worldwide reputation for our thoroughness and meticulous attention to detail. We have the same aim as clinicians, the benefit of the patient; and united we can extract the best from the manufacturing companies, eliminating dangerous and unsuitable products on the way. In case I am accused of merely fretting over my livelihood, I must point out that removing glucose completely from my laboratory's repertoire cuts our number of tests from about 120 000 to 105 000—and we have one of the smallest laboratories. To remove everything we do means a lot of work for someone somewhere, and is it really what the clinicians want?

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Self-monitoring of blood glucose in diabetic pregnancy

SIR,—We have read with great interest the article by Dr I Peacock and his colleagues (24 November, p 1333) and agree with them that diabetic pregnancy can now best be managed on an outpatient basis. Over the past 12 years we have adopted a very similar regimen for the 153 pregnancies with which we have dealt. The perinatal mortality when corrected for lethal congenital malformations has been 3%.

We have, however, on occasions been worried that the occasional patient was observing her strict dietary and insulin regimen only during the days when she was doing her own outpatient blood glucose monitoring; and more recently we have taken blood for glycosylated haemoglobin estimations each time the patient visits the hospital. We have found this estimation to be of great value in detecting those mothers whose blood glucose control is less than optimal. The mean glycosylated haemoglobin during the final trimester correlates well ($r=0.69$, $P<0.01$) with the cord blood C-peptide:glucose ratio, which has been shown¹ to indicate the degree of hyperresponsiveness of the fetal beta-cells induced by maternal hyperglycaemia. We have also found that the maternal glycosylated haemoglobin at the time of delivery correlates well with the mean blood glucose over the final trimester ($r=0.74$, $P<0.05$). We have further found that the glycosylated haemoglobin levels (estimated by the method of Fluckiger²) below 9% suggest that a good fetal outcome is likely and that maternal blood glucose control is good.

While supporting the principles outlined by Dr Peacock we do recommend the combination of outpatient blood glucose monitoring and regular measurement of maternal glycosylated haemoglobin levels as the best method of monitoring diabetic pregnancy.

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¹ Burke, B J, *et al*, *Lancet*, 1979, **1**, 1372.

² Fluckiger, R, Bergen, W, and Winterhalter, K H, *Diabetologia*, 1977, **13**, 393.

Interpretation of biochemical values

SIR,—I am writing in the hope that one of your readers may be able to clarify a mysterious and widespread medical practice—namely, drawing the conclusion that because an individual's value on some biochemical investigation, such as thyroxine estimation, is within the population normal range for the particular laboratory the function in question is de facto normal for that individual.

Elsewhere in biometrics, in particular in psychometry, practitioners distinguish between nomothetic (for example, the case in which the individual's value is compared with population norms) and idiographic (for example, the case in which the individual's present value is compared with his own personal norm or baseline) assessment. Thus it is not uncommon to diagnose pathological underfunctioning in the presence of values which may be above average for the population but which are known, or suspected on reasonable grounds, to fall below the norm for that particular individual.

Although writers such as Eastham¹ point to the lack of any necessary correlation between normal biochemical value and the patient's actual clinical state, I have yet to meet a doctor who appears to act on acceptance of the principle of idiographic assessment, even when baseline data obtained when the patient was asymptomatic are available. Even when the normal range for a particular substance is extremely wide, many complaints from patients whose values fall within that range often seem to be dismissed as unfounded. Can someone enlighten me why this is so?

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¹ Eastham, R D, *Biochemical Values in Clinical Medicine*. Bristol, John Wright, 1978.

Hypertension and general practice

SIR,—Dr H G Nicol's reaction (24 November, p 1368) to Dr D G Beever's little outburst (3 November, p 1137) prompted us to re-read the letter's words and to offer some history, some figures, and the following comments.

One should be very sure of one's facts before making sweeping generalisations. The truth is that all physicians were disgracefully unaware of the importance of detecting and treating hypertension for very many years. The life insurance companies were far ahead of us and long ago showed¹ that the early middle aged with diastolic pressures of 99 mm Hg and over had about a 15 times greater chance of having a stroke by their 65th year than the normotensive. By 1914 few could obtain a life insurance policy without having their blood pressure recorded.

