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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- ¹ Jopling, W H, and Morgan-Hughes, J A, *British Medical Journal*, 1965, **2**, 799.
- ² Noordeen, S K, *Leprosy in India*, 1972, **44**, 90.
- ³ Furness, M A, and Ranney, D A, *Leprosy Review*, 1972, **42**, 208.
- ⁴ Dongre, V V, Ganapati, R, and Chulawala, R G, *Leprosy in India*, 1976, **48**, 132.
- ⁵ Browne, S G, in *Topics on Tropical Neurology*, ed R W Hornabrook, Philadelphia, Davis, 1975.
- ⁶ McDougall, A C, *et al*, *Journal of Neurology, Neurosurgery, and psychiatry*, 1978, **41**, 874.

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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- ¹ Green, L H, Cohen, S I, and Kurland, G, *Annals of Internal Medicine*, 1976, **84**, 704.
- ² Opie, L H, *New England Journal of Medicine*, 1975, **293**, 941.
- ³ Scalf, J H, *New England Journal of Medicine*, 1976, **295**, 105.
- ⁴ Opie, L H, *New England Journal of Medicine*, 1976, **294**, 1067.
- ⁵ Noakes, T D, and Opie, L H, *Lancet*, 1976, **1**, 1020.
- ⁶ *Lancet*, 1978, **2**, 718.
- ⁷ Olivier, L R, *et al*, *South African Medical Journal*, 1978, **53**, 783.
- ⁸ Bassler, T J, *Annals of the New York Academy of Sciences*, 1977, **301**, 579.

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

depot hyperplastic obesity in studies in which visceral fat mass and adipocytes are not available for measurement. With the above background it does not seem too inappropriate to delete the word subcutaneous and instead use the more convenient term of hyperplastic obesity.

We agree with the authors that hyperplastic obesity is a rare condition constituting perhaps only a small proportion of adult, middle-aged populations. It is nevertheless a significant problem in obesity clinics, where these patients tend to "sediment" because they are, according to our experience, very difficult to treat by conventional therapy.

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An apple a day keeps Newton away

SIR,—Dr B Y Yankelowitz's excellent proof—or was it disproof?—of Newton's theorem (23-30 December, p 1775) made excellent Christmas reading, especially in an issue containing much wordy warfare between two statisticians and Lord Taylor's quotation of Bradford Hill's dictum that statistical treatment of the obvious is a waste of time.

I must, however, remind him of the dictum that "while all science is measurement, not all measurement is science." I feel also that he has neglected to consider both the effect of aerosols in the ionosphere on stalk growth and that of the lead content of the Los Angeles air on cerebration when he wrote his article.

Perhaps his next work might be better submitted to that excellent American publication, unfortunately not yet available over here, the *Journal of Irreproducible Results*.

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Myocardial ischaemia in patients with frequent angina pectoris

SIR,—We would like to congratulate Dr Andrew P Selwyn and his colleagues (9 December, p 1594) on their success in using the Medilog Mark 1 ambulatory electrocardiographic (ECG) recorder for the accurate registration of ST-segment shift in ambulant patients. Recordings of this kind are notoriously subject to baseline shift, and it is a measure of the quality of their recordings that they lost no more than 45 minutes out of each 24 hours as a result of artefact. However, it is remarkable that they found no significant difference in ST-segment shift when making comparisons with an Elema ECG recorder although the R-wave amplitude varied by $\pm 15\%$ and the comparison was between a system with a low frequency response of 0.15 Hz and a second system with a low frequency response of 0.05 Hz.

Our experience with the same equipment has not been so happy. We have found it necessary to devise a new electrode system in order to stabilise the baseline. Even so, we still find considerable ST-segment distortion, a result which was not unexpected.

Berson and Pipberger¹ have published figures showing the deleterious effects of phase distortion at low frequencies. A roll-off faster than 6 dB per octave is a more serious

source of error than a small difference in cut-off frequency. A roll-off faster than 6 dB per octave is inherent in direct recording systems and cannot be precisely defined, so there will be considerable variations from one recording to the other. It seems to us that accurate ambulatory ECG recordings for defining ST-segment shifts can only be obtained with the Medilog Mark I recorder by incorporating a carrier system in which both the cut-off frequency and roll-off are precisely defined, and we are working along these lines. As things stand we have been unable to produce the high standard of recordings claimed by Dr Selwyn and his co-authors and look forward to knowing how they surmounted these difficulties.

