prevent a patient who definitely has no abnormality from proceeding to venography.⁴

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- 1 Hopkins NFG, Wolfe JHN. Thrombosis and pulmonary embolism. *BMJ* 1991;303:1260-2. (16 November.)
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SIR,—N F G Hopkins and John H N Wolfe's article on thrombosis and pulmonary embolism in the ABC of Vascular Diseases contained several errors.¹

Firstly, the British Committee for Standards in Haematology, Haemostasis and Thrombosis Task Force of the British Society for Haematology suggests that after a baseline prothrombin time has been determined the usual induction dose of warfarin in adults should be 10 mg on the first day and 10 mg on the second day.² The large induction dose of 20-30 mg suggested in the article is no longer recommended, especially as there are theoretical concerns that such an approach may induce a transient hypercoagulable state due to rapid reductions in the vitamin K dependent anticoagulants, protein C and protein S.³

Secondly, the same body now recommends that treatment with warfarin should be aimed at keeping the prothrombin time at between two and three times the normal value, using international reference thromboplastin, in deep vein thrombosis and pulmonary embolism.² The lower value of 1.7 stated in the article is thus misleading.

Thirdly, after heparinisation the presently recommended value for the activated partial thromboplastin time is 1.5-2.0 times the upper limit of control values.⁴ Values 2-3 times control values are associated with significantly more bleeding complications and have no therapeutic benefits over the lower value.⁵

Fourthly, the article gives correct recommendations for conservative treatment of deep vein thrombosis in the calf but should also state that this is acceptable only if extension of the clot is excluded by repeated monitoring by ultrasonography, as 20-30% of thrombi in calf veins extend into the proximal veins.⁶

Finally, the illustration of the ischaemic limb induced by heparin states that this is due to thrombocythaemia. This should presumably read heparin induced thrombocytopenia.

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- 1 Hopkins NFG, Wolfe JHN. Thrombosis and pulmonary embolism. *BMJ* 1991;303:1260-2. (16 November.)
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AUTHORS' REPLY,—These comments on our article are well taken, but I am sure that the authors appreciate that an essential aspect of articles in

ABC series is their brevity and simplicity. We therefore pointed out the abnormalities that might be evident in chest x ray films and ventilation-perfusion scans without implying that these are always present. We made it clear that fibrinogen labelled with iodine-125 is useful only in a screening test and is not, therefore, part of the normal armamentarium in diagnosing deep vein thrombosis. Though labelled antifibrin monoclonal antibodies are of great interest to specialists, they are not yet widely available.

We are glad that the authors agree with our point about ultrasound but would point out that it is the Doppler probe that relies on the compression method and it is the ultrasound modality that is present in the duplex Doppler system.

We are glad of A M Robinson's comments about the dose of warfarin as we should have emphasised that the loading dose was 20-30 mg over three days and then daily, which is similar to the new recommendations of the British Committee for Standards in Haematology, Haemostasis, and Thrombosis. Again, the new suggested values for prothrombin time—2-3 times the normal value—vary only little from our suggestion of 1.7-3.0.

We entirely agree that calf vein thrombi should be treated conservatively only if they are not subsequently shown to extend into the popliteal and superficial femoral veins, but the essential point is that patients with popliteal thrombi should be mobilised and should not be admitted to hospital or treated.

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Age associated memory impairment

SIR,—John T O'Brien and Raymond Levy observed that "cognitive function fails with age."¹ They went on to consider clinical classification and treatment of older people who have experienced loss of memory and other cognitive abilities but who are not demented. The authors contrasted the diagnostic criteria for "benign senescent forgetfulness" proposed by Kral several decades ago² with those for "age-associated memory impairment" proposed by a National Institute of Mental Health work group that we had the privilege to cochair in 1985.³ O'Brien and Levy pointed out that the principal difference between the two approaches is that the Kral criteria describe people whose memory performance is impaired relative to that of other people of the same age, while the National Institute of Mental Health criteria describe people who report that their memory has declined since they were young adults and who perform at a level significantly lower than that of young adults. In discussing the development of treatments for memory loss in later life O'Brien and Levy argued that the National Institute of Mental Health criteria are too broad to be useful and that, as Kral proposed, memory impairment should be redefined using age standardised normal values, rather than those of young adults.

