remarked on by Professor W S Peart and Dr W E Miall (19 January, p 180) could well be an effect of social class rather than skin colour, for in Britain also there is a very steep gradient in stroke mortality by social class,² and a smaller one for ischaemic heart disease; so that here also one would expect a disproportionate effect on social classes IV and V in any controlled study.

General practitioners who have attempted systematic case finding, follow-up, and recall have mostly concentrated on those under 65 and above a diastolic threshold of 105 mm Hg. This policy was justified by evidence, but also pressed on us by circumstance. We simply did not have the time, the nursing and office help, or the clinical traditions to support any bigger undertaking. The group with diastolic pressure over 105 mm Hg represented about 4% of total practice populations at all ages, or about 7% of adults aged 20-64. As Dr D G Beevers rightly said (12 January, p 108), very few practices are attempting even as much as this; and about half of these people are still untreated, or at least uncontrolled. Now we are faced with a task three times as great.

I sympathise with Dr S L Barley's exasperation (12 January, p 109) with hospital programmes for screening and following up hypertensives in the community. Such schemes are wasteful and lead to further fragmentation of care and abdication from clinical responsibility in general practice. But I cannot share his outrage: if general practitioners in Brighton were already doing the necessary work, the hospital scheme would never have started. Such evidence as we have (from a study in the Lethian Health Board Area³) suggested that about 18% of general practitioners referred nearly all their hypertensives to hospitals and another 18% dealt with them almost entirely themselves; in uncomplicated cases of mild-to-moderate hypertension 84% followed up patients themselves after initial hospital assessment and 16% preferred shared care. Achievement of target pressures seems to be about equal between general practitioners and hospitals,⁴ and case finding and follow-up are about equally bad in general practice and in hospitals.^{5,6} It is futile for general practitioners (or anyone else) to claim exclusive rights to territory they are unable or unwilling to occupy.

I think and hope that we are more unable than unwilling, but time is running out. The job must be done by someone, and we are running out of legitimate excuses for inaction. We are not discussing an optional luxury that might reasonably be omitted from the threadbare residue of our National Health Service, nor is it any longer a suitable subject for token experiment by a few zealous enthusiasts. Either we shall get a large new public investment in a nationwide programme of expanded community care or the breach between what is done and what could be done will widen to an extent intolerable to an informed public—and we must see to it that the public is informed.

Such investment should be made through general practice, but on a scale that must change the nature of our contract. The genuine teamwork required and the shift from passive response to symptoms to active pastoral care imply fundamental changes (from both sides) in our relationships with our clerical staffs and our employed and attached nurses, with our colleagues in community medicine and specialised hospital departments, and with the communities we serve. If we cannot learn to relax our unrealistic claims to total autonomy

and immunity from peer or consumer criticism the investment will be made elsewhere, or (more probably) not at all.

Perhaps this is the jolt that will set us moving.

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¹ Hypertension Detection and Follow-up Program Cooperative Group. *JAMA* 1979;242:2562-77.

² Hart JT. *Practitioner* 1979;223:662-5.

³ Fulton M, Kellett RJ, Maclean DW, Parkin DM, Ryan MP. *J Roy Coll Gen Pract*; 1979;29:583-7.

⁴ Parkin DM, Kellett RJ, Maclean DW, Ryan MP, Fulton M. *J Roy Coll Gen Pract* 1979;29:590-4.

⁵ Heller RF, Rose G. *Br Med J* 1977;i:1441-2.

⁶ Heller RF, Rose G. *Br Med J* 1977;ii:1442-4.

SIR,—Dr D G Beevers (12 January, p 108) states that "research-minded general practices are few and far between," and for this reason widespread screening for hypertension in general practice is unlikely to be successful. The quality of general practice in inner cities and particularly London has declined to a level that the Royal Commission on the National Health Service reported that in such areas "the NHS is failing dismally to provide an adequate primary care service to its patients"¹ and recommended that research opportunities should be encouraged.

A study is under way in this area aimed at detecting and treating serious hypertension with a view to reducing the incidence in the community as a whole of hypertension-related morbid events—the City and East London pilot programme to detect serious hypertension. This study, designed by hospital-based physicians, clinical epidemiologists, and academic community physicians in co-operation with general practitioners and instigated by the department of community medicine at the area health authority, has been able to recruit 22 general practitioners from eight general practices in the East End of London. Research-minded general practitioners are available and perhaps it is a major function of community physicians to seek out and find them.

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¹ Royal Commission on the National Health Service. Report. London: HMSO, 1979:89.

Labile hypertension

SIR,—Your leading article (5 January, p 4) "Labile hypertension" is a problem in life assurance underwriting, since it is difficult to disregard an elevated reading even if it is suspected of being due to "examination apprehension."

Underwriters tend to have the blood pressure retaken—usually three readings—and work on an averaging basis, unless there are clear indications that the initial reading was what some have termed a "rogue" blood pressure reading. Rechecks often produce further doubts since, although some applicants may become more relaxed as they grow used to having their blood pressure taken, others, fearing that the recheck implies something wrong, grow more apprehensive and their recheck readings may be even higher.

