SIR,—I wonder how many readers are worried by the BMA's position with regard to the white paper. Its publication (31 January) was not an unreasonable attempt to deal with the problems of the NHS, which for so long have been a major topic of debate for most professionals in the service. Most of this debate had been critical of the efficiency of the service, and most informed sources realised that simply pouring more money into the existing system was not going to produce the service we all desired. For the ideal to be obtained, changes in some of our traditional practices would be required.

To suggest that we were being unduly pressurised to accept all the features of the white paper is a major distortion. Certainly, in our district there has been no such pressure to opt out. In fact, quite the contrary, as we were told at the early meetings that our hospital would not be a candidate as our managerial skill was below that required to opt out. Full and wide reaching debates took place and at one with at least 70 consultants I was the only person to have read the leaflet that the BMA sent to doctors' surgeries. The document, by any standards, is a distortion, and it surprises me that some of our elder doctor statesmen have not seen fit to comment on its distortion, which clearly cannot have done other than frighten a vulnerable public. It has provoked at least one resignation from the BMA and prevented the application of two potential members in my own circle.

The expensive advertising campaign is again a travesty of truth, and the latest advertisement naming Mr Clarke is not worthy of our profession. Rather than adopting reasoned argument we seem to have opted for blatant "shroud waving." It was with some relief therefore that I looked forward to reading in Ms Lois Quam's article on the NHS review[1] what boasted to be a scientifically proved alternative to the white paper. I read this article several times to find a true practical alternative, and still I cannot see how it is so different from the proposals in the white paper. It does suggest that money will be required to provide the computer back up for audit to assess "outcomes" effectively. I am not sure that Ms Quam is correct in stating that the government is allotting only £250 000. The paper was accepted for publication in May; I wonder how much time at the sharp end of the NHS Ms Quam, from Minnesota, spent producing her scientifically proved alternative since the publication of the white paper.

I would like to think the association is going to change its attitude. It surely cannot be correct to spend £1·8m on the current advertising campaign, which by its very nature is more likely to confuse what is already an extremely difficult problem.

G HARTLEY

Bramhall,
Stockport SK7 3DL

1 Quam L. Improving clinical effectiveness in the NHS: an alternative to the white paper. *Br Med J* 1989;**299**:448-50. (12 August.)

**The Secretary writes: "The association is not pressing for the maintenance of the status quo; it recognises that improvements in the National Health Service are necessary. Months ago it proposed that we should extend properly funded and clinically led medical audit as well as the resource management initiative, after proper evaluation of the latter. These will enable all those concerned with delivering services to the patient to make best use of all the available resources, and provide information for a system to have 'the money following the patient.' It will also strengthen the ability to ensure that the NHS is funded adequately, at the same time safeguarding teaching, research, and training. We need also to set up effective services for preventing and controlling disease and promoting health. Our critics have ignored these proposals and the government has changed the ground rules for public discussion, as the recent *Panorama* programme showed. It places less emphasis on reasoned argument now and more on advertising and message enhancement."—ED, *BMJ*.

Angiotensin converting enzyme inhibitors and renal artery occlusion

SIR,—Drs J Main and R Wilkinson described an early renal artery occlusion occurring after the use of enalapril in a patient with atheromatous renal artery stenosis.[1] For several years its sister drug captopril has been known to cause renal impairment in such patients.[2] This drug probably acts by preventing efferent arteriolar constriction and reducing glomerular filtration pressure.[3] The effect is usually reversible on stopping the drug, provided that thrombosis has not occurred. With renal scintigraphy this effect has been used in selected patients as a screening test for renal artery stenosis.[4]

In the case described initial difficulty in controlling the patient's hypertension, coupled with the subsequent deterioration of renal function after starting treatment with captopril, should be taken as diagnostic of near critical renal artery stenosis. Such a lesion is quite compatible with a normal appearance on intravenous urography, particularly in older patients, in whom the lesion is often bilateral. We advocate further investigation of these patients. Renal scintigraphy before and after withdrawal of the angiotensin converting enzyme inhibitor followed by digital subtraction renal arteriography would have shown the left renal artery stenosis. Surgery is still the treatment of choice since most lesions are atheromatous and ostial. Angioplasty may be performed in unfit patients and those with more distal stenoses of the renal artery.[5]

We recommend a high index of suspicion in cases of refractory hypertension, particularly in smokers and patients with evidence of peripheral or coronary vascular disease.[6] Early investigation and treatment may avert the lethal consequences of subsequent thrombotic occlusion and allow effective treatment of underlying disease.

G STANSBY J SCOBLE
J R NOVELL P SWENY
G HAMILTON

Departments of Surgery and Renal Medicine,
Royal Free Hospital,
London NW3 2QG

1 Main J, Wilkinson R. Early renal artery occlusion after enalapril in atheromatous renal artery stenosis. *Br Med J* 1989;**299**:394. (5 August.)
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Screening for antibodies to anaesthetics

SIR,—Drs J Watkins and A Milford Ward have suggested that the incidence of serious reactions to drugs during anaesthesia is between one in 350 and one in 700.[1] This figure is based on the assumption that they assay only 5-10% of all the reactions in the United Kingdom and therefore is of dubious value. In our department 32 547 anaesthetics were given between 1 January and 31 December 1987. We have an anonymous reporting system for incidents related to anaesthetics, and in 1987 we had five reported life threatening reactions attributed to anaesthetics. This would make our incidence of serious reactions to drugs during anaesthesia one in 6500. Even if only half the serious reactions in the department were reported on our incident forms the incidence would be one in 3250.

Before we embark on an expensive screening programme we need to evaluate accurately the real incidence of serious reactions as well as define a population, if one indeed exists, of high risk patients. To screen all patients would add £35m to the cost of the NHS, and as there is no evidence that screening would be either beneficial or cost effective it would seem sensible to await the results of further research.

M SINCLAIR

Nuffield Department of Anaesthetics,
John Radcliffe Hospital,
Oxford OX3 9DU

1 Watkins J, Ward AM. Screening for antibodies to anaesthetics. *Br Med J* 1989;**299**:326. (29 July.)

Bone mineral response to brief exercise test

SIR,—Mr Michael C Beverly and colleagues suggest that bone mineral content in the forearm may be increased by brief periods of stressful exercise,[1] but we wonder if their conclusion is valid.

They reported an increase in bone mineral content of 3·1% in the exercised forearm over a period of six weeks, which was significantly different from baseline. The bone mineral content of the unexercised forearm, however, also increased during this time, so these increases may actually be unrelated to exercise. As they state in the study design that the unexercised arm was to serve as a control, it seems that the correct evaluation of their results should be to compare the change in bone mineral content in the exercised forearm with that in the unexercised forearm: there seems to be no difference between the two. Furthermore, six months after exercise was stopped the bone mineral content of the exercised forearm seemed to be both lower than that in the control arm and lower than the baseline value, although the number studied was much lower. If true, this would clearly be a worrying trend.

It may be that all the changes in bone mineral content are artefactual as Mr Beverly and colleagues did not correct for forearm fat content or bone width. We cannot understand why they state that they did not expect a change in these variables during the brief experiment when they obviously expected a change in others—namely, the bone mineral content and the grip strength. Apparent changes in bone mineral content in the forearm have been found to be erroneous because of changes in the composition of the surrounding soft tissue.[2] Further studies are clearly needed to establish whether brief exercise affects bone mineral content.

JOHN C STEVENSON
BELINDA LEES

Cavendish Clinic,
London NW8 9SQ

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AUTHORS' REPLY,—Our primary message was that forearm bone mineral content reflects grip strength. We agree that the so called unexercised