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.

JOY PARKINSON

Department of English as Foreign Language,
Southwark College,
London SE1 3NU

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.

AUBREY LEATHAM

National Heart Hospital,
London W1M 8BA

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.

P S PARFREY

Department of Medicine,
Charing Cross Hospital Medical
School,
London W6 8RF

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-

stood Mendelian inheritance well. They found it virtually impossible to accept the concept of the healthy carrier. The usual response was to agree politely that the child was perfectly healthy and did not need any treatment, but that children should be tested as young as possible—"so that it can be treated in time."

Since completing this study we have been aware of the comparative simplicity of counselling families who come for testing, or have already been tested, if there is prior knowledge of their condition. They might, for example, have relatives with a haemoglobinopathy or trait, might have been tested themselves in pregnancy, or might have been referred by a doctor of their own ethnic background.

We conclude that mass screening for haemoglobinopathy is unjustified, at least in this country. We advocate instead further health education about haemoglobinopathies together with financial support for hospitals recognised as able to offer both adequate testing and counselling. The UK Thalassaemia Society's booklet contains a list of such hospitals in the London area.

MARY A ROSSITER
MARIA SOLOMON

Department of Paediatrics

G W MARSH

Department of Haematology,
North Middlesex Hospital,
London N18 1QX

JEANNE D REEVE

Department of Haematology,
Prince of Wales's Hospital,
London N15

CATHERINE S PECKHAM

Department of Community Medicine,
Charing Cross Hospital, London W6 8RF

Vaginal bleeding in early pregnancy

SIR,—At the end of your leading article "Mortality in the British rubber industries 1966-76" (16 August, p 471), you remark, "At a time of financial stringency money should be spent on preventive measures only when the evidence is sound and not simply because doubts have been raised." It is a pity that in your leading article "Vaginal bleeding in early pregnancy" (p 470) you did not think about that warning before advocating hospital antenatal care, serial oestrogen estimations, ultrasonography, and cardiotocography for every woman who bleeds in early pregnancy.

You rather reluctantly concede that some of this bleeding may be "physiological." In these women the extremely expensive surveillance suggested is not only unnecessary but also meddlesome. You might usefully have suggested ways in which we general practitioners could differentiate between those women in whom bleeding compromises the fetus and those in whom it does not, instead of making a recommendation which (if we acted on it) would further overload hospital antenatal clinics.

Those who preach and practise this sort of defensive obstetrics might find it interesting to join the end of the queue at their next antenatal clinic and find out for themselves what it feels like to be processed in the modern manner. We all have the safety of the mother and child at heart, but surely we should have very sound reasons indeed, certainly sounder than those your editorial advances, for asking a woman to submit to the delays, anxieties, and indignities of high-technology hospital antenatal care.

It is also an awful waste of money. The

general practitioner is paid £37.90 for a woman's antenatal care. I dread to think what the corresponding hospital costs would be.

J C BIGNALL

Health Centre,
Newport, Dyfed SA42 0TJ

SIR,—Your leading article (16 August, p 470) omits to mention that there are infective causes of bleeding in pregnancy, notably *Trichomonas vaginalis* infection and gonorrhoea. At the speculum examination vaginal and cervical swabs may well reveal an avoidable cause of "threatened abortion."

D C MACDONALD BURNS

Marlborough Clinic,
Royal Free Hospital,
London NW3 2QG

Screening for the small-for-dates fetus

SIR,—I note with great interest the comment on my letter made by Dr J P Neilson and others (12 July, p 147). I suspect that both sides are partially correct. If we use crown-rump length $\times$ trunk area (CRLA) we identify correctly 94% of small-for-dates fetuses, and with them a number of normal ones which depends on the prevalence of small-for-dates fetuses in the population under test. Using crown-rump length $\times$ trunk circumference (CRLC) we identify correctly 89% of small-for-dates fetuses and a rather smaller number of normals. If we ignore the test altogether, simply treating all fetuses as small-for-dates, we identify correctly 100% of small-for-dates fetuses and a very large number of normals. Which of these is the optimal policy depends on the relative risks of allowing small-for-dates fetuses to go to term and premature delivery of normal ones.

Space does not permit me to deal with the problems of applying monitoring to identify the false-positives generated at the first stage of testing. Instead I wish to submit a mathematical model relevant to the situation over which we are arguing for your readers' scrutiny and criticism.