Another difficulty is that readings which are only slightly raised, and are often not con-

sidered worth investigating and treating by medical practitioners, may be rateable from the underwriting viewpoint. The medical practitioner can "keep an eye on it," reacting as appropriate; but the life companies have only one opportunity and their decision, often for high, life-long cover, is then binding.

Labile, symptomless, untreated, arterial hypertension is the most resented of all the reasons for loaded premiums. I wonder if the research highlighted in your article indicates that the benefit of the doubt may be given more readily to applicants with labile hypertension?

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Proximal myopathy during beta-blockade

SIR,—The letter of Dr M Uusitupa and colleagues (19 January, p 183) quite rightly identifies the importance of occult thyrotoxicosis in the differential diagnosis of proximal myopathy and how this diagnosis may be masked by concurrent administration of beta-blocking drugs.

We must stress, however, that thyrotoxicosis was excluded in the patient reported by us (24 November, p 1331) by the finding (documented in the report) of normal levels of circulating thyroid hormones and, more importantly a normal plasma thyrotrophin response 20 minutes after intravenous thyrotrophin releasing hormone (200 µg) was given.

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SIR,—Your correspondent Dr R Stone (15 December, p 1583) rightly points out that many patients complain of muscle weakness while taking β -adrenoceptor-blocking drugs. While this may not be of importance when angina is the limiting factor for muscular exercise, the increasing use of beta-blockers in the treatment of hypertension means that many people who may feel perfectly well untreated are having symptoms when on treatment.

Current evidence suggests that this muscle weakness may be the result of an impairment of energy metabolism through retardation of the catecholamine-induced increase of muscle glycogenolysis. It is believed that the breakdown of glycogen to glucose in skeletal muscle is mediated via β_2 -receptors. One would predict therefore that the use of non-selective beta-blockers would inhibit this breakdown and thus reduce the supply of glucose for energy production in working muscle cells. Lack of substrate would be the explanation for the commonly seen leg fatigue during exercise during non-selective beta-blockade.

Franz and Lohmann¹ have shown marked differences between non-selective (pindolol) and selective (metoprolol) beta-blockers in hypertensive patients who exercised both moderately and to exhaustion. They found that blood glucose fell to hypoglycaemic levels during exercise in patients on pindolol, implying a considerable reduction in physical capacity. This impairment of physical fitness

was particularly marked in one patient, who had been following an intensive endurance training programme for several years. On pindolol he was unable to complete his training programme and complained of an insatiable appetite, presumably owing to low blood glucose levels. On metoprolol these changes were not apparent. Similar differences in terms of working capacity have also been shown by Fellenius *et al*² between the non-selective beta-blocker propranolol and metoprolol.

Fatigue is a commonly encountered problem in patients treated with beta-blockers. On the basis of these findings it would appear that in these circumstances the predominantly cardio-selective beta-blocker metoprolol may be preferable since it would appear to have less effect on energy metabolism during physical stress.

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¹ Franz IW, Lohmann FWZ. *Cardiology* 1979;68:503.
² Fellenius E, *et al*. Paper presented to 4th International Symposium on Biochemistry of Exercise, June 1979.

Smoking and doctors' mortality

SIR,—We have studied the recent paper by Mr P E Lee (15 December, p 1538), and offer the following alternative "summary and conclusions":

From 1951 to 1971 male doctors reduced their cigarette smoking more than did men in social classes I and II combined. During 1970-2 349 doctors died under the age of 65 from lung cancer, bronchitis, coronary heart disease, or stroke—diseases where smoking has a causal role. If their age-specific death rates for this group of smoking-related diseases had improved no more during the 20 years than did those of men in classes I and II, 445 doctors would have died in 1970-2; the actual total of 349 thus represents a "saving" of 96 deaths (22%), and includes a "saving" of more than a quarter of the lung cancer deaths that might otherwise have occurred. These smoking-related diseases accounted for a little over one-half of all deaths among doctors during 1970-2. For all other causes combined their death rates improved somewhat less than did those for men in classes I and II, and doctors fared much worse for the group of stress-related causes comprising suicide, cirrhosis of the liver, and accidents and poisonings: here they experienced a "loss" of 51 deaths (69%). There are no grounds for suggesting that doctors' reductions in smoking contributed to the coincidental rise in their death rates from stress-related causes—indeed, Doll and Peto¹ have shown that among doctors the death rates of light and moderate smokers for this group of causes are only just over half as high as those of heavy smokers, and that those of ex-smokers and non-smokers alike are less than half as high.

The marked relative improvement in mortality from smoking-related diseases among doctors contrasts with the relative worsening of their mortality from other causes, and provides clear evidence of the benefits derived from reducing or giving up smoking.

