a history of a sore throat, fever, and malaise. He had had two recent episodes of a sore throat treated with antibiotics. Examination showed oral candidiasis, enlarged asymmetrical cervical and axillary lymph nodes, a soft systolic murmur, and hepatosplenomegaly. Laboratory investigations showed anaemia (haemoglobin concentration 71 g/l), a platelet count of 128×10^6/l, a prolonged international normalised ratio, hypoalbuminaemia, and renal failure. The result of an HIV antibody test was positive. The CD4 cell count was 100×10^6/l. Throat swabs for bacterial pathogens yielded negative results, and serial antistreptolysin O titres were not raised. An electrocardiogram was normal and echocardiography showed a dilated left ventricle. Staining of cerebrospinal fluid for acid fast bacilli yielded a positive result, and empirical antituberculous treatment was started. He initially responded, with appreciable improvement in clinical, haematological, and biochemical variables.

Over the next five months he was admitted on three occasions with fever, arthralgia, progressive dyspnoea, and hypersplenism. On his final admission he had clinical signs of congestive cardiac failure, with cardiomegaly and pulmonary oedema evident on chest radiography. Repeat echocardiography showed a dilated hypokinetic left ventricle with a normal mitral valve. Despite conventional treatment for heart failure he developed progressive biventricular failure and died. At postmortem examination the gross findings included pronounced left ventricular dilatation, a normal mitral valve, pulmonary oedema, enlarged peripheral and retroperitoneal lymph nodes, and hepatosplenomegaly. Cardiac sections showed Aschoff's bodies in the myocardium and subendocardium.

This patient died of left ventricular failure secondary to rheumatic myocarditis. Patients with HIV infection are often unable to mount an appropriate serological response.[3] The absence of appreciable valve abnormalities agrees with a previous report of rheumatic heart disease in HIV infection.[2] Rheumatic heart disease should be considered in HIV positive patients from endemic areas who have evidence of heart disease. Clinical and laboratory features may be modified by immunodeficiency associated with HIV.

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Supplementation with folic acid

EDITOR,—In 1992 the Department of Health recommended that all women should take folic acid supplements around the time of conception to reduce the incidence of neural tube defects.[1] We undertook a survey of antenatal patients and those trying to conceive who attended our surgery in May and June 1994 to determine whether this advice was being followed. Of the 109 pregnant women surveyed, only 20 had taken supplements in accordance with the recommendations. In a further five cases the pregnancies were unplanned; thus the women could not have followed the recommendations fully, but they had started supplementation as soon as they thought that they were pregnant.

Women who had stayed on in education beyond age 16 were more likely to have taken supplements than those who had not stayed on (15/41 *v* 10/68, $P < 0.02$). Women who conceived in 1994 were more likely to have taken supplements than those who conceived in 1993, though the difference was not significant (15/48 *v* 10/61, $P < 0.5$). Eight women were identified who were trying to conceive. Six of them were not taking supplementation, of whom three were receiving treatment for infertility. The women surveyed were asked whether they had heard of the recommendations about folic acid. Seventy two were aware of them, and 44 of the 72 had learnt about supplementation from sources within our practice.

We found that 18% of the women had followed the department's advice; this figure is higher than that in three studies in hospital antenatal clinics in 1983, in which only 2·4% (15/613), 3% (14/507), and 5% (20/411) had done so.[2-4] We are disappointed with the low take up by our patients but believe that it would have been even lower but for a poster campaign we ran.

If all cereals and breads were fortified with adequate amounts of folic acid this would obviate the need to take tablets and solve the problem in the case of unplanned pregnancies. At the moment Britain's largest suppliers of cereals fortify their products with either 50 or 100 μg of folic acid per serving (an eighth or a quarter of the recommended level), but other manufacturers have much lower amounts in their cereals (personal communications). Fortification of bread also varies. We believe that the Department of Health should start a new campaign directed at both health care workers and the public to promote supplementation with folic acid. If this proves to be ineffective, fortification of cereals and bread should be made compulsory.

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Antibiotics carried in general practitioners' emergency bags

EDITOR,—S W S Menzies[1] highlights an important result in our survey that we were unable to elaborate because of restriction of space[2]; half of the general practitioners (1329 (50·4%)) still carried co-trimoxazole, and 694 (26·3%) carried ciprofloxacin. There are currently few indications for the first line use of co-trimoxazole (treatment and prophylaxis of *Pneumocystis carinii* pneumonia being one); even more disturbing is the increase in ciprofloxacin, which must have even fewer roles in the community.

We recommend that, in addition to parenteral benzylpenicillin, the following oral antibiotics should be kept in the emergency bag: amoxycillin for respiratory infections, trimethoprim for urinary tract infections, flucloxacillin for cellulitis and acute skin infections, and erythromycin succinate for people with sensitivity to penicillin and for suspected atypical pneumonia. Cephalexin or co-amoxiclav might be carried for use as a second line drug for urinary tract infection, particularly in older people in nursing homes.