In considering O'Brien and Levy's argument one might draw a parallel with other conditions in which prevalence is increased in later life. For example, presbyopia is defined by reference to normative standards for young adults and is so common among the elderly as to be considered "normal." Nevertheless, few clinicians would compare the vision of an 80 year old with norms established for other people of the same age and prescribe corrective lenses only to those whose visual performance falls outside those norms. As in the case of vision, considerable changes in memory

may occur with advancing age among healthy "normal" people. Deficits of 50% and more may be expected between age 25 and age 75 in the ability to perform such important everyday memory tasks as learning and remembering names of people to whom one is introduced or learning new verbal information.^{4,5} O'Brien and Levy described such problems as "just a benign inconvenience of growing old," but one might ask whether a cognitive deficit of 50% would be considered "benign" if seen in a young adult.

A well developed body of preclinical data⁶⁻⁸ and emerging clinical evidence^{9,10} suggest that memory deficits associated with normal aging—that is, age associated memory impairment—may be diminished through pharmacological treatment. The argument of O'Brien and Levy that this possibility should not even be investigated is difficult to justify scientifically and may be difficult to justify to the many older patients who might benefit.

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Mortality in meningococcal disease

SIR,—Keith Cartwright and colleagues recommend giving benzylpenicillin parenterally when meningococcal disease is suspected.¹ Their laudable aim is to reduce the case mortality of meningococcal disease by giving an effective antimicrobial drug at an early stage. They rightly point out that the initial rash in meningococcal disease may not be a "typical" petechial rash. Some patients do not show a rash of any type at any stage of their illness.²

Because an effective antimicrobial drug such as benzylpenicillin almost invariably clears the blood stream of meningococci rapidly, blood should be cultured before the first dose of benzylpenicillin is given. General practitioners should therefore consider carrying a blood culture kit in their emergency bag.

In South Australia we are faced with similar problems in bringing patients with suspected meningococcal disease to hospital, sometimes from considerable distances. The resuscitation team takes a blood culture before chemotherapy is started. In children systemic infection with other encapsulate bacteria, especially *Haemophilus influenzae* type b and pneumococci, may simulate

early meningococcaemia clinically, so a vigorous attempt should be made to recover the pathogen.

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1 Cartwright K, Strang J, Reilly S, White D. Mortality in meningococcal disease. *BMJ* 1992;304:116. (11 January.)

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Temporary pacing

SIR,—Adam Fitzpatrick and Richard Sutton make no mention of monitoring a supply of oxygen and resuscitation facilities as being necessary parts of a "suitable environment" for transvenous pacing.¹ In the past few years I have attended three cardiac arrest calls to patients undergoing pacing: in no case was any written record being kept of heart rate, blood pressure, or pulse oximetry readings, and in only one case was oxygen being given. Yet patients with slow heart rates or sick hearts, or both, are likely to have impaired cardiac output and reduced oxygen supply to the tissues.

I strongly recommend that oxygen saturation, pulse rate, and blood pressure are recorded every five minutes for all patients undergoing transvenous pacing (this is becoming standard practice in anaesthesia); there should be a low threshold for giving supplemental oxygen; and emergency drugs, suction equipment, and equipment to intubate and ventilate patients should be immediately to hand.

Transvenous pacing is an operation; a minor procedure can become a major performance if the suitable environment does not include adequate monitoring and resuscitation facilities.

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1 Fitzpatrick A, Sutton R. A guide to temporary pacing. *BMJ* 1992;304:365-9. (8 February.)

Costs of adverse effects

SIR,—Fiona Godlee emphasises the National Radiological Protection Board's concern¹ over the increasing contribution of x ray computed tomography to the total medical radiation dose for the population of the United Kingdom.^{2,4} This runs counter to the general medical perception that advances in equipment and techniques and closer radiological supervision have reduced the radiation dose and that radiological examinations are therefore safer than in the past.

For many clinicians the value of a radiological investigation is measured by the quality and relevance of the information it provides. Computed tomography does well on this scale, but the relatively high radiation doses entailed are not appreciated. A radiological examination is the responsibility of a radiologist and requires close consultation with the referring clinician before it is undertaken.

There are, however, other aspects of the National Radiological Protection Board's reports which should be seen in the context of the recently revised estimates of the risk of cancer associated with low dose and low dose rate whole body irradiation given by the International Commission for Radiation Protection after reappraisal of data from Hiroshima and Nagasaki.³ For both cancer induced by radiation and fatal cancer these are approximately 3.5 times higher than the previously quoted values and are now relevant to the doses received

by patients undergoing computed tomography. From Godlee's figures a computed tomographic scanner in the United Kingdom performs about 3000 examinations a year. At an average dose of about 5 mSv per examination this gives a total population dose of 15 person Sv. The costs of the adverse effects of this in fatal cancer induced by radiation have been calculated at roughly £150 000 a year, giving a total cost of £30m for the 200 scanners in the United Kingdom. The additional costs of non-fatal cancer induced by radiation have been calculated at 1.5 times greater than this. Furthermore, no account is taken of the additional risks of cancer and costs in children.