The difference between our conclusions and those of Mr Lee results mainly from different interpretations of the same basic data, but we would also like to comment on his methods. In his table I (and III) two schedules of standardised mortality ratios (SMRs) are presented which are each based on different standard populations. Direct comparison between two SMRs based on different standards is invalid, and even indirect comparison of relative differences can be misleading. For example, although the SMR for coronary heart disease in classes I and II fell

from 110 in 1949-53 to 89 in 1970-2 (his table III), age-specific death rates increased at all ages over that period.

We are unable to follow the rationale for the procedure carried out in Mr Lee's table II. Would it not have been more appropriate to compare observed deaths in 1970-2 with those expected if the 1949-53 rates had persisted by simply applying the age-specific 1949-53 rates to the 1970-2 age-specific population of doctors? We have made this comparison in the accompanying table (A), combining causes of death into three groups to obtain numbers sufficient to form the basis of age-specific rates.

Mortality in doctors

A. Observed deaths among doctors aged 25-64 during 1970-2, and expected deaths had doctors' age-specific death rates remained at their 1949-53 levels:

	Obs	Exp	Saving	Loss
Smoking-related causes	349	454	105	
Stress-related causes	125	90		35
Other causes	186	314	128	

B. Observed deaths among doctors aged 25-64 during 1970-2, and expected deaths (E3) if doctors had shown the same percentage improvements in age-specific death rates as men in classes I and II:

	Obs	E3	Saving	Loss
Smoking-related causes	349	445	96	
Stress-related causes	125	74		51
Other causes	186	173		13

We have also recalculated E_3 values in his table IV by obtaining the numbers of deaths expected among doctors if they had experienced the same percentage changes in age-specific death rates between 1949-53 and 1970-2 as men in social classes I and II. The results we obtained (table, B) are broadly similar to those of Mr Lee, but only because the four populations that he studied had comparable age structures. The use of successive manipulations of SMRs to obtain expected values yields results which should be interpreted with caution. His analysis would have been built on more secure foundations had he, for example, taken the basic data and applied the technique of direct standardisation.

In our opinion this paper is misleading. Its publication in the *BMJ* has doubtless lent it authority not only in the eyes of many readers but also in the eyes of the media. The author's conclusions were widely reported in the press, and the general public has been given the false impression that reducing smoking confers little benefit.

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¹ Doll R, Peto R. *Br Med J* 1976;iii:1525-36.

SIR,—We feel unqualified to comment on the statistical analysis in Mr P N Lee's paper "Has the mortality of male doctors improved with the reductions in their cigarette smoking?" (15 December, p 1538); however, his treatment of the relationship between smoking and stress has neglected some important points, and as a consequence is misleading.

The claim that "high-need smokers . . . may smoke to relieve stress" derives from a factor

analysis of smokers' self-reports of when and why they smoke. A look at comparable data suggests that some of the stress which is relieved by smoking has been induced by cigarette deprivation.¹ Behavioural experiments indicate that increases in cigarette smoking which often occur with stress are due to compensations for an associated increase in nicotine excretion, rather than to the direct ability of cigarette smoking to alleviate stress.² Although smokers who restrict their nicotine intake are more responsive to a noxious stimulus than are either unrestricted smokers or non-smokers, the unrestricted smokers show just as much annoyance as do the non-smokers.³ Smoking does not help these smokers cope better than the non-smokers do without benefit of tobacco.

Mr Lee proposes that "people who had reduced or who smoked cigarettes with lower deliveries . . . may no longer have found that cigarette smoking offered the protection against stress that it used to" (italics ours). Smoking should not be portrayed as a way to achieve protection from stress when evidence indicates that in high-need, addicted smokers smoking remedies mainly the stress for which it itself is responsible. Though smoking may sometimes directly reduce stress,⁴ the alternative, addiction-based account presented here should not be ignored. Not smoking may be hard on the addicted smoker, but this alone does not support the implication that one is protected from the hosts of common, non-chemical stresses by a steady diet of cigarettes.

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¹ Russell MAH, Peto J, Patel UA. *J R Statist Soc A* 1974;137:313-2.

² Schachter S, Silverstein B, Kozlowski LT, Perlick D, Herman CP, Liebling B. *J Exp Psychol: Gen* 1977; 106:3-40.

³ Schachter S. In: Krasnegor NA, ed. *Cigarette smoking as a dependence process*. NIDA Research Monograph 23 (DHEW publication No (ADM) 79-800), 1979:123-33.

⁴ Gilbert DG. *Psychol Bull* 1979;86:644-57.

Carbon monoxide yield of cigarettes

SIR,—I find it very difficult to understand Mr P N Lee's reluctance to see the publication of carbon monoxide levels (15 December, p 1584), unless it is that the Tobacco Advisory Council does not wish to have further attention drawn to the presence of a known toxic substance in cigarette smoke. Although Mr Lee states that cigarette tar levels were lowered before publication of the Government tar and nicotine tables, it is probable that anticipation of publication stimulated the industry to lower them. If impending publication did the same for carbon monoxide this would be all to the good.

Mr Lee's arguments for the Government not even informing the medical profession of the levels of a known toxic gas in cigarette smoke are most unconvincing. If Sweden can publish them why can't we?

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