in SI units, with comparative old units and conversion factors alongside. As well as this the equivalent result in old units was included on the pathology reports alongside the SI result for several months and even now the normal range is still printed on the form. As far as the patients are concerned they have probably benefited, since for each result one consults the normal range (until it is familiar) and perhaps converts the value to old units, thus having two to compare, although this exercise rapidly becomes unnecessary.

We do not particularly like the change. But we know of no accident occurring through misinterpretation, and despite a few snags initially the system is soon used fluently—as was decimal currency.

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Penicillin-"sensitive" methicillin-resistant *Staphylococcus aureus*

SIR,—The last sentence in Mr D F J Brown's letter (8 November, p 344) must be disputed. He states that "methicillin-resistant strains should be reported penicillin-resistant even if apparently penicillin-sensitive when tested at 37°C." Perhaps I am naive in thinking that the purpose of the laboratory sensitivity testing is to predict the outcome of therapy in vivo. But surely to discount apparent sensitivity in vitro at 37°C makes all sensitivity testing superfluous? It is possible to make any organism appear resistant to almost any antibiotic in vitro by adjusting the conditions appropriately. Thus *Escherichia coli* is always resistant to neomycin at pH 5, pseudomonas sensitivity to gentamicin is adversely affected by divalent metal ions, and co-trimoxazole is inactivated by thymidine.

For laboratory sensitivity testing to be useful, conditions as appropriate as possible to the in vivo situation are desirable. There is certainly doubt as to the reality of methicillin resistance in vivo. It would seem wise to report such few strains as "doubtful" rather than resistant.

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Long-term postinfarction treatment with practolol

SIR,—With reference to the multicentre international study on this subject (27 September, p 735) we wish to make the following comments.

It is gratifying that our findings^{1,2} of a reduction in the incidence of sudden death after acute myocardial infarction using alprenolol are confirmed by an independent study in which practolol was used. The authors of the multicentre study consider that beta-blocker therapy is especially indicated in patients with anterior wall infarcts.

In order to be able to apply the results and conclusions drawn from a study to other groups of patients the original patients must be representative of the general population

of infarct patients. It is impossible to assess the representativeness of the patients in the multicentre study without knowing the numbers of and reasons for the exclusions. There might have been a selection of patients in this study, as reflected by the low total mortality.

The main end points used in the study were death and reinfarction. Neither of these terms is clearly defined by the authors. We are, however, uncertain where data on the patients who died more than 24 hours after the onset of symptoms and/or the last dose of practolol are presented. A large number of patients dropped out of the study and 39% of the patients who died did so after withdrawal.

If sudden death is accepted as the main end point table VI shows that it is more important to discriminate according to blood-pressure levels than site of infarct. The reduction in the incidence of sudden death was the same among patients with anterior wall infarcts and those with posterior wall infarcts. It is probable that patients with large infarcts, with a complicated clinical course and a poor prognosis, have lower blood pressures than others on discharge from hospital. The retrospective demonstration of the importance of the diastolic blood pressure seems questionable since the mortality in the group given placebo was the same in patients with high and low pressure. The meaning of results from a discriminant analysis such as this must be prospectively validated. The material presented, therefore, does not permit the conclusion that beta-blockade is especially favourable in cases of specific infarct location.

A reduction in sudden mortality has so far been shown for two beta-blockers only, practolol and alprenolol. Other beta-blockers should be used for this indication only when clinical trials have shown that they possess the same property.

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¹ Wilhelmsson, C E, *et al*, *Lancet*, 1974, 2, 1157.

² Vedin, A, Wilhelmsson, C, and Werkö, L, *Acta Medica Scandinavica*, 1975, suppl 575, p 1.

Rheumatic heart disease in South Africa

SIR,—The study of rheumatic heart disease (RHD) in Soweto (23 August, p 474) is an epidemiological classic. Dr M J McLaren and his colleagues have exposed the dimensions of a rampant but preventable illness. Small wonder that they should have added this monumental task to their already burdensome duties. As Professor Barlow's guest at both Johannesburg General and Baragwanath hospitals I was struck by the unbelievable numbers of quite young Black patients with every conceivable kind and complication of advanced rheumatic heart lesion.

In other Western countries we still see RHD mainly in the poor, but less and less of it and usually not in an advanced stage until at least the third decade. Because the decline in RHD began even before we had effective antimicrobials we can relate it in part to improved conditions for the urban poor, including better medical attention. Primarily, however, high prevalence of

streptococcal infection and its consequences remain among the many scourges of poverty, afflictions that can be literally rooted out by socioeconomic amelioration. At the same time the physician's duty is clear: to press for massive intervention with public health measures including community education, constant case finding, and preventive treatment.