These costs of cancers and deaths induced by radiation are concealed and therefore do not feature in calculations of the revenue consequences of a computed tomography service. They are also ignored when the costs of computed tomographic and magnetic resonance scanners are compared, greatly to the detriment of magnetic resonance scanners. Were they more generally appreciated they would provide a strong incentive for district general hospitals to acquire magnetic resonance scanners. Britain lags even further behind other developed nations in its provision of magnetic resonance scanners than it does in its provision of computed tomographic scanners.

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Income distribution and life expectancy

SIR,—R G Wilkinson seems to have misunderstood our analysis of the relation between social inequality and inequality in health.¹ He states: "These [Wilkinson's] results should caution against using the lack of a close relation between national mortality and gross national product per head to infer that health inequalities within societies cannot be a reflection of income differentials." The reference given for such incautious use is our recent detailed analysis of life expectancies, age specific death rates, and causes of death for 22 European countries from 1954 to 1984, in which we used data from the World Health Organisation.² Like Wilkinson, we used gross national product per head and found many clear departures from any simple relation between health and poverty.

We did not, however, as implied, deny a relation between health inequalities and the distribution of wealth. We identified three policy options. One was the redistribution of wealth. We commented: "There is plenty of evidence in past history to demonstrate the enormous potential effect of an increase in living standards on health. That option is still relevant, particularly with regard to the causes and the health effects of child poverty. In many prosperous countries the lethal consequences of deprivation may have been prevented or reduced by welfare services and by modern technology, but physical and intellectual development may have

been hindered and health harmed." We cannot regard such words (or other comments throughout the paper) as a denial of the effects of income differentials on health inequalities.

We differed from Wilkinson in our emphasis on the large decrease in deaths from diseases known to be caused or aggravated by poverty (for example, deaths from infectious and respiratory diseases) and the large increase in deaths from such diseases as lung cancer and ischaemic heart disease due to smoking, sedentary living, and dietary intakes far removed from deprivation. Our analysis made clear that overconcentration on poverty ignored the great diversity of disease and its many social causes and the lethal nature of modern consumption behaviours with their varying relation to relative wealth and deprivation. A recent example of such overconcentration is Tony Delamothe's article on social inequalities in health, focused almost exclusively on relative poverty and the redistribution of income.³ Income distribution figured in our interpretation as one among many causes.

Our case was carefully reasoned and based on the analysis of a continent's mortality over 30 years. It would be sad if our conclusions, which implied a diverse and dynamic relation between social inequality and inequality in health, should simply be seen, because of an unfortunate reference, as a denial of the links between relative deprivation and health status.

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1 Wilkinson RG. Income distribution and life expectancy. *BMJ* 1992;304:165-8. (18 January.)

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SIR,—R G Wilkinson proposes the interesting hypothesis that relative income distribution is causally related to health, particularly in countries in which the gross national product per head is above \$5000.¹ The author's analysis covered Western, developed countries, but other developed countries—those of the former eastern European bloc—provide an additional context in which to explore the hypothesis.²

During 1975-85 small reductions in life expectancy at birth were recorded in Poland and Hungary but small increases were recorded in wealthier Czechoslovakia and the then German Democratic Republic. The increases in the last two countries were smaller than those achieved by Western nations, including the United Kingdom, in the same period. Given the political system and state monopoly that operated in countries in the Eastern bloc, the variation in income for different types of employment was small during the period in question. On the basis of the theory underlying the hypothesis, therefore, we should expect there to have been greater increases in life expectancy in those relatively wealthy countries with a more egalitarian distribution of income—namely, Czechoslovakia and the German Democratic Republic—than in the relatively non-egalitarian United Kingdom. In addition, Józán found that life expectancy of males varied among the 22 districts of Budapest from 64.2 years to 69.7 years.³ This was in a society in which we would expect the range of income distribution to be small and to vary little among the districts. In fact, as late as 1989, bus drivers in Budapest had an income at least as high as that of doctors.

Preliminary evidence from eastern Europe, with ostensibly egalitarian income policies, does not, therefore, support Wilkinson's hypothesis. Nevertheless, several caveats should be taken into account. The wealth of eastern European nations is hard to evaluate because of the former non-convertible status of their currencies, the dif-