patient relationship, many doctors from the EEC countries have hardly any knowledge of the basic structures of the language.

It took so long to establish the much-needed PLAB test and it would be sad to think that patient care came second to political issues.

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Newly acquired right bundle-branch block

SIR,—In your leading article on right bundle-branch block (14 June, p 1392) you refer to a paper entitled "Acquired complete right bundle-branch block without overt cardiac disease" and state that right bundle-branch block most commonly occurs in people who have or will soon develop clinically apparent cardiovascular disease. In the study which you quote, in all 37 pilots with isolated right bundle-branch block the investigations were said to reveal a mildly raised end diastolic pressure, suggesting a diffuse abnormality of the ventricular myocardium.

I doubt whether practising cardiologists who have followed up cases of isolated right bundle-branch block for many years would agree with this statement. Anyone with experience of routine electrocardiograms in pilots or in insurance work knows that the majority of symptomless patients thought to have isolated right bundle-branch block as the only abnormality remain healthy over long periods of time. I have looked at the paper to which you refer and I suspect that the findings of so-called elevated end diastolic pressure are due to erroneous measurements of presystolic *a* wave in the ventricular pressure pulse without appreciation of the fact that it is the pre-*a* pressure which is the true measure of left ventricular function.

This is not to say that right bundle-branch block may not appear early in the course of progressive fibrosis of the conducting tissue or of coronary disease; but careful history taking and examination should differentiate these two groups, and the differentiation on auscultation and echocardiography between proximal and distal right bundle-branch block may prove helpful.¹

There must be many healthy subjects with isolated right bundle-branch block who will now have considerable anxiety based on what is, in my opinion, a misleading statement in a widely read journal and I think that you should withdraw it or at least play it down.

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¹ Brooks N, Leech G, Leatham A. *Br Heart J* 1979;41: 637-46.

*Dr Leatham raises a particularly important clinical point. The Framingham study, however, showed that cardiovascular abnormalities occurred in 55 of the 70 patients with newly acquired right bundle-branch block.¹ This was noted either at the time of diagnosis or during the long follow-up period. The problem lies, as always, in applying these data to patients, and many, as Dr Leatham points out, will remain healthy over long periods. This does not, however, alter the conclusion drawn by the authors of the

Framingham report that "right bundle-branch block most commonly occurs in people who have or will soon develop clinically apparent cardiovascular abnormalities." Nevertheless, we agree with Dr Leatham that careful history and examination should reveal any apparent cardiovascular disease needing treatment; certainly in the absence of cardiovascular abnormalities patients should be reassured and not have their activities restricted.

¹ Schneider JF, Thomas HE, Kreger BE, McNamara PM, Sorlie P, Kannel WB. *Ann Intern Med* 1980; 92:37-44.

Sodium and potassium intake and blood pressure

SIR,—I have read with interest the recent paper by Dr Peter Burstyn and others (23 August, p 537), who during short-term alteration of sodium and potassium intake observed no change in blood pressure in normal subjects. They considered that the relatively small reductions in sodium intake and increases in potassium intake that might be achieved through propaganda and changes in food processing are unlikely to lower the mean blood pressure in Western countries. One would not disagree with this statement when applied to normotensive subjects but is it true for hypertensive subjects also?

It has been proposed that the steady-state relationship between mean arterial pressure and urinary sodium output is normally very steep and that in essential hypertension this relationship is shifted to higher arterial pressure levels and the slope of the relationship is depressed.¹ In other words, normally there is little change in blood pressure following alteration of sodium intake but in essential hypertension there is a significant change in blood pressure following a change in sodium intake. Work in progress in this hospital supports that suggestion as does previously published work on alteration of sodium intake in patients with essential hypertension.

