hospital), and that it is unfair and may be dangerous to leave decisions about follow up to these same mothers. He also proposes that children under 6 months should be seen monthly by health visitors. As a means of preventing infant deaths such visits remain controversial; they will certainly reduce the time available for health visitors to seek out children who do not attend at routine clinics.

Dr Valman deals at length with policies for secondary prevention but neither the recent multicentre study nor any other has convincingly defined sensitive and specific clinical predictors of sudden infant death. The report itself concludes that primary prevention might be most effective and suggests that the policies which are likely to improve postneonatal mortality are those aimed at reducing accidents and infections in infancy and smoking in mothers, encouraging breast feeding, and improving socioeconomic conditions.

DAVID JEWELL

PETER BURKE

ANN LOUISE KINMONTH

D J G BAIN

Aldermoor Health Centre,
Southampton SO1 6ST

- 1 Knowelden J, Keeling J, Nicholl JP. *A multi-centre study of post neonatal mortality*. London: Department of Health and Social Security, 1985.
- 2 Pollak M. Child care. In: Fry J, ed. *Trends in general practice 1977*. London: Royal College of General Practitioners, 1977.
- 3 Royal College of General Practitioners. *Morbidity statistics from general practice 1971-2. Second national study*. London: HMSO, 1979.
- 4 Hart C, ed. *Child care in general practice*. Edinburgh: Churchill Livingstone, 1982.
- 5 Wilson AD, Downham MAPS, Forster DP. Acute illness in infants: a general practice study. *J R Coll Gen Pract* 1984;34:155-9.

SIR,—When commenting on the Knowelden study of postneonatal mortality¹ Dr Bernard Valman (2 February, p 339) is incorrect to imply that there is no information on the prevalence of symptoms in infants. A preliminary report² documented symptoms in those who suffered cot deaths and controls²: 59% of cases and 26% of controls had minor symptoms in the preceding week (48% and 12% had major symptoms). With a low incidence of cot death, estimated for clinically "unexpected" and "terminal disease" cases at 28/year/10 000 children at risk,¹ this means that symptoms have minimal predictive value. Only one child in 23 000 with minor symptoms (and one in 4800 with major symptoms) can be expected to die in the following week. Obviously it is neither feasible nor desirable to admit every such baby to hospital, so parents, general practitioners, and health visitors are placed in great difficulty when managing mild illness in a baby under 6 months. The addition of risk factors does, however, give guidance towards early treatment and admission. For this reason alone there is good reason to retain risk scoring as a clinical tool for primary care workers, though such scores will need local adjustments.

The study argues that breast feeding is protective and smoking in pregnancy causative for cot deaths. The case remains unproved for both. Bottle feeding has a low relative risk and is heavily confounded by social factors. Smoking has a higher risk but no dose-response relation. Though it lowers birth weight, this is not how smoking affects mortality.¹ Pregnant women are now choosing to smoke less; probably those who continue to smoke are an exceptional group and so smoking in pregnancy

is becoming a stronger marker for social disadvantage.

Four national newspapers, aided by an uninformative DHSS press release, stated that the study proved that bottle feeding and smoking caused "cot deaths." After dismissing child abuse as a substantial factor it would be unfortunate if the study only increased the guilt of smoking or bottle feeding mothers who had suffered a tragedy.

This must not be the end of cot death studies. The authors have completed a very difficult task. Their reward should be to see their work used as a model for every health district. I would nominate Sheffield to coordinate such an enterprise and hope the DHSS will foot the bill.

ANGUS NICOLL

London School of Hygiene and Tropical Medicine,
London WC1E 7HT

- 1 Knowelden J, Keeling J, Nicholl JP. *A multi-centre study of post neonatal mortality*. London: Department of Health and Social Security, 1985.
- 2 Stanton AN, Downham MAPS, Oakley JR, Emery JL, Knowelden J. Terminal symptoms in children dying suddenly and unexpectedly at home. *Br Med J* 1978;iii:1249-51.

SIR,—I would like to comment on Dr Bernard Valman's leading article (2 February, p 339). Despite the lead taken by the Royal College of General Practitioners, child health services in the UK remain fragmented. Many infants are weighed, immunised, and seen with non-acute medical problems in child health clinics, seeing the general practitioner only with acute illnesses. Often communication between the general practitioner and the clinical medical officer or health visitor is poor, and consequently the information about these infants is fragmented, making identification of those at risk of postneonatal death more difficult.

