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'Keefe, B, et al, *Lancet*, 1978, **2**, 1208.

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

⁶ O'Keefe, 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

psychiatric morbidity looming large on the horizon.

A comprehensive psychiatric assessment and management at this point of crisis in a patient's life can have a very important preventive role, as well as providing for early detection of psychiatric illness in the making before the prognosis becomes unfavourable and unnecessary demands are made on psychiatric services, including long-term care.

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¹ Kreitman, N (editor), *Parasuicide*. New York, Wiley, 1977.

² Kreitman, N, *British Journal of Psychiatry*, 1979, **135**, 275.

Carbon monoxide poisoning

SIR,—I was interested to read the timely warning by Drs A J Crisp and K M Sherry (1 December, p 1438) regarding poisoning by carbon monoxide. It should be remembered that carbon monoxide is produced by the incomplete combustion of any of the common fuels, and that such incomplete combustion is likely to be due, in the domestic scene, to insufficient ventilation.

May I add a rider to the warning from Drs Crisp and Sherry? I am concerned that cases of subacute or chronic carbon monoxide poisoning may be going undetected. The symptoms—"headache, nausea, want of energy, a tired feeling, lack of concentration, irritability, breathlessness, tachycardia, giddiness, gastrointestinal disturbances, and ataxia"¹—are so all-embracing that patients presenting with this type of picture are likely to be regarded as neurotic and receive short shrift at the hands of casualty officers and others.

I would recommend inquiry about fires and other heating appliances in such cases. The diagnosis is easily made today by the use of the Dräger respiratory carbon monoxide test tube to analyse the patient's exhaled air. This device will give an instant, accurate estimation of the carbon monoxide content of exhaled air, from which the percentage of carboxyhaemoglobin in the blood can be derived. It is accurate over the range 2-35%—and it is also remarkably helpful in educating smokers.

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¹ *British Encyclopaedia of Medical Practice*, 1953, **12**, 126.

Carbon monoxide yield of cigarettes

SIR,—Dr Keith Ball's (22 September, p 731) suggestion that the Government should publish carbon monoxide (CO) yields of individual brands of cigarettes is based on three conclusions of doubtful validity.

Firstly, in stating that the evidence on CO as an important health hazard for smokers is growing he ignores the recent important retraction by Astrup¹ of his earlier studies² of the effect of CO exposure on normolipidaemic rabbits. Though there is evidence that CO can increase the severity of coronary artery disease in cholesterol-fed pigeons, rabbits, and monkeys, there is now no evidence that CO exposure in animals or birds fed on normal diets has any effect.³

Secondly, Dr Ball's conclusion that CO

levels have increased owing to the switch to filter cigarettes, which occurred mainly in the 1960s,⁴ is based on comparisons of yields of filter and plain cigarettes made much more recently and is in fact likely to be incorrect. The CO yield of unventilated cigarettes is strongly related to the permeability of the cigarette paper⁵ (and also to the size of the cigarette), and cigarette paper used currently in either filter or plain cigarettes is much more permeable than that of the typical old plain cigarette of 20 or more years ago. Gori⁶ has recently concluded that CO yields in US brands appear to have declined and it seems likely that considerable reductions have occurred in UK brands also—a view strengthened by the observation that a brand manufactured in 1937 delivered over 43 mg CO per cigarette, more than twice that delivered by present-day cigarettes.⁶ Dr Ball's fears that the "higher" CO levels may have led to more deaths from coronary heart disease are in any case at variance with the epidemiological evidence, which shows a reduced risk of coronary heart disease in smokers of cigarettes with reduced tar and nicotine levels⁷ and in smokers of filter rather than plain cigarettes.⁸

Finally, Dr Ball's statement that the publication of the tar tables stimulated the industry to lower tar levels of cigarettes ignores the fact that by far the major part of the tar reduction occurred *before* the publication of the first tar table in 1973 on the industry's own initiative. Average tar levels were 31.4 mg in 1965, 18.7 mg in 1973, and 17.4 mg in 1978.

