were hypertensive (>96 mm Hg). Of these, 447 981 (74·4%) had previously been detected, 36 626 (56·6%) were on therapy, and 25 857 (40·1%) were on therapy and controlled. Even if we set aside the possibility of natural attrition (levels of blood pressure and cholesterol are lower in the Tecumseh population study) and the contribution of North American enthusiasms for jogging, dieting, and stopping smoking, it is difficult to escape the conclusion that programmes of detection and control of high blood pressure are having a considerable effect here which is not yet apparent in the UK, and that the sooner more is done about it the better.

Our studies in general practice in Scotland[6] have confirmed the fact that 85% of patients on NHS lists consult their family doctor over a three-year period. A nurse or trained medical secretary can take blood pressure and even repeat the observations after four weeks before troubling the busy doctor, and in so doing achieve as nearly 100% cover in five years as would not matter. As to time and effort, our doctor colleagues told us that it took only a minute or so to take blood pressure and it was not worth troubling the nurse or receptionist.

The Hypertension Detection and Follow-up Program[7] has just reported that the systematic effective management of hypertension has great potential for reducing mortality for the large number of people with high blood pressure in the population, including those with "mild" hypertension. The North Karelia Study[8] has reported similarly. The outcome of the continuing UK trial of mild-to-moderate hypertension[9] is awaited with special interest as being based on a population among whom the incidence of coronary heart disease is rising—as opposed to declining in the other two studies. What is needed now is a clarion call to more action and a deaf ear to doubt.

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Surgery in outpatients

SIR,—The authors of your review article on day-stay surgery (8 December, p 1459) advocate studies of patients' reaction to this type of care. This work has already been undertaken, independently, by several groups of anaesthetists in this country.[1 2] The results should temper excessive enthusiasm for short-stay care of patients having general anaesthesia. Roughly a quarter of my patients undergoing termination of pregnancy had either headache, drowsiness, or uterine colic the next day and half did not feel like returning to normal activities at that time. Similar results have been found by other workers, using different anaesthetic combinations, in dental patients and in minor gynaecological surgery.[2] It is now my practice to offer patients overnight stay where the home circumstances are not suitable for early postoperative discharge.

Two groups of patients have been recommended for day-stay surgery who particularly warrant overnight care. They are children undergoing tonsillectomy and patients having laparoscopy.[3] The rapidity with which haemorrhage can kill a child after tonsillectomy means that expert postoperative care is essential for at least the first 24 hours after operation. I have found that pelvic laparoscopy can give rise to referred diaphragmatic pain lasting for up to three days after operation, and frequently this is distressing to the patient. Occasional patients request day-stay laparoscopy and I have never refused, on anaesthetic grounds, to allow them to go home. However, the likely pattern of recovery is always explained to the patient as part of my technique to minimise postoperative morbidity,[4] and I have found that half of these patients requesting day stay actually remain in hospital overnight.

I believe that we should use our knowledge of patients' recovery patterns to provide the best care for them and their sojourn in hospital should be decided on this basis.

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Indomethacin treatment of postural hypotension in autonomic failure

SIR,—We would like to confirm and extend the interesting and important findings of Professor G Abate and others (8 December, p 1466) on the increased systemic vascular resistance produced by indomethacin in autonomic failure.

Indomethacin is not always successful in the treatment of postural hypotension[1] and in an earlier paper that the authors refer to (reference 6) the diagnosis was not proved.[1] They stated that their patients had idiopathic Parkinsonism; but all seven males (though admittedly elderly) were impotent, three patients had defective sweating, and eight had bladder dysfunction. The fall in mean blood pressure of 34·8 mm Hg is greater than the blood pressure fall in patients with uncomplicated idiopathic Parkinsonism.[2] All these findings suggest that some of their patients had autonomic failure and multiple system atrophy (Shy-Drager syndrome). Although classification may be difficult, it is important to observe[3] the clinical, physiological, and biochemical criteria for differentiating between idiopathic Parkinsonism, idiopathic Parkinsonism with autonomic failure, and autonomic failure with multiple system atrophy. Professor Abate and his colleagues did not state the frequency, time, or duration of standing for blood pressure measurement. Patients with autonomic failure have such labile blood pressures that the precise conditions of measurement are important.