Dr Valman comments that sinister symptoms are an indication for a child to be seen by a member of the paediatric unit, who may or may not admit the child. Unfortunately, outside the teaching hospitals and centres of excellence such as Northwick Park these units are staffed almost exclusively by senior house officers with cover from the consultants. Are the paediatric consultants in these units prepared to attend all these infants in the receiving room and decide who should be admitted for observation overnight? More often than not the practitioner referring the child has more paediatric experience than the senior house officer assessing the infant, and if all those with sinister symptoms were admitted for observation the present facilities would be overwhelmed. Unfortunately even admission to hospital does not always prevent the sudden infant death syndrome.

Obviously all doctors dealing with infants should be taught how to assess them objectively for serious disease. I would suggest, however, that the study confirms the need for general practitioners with a special interest in paediatrics, as advocated in the Court report of 1977. Within group practices these doctors would organise child welfare clinics along with attached health visitors for their practice population. Information would not be fragmented, and those infants thought to be of high risk could be identified, and, one hopes, some of these unfortunate deaths prevented.

STEPHEN HODGES

Headingley Medical Centre,
Leeds LS6 3DS

Failure of single dose amoxycillin as prophylaxis against endocarditis

SIR,—The recent report of an alleged case of failure of amoxycillin prophylaxis against endocarditis has stimulated correspondence about penicillin tolerant streptococci causing endocarditis after dental procedures. Dr D W Denning and Dr W S Hillis (26 January, p 317) are mistaken to imply that giving probenecid concurrently with single dose amoxycillin is not relevant to the problem of endocarditis caused by streptococci tolerant to amoxycillin.

A difficulty arises when a patient has not had penicillin recently and an "unexpected" bacteraemia with amoxycillin tolerant streptococci follows a dental procedure. Single dose amoxycillin sometimes still prevents endocarditis in this situation as the antibiotic may interfere with the tolerant streptococci sticking to and multiplying on a heart lesion. However, when an enormous inoculum of tolerant streptococci enters the blood the chances of prophylaxis being successful are increased by prolonging the period during which tolerant bacteria are inhibited, as has been shown in some animal studies.^{1,2} The concurrent administration of 1 g oral probenecid with a single 3 g oral dose of amoxycillin results in more persistent serum amoxycillin concentrations.³ Therefore, if probenecid were to be recommended together with the single dose of amoxycillin one hour before dental extractions successful prophylaxis against endocarditis might be expected even when a large dose of amoxycillin tolerant streptococci enters the blood.

Incidentally, the simplicity, clinical effectiveness, and lack of toxicity of amoxycillin given together with probenecid has been shown in another situation where organisms with reduced sensitivity to penicillins have to be considered—for the single dose treatment of patients with gonorrhoea.

D C SHANSON

Department of Medical Microbiology,
Charing Cross and Westminster Medical School,
London SW1P 2AR

- 1 Glauser MP, Bernard JP, Moreillon P, Francidi P. Successful single-dose amoxycillin prophylaxis against experimental streptococcal endocarditis: evidence for two mechanisms of protection. *J Infect Dis* 1983;147:568-75.
- 2 McGowan DA, Nair S, MacFarlane TW, MacKenzie D. Prophylaxis of experimental endocarditis in rabbits using one or two doses of amoxycillin. *Br Dent J* 1983;155:88-90.
- 3 Shanson DC, McNabb R, Hajipieris P. The effect of probenecid on serum amoxycillin concentrations up to 18 hours after a single 3 g oral dose of amoxycillin: possible implications for preventing endocarditis. *J Antimicrob Chemother* 1984;13:629-32.

* * * This correspondence is now closed.—ED, *BMJ*.

Can we afford screening for neural tube defects?

SIR,—Two reports have questioned the wisdom of continuing regional screening programmes for neural tube defects based on maternal serum α fetoprotein values.^{1,2} One centre, in an area of low incidence, found serum α fetoprotein values to have poor predictive value in detecting open neural tube defects and abandoned the programme after screening 3479 pregnancies.¹ The other study, in a high incidence area, found an efficacy (proportion of open neural tube defects in fetuses of women who underwent terminations

as a consequence of the screening programme) of only 56.1% (anencephaly 66.6% and open spina bifida 40.7%).² Poor efficacy was attributed to poor compliance, failure of the screening test to detect open neural tube defects, and decisions against termination. These workers concluded that it was doubtful whether efficacy levels above 65% for open spina bifida could be achieved in practice.