The antibiotics carried would obviously depend on patterns of resistance in the area where the general practitioner works. Family health services authorities or even the Royal College of General Practitioners should consider whether a central and national policy on emergency drugs carried by general practitioners might improve practice.

We did ask about chloramphenicol in our questionnaire (general practitioners were asked to tick whether the drugs that they carried were oral, parenteral, or topical), and none carried parenteral chloramphenicol. We accept that chloramphenicol can be used as an alternative in cases of known allergy to penicillin, as noted by Annette Wood and Sarah O'Brien[1] and Vivien Hollyoak and Alice Gunn,[1] particularly in the primary care setting; we recommend, however, as most authorities in Europe and North America do, a third generation cephalosporin such as cefotaxime or ceftriaxone.[3] Third generation cephalosporins have the advantages of high activity against all common major meningeal pathogens except *Listeria monocytogenes*; a lack of resistance currently; excellent penetration into the cerebrospinal fluid; and ease of use, being single agents that can be given three times daily (cefotaxime) or once or twice daily (ceftriaxone).

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Units of measurement of central venous pressure

EDITOR,—While reading the Grand Round on nocardia pericarditis I was stopped in midsentence by the use of kilopascals as units of central venous pressure.[1] Central venous pressure is measured in a mixture of units according to circumstance. Thus when it is estimated at the bedside as centimetres above the clavicle the unit of measure is centimetres of blood; when a simple manometer is used the unit is centimetres of water or, more accurately, centimetres of isotonic saline solution; and when a pressure transducer is used the unit is millimetres of mercury. The conversion is 1 cm blood ≈ 1 cm H_2O ≈ 1 cm isotonic saline ≈ 0·74 mm Hg. Most clinicians use these different units in an (incorrect) interchangeable mish-mash. This, however, seldom matters as the differences are modest compared with variations in central venous pressure among patients. Furthermore, especially in an intensive care environment, central venous pressure is most commonly used as a component of sequential evaluations of haemodynamic status, when what matters most is the trend rather than the absolute value.

Kilopascals are the SI unit of pressure, but it is

nonsense to use them to measure intravascular pressures. Arterial blood pressure is measured in millimetres of mercury because most manual sphygmomanometers are filled with mercury. In the Grand Round the confusion is compounded because the offending sentence reads: "the patient . . . was . . . hypotensive (90/40 mm Hg) with 15 mm Hg paradox, central venous pressure was 1·86 kPa." Thus two separate units of intravascular pressure are used in the same sentence. As 1 mm Hg = 0·133 kPa, a central venous pressure of 1·865 kPa equates to 13·98496 mm Hg. I therefore suspect that the patient's central venous pressure was measured as 14 mm Hg and converted into kilopascals later. Certainly, I have never seen a central venous pressure manometer calibrated in kilopascals. A further problem lies in the computation of vascular resistance, for which all pressures must be in the same units. There is therefore an overwhelming case for ignoring the SI units where intravascular pressures are concerned.

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1 Tabrizi SJ. Nocardia pericarditis. *BMJ* 1994;309:1495-7. (3 December.)

**The *BMJ* accepts that it was wrong in its use of kilopascals as units of central venous pressure. When pressure is measured as a column of mercury the SI units are mm Hg.—EDITOR

Hepatitis C in asymptomatic blood donors

EDITOR,—In their response to a letter about their paper on hepatitis C in asymptomatic blood donors D J Multimer and E Elias state that "guidelines on counselling and investigating these donors do not exist."[1] They are no doubt referring to clinical guidelines for doctors dealing with these people once they have been referred for further investigation by specialist hepatology units. The National Blood Service has had detailed guidelines for confirming hepatitis C, counselling donors, and referral since screening was initiated in 1991.

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1 Multimer DJ, Elias E. Hepatitis C in asymptomatic blood donors. *BMJ* 1995;310:260. (28 January.)

Clinical Negligence Scheme for Trusts

Department of Health's view

EDITOR,—I wish to correct a few misconceptions in Paul Fenn and Robert Dingwall's editorial on the new Clinical Negligence Scheme for Trusts.[1]

Firstly, the Department of Health's actuaries have not assumed that all claims for clinical negligence pending against the NHS will result in financial settlements; they have assumed a drop out rate of around 30-40% (but lower for the high value claims, which make up a disproportionate share of the total cost). It is unfortunate that Fenn did not take up an earlier invitation to check the facts before repeating such a misleading assertion. While I welcome informed debate on the likely future costs of clinical negligence in the NHS—the scheme's designers would not claim to know all the answers—it would be better if debate started from a more accurate understanding of the assumptions made.