Dr McLaren and his colleagues propose a comprehensive preventive campaign against RHD while improving the lot of the Black community. For this these outstanding humanitarian scientists need no further documentation. Yet I suspect that they may need powerful support from their colleagues abroad because, as I have personally witnessed, the situation is appalling despite the efforts of the already overworked Johannesburg physicians.

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Merrison Report and overseas doctors

SIR,—Although the Merrison Report¹ was published some months ago there has been singularly little informed discussion in public about it and its recommendations. Some interested organisations have readily accepted it and the British Medical Association wants the implementation of its recommendations. Controversial measures have already been taken on its recommendation which may have far-reaching consequences for the NHS.

I would like to draw the attention of your readers to paragraphs 181-5 in part C of the report. These deal primarily with the assessment of overseas doctors—a euphemism in this report for coloured doctors from the New Commonwealth and Middle Eastern countries—and the term is often used pejoratively. The methods of assessment and conclusions are open to serious objection on the following grounds.

(1) There was not a single member from the minority ethnic groups on the committee. (2) None of the medical members of the committee had any reasonable and personal knowledge of the cultures of the overseas doctors or personal experience of working with them in Britain or abroad. (3) Out of 140 persons and organisations listed in appendix A from whom evidence was received, there was not one single immigrant doctor or organisation representing solely overseas doctors. (4) The objective and subjective evidence described in the report would not satisfy any careful and impartial investigator. This evidence was received from chosen sectors of society—that is, the royal colleges, whose membership is well known for preconceived notions and rigid attitudes against immigrants generally—but completely ignored the consumers, the patients whom both British and overseas doctors treat. In other words, complaints were received from all other interested sources except from patients or their organisations. (5) The deputising services and other agencies which have considerable experience of working with overseas doctors will not wholly agree with the "inescapable conclusions" reached by the committee. (6) The number of complaints to family practitioner committees do not show any unduly large proportion against overseas

doctors. The management committees of hospitals will also substantiate this figure.

The Merrison Committee has acted as prosecutor, jury, and judge of the overseas doctors, who now constitute 16% of general practitioners and 40% of junior hospital doctors in Britain. It is felt that the committee has given in to the pressure groups who wish to restrict immigration of doctors for political reasons. It is well known that there has always been a vocal group who felt that their bargaining position was weakened by the presence of overseas doctors here.

Is it too much to expect the Department of Health and Social Services and the BMA to dissociate themselves publicly from these findings?

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¹ Report of the Committee of Inquiry into the Regulation of the Medical Profession, Cmnd 6018. London, HMSO, 1975.

Miliary tuberculosis presenting with polymyalgia rheumatica

SIR,—Dr C R McGavin's letter (4 October, p 44) asks for a clarifying reply. The term "polymyalgia rheumatica" literally means "many aching muscles"; these are normally central and proximal limb muscles mainly around the limb girdles, and the presence of the syndrome is confirmed when the appropriate spinal and proximal muscles or joints are found to be restricted. The syndrome has many causes, most of which produce elevation of the erythrocyte sedimentation rate (ESR). In the case described (miliary tuberculosis) the syndrome was suspected from the history and signs before the ESR became elevated. Once its cause was found to be widespread tuberculous infection steroids were not indicated and not given. The symptoms and signs resolved following a course of antituberculosis therapy.

The fact that polymyalgia rheumatica is a syndrome with many causes needs emphasis. As many patients arrive in clinics without blood test results it is preferable to confine definition of the syndrome to its clinical features.

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Renal stones and coronary heart disease

SIR,—Coronary heart disease and renal stones are both disorders increasing in frequency in the Western world, to some extent presumably owing to the raised standard of living. An association between the two diseases has been reported, with a higher stone prevalence in patients with myocardial infarction.^{1,2} Risk factors for coronary disease have not been systematically investigated in renal stone formers, but raised serum cholesterol¹ and high blood pressure³ have been reported. Over-consumption of vitamin D has also been suggested as a precipitating cause.²

In a health survey of more than 2000 middle-aged men in Uppsala, Sweden, 13.7% were found to have a history of previous renal stone disease.⁴ Some of the clinical and laboratory findings in stone

