Arch Dis Child* 1991;66:986-7.
2 Garson A. Medicolegal problems in the management of cardiac arrhythmias in children. *Pediatrics* 1987;79:84-8.

Accidents on hospital wards

SIR,—Levene and Bonfield have emphasised the problem of accidents occurring to children on hospital wards and have also demonstrated how information, which previously had been collected solely for medicolegal purposes, could be used for preventative work.¹ A similar case could be made for studying accidents to children in health centres and baby clinics.

By inspecting incident log books maintained by our community unit, it was discovered that for the period 1989-90 inclusive, 17 preschool children in this district were reported as having had accidents while on community health premises.

The commonest reported incident was head injury sustained as a result of a fall; four infants fell from changing mats during weighing sessions and 10 children fell while playing in waiting rooms. Two children had hand injuries due to trapped fingers in swinging doors and one child ingested the boric acid crystals contained in a urine container.

Although none of these incidents resulted in serious injury, the potential for more serious outcome was present. In particular, the importance of injuries to the head as a major cause of childhood mortality and morbidity should be remembered.² In retrospect most of the accidents could have been avoided by greater supervision and some modification of clinic premises. In some respects, health centres may be more hazardous to children than hospital wards as they are busy places and supervision of young children is not easy especially in overcrowded waiting rooms. Nevertheless, after our investigation we have endeavoured to increase awareness of child safety among clinic staff and have urged particular caution with regard to the supervision of babies while they are on changing mats.

Child accident prevention should be a key issue for every health authority. By ensuring that children are as safe as is reasonably possible while on health authority premises, we are setting a good example for parents and are ensuring that opportunities for health promotion are not missed.

In addition, it should be remembered that children also have accidents on premises maintained by social services, local education authorities, and borough councils. Perhaps collation of accident data kept by these authorities with appropriate intervention should also form part of every district's child accident prevention strategy.

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1 Levene S, Bonfield G. Accidents on hospital wards. *Arch Dis Child* 1991;66:1047-9.
2 Sharples PM, Storey A, Aynsley-Green A, Eyre JA. Causes of fatal childhood accidents involving head injury in Northern region, 1979-1986. *BMJ* 1990;301:1193-6.

Hazard from the exposed terminals of a battery driven transport incubator

SIR,—In common with most regional intensive care units we offer a retrieval service, collecting

about 75 babies annually. Recently an experienced team were collecting an extremely sick baby with pulmonary hypoplasia who required maximal support and monitoring. The baby was transferred to the transport incubator before departure and a pulse oximeter was inadvertently placed on top of the 12 volt car battery used to power the incubator. Sparks and flames ensued, which were extinguished by a quick thinking senior house officer using a carbon dioxide fire extinguisher. The accident would obviously have been much worse if the team had left the hospital.

On close inspection afterwards the metal terminals of the battery were found to be exposed (figure). Current had passed through the casing of the pulse oximeter which still worked. The plastic coverings originally supplied with the battery had long since been mislaid. We report this incident as we feel that other units with old transport systems may also have lost the battery coverings, and suggest that the batteries used to power incubators should be completely encased. Consideration might also be given to inclusion of a fire extinguisher in the flying squad equipment.

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Exposed terminals and damage to battery.

Treating neonatal jaundice with phenobarbitone: the inadvertent administration of significant doses of ethyl alcohol

SIR,—One indication for the administration of phenobarbitone is neonatal jaundice: the drug acts on the immature liver to induce microsomal enzymes and may also have a direct protective effect on the central nervous system.

Phenobarbitone can be given orally as an elixir. The hospital pharmacy will prepare this, on request, as an aqueous solution. In this form, the elixir keeps poorly and cannot be stored on the ward. Stock phenobarbitone elixir on the ward, however, is made with ethyl alcohol to prolong its shelf life. Elixirs in this form are purchased directly from the drug companies and are 40% ethyl alcohol by volume.¹

Regular dosing with the alcoholic elixir can result in neonatal alcohol toxicity. It should not be taken for granted that the elixir will be aqueous. We would therefore advise that the solvent is also specified on the drug chart when prescribing oral phenobarbitone elixir for neonatal jaundice.

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1 *The EBL guide*. Aylesbury: Edwin Burgess, 1988:178.

Dr Leach comments:

Drs Colquhoun-Flannery and Wheeler are perfectly correct in saying that elixir phenobarbitone contains high amounts of alcohol: the current preparation included in the *British National Formulary* (September 1991) contains 'phenobarbitone 15 mg/5 ml in a suitable flavoured vehicle, containing alcohol 38%'. The current edition of *The Extra Pharmacopoeia* (29th edition, 1989) includes phenobarbitone elixir BP (phenobarbitone oral solution) and states that it is a solution containing phenobarbitone 0.3% and alcohol 38%—that is, the same formulation as the elixir in the *British National Formulary*.

