Numerous surveys have shown that for acute and for chronic debilitating diseases virtually all urban dwellers readily seek medical attention. At this hospital ulcerative colitis is diagnosed on the basis of clinical, endoscopic, radiological, and histological evidence. The possible roles of infective dysenteries and amoebiasis are, of course, taken into account.

The writers state that "varicose veins are seen not infrequently during village surveys in Africa, but people with this disorder are less likely to seek treatment than in Western cities." Locally the same relatively low prevalence in black adults is observed, whether one studies outpatients at hospital or persons in random house-to-house or kraal-to-kraal visits.

We therefore regret the impression that much of the evidence cited in support of the fibre-depletion hypothesis is hearsay, anecdotal, or uncritically acquired. It is interesting that while some consider Western diseases in developing populations to be underdiagnosed, the Gilbertian situation is such that some diseases in Western populations almost certainly are overdiagnosed. In the USA the prevalence of appendicectomies has been reported to have fallen considerably (40%). Could it be that this phenomenon (not explicable on the fibre-depletion hypothesis) is related to the very high frequency of total operations in that country and that the reduced rate of appendicectomies is now on par with that prevailing in, say, the UK? Prevalences of tonsillectomies in groups of South African white pupils of 16-18 years average about 65% (at one school it was 74%). In groups of urban black pupils of the same age the prevalence is approximately 3%. Yet, as to ill health, school attendance records of black pupils are as good as or often better than those of white pupils. Perhaps physicians of white children are looking too hard.

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Greater auricular nerve in diagnosis of leprosy

SIR,—The reminder of Major Peter Lynch (11 November, p 1340) that palpable enlargement of a greater auricular nerve is not pathognomonic of leprosy is both timely and salutary. The cutaneous branch of the radial nerve above the wrist and the unnamed cutaneous fibrils coursing over the dorsum of the hand may also be palpable in thin subjects. These palpable nerves are almost never tender in the absence of leprosy. Some abnormality of a peripheral nerve trunk, especially at a site of predilection, is very characteristic of leprosy, and "enlargement, hardness, and tenderness" are the commonest combination of deviations from the normal. Such abnormalities may precede, sometimes for many years, the appearance of some skin change, and may be the sole manifestation of leprosy¹⁻⁴ in up to a sixth of patients in some areas in India.

Experienced leprologists who have conducted clinical examinations of contacts of leprosy patients, recruits to the armed Forces, and

individuals exposed to leprosy in the community would confirm the report by Major Lynch and my own remarks:⁵ "The observation of enlarged peripheral nerves in areas of high leprosy prevalence is of doubtful diagnostic value without supporting histologic evidence. The sensitive finger can often detect sensory nerves (for example, the posterior auricular) in thin healthy subjects."

Relevant histological evidence is reviewed in a recent useful paper by McDougall *et al*,⁶ who suggest that a confirmatory or suggestive picture may be found in sections taken from enlarged nerves in subjects with no typical skin changes due to leprosy.

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Heat stroke in a "run for fun"

SIR,—The recognition of the hazards of heat stroke in long-distance running is long overdue (21 October, p 1158). Much of the delay has been due to errors in reporting such deaths. Our organisation has maintained forensic surveillance over marathon runners during the past 11 years. We have acted as a "clearing house" for reports of deaths in runners who have covered the 42-km distance at least once. Several heat-stroke deaths have reached the medical literature as deaths due to "ischaemic heart disease":

- (1) During the 1973 Boston Marathon a well-conditioned runner suffered brain death while racing under hot and humid conditions. Although the coronary arteries were found to be widely patent at necropsy the case was reported as "fatal myocardial infarction."¹
- (2) Fifteen years ago a 19½-year-old runner died during a marathon race in South Africa. Although the pathologist found normal coronary arteries the district surgeon wrote "coronary atheroma" on the death certificate.²
- (3) A 35-year-old fully-trained runner died a few days after a 40-mile (64-km) training run. The electrocardiogram was consistent with heat stroke.³ Although a necropsy had not been done, detailed necropsy findings were published, giving "ischaemic heart disease" as the cause of death.⁴ This was subsequently corrected.²
- (4) A 47-year-old died during a mountain race. No necropsy was performed, but the runner was known to have had a clinical heart murmur and a normal electrocardiogram. This case is included with cases (2) and (3) above as another case of "sudden death"⁵ with no mention of the hazards of exertion-induced heat illness.

As reported, these cases would not prompt the amateur athletic sanctioning bodies to change their racing policies. "Ischaemic heart disease" has become a popular euphemism for sudden death. The early signs of exertion-induced heat illness can easily be mistaken for signs of ischaemic heart disease.⁶ In one report 23 of the 48 marathon runners examined after a 90-km race were determined to have "early ischaemic heart disease" on the basis of enzyme and electrocardiographic changes.⁷ Such clinical findings do not reflect anything other than "prolonged exertion and heat load."⁸

In our view only the correct cause of death

in each case will give the sanctioning bodies cause to alter their policies. We will continue to review reports for runners above the marathon threshold.⁸ We hope that these reports will increase the safety of this rapidly growing sport.

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Does adipocyte hypercellularity in obesity exist?

SIR,—Dr R T Jung and his colleagues (29 July, p 319) questioned the existence of adipose tissue hypercellularity in obesity. It is not possible to go into detailed criticism of their paper and conclusions in the limited space allowed for a letter in the *BMJ* and therefore the following points constitute a summary of the criticisms which we will submit for publication elsewhere.

(1) Of the patients studied only three were severely obese (190-225% overweight). Their body fat was probably underestimated owing to difficulties with skinfold measurements in such patients.

(2) The control group was small, selected, and defined according to rather old standards from another continent.

(3) Omental fat cells were measured only in moderately obese patients. The above three considerations prevent conclusions being drawn about adipose tissue cellularity in general and hypercellularity in obesity in particular.

(4) It is true that visceral fat cells are difficult to measure, and the authors confirm previous reports that these cells are smaller than subcutaneous cells. The problem is that the relationship between visceral fat and subcutaneous fat is almost impossible to obtain accurately. In order to invalidate the concept of adipocyte hypercellularity one is, however, faced with the problem that hyperplastic obese subjects must have a much larger proportion of their fat cells in the subcutaneous fat than the controls. This is best explained by an example utilising the figures from the paper by Dr Jung and his colleagues. If all the obese subjects are analysed and not only the moderately obese part of the material, which for some reason has been selected for analysis by the authors, then one obtains a total number of about 10×10^{10} fat cells (body fat approximated by us to about 50 kg, fat cell sizes given in the paper, proportion subcutaneous:visceral fat 2:1 suggested by the authors). We then know from their report that controls have 16-kg body fat cell weights of 0.43 and 0.16 μ g of triglyceride in subcutaneous tissue and viscera respectively. In order to reach the number of fat cells of the obese the controls must have *all* their fat cells in the viscera. This is obviously a very unlikely situation.

To be correct, hyperplastic obesity should of course instead be called subcutaneous