	Stone formers	Controls
Serum cholesterol (mmol/l)	6.1 ± 1.1	6.0 ± 1.0
Serum triglycerides (mmol/l)	17.5 ± 10.0	17.9 ± 11.1
Serum urate (mmol/l)	0.25 ± 0.06	0.26 ± 0.06
Glucose tolerance (K value)	1.78 ± 0.70	1.68 ± 0.69
Weight index (actual/ideal weight)	1.09 ± 0.15	1.09 ± 0.13
Systolic blood pressure (mm Hg)	134 ± 19	131 ± 15
Diastolic blood pressure (mm Hg)	85 ± 12	83 ± 10

Conversion: SI to traditional units—cholesterol: 1 mmol/l $\approx$ 38.6 mg/100 ml; triglycerides: 1 mmol/l $\approx$ 88.5 mg/100 ml; urate: 1 mmol/l $\approx$ 16.8 mg/700 ml.

formers and their matched controls are listed in the table. Only in diastolic blood pressure was a significant difference found ($P < 0.05$). This was primarily due to a greater number of individuals in the highest ranges. Thus 7.4% of stone formers had an untreated diastolic pressure of 105 mm Hg or more compared with 2.0% of controls. Stone prevalence was also investigated in a consecutive series of 102 male survivors of myocardial infarction and was found to be 12.8%, which is much the same as in the health survey, though the survivors were somewhat older (mean age 59 years). The vitamin D intake in survivors of myocardial infarction, renal stone formers, and their matched controls was also studied. The average daily consumption was below 10 μ g in all groups and there were no differences between them.

In conclusion, the coronary risk profile was similar among renal stone formers and carefully matched controls in this study of a well-defined male population. Furthermore, individuals surviving myocardial infarction did not have a higher prevalence of renal stones than expected. This is in contrast to some other reports. Compared with the Norwegian studies^{1,2} cited above differences in dietary habits, including vitamin D consumption, could be part of the explanation for this contrast.

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Health Service administration

SIR,—Mr P H Lord (15 November, p 405) is unfortunately not alone in talking of the "tiers" of district, area, region, and Department of Health and Social Security as though these were comparable bodies at different levels. Mr Lord may be equating "districts" with the previous hospital management committees. The only levels of authority are the Secretary of State, the regional health authority, and the area health authority. "District" is a geographical part of an area in which the services are managed by the district management team—a group of officers and clinicians who are accountable to the AHA.

Mr Lord describes the area as a "political gimmick." Areas were deliberately created coterminous with the new counties (or in conurbations with the metropolitan districts) to facilitate collaboration with the

local authorities with which the NHS shares responsibility for services for large groups of patients with real needs, such as the elderly, psychiatrically ill, and handicapped. The AHAs (with their family practitioner committees) have replaced a much larger number of local health authorities, hospital management committees, and executive councils. To abolish the lowest level of authority, as Mr Lord suggests, would make control remote indeed. It would also mean a large number of senior officers directly accountable to the RHA—a daunting prospect even in a region as small as the one in which he and I work.

Finally, Mr Lord assumes that the demise of the HMC has resulted in the loss of the voice of the consumer. Has he overlooked the new community health councils, whose sole job is to represent the viewpoint of patients in each district?

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Antibiotic diarrhoea

SIR,—In your leading article on this subject (1 November, p 243) you omitted to mention a recent report¹ on the successful use of cholestyramine in lincomycin- and clindamycin-induced pseudomembranous enterocolitis (PME). These drugs cause diarrhoea in 10-50% of patients treated^{2,3} and PME allegedly in 10%.⁴

These are such widely used and useful drugs, especially in the treatment of Gram-negative anaerobic infections, that it would be worth while doing a double-blind study to investigate the use of bile-salt-binding resins and their mode of action in antibiotic-induced diarrhoea and PME.

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Clonidine overdose

SIR,—A man aged 44 years was given 50 tablets of clonidine, each of 100 μ g—that is, a total of 5000 μ g—dissolved in his soup. He slept heavily that day and sweated a lot. The following four days were troubled by diarrhoea, after which he consulted me. Clinical examination revealed no abnormality and his blood pressure was 120/80 mm Hg. His usual range was 180/110-150/100 mm Hg on 100 μ g of clonidine three times a day.

The *British National Formulary* includes constipation and fluid retention among the side effects of clonidine. This case suggests that the reverse may also be true. Forced frusemide diuresis in clonidine overdosage was recommended by Dr S N Hunyor and others (4 October, p 23). I support the view of Dr L M H Wing and his colleagues (15 November, p 408) that this is unnecessary as the excess clonidine is excreted in the urine and eliminated extrarenally.

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