Phenobarbitone has only a low solubility in water (1 in 1000) but is soluble in alcohol (1 in 10). It is therefore necessary to include a high proportion of alcohol in the formulation to achieve adequate solution of the phenobarbitone. An aqueous preparation would require the use of phenobarbitone sodium which is water soluble.

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Parenteral lipids and free radicals in preterm infants

SIR,—Professor Cooke describes an interesting association between the use of parenteral lipids and chronic lung disease in preterm infants.¹ The study nicely confirms and extends the results of Hammerman and Aramburo.² The author mentions lipid peroxidation and generation of free radicals in the lipid solution as one possible mechanism by which parenteral lipid solutions may injure preterm infants.¹ We should like to draw your readers' attention to the following: free radical induced lipid peroxidation in parenteral lipid solution has actually been described both in vitro and in vivo upon infusion in preterm infants.^{3,4} Although the article referred to by the author does not concern lipid peroxidation and free radicals,⁵ we agree with Professor Cooke's conclusion that the advantages of parenteral lipid infusion should be carefully weighed against its potential for harm. The possibility of such adverse effects should not be ignored, particularly when using parenteral nutrition in small premature infants. Work with these patients indicates a close association between free radical induced lipid peroxidation and chronic lung disease.⁶

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1 Cooke RWI. Factors associated with chronic lung disease in preterm infants. *Arch Dis Child* 1991;66:776-9.

2 Hammerman C, Aramburo MJ. Decreased lipid intake reduces morbidity in sick premature infants. *J Pediatr* 1988;113:1083-8.

3 Whispe JR, Bell EF, Roberts RJ. Assessment of lipid peroxidation in newborn infants and rabbits by measurements of expired ethane and pentane: influence of parenteral lipid infusion. *Pediatr Res* 1985;19:374-9.

4 Pitkanen O, Hallman M, Andersson S. Generation of free radicals in lipid emulsion used in parenteral nutrition. *Pediatr Res* 1991;29:56-9.

5 Pereira GR, Fox WW, Stanley XA, Baker L, Schwartz JG. Decreased oxygenation and hyperlipemia during intravenous fat infusions in premature infants. *Pediatrics* 1980;66:26-30.

6 Pitkanen OM, Hallman M, Andersson SM. Correlation of free oxygen radical-induced lipid peroxidation with outcome in very low birth weight infants. *J Pediatr* 1990;116:760-4.

Antenatal diagnosis of inborn errors of metabolism

SIR,—The authors of this paper state that they are unaware that prenatal diagnosis has been attempted for hyperlysinaemia.¹ After the diagnosis of a previously affected child such an attempt has been made in a subsequent pregnancy.² The presence of an unaffected fetus was demonstrated by normal amniotic fluid amino acid analysis and normal oxidation of lysine by cultured amniotic fluid cells. Thereafter, the parents refused further attempts at antenatal diagnosis and three affected infants were born, the management of whom has been reported in this journal.³

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1 Cleary MA, Wraith JE. Antenatal diagnosis of inborn errors of metabolism. *Arch Dis Child* 1991;66:816-22.

2 Gray RGF, Bennett MJ, Green A, et al. Studies on a case of persistent hyperlysinaemia with a possible method for prenatal diagnosis. *J Inherited Metab Dis* 1983;6(suppl 2):115-6.

3 Gregory JW, Beail N, Boyle NA, Dobrowski C, Jackson P. Dietary treatment of hyperlysinaemia. *Arch Dis Child* 1989;64:716-20.

Transient gluten intolerance

SIR,—Burgin-Wolff *et al* report that 11% of children (15/134) who were originally diagnosed as having coeliac disease, with a flat small intestinal mucosa and clinical response to a gluten free diet, had a normal small intestinal mucosa after four to 15 years of regular gluten challenge.¹ Thus there had been no histological or clinical relapse after this interval making it possible that the original diagnosis in these children was transient gluten intolerance rather than coeliac disease. None the less Polanco and Larrauri have reported five children with coeliac disease who took up to nine years to relapse after gluten challenge having been shown to have normal small intestinal mucosal biopsies four to five years after gluten challenge.² Thus some of the children reported by Burgin-Wolff *et al* may ultimately relapse. Nevertheless it is of great importance that all 15 children who have not relapsed were aged less than 2 years at initial diagnosis. This finding provides firm data to support the revised European Society for Paediatric Gastroenterology and Nutrition (ESPGAN) criteria for coeliac disease³ recommendation that children originally diagnosed as coeliac disease under the age of 2 years should always have the diagnosis established by means of serial biopsies coupled with gluten elimination and challenge as originally recommended.⁴ No child over this age failed to relapse in the study of Burgin-Wolff *et al*.

What still remains unknown is the gamma-delta status of the intraepithelial lymphocytes of biopsy specimens from children diagnosed as having transient gluten intolerance.