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

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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 hypercoaguable 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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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 ago[2] 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 data[6-8] 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