# LETTERS TO THE EDITOR

## Pressures used to flush central venous catheters

EDITOR,—It is common practice to flush a central venous catheter (CVC) manually when occlusion is suspected. The manufacturers of one silastic CVC commonly used in neonatal units recommend a maximum flushing pressure of 1.2 bars. (Vygon GmbH & Co KG. Epicutaneo-cave-catheter product insert; 1994.) Excessive pressure may lead to CVC rupture with associated infection, or to avulsion of the distal catheter. We set out to assess whether our current practices are safe. In vitro studies showed that the typical burst pressure of 4 CVCs was between 5.2 and 7 bars.

We then asked 22 doctors and 14 nurses to flush an occluded, fluid filled intravenous pressure line attached to a manometer. Each participant was asked to exert the maximum pressure which they would apply when flushing a CVC. The first 20 subjects were asked to flush using 2.5 ml and 5 ml syringes, and the remaining 16 subjects used only 2.5 ml syringes. Two attempts were made with each syringe, and the higher reading was analysed. Results were expressed in geometric mean (95% CI).

We found that:

(i) maximum pressures were significantly lower using a 5 ml than a 2.5 ml syringe, being 0.76 (0.56, 1.01) bars and 1.0 (0.71, 1.41) bars, respectively ($p<0.01$).
(ii) doctors exerted significantly higher pressures than nurses ($p<0.05$).
(iii) for the 22 doctors using a 2.5 ml syringe (the normal situation on our unit) the 97th centile for maximum flushing pressure was 12.2 bars. Fifteen (68%) exceeded the recommended maximum pressure and four (18%) exceeded the likely burst pressure.

Smaller syringes exert higher output pressures. A survey of American CVC product leaflets reported that nearly all recommend a 10 ml syringe as the minimum size for flushing.[1] Although there would be a reduced risk of rupture if 5 ml rather than 2.5 ml syringes were used, we recommend that training with a manometer should be given to all staff who are likely to flush CVCs. We also suggest that British product leaflets should emphasise the hazards of using smaller syringes to flush CVCs.

R H PRIMHAK
*University Department of Paediatrics*
*Sheffield Children's Hospital*
*Western Bank*
*Sheffield S10 2TH*

N GATHERCOLE
H REITER
*School of Materials Science*
*University of Bath*

1 Conn C. The importance of syringe size when using implanted vascular access devices. *Journal of Vascular Access Nursing* 1993;**3**:11-18.

## Insertion of umbilical venous catheters past the ductus venosus using the double catheter technique

EDITOR,—We describe the use of the double catheter technique for umbilical venous catheterisation. Such a technique has been described before for the cannulation of the umbilical arteries,[1] but to our knowledge has not been described for umbilical venous catheter (UVC) insertion.

One of the major problems with insertion of UVCs is failure of the catheter to negotiate the ductus venosus, thereby preventing it traversing the inferior vena cava (IVC).[2 3] If this occurs the double catheter technique can be used as follows. After the UVC is inserted in the standard way and an *x*-ray picture shows the catheter tip has lodged either in the portal vein (or tributaries) or the left or right hepatic portal veins (or branches), then a second catheter is inserted in the UV whilst the first remains in situ. The first catheter is then withdrawn and the second fixed in place. An *x*-ray picture is then repeated.

We used the double catheter technique on two occasions in 1996: in a 4350 g term baby with group B streptococcus sepsis. The first UVC lodged in the liver. A second catheter placed down the side of the first resulted in successful negotiation of the ductus venosus and right atrium. The second occasion was in a shocked 31 week gestational age infant. The first UVC was seen lodged in the portal vein, and the second UVC passed through the ductus venosus and IVC to end up in the right atrium. There were no known complications of the procedure in either case.

Our explanation for the success of this technique is as follows. The UV ends in the left hepatic portal vein opposite the entrance to the ductus venosus.[2 4] Failure of the catheter to enter the ductus venosus occurs because, firstly it is narrowest at its origin, and functional closure occurs here soon after birth, and secondly the ductus venosus inlet may not be aligned on the opposite side of the left hepatic portal vein.[2] If the catheter fails to enter the ductus venosus it will then enter the left hepatic portal vein and either become lodged in the liver or in the portal vein (or its tributaries). The first catheter takes the course of least resistance and blocks this undesirable route. The second catheter inserted down the side of the first then has a far greater chance of entering the ductus venosus to continue on to its more desirable location.