We are able to extend these observations (paper submitted for publication). In four patients with autonomic failure and multiple system atrophy, blood pressure was measured at 0600 and 1800 hours (times at which postural hypotension was greatest and least respectively) for seven days on no drugs and for seven days on indomethacin (25 mg thrice daily for four days and 50 mg subsequently). For the seven days before indomethacin, the average mean blood pressure in mm Hg ($\pm$SD) was: at 0600 hours—supine 92 ± 11, standing (5 minutes) 69 ± 15; and at 1800 hours—supine 102 ± 16, standing 76 ± 17. Indomethacin increased only the average mean supine pressure at 1800 hours (139 ± 23, $p=0{\cdot}05$, paired t test); other values did not change. The pressor sensitivity of our patients to intravenous noradrenaline and angiotensin II was increased by indomethacin. There were no detectable changes in blood volume.

Increased vasoconstriction caused by endogenous angiotensin II and noradrenaline from remaining nerve endings is probably one cause of the increase in systemic vascular resistance. Inhibition of prostaglandin synthesis may also be important for, in our patients, urinary prostaglandin excretion was greater than in normal subjects and was decreased by indomethacin. In cases of autonomic failure indomethacin may augment the beneficial effects of fludrocortisone,[4] since both drugs can increase blood volume and vascular smooth muscle sensitivity to noradrenaline.[5]

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Dietary fibre and blood pressure

SIR,—The paper "Dietary fibre and blood pressure" by Angela Wright and others (15 December, p 1541) fails to consider a more important dietary control of blood pressure—namely, salt intake.

It is possible to question the role of sodium (and other cations) as was done in an editorial[1] which confused the issue by discussing severe renal failure as well as essential hypertension and by not considering the normal person. There was already evidence[2 3] showing the importance of sodium intake in animal blood pressure control, and a well-controlled clinical study[4] had clearly shown a mean decrease in blood pressure of 7·7/4·4 mm Hg with very modest salt restriction. This fall, of the same order as that now reported by the Southampton group, was similar to that found[5] with an even less stringent sodium restriction (to a mean of 157 mmol(mEq)/day in 24-hour urine samples).[5] A larger fall (mean 13/11 mm Hg) was found in another series[6] when dietary sodium was reduced to 51 mmol/day without urinary control. Freis[7] reviewed the epidemiological evidence with some physiological and clinical studies and concluded that reduction of dietary salt to below 34 mmol/day would result in the disappearance of essential hypertension. We believe he is more nearly correct than the Southampton workers, who pay no

attention to sodium. We suggest that the changes in blood pressure that they saw were due to changing the dietary sodium, and that 24-hour urine collections would have revealed this.

Dietary fibre may not be irrelevant. Varying fibre amount and type might have altered the availability of cations for absorption in the bowel. This, however, is speculation and could not be detected by comparing tables of dietary sodium content. In any event, there is no case for relating this study to Burstyn's experiments with fat-enriched diets for rabbits (references 6-9 in the paper) until the major confounding variable of effective cation intake has been taken into account in both species.

The report also omits any mention of a search for an effect of age on blood pressure in the subjects, and does not refer to the age distributions of the various experimental groups. It is clear that blood pressure was measured in many different ways (at home, at work, at a health centre; with a standard and a random zero instrument; thrice weekly and on a single occasion) but we are not told how the various methods were distributed between the groups and during the crossover experiment.

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SIR,—Angela Wright and others (15 December, p 1541) recently reported that a high-fibre diet exerted a hypotensive effect on the blood pressure of normal volunteers, the mechanism of which is not known. The diet of primitive societies with a low prevalence of hypertension is not only high in fibre content but low in sodium and rich in potassium. Modest modification of sodium intake may be helpful in the treatment of mild hypertension[1] and potassium supplementation in the diet may exert an ameliorating effect on the development of hypertension and protect from hypertensive complications.[2] Wholemeal bread has double the potassium content of white bread and All Bran has a much higher potassium content than cornflakes.[3] It is possible that alteration of sodium and potassium intake by the volunteers in this study could have caused the blood pressure changes observed. In addition to information concerning the electrolyte content of the diets prescribed, any information on the changes in weight in these volunteers would be of interest, especially as potassium may have a natriuretic effect.[4]

It is surprising to observe such low standard errors in blood pressure measurement as those quoted in this paper when one notes the age range (18-60 years) of the volunteers and the fact that exercise was undertaken between each inflation of the cuff. On a low-fibre control diet the systolic blood pressure of 17 of their volunteers was 121·2±1·6 mm Hg and the diastolic blood pressure was 78·5±1·7 mm Hg. Using a London School of Hygiene "blind" sphygmomanometer we found that the supine systolic blood pressure of 19 male medical students, aged 20-22 years, was 123·4±2·4 mm Hg and the supine diastolic blood pressure was 66·4±2·3 mm Hg. Bigger standard errors are normally found in groups of subjects with the age range given in the paper.