Secondly, if the scheme does collect more in contributions than are needed in any year the surplus will be refunded to members as soon as the scheme's managers judge prudent—if necessary, within the financial year in which it is realised that a surplus arose. I have no wish to see funds tied up that could have been used for patients' care; that is one of the scheme's main motivations.

Thirdly, the current arrangement, under which trusts can take out a "loan" from the department against large clinical negligence losses, may seem (in the short term) like a free lunch for the individual trust; it is not for the NHS as a whole. The funds needed for the loan will have to be top sliced from allocations available to purchasers generally and are therefore lost to patients' care in just the same way as when trusts (or the scheme) make the payments.

Having said this, I agree with Fenn and Dingwall's advice that any trust still undecided about the scheme should carefully consider the fine print; we have nothing to hide. For some trusts, using self insurance for the smaller claims and the loan scheme for the larger claims may be a satisfactory solution. Alternatively, they could join the scheme but choose a relatively high value of the excess, covering the cost of the more frequent (and therefore more predictable) low value claims themselves while using the scheme to even out the incidence of the relatively rare high value claims. I am confident that those trusts that have chosen to join the scheme will find it a valuable way of reducing future financial uncertainties while using the available funds to the maximum extent for patients' care.

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1 Fenn P, Dingwall R. Mutual trust? *BMJ* 1995;310:756. (25 March.)

Authors' reply

EDITOR,—We had no knowledge of what actuarial calculations had been performed before the scheme was launched and did not comment on them. What we referred to was the illustrative projections in the documentation for the induction seminar at which the scheme was presented to the trusts. Figure 4 of this document suggests that ultimately a trust with 300 doctors would have to meet financial liabilities of £2·25 million a year. This is the result of multiplying the assumed number of claims opened each year (45) by the assumed average size of the claim (£50 000). It now seems that the latter figure is supposed to take into account the fact that some claims are not paid. But if it is true that the scheme's actuaries assumed on average a "drop out rate of around 30-40%" then this is inconsistent with the evidence. As we pointed out in our editorial, all of the available evidence points to a settlement rate of around 25-33%, which in turn implies a drop out rate of 65-75%. We are unaware of any published information suggesting that this drop out rate varies with the size of the claim. If the scheme assumes a drop out rate of 30-40% it will incorporate subscriptions that are twice as high as needed. The fact that the resulting surplus will be returned to member trusts as soon as possible does not, to our mind's, justify the error.

Secondly, we did not imply in any way that the loan arrangements at present were a "free lunch." We did imply that they at least ensured a direct equivalence between the provisions made by trusts and the cost of claims settled and therefore that there was less likelihood that funds would be unnecessarily diverted from current patient care.

We are not trying to minimise in any way the severity of the problem of clinical negligence for trusts or the importance of an effective system for dealing with pooling risk and risk management. The points we made are based on information we have made public over many years and reflect our view that this is an area in which good data are in short supply. We are heartened that the minister agrees with us that a fully informed decision is a good decision.

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Ocular injuries due to alkaline substances

EDITOR,—Within the past few weeks several people with severe ocular chemical injuries have presented to the accident and emergency department at Birmingham and Midland Eye Hospital. The wounds have been due to deliberate splashing of alkaline substances into the victims' eyes. Robbery and violent assault seem to have been the motives in most cases.

In these injuries massive corneal and conjunctival epithelial loss occurs within seconds. Necrosis of corneal epithelial stem cells may ensue, resulting in delayed and cicatricial healing of the ocular surface. Alkali burns to the eye trigger a cascade of proteolytic events, causing varying degrees of destruction depending on the strength of the alkali. Zonal or diffuse opacification of the cornea, cataract, and secondary glaucoma may follow. The eye may ultimately become irreparably damaged, and a blind, painful eye may result in the worst cases.

Severe ocular chemical injuries necessitate prolonged admission to hospital and intensive and long term treatment, requiring multiple outpatient visits. Recovery and rehabilitation may take many months. As a result of loss of vision in one or both eyes the patients may lose their ability to drive, lose their job, or become dependent.

The consequences of an action that takes seconds to execute have devasting implications for the victim. The assailants, many of whom are children and young adults, may be unaware of the effects of alkali on the eyes. Indeed, many health workers may be unaware of the full importance of these injuries. Members of the public are less likely to be aware of the severe ocular toxicity of alkaline substances. We need to increase general awareness of the danger of using these substances during an assault.

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Correction

Diagnosing pulmonary embolism

An editorial error occurred in the final sentence of this letter by J Richard Harding (18 February, p 467). The sentence should have read: "In institutions where lower limb venography remains the usual investigation for deep vein thrombosis, use of liquid crystal thermography has the added advantage that the risks due to ionising radiation or allergy to contrast medium are avoided in 36% of patients."