One of us (MC) has a letter in the files of 15 years ago from a consultant physician advising that no treatment was necessary for a patient with a blood pressure of 220/115 mm Hg because a well-known professor of cardiology (named) advised no therapy until the diastolic pressure was 120 mm Hg or above. This was the teaching which most general practitioners (and consultants) then received. It is only within the last 10 years that the mass of opinion has changed. The interest of one of us (MC) was originally aroused when he realised that so

many hypertensives were presenting initially with a stroke. Could early detection prevent this? We have now known for some time that it can.^{2 3}

What are the present facts? Some light may be thrown on this question by the analysis of the screening figures obtained by one of us (GB), who is closely concerned with the present Medical Research Council treatment trial for mild-to-moderate hypertension. In 72 general practices 194 801 people aged 35-64 years were screened (the response rate being 74%). The total found to be mildly hypertensive (systolic pressure <200 mm Hg, diastolic ≥ 90 mm Hg <110 mm Hg) was 14 611 or 7.5% after four readings, while those with severe hypertension (systolic pressure ≥ 200 mm Hg or diastolic ≥ 110 mm Hg, or both) were only 0.95%. Moreover, of the total screened (194 801) 6.4% were found to be already on treatment and of these half had diastolic pressures of under 90 mm Hg.

Surely this does not show general practitioners to be disgracefully underdiagnosing or undertreating hypertension, especially with regard to detection, as a great number of these people had not seen their doctor for some considerable time. Let us therefore stop throwing brickbats and all work towards improving the detection of hypertension (and occult disease) in our general or hospital practices. This, of course, would be aided by any uninvolved general practices inquiring about the MRC trial (Epidemiology and Medical Care Unit, Northwick Park Hospital, Harrow, Middx HA1 3UJ) and maybe taking part if they are suitable.

A little more age and experience will change, we hope, Dr Beever's views.

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¹ Metropolitan Life Insurance Survey. New York, 1961.

² Leishman, A W D, *Lancet*, 1963, **1**, 1284.

³ Breckenridge, A, Dollery, C T, and Parry, E H O, *Quarterly Journal of Medicine*, 1970, **39**, 411.

Blood pressure measurement

SIR,—In the course of seven articles Dr Eoin O'Brien and Professor Kevin O'Malley consider multifarious sources of error in estimating—what? At times it is the instantaneous intra-arterial pressure (systolic and diastolic); at other times a casual reading is maligned and a mean or "representative" blood pressure, from continuous or from home recording, is preferred. Surely the errors resulting from variable definition are greater than those (fortunately often random or mutually opposed) which are addressed in the articles.

If we exclude continuous recordings of home recordings as impractical for the majority of diagnoses and follow-ups, should blood pressure be defined from samples of one, two, or more sets of readings? Their answers vary, though we are consistently advised to take three readings (one systolic and two diastolic) in each set. There are indeed good reasons for three—a single quirky value can be identified; and in this case three values are readily obtainable, with less bias too than if they were supposedly identical.

We also need to know, however, at what

intervals to repeat the sets, and whether they are preferably taken under identical or disparate conditions: let us say, one taken by a doctor, one by a nurse, and if both these are female then the third by a male—all in different premises and under different auspices. Three retrospective sets would be a reasonable, less satisfactory alternative and in fact such a matrix of readings—from the insurance doctor, the GP, and the specialist physician—is probably the commonest diagnostic base used in practice. A minimum of three sets would be needed too in view of selection effects when high values are screened for: "regression to the mean" is an important matter not even considered by Dr O'Brien and Professor O'Malley. In this case the last set of the series is the most significant.

A 3×3 blood pressure matrix may be the maximum which can be studied without unmanageable complexity. As the authors indicate, the readings taken with a patient standing give little extra information. The same can be said of exercise. The next requirement is to relate the blood pressure, redefined, to outcome in the short and long term, and with and without intervention. This would check not at first accuracy but relevance: a thing far more to be desired. Comparison with intramuscular manometry or continuous recording, or both, is also of interest but "validation" probably proceeds the reverse way to what we imagine.

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Proximal myopathy during beta-blockade

SIR,—Your correspondents Dr J C Forfar and others (24 November, p 1331) have drawn scientific attention to the proximal myopathy associated with beta-blockers. They give the impression that they feel this is a rare complication and the purpose of this letter is to correct this.

It is my experience that inquiry will elicit symptoms of weakness of quadriceps and sometimes of other muscles in buttocks and legs in at least 30% of patients having full beta-blockade. Many patients find that they are unable to walk up hills or even to hurry upstairs, and this applies even to the so-called cardiospecific beta-blockers. It is so frequent a complication that it should be a cause for inquiry in any patient receiving beta-blockers.

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Circulatory collapse after disopyramide and practolol

SIR,—We were interested in the report by Drs A D Cumming and C Robertson (17 November, p 1264) of circulatory collapse following injection of both practolol and disopyramide.

The adverse effects of both beta-blockers and disopyramide on the circulation have been widely reported in the literature¹⁻³ and caution has been advocated in the use of intravenous disopyramide in circumstances where cardiac function is already compromised.⁴ This category must certainly include patients with coronary disease and supraventricular tachy-

cardia who have already received large doses of practolol. An additive deleterious effect is only to be expected, and can hardly be regarded as a specific interaction between the two drugs.