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**We sent copies of these letters to Dr Burstyn, whose reply is printed below.—ED, *BMJ*.

SIR,—The letter of Dr Williams and his colleagues suggests that the effect we report (15 December, p 1541) may have been due to changes in the sodium intake of our subjects. Although we did not measure the sodium intake of the people participating in our study, we have no reason to suppose that they should synchronously change their food salting habits to confound our experiment. The high-fibre bread we gave our subjects would have supplied them with 31 mmol(mEq) sodium a day, while the low-fibre bread supplied 38 mmol/day (based on their mean intake of about 1 kg of bread a week). This difference of 7 mmol/day represents 8% of the rather low sodium intake recommended by the US Senate Select Committee on Nutrition[1] and less than 5% of the sodium intake typical of Western man, hardly sufficient to account for the effect we report.

Dr Williams and his colleagues are also dissatisfied with our blood pressure measurement techniques. All of the blood pressures apart from those of our 12 hypertensive patients were measured with Hawksley Random Zero sphygmomanometers in an office at the university. The two observers standardised their techniques carefully. Only one of these observers measured the blood pressures of the hypertensive patients at their own homes using an ordinary Accoson sphygmomanometer. We did not have large enough numbers of participants over the age of 30 to look for any effect of age on blood pressure. The average age of our subjects (with the exception of the hypertensive patients) was 26 and did not differ between experimental groups.

The letter of Dr Parfrey and his colleagues quite correctly asks how we managed to keep our standard errors small. The precision of our measurements was achieved by taking blood pressure readings three times weekly, each of which was itself the mean of three sphygmomanometer inflations. Between inflations subjects were asked to clench their fist sufficiently to prevent pooled blood from distorting subsequent diastolic pressure values. Furthermore, a group of 36 people whose systolic pressures were measured three times on a single occasion yielded the following result: first measurement—128·7±2·3; third measurement—123·5±2·2. These were compared with the blood pressures of 36 of our experimental volunteers (who were age matched with the above group) towards the end of their control periods (unfortunately, we had not preserved the three individual blood pressure values of our volunteers from the beginning of their control periods): first measurement—117·8±1·7; third measurement—117·8±1·6. The systolic pressures of people who are unaccustomed to blood pressure measurements drops significantly ($p < 0·001$) in the four minutes between the first and third measurement. This is not the case with the "trained" volunteers who have had two weeks of blood pressure measurements. The systolic pressures of the "untrained" people were higher than those of the "trained" people (first measurement—$p < 0·001$; third measurement—$p < 0·05$), although the diastolic pressures of the two groups were very similar. Finally, the standard errors for the blood pressure readings are considerably larger for the "untrained" group (which are similar to those of Dr Parfrey and others) than the "trained" group. It appears that repeated blood pressure measurements over a period of days improve precision in two ways: (*a*) by allowing the observer to take mean values and (*b*) by accustoming the individual to the measurement and hence reducing the variability of this measurement (that is, producing lower systolic pressures in the third reading of "untrained" people and lower mean systolic pressures in "trained" people).

The suggestion by Angela Wright and her colleagues that potassium may have played a part in producing our results is an interesting one. Our bran-enriched wholemeal bread contained more potassium than our white bread and would have provided (with the 5 g bran a day given to the volunteers) 18 mmol(mEq)/day potassium, compared with 5 mmol/day provided by the low-fibre bread. This difference of 13 mmol/day amounts to 20% of the average daily potassium intake.[2] That this may have affected our results is undeniable, although it required 135 mmol/day potassium to reduce the blood pressure in the two cases of salt-induced hypertension cited by Meneely and Battarbee.[3]

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Sclerosant treatment for hydroceles and epididymal cysts

SIR,—My experience of sclerosant treatment in the management of hydroceles and epididymal cysts differs widely from that reported by Mr H Thomson and Mr M Odell (22 September, p 704).

As a result of Moloney's[1] enthusiastic article[1] I set up a prospective study and treated approximately 75 patients between 1 January 1976 and 31 December 1978. The results in the first 56 patients were reported in detail earlier this year.[2] In summary, assessment one to two years after treatment showed 95% cure for hydroceles and 100% for epididymal cysts. Side effects and complications were minimal, none requiring surgical intervention. The few complications