We believe this technique is useful for those of us who provide neonatal intensive care. While the adverse effects of this technique remain largely unstudied, it would be prudent to use it only in those infants in whom an umbilical venous catheter is absolutely necessary.

M W DAVIES
D W CARTWRIGHT
*Grantley Stable Neonatal Unit*
*Royal Women's Hospital*
*Herston, Brisbane*
*Queensland, Australia, 4029*

1 Schreiber MD, Perez CA, Kitterman JA. A double-catheter technique for caudally misdirected umbilical arterial catheters. *J Pediatr* 1984;**104**:768-9.
2 Rosen MS, Reich SB. Umbilical venous catheterization in the newborn: Identification of correct positioning. *Radiology* 1970;**95**:335-40.
3 Seguin J, Fletcher MA, Landers S, Brown D, Macpherson T. Umbilical venous catheterizations: Audit by the study group for complications of perinatal care. *Am J Perinatol* 1994;**11**:67-70.
4 Sanders CF. The placement of the umbilical venous catheter in the newborn and its relationship to the anatomy of the umbilical vein, ductus venosus and portal venous system. *Clin Radiol* 1978;**29**:303-8.

## High concentrations of GRO-α and MCP-1 in bronchoalveolar fluid of infants with respiratory distress syndrome after surfactant

EDITOR,—Recent work has related the severity of lung damage after neonatal respiratory distress syndrome (RDS) to the early inflammatory response and the degree of matrix degradation.[1 2] Although treatment with exogenous surfactant reduces the incidence of bronchopulmonary dysplasia (BPD), it is not known whether it attenuates this inflammatory response.

This response has largely been investigated by looking at cells, cytokines, and chemokines in the bronchoalveolar lavage fluid (BAL) of ventilated infants. Increased concentrations of the macrophage cytokines tumour necrosis factor α and interleukin-1β, the CC chemokine MIP-1α and the CXC chemokine IL-8 have all been detected in the BAL fluid of infants who subsequently develop BPD.[1 3] Treatment with dexamethasone decreases inflammatory cell numbers and the concentrations of pro-inflammatory mediators, as well as improving clinical outcome, although at considerable metabolic cost.[1 3]

The CC chemokine MCP-1 and the CXC chemokine GRO-α have not been investigated in RDS. They may both have a central role in pulmonary inflammation and they have been implicated in the pathogenesis of experimental pulmonary fibrosis and adult fibrotic disease.[4 5] We measured these chemokines in the BAL fluid of 15 infants with RDS (median birthweight 1001 g, range 700–1340 g; gestation 27 weeks, range 25–31 weeks), who had been treated with early exogenous surfactant. BAL was performed in the first six days of life, after surfactant treatment, as described before.[3] Of the 15 infants, 10 had been treated with porcine surfactant (Curosurf, Serono) and five had been treated with synthetic surfactant (Exosurf, Wellcome) None had received postnatal steroids at the time of study. Ten of the infants subsequently became oxygen dependent at 36 weeks of equivalent gestation, fulfilling current criteria for the diagnosis of BPD.

The median concentration of MCP-1 was 14.72 ng/ml (95% CI 10.83–19.08) and of GRO-α was 4.32 ng/ml (2.80–6.91). These concentrations are far in excess of those found in any previous study of pulmonary inflammation.[4 5] Our results suggest that the intense early pulmonary inflammatory response in RDS is not attenuated effectively by surfactant treatment, and they confirm that four potent chemokines are produced in high concentration in this condition. Work in animal models has shown that pre-treatment with cytokine antagonists can prevent experimental pulmonary fibrosis, whereas subsequent treatment is ineffective. RDS is probably the only human inflammatory condition in which such pretreatment is possible. We suggest that the time has come for similar studies of cytokine antagonism in infants ventilated for RDS.

