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.

JANET M RENNIE
ED BROADHURST
Rosie Maternity Hospital,
Robinson Way,
Cambridge CB2 2SW

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.

W COLQUHOUN-FLANNERY
R WHEELER
Wessex Regional Centre
for Paediatric Surgery,
Southampton General Hospital,
East Wing, Shirley,
Southampton SO9 4XY

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.

FRANK LEACH
North Western Regional Health Authority,
Regional Drug Information Service

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

STURE ANDERSSON
OLLI PITKANEN
Children's Hospital,
00290 Helsinki, Finland

MIKKO HALLMAN
University of California,
Irvine, CA 92668, USA

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

J W GREGORY
Department of Child Health,
The Medical School,
Framlington Place,
Newcastle upon Tyne NE2 4HH

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.

T lymphocytes expressing gamma-delta are invariably increased in the gut epithelium in coeliac disease and occasionally in other enteropathies, but only in infancy.⁵ It is not possible at present to use monoclonal antibodies for these markers on paraffin sections so old biopsy specimen in paraffin blocks cannot be used. So it will thus take some years to determine this point.

Finally it is disappointing that the authors¹ use the term transient coeliac disease rather than transient gluten intolerance as recommended by ESPGAN since 1970.⁴ Coeliac disease, although perhaps expressing itself in different ways in the small intestinal mucosa at different times of life, is by definition a permanent lifetime disorder.

J A WALKER-SMITH
Academic Department of Paediatric
Gastroenterology,
Queen Elizabeth Hospital for
Children,
Hackney Road,
London E2 8PS

- 1 Burgin-Wolff A, Gaze H, Hadziselimovic F, et al. Antigliadin and antiendomysium antibody determination for coeliac disease. *Arch Dis Child* 1991;66:941-8.
- 2 Polanco I, Larrauri J. Does transient gluten intolerance exist? In: Kumar J, Walker-Smith JA, eds. *Coeliac disease: 100 years*. Leeds: University of Leeds Printing Service, 1990: 226-8.
- 3 Walker-Smith JA, Guandalini S, Schmitz J, Schmerling DH, Visakorpi JK. Revised criteria for diagnosis of coeliac disease. *Arch Dis Child* 1990;65:909-11.
- 4 Meeuwisse GW. Diagnostic criteria in coeliac disease. *Acta Paediatr Scand* 1970;59:461.
- 5 Spencer J, Isaacson PG, MacDonald TT, Thomas AJ, Walker-Smith JA. Gamma/delta T cells and the diagnosis of coeliac disease. *Clin Exp Immunol* 1991;85:109-13.

Long term survival after heart transplantation for doxorubicin induced cardiomyopathy

SIR,—We read with interest the case report of the child with severe cardiomyopathy requiring cardiac transplantation after the administration of doxorubicin.¹ We wish to emphasise the increasing problem of anthracycline induced cardiomyopathy not only in the short term, as is the reported patient, but also many years after its use.

Case report

This girl presented at the age of 4·8 years with a Wilms' tumour. She initially underwent laparotomy and removal of the tumour and was found to have stage III disease. Chemotherapy with vincristine, actinomycin D, and doxorubicin (Adriamycin) was commenced. She went on to receive radiotherapy in a dose of 2000 cGy to the renal bed; the field did not include the heart. Chemotherapy was continued with cycles of three weeks for a further 11 months; she received a total dose of 360 mg/m² of doxorubicin.

She remained well until December 1990, when at the age of 15·3 years she presented with a three week history of malaise and lethargy with abdominal pain and vomiting. Four days before her admission she developed symptoms of breathlessness, orthopnoea, and weight gain. She was found to have severe congestive cardiac failure confirmed on echocardiography, which also revealed an extensive mural thrombus in the left ventricle.

Despite an initial response to conventional treatment with diuretics and angiotensin converting enzyme inhibitors, her myocardial function remained poor and she underwent orthotopic cardiac transplantation in March 1991. She remains well.

