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 psychologists 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