DAVID P INWALD
KATE COSTELOE
SIMON H MURCH
*Garden House Children's Unit*
*Royal London Hospital*
*Whitechapel Road*
*E1 1BB*

1 Groneck P, Speer CP. Inflammatory mediators and bronchopulmonary dysplasia. *Arch Dis Child* 1995;**73**:F1–F3.

2 Murch SH, Costeloe K, Klein NJ, *et al*. Mucosal TNF-α production and extensive disruption of sulfated glycosaminoglycans begin within hours of birth in neonatal respiratory distress syndrome. *Pediatr Res* 1996;**40**:484–9.
3 Murch SH, Costeloe K, Klein NJ, *et al*. Early production of MIP 1 α occurs in RDS and is associated with poor outcome. *Pediatr Res* 1996;**40**:490–7.
4 Smith RE, Stieter RM, Zhang K, *et al*. A role for C-C chemokines in fibrotic lung disease. *J Leuk Biol* 1995;**57**:782–7.
5 Villard J, Dayer-pastore F, Hamacher J, *et al*. GRO-α and IL-8 in Pneumocystis carinii pneumonia or bacterial pneumonia and adult respiratory distress syndrome. *Am J Respir Crit Care Med* 1995;**152**(5 pt 1): 1549–54.

### 677→CT mutation on the methylenetetrahydrofolate reductase gene is not a risk factor for neural tube defects in Turkey

EDITOR,—Recent studies have shown that periconceptual folic acid supplementation reduces a woman's risk of having a baby with neural tube defects (NTD). Mothers of infants with NTD have increased homocysteine activities. People with a thermolabile form of the enzyme 5,10 methylenetetrahydrofolate reductase (MTHFR) have reduced enzyme activity and increased plasma homocysteine which can be lowered by supplemental folic acid. Thermolability of the enzyme is caused by a common mutation (677→CT) in the MTHFR gene. In different populations the 677→CT mutation has been implicated in susceptibility to NTD.[1] We studied the 677→CT mutation as a risk factor for spine bifida in a group of Turkish patients with NTD, and in their parents.

Blood for mutation analysis was obtained after written informed consent from cases with NTDs, and their parents. The study protocol was approved by the ethics committee of Hacettepe University Faculty of Medicine. The study population comprised 49 subjects with NTDs, 40 mothers, and 33 fathers. The control group consisted of 93 healthy adults of Turkish origin.

We genotyped blood samples using the polymerase chain reaction and allele specific restriction digestion, according to the method described by Frosst *et al*.[2] Our data showed no evidence for an association between the 677→Thomozygote genotype and the occurrence of NTD (table1). The homozygote TT prevalences were, respectively, 8.2% for NTD cases, and 7.5% for controls (OR 0.916, 95% CI 0.255–3.294; p=0.893). The TT genotype was more rare among the parents (12.3%, n=9) of NTD cases than among the control group (OR 1.728, 95% CIl 0.611–4.885; p=0.298).

The prevalence of the 677→T allele differs among different populations. It has been noticed that the prevalence is relatively low in controls in those countries where the MTHFR polymorphism has been implicated in susceptibility to NTD.[3] Although the prevalence of the 677→T allele in the Turkish control group (0.28) is very close to that of Dutch and Irish populations,[1] where the MTHFR mutation has been implicated in a predisposition to NTD, we found no evidence for the association between the MTHFR mutation and NTD in the Turkish population studied.

It has been suggested that 677→CT mutation might not be responsible for a large percentage of folic acid preventable NTD cases[1] as there are methodological problems in the studies indicating the association between the mutation and NTD. As the studies implicating [1 4] and refuting[3 5] 677→CT as a risk factor for NTD used almost identical methods, it is unlikely that the different results in different populations were due to methodological issues. We suggest that the different results reflect the real genetic variation between the populations.

The results of this study indicate that 677→CT mutation is not responsible for NTD in Turkish patients. Further investigation is needed to elucidate the role of other mutations in either MTHFR or other folate related enzyme genes, which might be responsible for NTD.

This study is supported by The Scientific and Technical Research Council of Turkey with a grant encoded "SBAG-1693".

