encompassing the group genetically at high risk. Detailed analysis of individual deaths during treatment does not favour any severe risk, particularly of non-accidental violent death²; complete abolition of coronary heart disease would increase life expectancy by only three years, premature mortality and morbidity being the key³; and a naturally low cholesterol concentration may increase the risk of stroke while reducing the risk of coronary heart disease. It is all a question of balance.

It is a proper concern that treatment may on balance be harmful in some patients, although any mechanisms are wholly speculative, but we also know that the special minority do badly without treatment. At a meeting organised by the Royal Statistical Society recently Michael Oliver was emphatic that young, middle aged, or clinically affected men with cholesterol concentrations above 7.5 mmol/l were potential candidates for lipid lowering drugs (a lower intervention point than many propose), endorsing the thrust if not the style of his editorial.⁴ A blanket moratorium on treatment is nonsense, but we do need to sharpen up perceptions of when and when not to introduce lipid lowering as a part of a sensible treatment plan.

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SIR,—George Davey Smith and Juha Pekkanen inappropriately include the expanded clinical evaluation of lovastatin (EXCEL) study¹ in their meta-analysis of cholesterol lowering interventions for primary prevention of coronary disease and misinterpret it to support their argument that cholesterol lowering treatment may not reduce mortality.² The objective of the EXCEL study was to evaluate the safety of the 3-hydroxy-3-methylglutaryl coenzyme A reductase inhibitor lovastatin in a large trial. It was not designed to show primary prevention: the 48 weeks of treatment was much too short for this purpose, and 28% of the patients had known ischaemic heart disease at baseline. No study of primary or secondary prevention with a 3-hydroxy-3-methylglutaryl coenzyme A reductase inhibitor has yet been completed.

The EXCEL study randomised 8245 patients into five equal groups taking lovastatin 20 mg daily, 40 mg daily, 20 mg twice daily, or 40 mg twice daily, or placebo. There were nine, five, 10, nine, and three deaths, respectively, in the five groups. These differences did not approach significance ($p>0.2$ by the Kaplan-Meier method). As the authors reported, of the 36 deaths, 31 were due to coronary disease, two to a pulmonary embolism after surgery, and one each to a ruptured aortic aneurysm, a haemorrhagic stroke, and viral pneumonia. Twenty five of these 36 patients had known ischaemic heart disease at baseline.

An adverse effect of lovastatin on coronary mortality is highly improbable for several reasons. Firstly, there was a trend towards fewer non-fatal myocardial infarctions in the groups given lovastatin (10, three, 13, 11, and 18 patients, respectively). Secondly, lovastatin when given concomitantly with a bile acid sequestrant slows the progression and induces regression of atherosclerotic coronary lesions.³ Thirdly, as Davey Smith

and Pekkanen's meta-analysis shows, there is good evidence that lipid lowering treatment reduces deaths from coronary heart disease.

A point more relevant to the issues that Davey Smith and Pekkanen raise is that no traumatic deaths occurred in the EXCEL study. The same is true for a five year study of lovastatin in 745 patients⁴ (lovastatin study groups, unpublished results); and in a one year study in 2361 patients of the closely related simvastatin one accidental death occurred.⁵ These three studies together provide roughly 12 000 patient years of vigorous lipid lowering treatment with only one death due to an accident and none due to suicide or homicide. Only the lovastatin five year study is long enough to be informative on the incidence of cancer, which was well below that expected on an actuarial basis (lovastatin study groups, unpublished results). Whatever the explanation for the differences in non-cardiac mortality reported in some of the studies of older drugs, there is no evidence that lovastatin or simvastatin has any effect on non-cardiac mortality.

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Managing hypertension

SIR,—We were interested in the results of the Medical Research Council's trial of treatment of hypertension in older adults and their implications for the treatment of hypertensive patients of all ages, particularly the prevention of stroke.¹ Individual studies and meta-analyses show that about 40% of stroke in hypertensive patients can be prevented by drug treatment.^{2,3}

In the Western Infirmary we recently established an acute stroke unit, which admits around 300 unselected patients each year from a catchment population of 220 000. Blood pressure immediately after stroke may increase temporarily. We have information on previous diagnosis and treatment for 351 patients (159 men (median age 67) and 192 women (median age 74)). Of these, 117 were aged 70-79 and 80 were aged ≥ 80 . Hypertension had been diagnosed in 132 patients (information obtained from the patient, relatives, or general practitioner's referral letter), but only 62 were receiving drug treatment: 12 (44%) of the 27 aged ≥ 80 , 23 (49%) of the 47 aged 70-79, and 27 (47%) of the 58 aged < 70 . Thirty seven (60%) of the 62 receiving treatment for hypertension had at least one other major cardiovascular risk factor, as did 30 (43%) of the 70 not receiving treatment.

We do not know why antihypertensive treatment was not offered to or continued in patients at high risk of stroke and vascular disease. Some of the patients had been treated previously but had either stopped taking their drugs or had their treatment stopped weeks or months before presenting with stroke. Despite the clear evidence of the value of antihypertensive treatment in younger patients that has been available for many years it is surprising that the "rule of halves" still holds: up to half of

all hypertension is unrecognised and only half of detected hypertension is treated. It remains to be seen whether further evidence of the benefits of treating elderly hypertensive patients will influence medical practice. If it does not many strokes that are preventable will occur, with resultant disability and death.

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Thrombosis and pulmonary embolism

SIR,—In their article on thrombosis and pulmonary embolism N F G Hopkins and John H N Wolfe describe chest radiographic findings in pulmonary embolism without making it clear that, in most cases, there is either no abnormality or minimal, non-specific change in the plain chest film.¹

In their discussion of ventilation-perfusion lung scans they do not mention that scans are classified as showing a normal or very low, a low, an intermediate or indeterminate, or a high probability of embolism. Though the first and last of these groups can be taken at face value (although even they are not 100% guarantees), patients with scans showing a low probability were shown by the prospective investigation of pulmonary embolism diagnosis to have a 14% incidence of embolism.² Either one has to do pulmonary arteriography on, at least, all patients with scans showing an indeterminate probability or one adopts the policy that in the absence of a scan showing a high probability and of persisting venous thrombosis (diagnosed by whatever means) pulmonary embolism, even if present, does not require treatment. One cannot, however, just take ventilation-perfusion scanning as a test that will in every case tell for certain whether pulmonary embolism is present.

With respect to the investigation of deep venous thrombosis, as stated venography remains the standard test. Ultrasound can be used in several ways. Duplex Doppler ultrasonography will show venous patency accurately from the popliteal vein up, and the use of colour flow even allows assessment in the calf and will show non-occlusive thrombus. Without Doppler, however, ultrasound relies on the compression method. At best this is of use only in the groin and upper thigh and in the popliteal fossa (the vein being too deep in the adductor canal), and even in the groin non-compressibility can be normal where the long saphenous vein enters the femoral vein.³

Radioisotope studies such as those using labelled fibrinogen or plasmin show only forming thrombus, not that already present. Hence anticoagulant treatment cannot be started until imaging is complete. Labelled antifibrin monoclonal antibodies will, however, target formed thrombus. Another method, not referred to in the article, is light reflective rheography. Though this is non-specific (a positive result indicating venous abnormality but not necessarily thrombus), it is highly sensitive and could serve as a screening test to