vironmental and dietary factors, particularly salt.[1 2]

Our average salt consumption of 12 g per day is far in excess of physiological requirements. Expert committees in the United States and the United Kingdom have recommended reductions,[3 4] and evidence suggests that reducing salt consumption by 5 g per day would shift the population's blood pressure distribution by 9/5 mmHg,[2] more if extra potassium is added to the diet—for example, fruits and vegetables. Rose has estimated that a downward shift of the whole blood pressure distribution of a mere 2-3 mmHg would equal all the lifesaving benefits of current antihypertensive treatment.[5]

Dr Coope has concentrated exclusively on the high risk approach—that is, identification and treatment of patients with hypertension. I agree that basic changes are needed in general practice. Age-sex and hypertension registers are essential tools in managing hypertension. I also agree that doctors must learn to delegate tasks traditionally theirs. Nurses or other health personnel could detect and manage hypertension.

The high risk approach would seem reasonable for dealing with hypertension if there was a 100% detection and compliance. We know however that this is not so (24 March, p 903, p 906). In addition to the high risk approach, the general practitioner should advise patients to consume less salt and increase their consumption of potassium rich foods. In the long term this approach would seem to be the best way for controlling a mass disease like hypertension.

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3 US Senate Select Committee on Nutrition and Human Needs. *Dietary goals for the United States.* Washington: Government Printing Office, 1977.
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SIR,—I was interested to read the paper concerning the quality of care in managing hypertension by case finding in north west London by Dr G Michael (24 March, p 906) and also your leading article by Dr John Coope (p 880).

In 1981 and 1982 we carried out a systematic screening of all our men patients between the ages of 40 and 65 for hypertension. These patients were circularised by cyclostyled postcards after selection from the age-sex register. We set up a specific blood pressure surgery run twice weekly, where the blood pressures were recorded on a random zero sphygmomanometer by the wives of two of the partners who are state registered nurses.

Our practice consists in the main of social classes II and III and we have noticed in recent years increasing health consciousness among many of our patients. Despite this, we found that the take up rate for the invitation for blood pressure measurement was barely 50% of patients circularised, and perhaps more important was the fact that the patients who accepted our invitation were those very patients on whom we were more likely to have had a previous blood pressure measurement in any case. During 1983 we recircularised a random selection of the non-attenders in 1981-2 and, as expected, the response was negligible.

It seems therefore that to remedy poor blood pressure control in general practice patient education, perhaps by the mass media, will have to be adopted far more intensively so that the general public may realise the inherent dangers of sustained hypertension.

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Thyroid hormone concentrations after exogenous thyroxine

SIR,—Dr C J Pearce and Dr R L Himsworth (3 March, p 693) show that during conventional treatment of hypothyroidism with thyroxine a third of 122 patients who are well show raised total and free thyroxine concentrations. Their finding that only five out of 39 who had elevated total thyroxine also had elevated total triiodothyronine, and that eight out of 52 who had elevated free thyroxine also had elevated free triiodothyronine shows that supraphysiological thyroxine and free thyroxine concentrations are not necessarily associated with raised free triiodothyronine concentrations, and the correlation between the two is not impressive.

Recently we studied 20 hypothyroid women who had been on thyroxine treatment for at least three months.[1] All were clinically euthyroid. We noticed that the patients formed two groups: those with thyroid stimulating hormone secretion moderately suppressed, and those with thyroid stimulating hormone strongly suppressed (< 1 mU/l). Exogenous oral thyroxine raised within 30 minutes total and free thyroxine concentrations. The maximum was seen after two hours, when their concentrations were 10-15% higher than at the start. Total and free thyroxine concentrations exceeded the upper normal limits in five out of 20 and seven out of 20 patients respectively.

Because thyroxine does not seem to replace triiodothyronine from serum binding protein and because the conversion of thyroxine in peripheral tissues is evidently a slower phenomenon[2] and does not directly follow the fluctuation caused by daily exogenous thyroxine, it was not surprising that the serum thyroxine and triiodothyronine concentrations did not correlate well. Because Dr Pearce and Dr Himsworth made no attempt to relate the elevated total and free thyroxine concentrations with the timing of the thyroxine dose it is possible that at least in the marginally elevated cases (up to 15%) total thyroxine (26 out of 39) and free thyroxine (16 out of 52) concentrations can be explained because some patients had taken their thyroxine just before sampling.

We want to emphasise that it is essential to know when the patient has taken the thyroxine dose in relation to the blood sample. If the timing is not taken into account this increase may generate unnecessary concern in both the doctor and the patient. We suggest that the blood samples for thyroid hormone determination should be taken before the patient takes his thyroxine.

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Misleading laboratory findings

SIR,—The statements that a normal Schilling test excludes pernicious anaemia and that replacement treatment of hypothyroidism can be controlled by following the serum thyroxine concentration[1] have both been shown to be wrong in two recent *BMJ* articles. Dr D W Dawson and others (3 March, p 675) show that a normal Schilling test does not exclude pernicious anaemia. I have seen two patients recently with myxoedema, anaemia, low serum B_{12} concentrations, and normal Schilling tests. A normal Schilling test had been thought erroneously to exclude pernicious anaemia in these patients.

The second article is the one by Dr C J Pearce and Dr R L Himsworth (3 March, p 693). Adequate thyroxine replacement suppresses thyroid stimulating hormone production and both the thyroxine and triiodothyronine that may be produced by the failing gland. The thyroxine estimation in hypothyroid patients on thyroxine is measuring only the exogenous thyroxine. The sole function of the test is to confirm compliance (very important in itself).

A man aged 69 with myxoedema and Hoffman's syndrome was treated with thyroxine 200 μg daily. On this dose he was clinically euthyroid although the free thyroxine was 37·8 pmol/l (2·6 ng/100 ml) and 30·9 pmol/l (2·4 ng/100 ml) (normal range 9-23 pmol/l (0·6-2·2 ng/100 ml)). He spontaneously mentioned that he felt "really well—just like my old self" only for a short period after taking diclofenac for a tenosynovitis. Fenclofenac competes for thyroid binding sites

Effect of thyroxine ingestion on serum thyrotrophin and total and free thyroxine concentrations in hypothyroid patients. Values are means (SD)

	Group 1 (n = 11)		Group 2 (n = 9)		Normal values
	Initially	After 2 hours	Initially	After 2 hours	
Thyroid stimulating hormone (mU/l)	10·0 (5·4)	7·7 (5·9)***	<1·0	<1·0	<7·0
Thyroxine dose (μg) (range)	132 (40) (50-200)		178 (106) (50-400)		
Thyroxine (nmol/l)	119 (23)	126 (28)	138 (40)	155 (42)**	70-150
Free thyroxine (pmol/l)	18·6 (2·6)	20 (3)**	27 (10)	32 (13)*	9-23

*p < 0·05, **p < 0·01, ***p < 0·001 with paired *t* test.
Conversion: SI to traditional units—Thyroxine: 1 nmol/l ≈ 0·07 μg/100 ml. Free thyroxine: 1 pmol/l ≈ 0·07 ng/100 ml.