KORAY BODUROGLU
MEHMET ALIKASIFOGLU
BURCU ANAR
ERGUL TUNCBILEK
*Hacettepe University*
*Department of Pediatrics*
*Clinical Genetics Unit*
*06100 Samanpazari*
*Ankara*
*Turkey*

1 Posey DL, Khoury MJ, Mulinare J, Adams MJ, Ou CY. Is mutated MTHFR a risk factor for neural tube defects? *Lancet* 1996;**347**:686-7.
2 Frosst P, Blom HJ, Milos R, *et al*. A candidate genetic risk factor for vascular disease: a common mutation in methylenetetrahydrofolate reductase. *Nature Genet* 1995;**10**:111-13.
3 Papapetrou C, Lynch SA, Bum J, Edwards YJ. Methylenetetrahydrofolate reductase and neural tube defects. *Lancet* 1996;**348**:58.
4 Kirke PN, Mills JL, Whitehead AS, *et al*. Methylenetetrahydrofolate reductase mutation and neural tube defects. *Lancet* 1996;**348**:1037-8.
5 de Franchis R, Mandato C, Buoninconti A, *et al*. Neural tube defects (NTD): investigation of genetic factors involved in homocysteine metabolism. *Med Genetik (Suppl)* 1997;**9**:82.

*Table 1 Genotype and allele frequencies of MTHFR 677C→T in Turkish subjects*

| | *Genotype frequencies (%) (N)* | | | *Allele frequencies* | |
|---|---|---|---|---|---|
| | *C/C* | *C/T* | *T/T* | *C* | *T* |
| NTD cases (49) | 40.8 (20) | 51.0 (25) | 8.2 (4) | 0.66 | 0.34 |
| Mothers (40) | 42.5 (17) | 40.0 (16) | 17.5 (7) | 0.62 | 0.38 |
| Fathers (33) | 42.4 (14) | 51.5 (17) | 6.1 (2) | 0.68 | 0.32 |
| Controls (93) | 50.6 (47) | 41.9 (39) | 7.5 (7) | 0.72 | 0.28 |

### Quinupristin/dalfopristin in neonatal *Enterococcus faecium* meningitis

EDITOR,—Antimicrobial resistance in enterococci, particularly *E faecium*, may greatly restrict the choice of treatment. We report the case of a neonate with vancomycin resistant *E faecium* (VRE) meningitis successfully treated with quinupristin/dalfopristin (a semisynthetic injectable streptogramin) and chloramphenicol.

The patient underwent repair of obstructed infradiaphragmatic total anomalous pulmonary venous drainage on the first day of life. Recovery was complicated by an episode of necrotising enterocolitis for which cefotaxime and metronidazole were given. At the age of 13 days abdominal signs had resolved but the patient was febrile. Over the next three days six blood cultures yielded *E faecium* resistant to vancomycin, amoxycillin, erythromycin, rifampicin, and sensitive to chloramphenicol. *E faecium* with the same susceptibility pattern was grown from three intravascular lines removed at this time. Two doses of vancomycin and a single dose of gentamicin had been given when the lines were removed. There was only modest clinical improvement so treatment with chloramphenicol (30 mg/kg/day) was started. There was no evidence on echocardiography of intracardiac infection.

*Table 1 Minimum inhibitory concentration (mg/l)*

| *Antimicrobial* | *Blood culture* | *CSF isolate* |
|---|---|---|
| Ampicillin | >128 | 64 |
| Erythromycin | >128 | >128 |
| Vancomycin | >128 | 128 |
| Teicoplanin | 8 | 2 |
| Rifampicin | 128 | >128 |
| Chloramphenicol | 4 | 4 |
| Ciprofloxacin | >32 | >32 |
| Gentamicin | 8 | 16 |
| Tobramycin | 64 | 128 |
| Streptomycin | 2048 | 2048 |
| Quinupristin/ dalfopristin | 0.25 | 0.125 |

During the first three days of chloramphenicol treatment three more blood cultures yielded VRE. Teicoplanin was added—a loading dose of 16 mg/kg followed by 10 mg/kg daily. After an initial improvement the patient again became febrile and lumbar puncture was performed. Cerebrospinal fluid contained 16 million red cells/l and 380 million white cells/l. Culture yielded VRE. Minimum inhibitory concentrations of a range of antibiotics were measured by broth microdilution technique for the cerebrospinal fluid and blood isolates (table 1). The dose of chloramphenicol was increased to 75 mg/kg/day and the patient enrolled (with informed parental consent) in a compassionate use programme to receive quinupristin/dalfopristin, 7 mg/kg/day in three divided doses. Teicoplanin was discontinued. A second sample of cerebrospinal fluid obtained after seven days contained <1 million red cells/l, 15 million white cells/l, and was sterile. Chloramphenicol was continued with quinupristin/dalfopristin for 13 days. No isolates were detected in the stools of patients in the ward screened for the presence of VRE. The patient made a full recovery with no adverse events.