The acute cardiac toxicity of anthracyclines is well recognised with the recent reports of doses as low as 40 mg/m² causing some degree of cardiac dysfunction.² We are now seeing late toxicity with increasing frequency and it is vital that these children remain on regular follow up, and that there is good liaison with adult physicians who also require access to the patient's records and details of chemotherapy received.

With the improved survival of many of the childhood malignancies, it is essential to consider whether the more frequent use of anthracyclines is justified.

M E M JENNEY
P H MORRIS JONES
Department of Paediatric Oncology,
Royal Manchester Children's Hospital,
Pendlebury,
Manchester M27 1HA

- 1 Arico M, Pedroni E, Nespoli I, Vigano M, Porta F, Burgio GR. Long term survival after heart transplantation for doxorubicin induced cardiomyopathy. *Arch Dis Child* 1991;66: 985-6.
- 2 Lipshultz S, Colan S, Gelber R, et al. Late cardiac effects of daunorubicin therapy for acute lymphoblastic leukaemia in childhood. *N Engl J Med* 1991;324:808-15.

Aristotle, Francois Mauriceau, and plagiarism

SIR,—I write to defend a venerable colleague against the charges of plagiarism raised against him by Professor Dunn in January issue of this journal.¹ I refer, of course, to Francois Mauriceau (1637-1709), who was accused by Professor Dunn of having 'lifted' his recommendation of the dorsal recumbent delivery position from an ancient obstetrical treatise by Aristotle, which was cited in an 18th century translation called *Aristotle's Experienced Midwife*.

There are, however, no genuine extant obstetrical treatises written by Aristotle. On the contrary, towards the end of the 17th century there began to appear a number of anonymous medical and sexual 'self help'

books or 'marriage manuals' attributed to Aristotle. They were reprinted extensively throughout the 18th and 19th centuries and even into the 20th century. While they have their roots in medieval and renaissance lore, they freely plagiarised from 'contemporary' medical works at the time in which they were written. Among the most notable of these works are *Aristotle's Compleat Masterpiece in Three Parts, Displaying the Secrets of Nature in the Generation of Man*; *Aristotle's Book of Problems . . . Touching the State of Man's Body*; *Aristotle's Last Legacy Unfolding the Mysteries of Nature in the Generation of Man*; and Professor Dunn's 'Aristotleian' treatise, *Aristotle's Compleat and Experienced Midwife*.² This source dates from the mid-18th century and was published well after Hugh Chamberlen's English translation of Mauriceau's work on obstetrics in 1673.

The similarities in style are best explained by the anonymous 'Aristotle' lifting his text from Chamberlen's translation of Mauriceau, not the other way around. Rather than being the culprit, Mauriceau is the victim in this instance. Professor Dunn owes him an apology!

L LEWIS WALL
Section of Gynecology and Obstetrics,
Emory University School of Medicine,
Atlanta, Georgia, 30322, USA

- 1 Dunn PM. Francois Mauriceau (1637-1709) and maternal posture for parturition. *Arch Dis Child* 1991;66:78-9.
- 2 Bouce PG. Some sexual beliefs and myths in eighteenth-century Britain. In: Bouce PG, ed. *Sexuality in eighteenth-century Britain*. Manchester: Manchester University Press, 1982:28-46.

Professor Dunn comments:

Plagiarism, or the passing off of the ideas of another as one's own, is something we all do to a greater or lesser extent. As Wilson Mizner has pointed out: 'If you steal from one person it's plagiarism; if you steal from many, it's research'! Dr Wall's speculation that Mauriceau was himself plagiarised by the anonymous 18th century author or editor of *Aristotle's Compleat and Experienced Midwife*, rather than that he plagiarised Aristotle as I suggested in my article, is of considerable interest. If he is correct, as he may well be, then I certainly owe Mauriceau an apology. Even more so I would need to apologise to the reputation of Aristotle for incorrectly asserting that he, alone among the classical workers, had advocated that women adopt the unphysiological dorsal position for delivery. If I were Dr Wall's 'venerable colleague' I would much prefer to be found guilty of plagiarism than of being responsible for introducing into Western obstetrics a practice which has made childbirth more painful, more difficult, and more dangerous for both mother and child.