There seems no strong case for publication of CO yields.

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¹ Stender, S, *et al*, *Atherosclerosis*, 1977, **28**, 357.

² Astrup, P, *British Medical Journal*, 1972, **1**, 447.

³ Turner, D M, *et al*, *Atherosclerosis*, in press.

⁴ Lee, P N, *TRC Research Paper No 1*. London, Tobacco Research Council, 1976.

⁵ Gori, G, and Ellis, R L, *Preventive Medicine*, 1979, **8**, 358.

⁶ Spincer, D, and Evans, N, Imperial Tobacco Limited Report No R417, August 1976.

⁷ Hammond, E C, *et al*, *Environmental Research*, 1976, **12**, 263.

⁸ Dean, G, *et al*, *TRC Research Paper No 14*, part I. London, Tobacco Research Council, 1976.

Rectal dosage of metronidazole

SIR,—May I draw the attention of your readers to an error in the ninth edition of *A Paediatric Vade-mecum*¹?

The rectal dose of metronidazole is incorrectly given (on p 191) as 500 mg three times daily for ages 2 weeks to 7 years. This is a toxic dose at the lower age range and should not be given. The May and Baker data sheet states that under 1 year the dose should be 125 mg (a quarter of a suppository), from 1 to 5 years 250 mg (half a suppository), and from 5 to 12 years 500 mg (1 suppository) eight hourly.

The suppositories are difficult to subdivide accurately and there is probably a need for lower-dose suppositories to be made available.

I am very grateful to Dr J Goulton of Messrs May and Baker for drawing my attention to this error and apologise to readers for its occurrence.

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¹ Wood, B (editor), *A Paediatric Vade-mecum*, 9th edn, p 191. London, Lloyd-Luke, 1977.

Addiction to smoking and drinking—our most serious preventable diseases

SIR,—Smoking and drinking give rise to poisoning of the organism by tobacco and alcohol. Poisoning of the organism constitutes a disease: a "morbid condition" of the body (Oxford English Dictionary). And because of their prevalence the addictions to tobacco and alcohol are by far the most serious avoidable diseases in the Western world—for both are preventable and curable. Yet we continue to evade taking effective action, on the misconception that they are merely habits and not addictive diseases. We accept unquestioningly as diseases their several complications, such as bronchial carcinoma and cirrhosis, but still fail to indict and control the primary addictive conditions.

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Trial of trimipramine for depression

SIR,—I never expected to see such an article as that by Dr J P R Young and others (24 November, p 1315) emanating from Dr William Sargent's old hospital.

It is surprising to find that anybody would nowadays take "a large, unselected group of depressed outpatients" for treatment without sorting them into recognised clinical categories.

There are many reports which indicate that the monoamine oxidase inhibitors (MAOIs) are the treatment of choice in so-called atypical depression. Does this investigation tell us whether trimipramine is as good as, better, or worse for the treatment of this group of patients? Patients are individuals and they come to us not to have their Hamilton or Beck or MRC scores lowered but to be made to feel better. Does this study show us how to do this?

The authors conclude that neither MAOIs nor MAOIs combined with trimipramine is the treatment of first choice in unselected patients with depression. Those of us who were treating patients in the early 1960s learned that. Since then we have learned something about the selection of patients.

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Management of haemorrhoids

SIR,—The advocates of stretching, banding, and freezing of haemorrhoids are many, but rarely do they define exactly the severity of their patients' symptoms, nor do they compare their results with management by injection therapy and haemorrhoidectomy.

The latest paper by Mr M R B Keighley and others (20 October, p 967)¹ is an example of this. An attempt has been made to divide patients into two groups on the basis of pressure studies, but their definition of the two groups is rather curious. Their first or high-pressure group were mainly men (99/108) and pain and bleeding were the principal symptoms (92/108), but their second or low-pressure group—"usually women with prolapse as a principal symptom"—contained 55 men and 53 women, while 56 had bleeding as a