The baby probably developed intravascular line infection, possibly from an intestinal source, which failed to be controlled by removal of the lines and administration of chloramphenicol and teicoplanin, and this resulted in meningitis. Although enterococcal meningitis has been described in neonates with bacteraemia associated with central venous lines,[1] meningitis with *E faecium* is rare.[2] Central nervous system infections with VRE present a particularly difficult therapeutic problem as β lactams are inactive and teicoplanin penetrates cerebrospinal fluid poorly[3] and in any event has little activity against some VRE phenotypes. Chloramphenicol is not bactericidal and there are few data on its use in VRE meningitis.

We were unable to measure the concentration of quinupristin/dalfopristin in the patient's cerebrospinal fluid to assess penetration, but our clinical experience confirms that the combination has a role in the treatment of serious infection with vancomycin resistant *E faecium*; furthermore, it complements in vitro evidence that quinupristin/dalfopristin has an

additive effect in the presence of chloramphenicol. (Messick CR, Pendland SL. Abstract EO111 presented at the 36th Interscience Conference on Antimicrobial Agents and Chemotherapy, New Orleans, 1996.)

W R GRANSDEN
ANNA KING
DALMA MAROSSY
*Department of Microbiology*
*Guy's Hospital*
*London SE1 RT*

E ROSENTHAL
*Department of Paediatric Cardiology, St Thomas' Hospital*

1 Dobson SRM, Baker CJ. Enterococcal sepsis in neonates: features by age at onset and occurrence of focal infection. *Pediatrics* 1990;**85**:165-71.
2 Barker DP, Buss PW, Marlow N, Brown NM, Millar MR. Enterococcus faecium meningitis. *Arch Dis Child* 1994;**70**:F78-9.
3 Stahl JP, Croize J, Wolff M, *et al*. Poor penetration of teicoplanin into cerebrospinal fluid in patients with bacterial meningitis. *J Antimicrob Chemother* 1987;**20**:141-2.

# BOOK REVIEWS

**Fetal & Neonatal Brain Injury. Mechanisms, management and the risks of practice.** David K Stevenson, Philip Sunshine, eds. [Pp 665; £175 paperback.] Oxford University Press, 1997. ISBN 0-19-262640-X

I enjoyed delving into this book. The editors acknowledge that the series of chapters by individual authors had resulted in some overlap and repetition but that it allowed individual prose styles to come through. That is indeed the case and makes the narrative style easy to read.

The foreword took me somewhat by surprise: the rationale is not how we best prevent or manage neonatal injury, nor is it a state-of-the-art review of multicentre trials telling us how to modify our management protocols; rather, it is a book for the courtroom "where these complex neurologic issues are regularly publicly debated. Opposing neuroscientists are in great demand as expert witnesses." I would question whether this book really does serve that purpose. It might have achieved it rather better had some of the subjects been reviewed as a critical evaluation of the available scientifically valid studies. For example, there is scant recognition of the debate that rages over the treatment of neonatal electrographic seizures without clinical accompaniment. Do we really know whether treatment of these is of definite benefit?

The final chapter on the appropriateness of intensive care application made particularly interesting reading and especially so in the light of the recently published Royal College of Paediatrics and Child Health guidelines on *Withholding or withdrawing life saving treatment in children*. It seems clear that financial considerations are beginning to figure more prominently in these matters, and in this country we are behind in that debate. The author admits that, in the USA, practical considerations such as litigation or even adverse media publicity may prevail over moral and medical judgment of physicians and, hence, parents.

This book made me think about neonatal brain injury in some detail and examine my own understanding and practice. To this end it will be valuable to those of us privileged to be invited to attend the courtroom to discuss such issues. Of course, not all the answers are there: it is for paediatricians to provide those answers through peer reviewed research and not let legal precedents take the lead.

MARTIN KIRKPATRICK
*Consultant paediatric neurologist*
*Ninewells Hospital and Medical School, Dundee*

**An atlas of neonatal brain sonography.** P Govaert, L S de Vries eds. (Pp 363; £50 hardback) Cambridge University Press, 1997. ISBN 1-898683-09-3

In the early 1970s intracranial haemorrhage in prematurely born babies was thought to be predominantly fatal, with the few survivors developing post haemorrhagic hydrocephalus. With the introduction of computed tomography brain scanning of surviving premature infants, it was found that far from being fatal, most infants who had intraventricular haemorrhage survived—and often with few or no abnormal neurological signs. In the late 1970s real-time ultrasonography was shown to be a convenient, safe, and reproducible technique for imaging the newborn baby's brain and improvements in technology now mean that every neonatal unit in the developed world has access to high quality ultrasound imaging.