Studies emanating from the London Hospital have shown that a high-potassium diet with no added sodium for three months lowers blood pressure in patients with essential hypertension, a response that does not occur in normal subjects.² Similar results have been observed in the sons of hypertensive individuals but not in the sons of normotensive ones. These results and those of Dr Burstyn and his colleagues are at variance with those of Skrabal *et al.*,³ who observed that moderate salt restriction and high potassium intake did lower blood pressure and ameliorated stress-induced blood pressure rises in normotensive subjects, although no allowance was made for genetic susceptibility in the latter study. None the less it is possible that increase of potassium intake may have a greater hypotensive effect in patients with essential hypertension than in normal subjects.

Dr Burstyn and his colleagues have utilised epidemiological evidence of the lack of correlation between blood pressure and renal sodium excretion in some Western populations to support their conclusions. However, there are no well-documented examples of populations with either a high salt intake and low prevalence of hypertension or a low salt intake and a high prevalence of hypertension. Furthermore, Gleibermann⁴ reviewed the literature on a cross-section of the world's population and obtained a direct linear relationship between level of blood pressure and sodium intake. The inability to obtain clear relationships between blood pressure and sodium intake in adult individuals within one population can be explained in a number of possible ways. Firstly, the results from subjects susceptible to hypertension may have been swamped by the large number of individuals without any susceptibility to hypertension; secondly, in vulnerable subjects

salt may exert its pathological effect below the daily salt intake usual in Western society; thirdly, the kidney of the prehypertensive person may be susceptible to excess salt intake up to a certain age, after which other factors become more important; and fourthly, the pattern of salt appetite and intake may change with severity of hypertension. The cultural practices of civilised peoples involve not only high sodium but also low potassium intake. Recently a highly significant relationship between urinary potassium excretion and blood pressure was found in the United States, and no such association was discovered for sodium.⁵

In conclusion, it may be true that small reductions in sodium intake and increases in potassium intake do not lower blood pressure in normal subjects but may do so in essential hypertension. Then propaganda, changes in food processing, and partial substitution of potassium for sodium in condiments may have very beneficial effects on the blood pressure of subjects susceptible to essential hypertension.

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¹ Parfrey PS. *Br J Hosp Med* 1980;24:58-65.

² Parfrey PS, Vandenburg MJ, Wright P, *et al. Clin Sci* 1980;59:2P.

³ Skrabal F, Auböck J, Hörtnagl H. *Clin Sci* (in press).

⁴ Gleibermann L. *Ecol Food Nutr* 1973;2:143-56.

⁵ Gordon Walker W, Whelton PK, Saito H, Patterson-Walker R, Herman J. *Hypertension* 1979;1:287-91.

Education, screening, and genetic counselling in thalassaemia

SIR,—Drs E Silvestroni and I Bianco (12 July, p 149) commend a strategy of routine screening of schoolchildren for thalassaemia trait. Dr B Modell (7 June, p 1347), commenting on this, in fact referred to our small pilot study in the London Borough of Haringey, following which we abandoned plans to set up routine screening. Instead we advocated ready access to suitable hospitals.

In 1977, haemoglobinopathy screening was offered to 84 14-year-old children of relevant ethnic background at one school, at the same time as BCG. Sixty-three children took up the offer; 15 had suspected haemoglobinopathy, and this was confirmed in 12. The 15 children and their families were offered counselling twice at one-month intervals, and then six months later structured questionnaires were submitted to them. Only six children and families returned to complete the final questionnaires. The numbers were too small for statistical analysis, but we felt able to draw the following conclusions.

(1) Although the children, mainly born in England and with English as their mother tongue, could understand the counselling, their families had great difficulty. Views about racial prejudice, marriageability, etc, were not frequent, but when present were expressed with some vehemence.

(2) Even in this small sample we found an unexpected variety of types of haemoglobinopathy (for example, β -thalassaemia trait, haemoglobin-C trait, sickle-cell trait with α_1 -thalassaemia) among the various ethnic groups (seven West Indian, two Asian, three Turkish Cypriot, three Greek Cypriot). This made counselling particularly difficult; we had, for instance, to explain to a West Indian girl with β -thalassaemia trait and no knowledge of biology that she was at risk of having a child with sickle-cell- β -thalassaemia.

(3) Most children and their families under-