Professor B M Hibbard and colleagues (26 January, p 293) have carried out a cost benefit analysis using the efficacy data of their previous study² and have inferred that screening for neural tube defects in areas in which prevalence falls below 5 per 1000 is not cost effective, since prevalence is the biggest determinant of cost.

Our four year study in an area of low incidence (1.95 per 1000), screening 31 976 pregnancies, paints a different picture. In our hands efficacy for all open neural tube defects is 78% (open spina bifida 70% and anencephaly 85%), and the sensitivity of the screening programme in detecting anencephaly in the screened population is 97% (95% for open spina bifida, 50% for closed spina bifida). Our screening programme reaches 88% of the pregnant population, and 93% of these are screened at 16 to 18 weeks. It is surprising that in such a high incidence area as south Wales only 70% of the population took up screening. Another surprising feature of the Cardiff data is the termination of two normal fetuses per 10 000 pregnancies. In four years of screening in our area (serving the patients of nine consultant obstetricians) not one normal fetus has been terminated.

Our experience shows that in an area with a low incidence a high detection rate, good patient compliance, and good counselling result in efficacy levels of over 75%. Our cost benefit equation is not dictated by the prevalence of the disorder but by the detection rate and patient compliance. Regions should be careful in interpreting cost benefit data from centres in which detection rate and patient compliance are poor. Our screening programme is highly cost effective, since if our efficacy of 87% for open spina bifida is put into the calculation of Professor Hibbard and colleagues in place of their figure of 50% the cost per case of open spina bifida avoided using the initial serum α fetoprotein screening strategy (option 1) would be £6152 for a prevalence of 5 per 1000—that is, a 43% reduction in their cost data. Clearly prevalence is not the only factor determining screening costs: efficacy and detection rate are equally important factors. What Professor Hibbard and others also fail to take into account are the psychological benefits of identifying non-viable pregnancies and the fringe benefits from detecting other abnormal pregnancies such as gastroschisis, Turner's syndrome, Potter's syndrome, Mickel's syndrome, and congenital nephrosis, plus, in our hands, 50% of all cases of closed spina bifida. All of these factors need to be considered in any cost benefit equation.

Clearly with good analytical precision, highly motivated and well coordinated clinicians and nurses, and sympathetic counselling, screening can be highly cost effective in a low incidence area. Health service resource planners should take note of what can be achieved by other centres.

K SPENCER
P CARPENTER

Biochemistry Department,
Oldchurch Hospital,
Romford, Essex RM7 0BE

- 1 Standing SJ, Brindle MJ, Macdonald AP, *et al*. Maternal alpha-fetoprotein screening: two years' experience in a low-risk district. *Br Med J* 1981; 283:705-7.
- 2 Roberts CJ, Hibbard BM, Elder GH, *et al*. The efficacy of a serum screening service for neural-tube defects: the South Wales experience. *Lancet* 1983; i:1315-8.

SIR,—Professor B M Hibbard and colleagues report that the financial costs to the NHS of a screening programme for open neural tube

defects range from £656 100 to £735 600 per 100 000 population (at 1980 prices), depending on the screening strategy adopted. If the incidence of open spina bifida were 1.25 per 1000, which is probably close to the true incidence in Great Britain as a whole,¹ such a screening programme would detect 125 cases of open spina bifida, giving a cost per case detected of about £5200 to £5900. Calculations have suggested that the lifetime resource savings from avoiding the birth of one child handicapped by open spina bifida range from £7100 (assuming "replacement" one year later by a non-handicapped child, and discounting future costs at 10% a year) to £19 900 (assuming no replacement and discounting future costs at 4% a year) at November 1979 prices.²

Comparing these two sets of figures suggests that a screening programme would produce a net saving to society of between about £2000 and £16 000 (at 1980 prices) per birth avoided. Hence from society's perspective perhaps the question should be re-phrased: Can we afford not to screen for open neural tube defects? Moreover in higher risk areas, such as Northern Ireland and Wales, if the incidence of open spina bifida were 2.5 per 1000 or greater the net saving to society would be greater, ranging from around £5000 to £20 000 per birth avoided. (On top of these resource savings there are, of course, the intangible benefits to the parents from being able to avoid the birth of a handicapped son or daughter and to try again for a non-handicapped child much sooner, which must be weighed against the intangible costs of anxiety and distress caused by false positive and false negative test results and occasional loss or damage of non-handicapped fetuses caused by false positive indications for amniocentesis.)