There can be little doubt that this title represents the definitive work on neonatal cranial ultrasound. The authors have had extensive experience in the use of ultrasound scanning the neonatal brain for almost as long as ultrasound has been used to investigate intracranial pathology on the neonatal unit; their combined experience is most impressive. The book covers every aspect of ultrasound brain imaging currently available and discusses variations of every form of pathology evident by this technique.

The illustrations are extremely good and considerable pains have been taken to ensure that they are reproduced to best effect. One problem, as the authors readily admit, is knowing the clinical significance of some of the appearances they describe. Do they represent pathology or are they normal or developmental variants?

Unfortunately, the strength of this book is also its weakness. By showing examples of so much pathology, with many of the abnormalities being relatively subtle, begs the question of whether more information could be obtained from other imaging modalities. In recent years magnetic resonance has become a very important technique for imaging the brain, and to some extent, ultrasound and magnetic resonance are complementary. In other indications, particularly in mature infants, magnetic resonance is the best technique for recognising many forms of pathology. By concentrating on the minutiae of ultrasound imaging, the reader misses the point as to what is the best way of diagnosing abnormalities. The art of modern imaging is not to expect a great level of skill in one technique but the best selection of imaging tools from the range of techniques available. It is a great pity that this excellent book did not include more magnetic resonance or computed tomography images with comments on the advantages and disadvantages of each. The authors acknowledge this criticism in the second sentence of their preface, but do not explain why they did not extend the scope of the book.

This book tells you everything you need to know about neonatal cranial imaging with superb illustrations, but it does not tell the clinician what s/he really needs to know, which is the limitation of ultrasound as a diagnostic technique and how other modalities can aid in more accurate diagnosis.

MALCOLM LEVENE
*Professor of Paediatrics, General Infirmary, Leeds*

**Neonatal hematology and immunology III.** J A Bellanti, R Bracci, G Prindull, M Xanthou, eds. (Pp 252; $US 172 hardback). Elsevier Science, 1997. ISBN 0-444-82573-8

This book is a compilation of papers presented at the third meeting of the European Society for Pediatric Haematology and Immunology in 1996. As the editors themselves admit, the book does not cover the entire area of either haematology or immunology but "reflects the particular interests of the colleagues who attended the symposium." This raises the important question as to whether these participants were indeed invited speakers representing their expert areas or whether they represent authors who had submitted abstracts to the symposium.

The book is divided in two sections—immunology and haematology—with each section further divided into five areas of interest. The immunology section includes microbial host-cell interaction, immunological enhancement of neonates, viral infections and food allergy. The haematology section includes the use of erythropoietin, coagulation, stem cell function, and immunologically mediated cytopenia and anaemia.

Each subdivision has three to four short papers of three to six pages including references. Some important subject areas are covered, including the use of immunoglobulins in neonatal sepsis, the use of erythropoietin for anaemia of prematurity, and the use of G-CSF for neutropenia. The quality varies considerably between chapters. Some argue rather strongly for the use of their selected therapeutic modality despite insufficient published data while others argue more objectively.

It was interesting to read about the use of immunoglobulins in respiratory syncytial virus in post-neonatal infants (surprisingly in a neonatal book) but rather repetitive to read for the third time in three consecutive chapters the adverse effects observed with formalin inactivated RSV vaccine when it was introduced in the 1960s.

Furthermore, the chapters have varying fonts as well as styles, making this book rather difficult to read. The large number of spelling mistakes only makes this worse. The style for each chapter presumably reflects that of the author's with no editorial uniformity. Helpfully, some chapters have a summary or abstract, but most do not.

The book is unlikely to be of use to the general paediatrician or neonatologist as the introduction does not give sufficient information on the topic being discussed. Nor do the chapters give sufficient detail to permit the reader to decide whether to use the treatment or intervention being discussed.

SAILESH KOTECHA
*Senior lecturer in child health*
*University of Leicester*