Definition of terms

k is the proportion of the tested population with the disease (prevalence);
 T is a general test with only two outcomes—positive and negative;
 S is the sensitivity of T , defined as the proportion of a population consisting entirely of positives that T will identify as such;
 s is the specificity of T , defined as the proportion of a population consisting entirely of negatives that T will identify as such;
 r is the probability of a morbid event occurring in a subgroup of the main population which is defined by the suffix of r ;
 D is the total number of morbid events occurring in a population of unit size.

Suffixes

1 and 2 are suffixes of T , S , s , and D referring to two tests. The suffixes defining the subgroups to which risks r pertain are:
 dis, Rx The group which has the disease and to which treatment is given.
 $-Rx$ The group which does not have the disease, but to which treatment is given.
 $dis,-$ The group which has the disease, but to which no treatment is given.
 $-,-$ The group which does not have the disease and to which no treatment is given.

If $S_1 > S_2$ and $s_1 > s_2$, T_1 is a better criterion of

management under all circumstances than is T_2 . The problem to which this model addresses itself is the situation in which $S_1 > S_2$ but $s_1 < s_2$. The object of management on the basis of T_1 and T_2 is to minimise D . We shall investigate under what circumstances T_2 is a better criterion of management than T_1 , even though it is less sensitive. We can identify four groups, and their sizes and their associated risks for the general test T .

Group	Size	Associated risk
True+	kS	$r_{dis,Rx}$
False—	$k(1-S)$	$r_{dis,-}$
True—	$(1-k)s$	$r_{-,-}$
False+	$(1-k)(1-s)$	r_{-Rx}

$$\text{Thus } D = kSr_{dis,Rx} + k(1-S)r_{dis,-} + (1-k)sr_{-,-} + (1-k)(1-s)r_{-Rx} \quad (1)$$

Writing two such equations for T_1 and T_2 respectively, subtracting and simplifying we obtain

$$D_1 - D_2 = k(S_1 - S_2)(r_{dis,Rx} - r_{dis,-}) + (1-k)(s_1 - s_2)(r_{-,-} - r_{-Rx}) \quad (2)$$

For T_2 to be a better criterion of management than T_1 , $D_1 - D_2 > 0$.

Making this substitution in (2) and simplifying we obtain

$$((1-k)(s_2 - s_1))/k(S_1 - S_2) > ((r_{dis,-} - r_{dis,Rx})/(r_{-Rx} - r_{-,-})) \quad (3)$$

provided $r_{-Rx} > r_{-,-}$. The inequality is reversed if T_1 is a better parameter than T_2 . For simplicity, we shall define the right-hand side of (3) as a risk ratio, R . Rearranging to make k the subject yields

$$k < \frac{(s_2 - s_1)/(S_1 - S_2)}{R + (s_2 - s_1)/(S_1 - S_2)} \quad (4)$$

It is thus possible to determine the prevalence below which greater specificity is at a premium over greater sensitivity.

Applying (4) to the parameters CRLA (T_1) and CRLC (T_2), we find that $(s_2 - s_1)/(S_1 - S_2) = 0.6$. As workers in the relevant field, Dr Neilson and his colleagues are better placed than I to estimate R , and thus to determine whether CRLA or CRLC is the better criterion of management at the prevalence of small-for-dates fetuses in their experience.

R H KLIPSTEIN

Neurology Department,
Whittington Hospital,
London N19 5NF

Fetal subdural haemorrhages presenting as hydrocephalus

SIR,—In their recent article Dr M J Robinson and others (5 July, p 35) suggest that hydrocephalus may result from subdural haemorrhage in utero secondary to maternal anticoagulation with warfarin. We are concerned about the lack of confirmatory information for the diagnosis of hydrocephalus. In addition, recent data from this institution prompts us to caution against the assignment of specific factors in the development of intracranial haemorrhage and hydrocephalus.

The determination of hydrocephalus was apparently made on fetal biparietal diameter alone. There was no mention of other ultrasound parameters which are required for this determination, nor were confirmatory necropsy findings reported. The diagnosis of fetal and neonatal hydrocephalus requires the evaluation of the ventricular:cortical ratios for the lateral ventricular system. An abnormally enlarged biparietal diameter is not diagnostic of hydrocephalus. In this case the biparietal diameter of 10.4 may have been the result of the proposed intrauterine subdural haematoma or several other possible factors.

We have been impressed with the advantages