The figures reported by Professor Hibbard and colleagues of the cost of avoiding the birth of a child with open neural tube defect who is likely to survive beyond 24 hours after birth seem to suggest a very low survival rate. The figures for the savings, quoted above, assumed that 14% of the children with prenatally detectable open spina bifida would be stillborn but that 36% would survive at least until their first birthday. The savings were calculated to arise from reduced expenditure on education and the extra output produced at work, as well as reduced health service expenditure. So while NHS health authorities might not realise a financial "profit" on screening for open neural tube defects, there is reason to believe that the exchequer might, and the nation would.

JOHN B HENDERSON

Department of Community Medicine,
University of Aberdeen,
Aberdeen AB9 2ZD

- 1 Carstairs V, Cole S. Spina bifida and anencephaly in Scotland. *Br Med J* 1984; 289:1182-4.
- 2 Henderson JB. Measuring the benefits of screening for open neural tube defects. *J Epidemiol Community Health* 1982; 36:214-9.

Value of computed tomography in patients with stroke

SIR,—We read with interest the paper by Dr Peter Sandercock and his colleagues (19 January, p 193) dealing with the value of computed tomography in patients with stroke. They suggest that anticoagulation would be unsafe in at least 15% (seven out of 46) of the

patients who had atrial fibrillation at the time of the first stroke. Did any patients among these seven have rheumatic heart disease as the cause of their atrial fibrillation, because these patients would have received anticoagulants irrespective of the risk of haemorrhage or haemorrhagic infarct? Did any of the four patients with intracerebral haemorrhage have uncontrolled hypertension in addition to atrial fibrillation, because hypertension is present in roughly half of patients with chronic atrial fibrillation? Most physicians would withhold anticoagulants in patients with uncontrolled hypertension. It may well be that some of these 15% patients at risk by anticoagulation would not have received anticoagulants while others would have received them irrespective of the risk of haemorrhage.

Non-valvular atrial fibrillation is the commonest source of cardiogenic cerebral embolism² and it has been suggested that anticoagulation should be undertaken in these patients.³ Is there a place for anticoagulating patients with non-valvular atrial fibrillation if they are selected properly, or do the risks of anticoagulation outweigh the benefits in these patients?

M JOGLEKAR

Bridgend General Hospital,
Bridgend, Mid Glamorgan

K MOHANARUBAN

Cardiff Royal Infirmary,
Cardiff

- 1 Kannel WB, Abbott RD, Savage DD, McNamara PM. Epidemiologic features of chronic atrial fibrillation. *N Engl J Med* 1982; 306:1018-22.
- 2 Easton JD, Hart RG, Sherman DG, Kasre M. Diagnosis and management of ischaemic stroke part I. *Curr Probl Cardiol* 1983; 8:3.
- 3 Anonymous. Cerebral embolism [Editorial]. *Lancet* 1985; i:29.

*.*The authors reply below.—ED, *BMJ*.

SIR,—In reply to Dr Joglekar's and Dr Mohanaruban's first question, none of the seven patients with atrial fibrillation and stroke due to either primary intracerebral haemorrhage or haemorrhagic cerebral infarction had rheumatic heart disease. We disagree strongly with their contention that patients with stroke due to primary intracerebral haemorrhage or haemorrhagic cerebral infarction should be anticoagulated if they have atrial fibrillation and a history of rheumatic heart disease; doing so may result in fatal intracranial haemorrhage.¹

In reply to their second question, all four of the patients with primary intracerebral haemorrhage had hypertension (blood pressure >160/90 mm Hg recorded twice or more before the stroke). The blood pressures which we measured at the time of the stroke in the four patients (mean of two readings taken in the "non-stroke" arm) were: 214/99, 170/70, 155/97, 190/115 mm Hg. The first two patients were on antihypertensive drugs at the time of the stroke. While many clinicians might regard our definition of hypertension as very stringent, even relatively mild hypertension may increase the risk of intracranial haemorrhage.² Furthermore, blood pressures measured soon after a stroke may be affected by many factors and do not give a reliable estimate of the prevalence of "uncontrolled hypertension" in patients with acute stroke.³

However, we do agree that for patients with stroke in atrial fibrillation the presence of clinically obvious contraindications (other than primary intracerebral haemorrhage or haemorrhagic cerebral infarction) may preclude the use of anticoagulants and hence avoid the need to perform computed tomography. We are analysing data on a larger number of cases to determine the proportion of cases with stroke and atrial fibrillation not due to rheumatic heart disease that might be suitable for