when groups of people with pneumoconiosis are compared with others the differences in symptoms, function tests, and expectation of life are small. What I object to is the assumption that all patients with simple pneumoconiosis respond to dust in their lungs in the same way. Can the statisticians tell us how many may, for example, develop secondary emphysema and disability without upsetting the statistical conclusions that, as a group, they are no worse off than non-pneumoconiotics? It is time we accepted that the post-mortem studies of Lyons *et al.*¹ are the best available instead of trying to discredit them, mainly, it seems, because they do not support current dogma. Does Professor Morgan really believe that they are more biased than some of his studies in which ex-miners and retired miners are not included?

Most of the letter from Dr. McGowan and his colleagues deals with the aetiology of chronic bronchitis in miners and is a distraction from my main theme. As they seem to feel that my criticisms of panels are unjust I would like to offer four helpful suggestions which they have the power to implement.

(1) That when recording the results of ventilatory tests the expected readings should be added. This should reduce the frequency with which abnormal readings are reported to be normal. Dr. T. J. G. Phillips may recall a recent case in which readings of 70% of the expected were repeatedly claimed to be normal.

(2) That the frequent practice of writing statements such as "disability slight" under the readings should be abandoned—that is, disability should not be determined solely by these.

(3) That chronic bronchitis of various degrees of severity should not be diagnosed on naked-eye examination of post-mortem material unless criteria for making such a diagnosis and distinctions can be given.

(4) That when rejecting pneumoconiosis as a factor in disability or death panels should not use as the main argument such statements as "this degree of pneumoconiosis should not prevent him from carrying out his regular occupation" and "insufficient pneumoconiosis was present to have caused or accelerated death." The present philosophy is that pneumoconiosis does not matter until x cm of progressive massive fibrosis are present; x is bad, x-1 is harmless. In the latter case there may be a lot of scratching around for alternative explanations and practice (see (3)) is then often invoked.—I am, etc.,

D. DAVIES

Ransom Hospital,
Rainworth,
near Mansfield, Notts

¹ Lyons, J. P., *et al*, *British Medical Journal*, 1972, 1, 713.

Traumatic Hindquarter Amputation

SIR,—Your leading article (5 October, p. 4) states that "there has . . . been one case of traumatic hindquarter amputation, and the patient survived." This presumably refers to Gordon-Taylor's series, but it could be interpreted as indicating that you know of only one case of traumatic hindquarter amputation in which the patient has survived. McLean¹ was under a similar impression when he reported his case in 1962.

Recently at a meeting of the Holdsworth Club I showed a patient of my own who had survived a traumatic hindquarter amputation and mentioned another case treated by Mr. Bruce McCulloch in Bishop Auckland. Neither of these cases had previously been recorded. Two members of the Holdsworth Club stated that they had seen similar injuries. McPherson² reported one case in 1960, Wade and Macksood³ reported two cases in 1965, and a single case was reported from Budapest in 1969 by Lasslo.⁴ The most recent case reported is that of Ganapathy⁵ in 1973.

This injury is perhaps not so common as we had previously thought.—I am, etc.,

GILBERT PARKER

Middlesbrough General Hospital,
Middlesbrough, Cleveland

¹ McLean, E. M., *Journal of Bone Joint and Surgery*, 1962, 44B, 384.

² McPherson, J. H. T., jun., *Journal of the Medical Association of Georgia*, 1960, 49, 494.

³ Wade, F. W., and Macksood, W., *Journal of Trauma*, 1965, 5, 554.

⁴ Lasslo, P., *Orvosi Hetilap*, 1969, 17, 970.

⁵ Ganapathy, D. H., *Injury*, 1973, 5, 51.

