The figure compares the costs inherent in each mode. One third of a consultation's costs are allocated to administration; this is taken as one cost unit, which happens to approximate to the average cost of one item on prescription. No account is taken of opportunity costs by the patient through loss of earnings or by the fact that the specialist clinic continues to function. Nevertheless, the model shows that referral as an outpatient, for example, may be seen to be a highly expensive management decision.

Morbidity studies of general practice show that the referral option is exercised with a high degree of discrimination (about 1 in 10). The general practitioner thus plays a key role in "sparing" expensive specialist resources. Nevertheless, there is a wide range of variation, suggesting that there may be scope for creating a greater awareness of cost effectiveness in primary care. Issuing a prescription may be obviated by using the counselling mode, but at the expense of consulting time. The figure shows the relative insignificance of prescribing as a cost, yet this management option has received so much attention. We need a reappraisal of primary medical care costs in a fuller context, to include the general practice-hospital interface and the public.[3]

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SIR,—There are many reasons why the concept of clinical budgeting is misguided, but the fundamental one is easily identifiable in Mr John Appleby's leading article (7 February, p 326). Mr Appleby is a district health economist and seems to be unaware of the role of the children. He states that "the separation of clinical judgment from financial responsibility will soon end." This notion is based on a confusion, evident when he describes the clinician as making decisions about "using society's resourses."

As a clinician, the only activity I am engaged in is doing the best for the person with whom I have an implicit contract: the patient. To describe what the clinician, qua clinician, is doing in any other way is to be confused and will lead to dilemmas and conflict for those who imagine that they can wear a clinician's hat at the same time as the budget holder's. To weigh the virtue of doing the best for the patient against that of saving money is not only immoral but will lead to a lowering of standards and, sooner rather than later bad medicine.

There is no question that money can be saved; paradoxically, this can be achieved even by improving medical treatment. How much of what we do is ritual? How often are our clinical judgements examined critically and the usefulness of what we do questioned? Instead of spending time on administration, committees, and trying to run the service (our aptitude for and success in these endeavours over the years is far from obvious) we would be better occupied in discussing clinical problems, sorting out how best to deal with the common conditions that comprise most of our work, and regaining our optimism about our chosen subject: clinical practice.

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Moderate sodium restriction with angiotensin converting enzyme inhibitor in essential hypertension

SIR,—The study by Dr Graham A MacGregor and others (28 February, p 531) shows that reduced dietary sodium is associated with a fall in blood pressure in hypertensive patients treated with an angiotensin converting enzyme inhibitor. The authors failed to comment, however, on the fact that treatment with slow sodium increased urinary sodium excretion by 50 mmol/day, a value identical with the reduction during placebo treatment.

The trial compared the effects of sodium restriction and sodium loading on blood pressure in patients treated with captopril. It was implied that the response to sodium restriction was greater in patients treated with captopril (13/9 mm Hg, supine) than in untreated patients (10/5 mm Hg), these figures being taken from an earlier report.[1] No evidence of the precision of these estimates was given, however, and it is difficult to be sure that the small difference between the experiments was not merely due to chance.

The findings are used by Dr MacGregor and colleagues to advocate managing mild hypertension with salt restriction followed by treatment with an angiotensin converting enzyme inhibitor. This policy was not evaluated in their study, and we cannot be certain that the addition of treatment with an inhibitor to salt restriction would produce an effect on blood pressure comparable to that observed. Furthermore, treatment of patients who have a restricted salt intake with such an inhibitor might cause a significant incidence of first dose hypotension.[2] Before this approach can be recommended for general use its efficacy and safety must be evaluated extensively. More pertinently, it will be necessary to show that such a regimen is superior to current practice (stepped care based on thiazide diuretics and β blockers), which large trials have shown to be safe and to prevent cerebrovascular events.[3] Experience with angiotensin converting enzyme inhibitors is much more limited, and concern that they may not prevent strokes has been expressed.[4]

Until we have evidence that new treatment policies have significant clinical advantages over stepped care, hypertension should continue to be treated by measures proved to have a beneficial influence on long term outcome.

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Inadequacy of oleic acid in erythrocytes as a marker of malignancies

SIR,—We believe that several points on the use of stearic:oleic acid ratios as markers of malignancy need clarification. In 1985 Wood *et al* suggested that the erythrocyte ratio was a very good tumour marker, giving a positivity of almost 100% in disease.[1] More recently, this hypothesis has been questioned by Dr O Søreide and coworkers (28 February, p 548), who stated that there were no differences between the erythrocyte ratios in normal subjects and cancer patients. Questions arise about the methods used by both groups. Even after careful examination of all the references cited by Wood *et al* we could not ascertain the details of the methods they used. The methods quoted by Dr Søreide and others were equally perplexing—for example, no details of interbatch and intrabatch variations or recoveries were given. In addition, Dr Søreide and others stated that "oleic and stearic acids were separated almost completely by gas liquid chromatography." What exactly "almost completely" means in relation to gas liquid chromatography is difficult to understand and accept. Furthermore, the quoted erythrocyte stearic:oleic acid ratio for the control group was 0·68, which contrasts strongly with the values usually obtained.[2]

We have also assessed the use of the erythrocyte stearic:oleic acid ratio as a marker of malignancy. Our work differs from that of Wood *et al* and Dr Søreide and coworkers in that we evaluated the method used to determine erythrocyte ratios before assessing its use as a tumour marker.[3] We found the erythrocyte stearic:oleic acid ratio to be of no use in the diagnosis and monitoring of patients with malignant tumours. There was a small, but significant, difference between the mean (SD) erythrocyte ratio in 21 patients with untreated bronchogenic carcinoma (1·23 (0·17)) and that in 27 healthy adults (1·38 (0·14)). Six patients with lymphoma also had lower ratios (1·11 (0·19)), while five patients with hepatoma showed no difference (1·46 (0·44)). Using a cut off value of 1·00, the erythrocyte stearic:oleic acid ratio would have a sensitivity of less than 12·5% in the diagnosis of malignancy. Furthermore, the ratio may be lower in patients with other diseases, such as diabetes.[4]

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Dose dependent response of symptoms, pituitary, and bone to transdermal oestrogen in postmenopausal women

SIR,—Though Drs Munro Peacock and Peter Selby (14 February, p 440) responded to some of our criticisms (17 January, p 181), most were not answered.

In Coope's study group[1] the average weekly number of hot flushes was 44 before treatment; the average one, two, and three months after withdrawal of oestrogen was 24, 56, and 60, respectively. Thus one month without oestrogen was insufficient for the pretreatment value to be regained. We do not understand the comment by Drs Peacock and Selby about the values in Coope's placebo group as we were discussing the effects of oestrogen withdrawal, not placebo treatment. Washout periods ranging from four to six weeks are clearly inadequate.[2-4]

Drs Peacock and Selby have misrepresented our study.[5] In agreement with all other investigators, we