When cardiotoxicity due to disopyramide occurs, atropine has been shown experimentally to be without beneficial effect⁵ and isoprenaline in relatively large doses has been considered the best available antidote.⁶

In cases of supraventricular tachycardia when it is judged urgent to restore normal rhythm, we would not recommend combinations of cardiodepressant drugs. It is our practice to use verapamil 0.15 mg/kg by intravenous injection, followed by synchronised DC shock in refractory cases.

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¹ *British Medical Journal*, 1978, **1**, 1010.

² Desai, J, et al, *Circulation*, 1978, **57-8**, suppl ii, 11.

³ Hayler, A M, Holt, D W, and Volans, G N, *Lancet*, 1978, **1**, 968.

⁴ O'Keeffe, B, et al, *Lancet*, 1978, **2**, 1208.

⁵ O'Keeffe, B, et al, *Cardiovascular Research*, 1979, **13**, 630.

⁶ O'Keeffe, B, et al, in *Proceedings of the Sixth World Symposium on Cardiac Pacing*, ch 6, 1979.

Prognosis after myocardial infarction

SIR,—Your leading article (24 November, p 1311) says that the common factor underlying all the physical signs that contribute to prognostic indices is the size of the infarction. This oversimplification makes it difficult to understand why some patients with apparently small infarcts are crippled by their illness and why some with large infarcts can be trained to be physically more able than they were before the illness.

It is probably more helpful to regard the physical signs as indicators of left ventricular dysfunction, which depends both on the severity of the acute infarction and on the amount of coronary arterial disease and myocardial fibrosis already present. The net effect of the new and old lesions can be gauged very well from the physical signs: an atrial gallop suggests that the mean left atrial pressure is 10 mm Hg or less, a pure third sound gallop that it is 20 mm Hg or more. Intermediate pressures are associated with intermediate types of low-frequency chest wall vibrations over the apex of the heart.¹

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¹ Taylor, D J E, and Nixon, P G F, *British Heart Journal*, 1972, **34**, 905.

Complete detachment of a Björk-Shiley prosthesis

SIR,—Dr D J Gawkrödger and Mr A K Deiraniya's report (1 December, p 1401) of a case of complete detachment of a Björk-Shiley prosthesis is an interesting addition to the growing catalogue of problems with this type of prosthetic valve.

The tragedy of the reported case is that the diagnosis was made at necropsy. The

radiograph accompanying the paper is said to show an abnormal position of the prosthesis, although this is unfortunately not clear in the reproduction. This feature may have been noticed only retrospectively, but even if this vital clue was ignored the diagnosis could almost certainly have been made by fluoroscopic screening of the valve. Björk-Shiley prostheses are well known to "stick," especially if anticoagulant control is inadequate; and valve dysfunction should always be suspected when pulmonary oedema develops suddenly in these patients. X-ray screening will demonstrate proper function of the prosthesis more accurately than simple auscultation.

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Policies on self-poisoning

SIR,—I would like to thank you first for the informative and factual, up-to-date leading article (3 November, p 1091) on the policies on self-poisoning applied in various hospital set-ups in this country. However, I was rather surprised to see that Dr N B Kreitman's view and work had not been well represented in your article. He has contributed papers based on his own work in Edinburgh to various scientific journals and has edited a book on parasuicide.¹ Neither did I see any mention of the splendid article he wrote in the *British Journal of Psychiatry* recently.² Is it because he is suggesting that psychiatrists must be involved in the treatment and assessment of patients presenting with one of the most challenging psychiatric phenomena of this decade? Has it now become unfashionable for the psychiatrists or psychiatrists in training to see the patients just because there are too many of them? I am also somewhat at a loss to understand how consultant psychiatrists of tomorrow will be able to train others in assessing such patients when they are not supposed to see these patients when they are in training themselves. I hope your readers will forgive me for asking one other naive question—is the concept of teamwork also dead or are only the junior psychiatric doctors chosen for exclusion with a specific purpose?

I am delighted by the finding of a few teaching units (which bear little resemblance in staffing either in number or in composition to the vast majority of district general hospitals) that the junior medical staff in general, social workers, and psychiatric nurses can be trained within a short time how to assess suicide risks of patients presenting with parasuicides. However, I would like to raise a couple of rather important issues for further debate within our profession. Firstly, diagnosis and treatment of a large number of common medical conditions can be taught to non-medically trained personnel, as is being done in some Third World countries by flowcharts. Should we introduce the same practice in this country too? Secondly, however much we are under pressure through the rising tide of parasuicide patients, I feel that we should not think of bypassing the problem by pushing it sideways. Instead, we have to face the challenge and try to establish the causes of this rising trend, for finding effective and constructive remedies for stemming the tide; and also acknowledge the fact that often parasuicide is the first storm cloud signalling future