Cold and the Heart

SIR,—Your leading article (17 August, p. 430) gives inadequate advice, I believe. True, the hypertensive stress reaction to sudden cold may cause myocardial hypoxia in a person with ischaemic heart disease, and avoidance of sudden exposure to cold will, of course, prevent such occurrence. Also I agree that those who use nitrates would do well to take an extra dose before exposure and perhaps they should indeed increase their daily measure of beta-blocking medication during the cold months. But such advice is stop-gap, remedial, and fails to make any attempt at correcting the heart's ability to cope with the increased work load stress evidently demands.

Anginal patients will experience myocardial hypoxia during exertion. But do we advise them to stay in bed and take extra medicine when they must walk? On the contrary we urge them to strengthen the heart muscle and to improve coronary oxygen supply by gradually increasing exercise. The same applies with regard to temperature changes and needs attention when one must face winter each year, as in the U.K. By routinely taking a cool to cold shower after a warm bath the circulatory stress response itself will become attuned or numbed to the sudden cold stimulus and the heart will be trained to cope with the response. In contrast the use of beta-blockers suppresses the heart's compensatory action and will thereby tend to weaken the heart. I believe that adaptation through training is a therapeutic principle of prime importance for the long haul, while avoidance and medication must be considered temporary, short-term necessities.

A second point: stepping out into the cold is one way of looking at this cold stimulus. But the other way is emerging from the heat. We are dealing here not so much with exposure to cold or winter as with the impact of a sudden change. I advise my patients to bring their indoor temperature in better harmony with the outdoors, both in summer and in winter, and to dress indoors accordingly. For the winter that means an indoor temperature in the low

60s and being warmly dressed. This greatly reduces the "insult" of stepping out into the crisp cold.

While there are yet other points to consider (did exposure to cold happen after a hearty meal or on an empty stomach; did the patient drink coffee or tea or whiskey; is he obese or lean; did he argue with his wife?), adapting oneself to the seasonal temperatures and training the adaptation process deserve, I believe, our major attention.—I am, etc.,

WM. F. KREMER

Dickerson,
Maryland, U.S.A.

Geriatric Policies

SIR,—Professor G. F. Adams says that "the geriatric physician with a high turnover and no longstay problem is . . . suspect as a gerontological spiv. Somebody, somewhere must carry the can for him" (28 September, p. 789). I find this denigration of colleagues who strive for high turnover in order to run their service without a waiting list regrettable.

The high turnover strategy does not make the longstay problem disappear, but it can give a more efficient service based on one's limited resources.^{1,2} Those of us who use this approach feel that the tougher discharge policy is a good exchange for abolition of the wait for admission which the patient, the relatives, the general practitioner, and the community services would otherwise have to bear. Somebody has to "carry the can" either way; what needs to be properly examined and discussed is which of the alternatives imposes the lighter burdens. Should we not argue the relative merits of the high turnover strategy and of the conventional system without unnecessary emotionalism or insults or implying that those of one's own persuasion have the monopoly of "warmth, feeling, compassion, humour, patience, integrity and understanding"?—I am, etc.,

H. M. HODKINSON

Geriatric Department,
Northwick Park Hospital,
Harrow, Middlesex

¹ Hodgkinson, H. M., and Jeffreys, P. M., *British Medical Journal*, 1972, 4, 536.

² O'Brien, T. D., Joshi, D. M., and Warren, E. W., *British Medical Journal*, 1973, 4, 277.

Experts and Child Abuse

SIR,—Dr. Selwyn M. Smith and Mrs. Ruth Hanson are I feel breaking new ground with their paper (14 September, p. 666) which draws together the psychological, community paediatric and psychiatric fields into the joint study of battered children. Professor S. D. M. Court and his colleagues (28 September, p. 801) have pointed out that the doctor cannot intervene uninvited. Hence it is all the more important for him to become invited by meeting the parents-in-need half-way, through the context of the ordinary and routine child health service. Dr. Smith and Mrs. Hanson report that many (50) of the 134 children studied had no doctor as a general practitioner, yet many were already "at risk" since developmental quotients were significantly lower for battered children of low birth weight or with failure to thrive. There were then clear reasons for parental concern. The authors