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¹ Berson, A S, and Pipberger, H V, *American Heart Journal*, 1966, 71, 779.

Squares, cubes, and power

SIR,—May I use your columns to correct my uncle's applied mathematics (16 December, p 1717)?

Posture is maintained by muscles in isometric contraction, and ideally then there is no movement in the structure. It is therefore not a matter of the power the muscles produce but of the force that they can exert, and this is surely proportional to their cross-section and thus to the square of the structure's linear dimension only.

In this respect are they not like girders and the bridges which they support?

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Glaucoma

SIR,—I beg to disagree with Mr P A Gardiner (16 December, p 1690) where he suggests that coloured haloes around lights are pathognomonic for glaucoma, for they occur in corneal oedema from any cause. I have had experience of miners exposed to hydrogen sulphide gas underground and the presenting symptoms were intense irritation and pain in the eyes, plus coloured haloes around lights.

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Myopia, or juvenile expansile glaucoma

SIR,—I hesitate to write disagreeing with so eminent a research worker as Mr P A Gardiner (2 December, p 1555), whose Children's Test Type I I use; but the thought of 2 million children going worse when successful treatment for arrest is available drives me to take issue with his paper regarding myopia.

Myopia is the second cause of blindness under 60 and demands the utmost concern. It leads to about 400 000 macular degenerations

and 10 000 retinal detachments as well as many glaucomas. But it can be arrested in 90% of cases by bifocals and in most of the remainder by a special type of hard contact lens—which can be used continuously for a week—combined with bifocals.¹

The basic cause is accommodation within 6 m (20 ft). This causes increased intravitreal pressure. In those who become myopic this is not released fast enough by relaxation for distant vision. So in over 90% the myopia is apparently self-inflicted. The remainder show an "active" increase in intraocular pressure due to anterior chamber anomalies and other causes not yet found. Congenital causes may be the origin rather than heredity. What has been missed and what Friedman pointed out in 1966² is that the increase from 10 to 20 mm Hg in the conventional intraocular pressure means a rise eight times greater in the shearing stress of the sclera, as in bursting a balloon. In the rare congenital myopia the expansion has happened in utero. There is usually no postnatal increase and the myopia can be reduced. So all young myopes really have a type of juvenile expansile glaucoma—inactive, stress, or active.

In the past bifocals have had success or failure depending on the arbitrary distance correction, the height, the segment size, and the strength of the additions. Oakley and Young³ in a detailed research series of 269 cases with 275 controls in natural Americans (Indians) and Caucasians, adopted the maximum segment and had a 90% success. Young⁴ in a brilliant follow-up implanted radiosensitive sources in the vitreous which confirmed the concealed pressure build-up.

I feel that if any treatment can arrest or cure this kind of disease, and does not affect the rest of the body, it should be carried out forthwith. The cause can be found at leisure. Since myopia can increase severely in a few months and 100% of cases go worse in four years, treatment should start immediately the disease is found. It seems probable that we may soon be able to anticipate myopia and eliminate our future 7 million within a generation.

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¹ Kelly, T Stuart-Black, Chatfield, C, and Tustin, G' *British Journal of Ophthalmology*, 1975, 59, 529.

² Friedman, B, *Eye, Ear, Nose and Throat Monthly*, 1966, 45, 59.

³ Oakley, K H, and Young, F A, *American Journal of Optometry and Physiological Optics*, 1975, 52, 758.

⁴ Young, F A, *Optician*, 1978, 176, 7.

Infection with Epstein-Barr virus

SIR,—We apologise for our late reply to the letter of Dr D A Warrell and others (9 September, p 774), but we have been at pains to verify the reasons for the discrepancies in our report (22 July, p 248).

The antibody titres to Epstein-Barr (EB) virus capsid antigen of 1/5 on 4 March 1978, 1/20 on 26 July, and 1/40 on 13 September were all determined by indirect immunofluorescence in the laboratory of Dr R N P Sutton in Manchester. The "EB virus IgG" found to be 1/512 on 9 March 1978 was also antibody to virus capsid antigen, determined by immunofluorescence in the laboratory of Dr Joan Edwards in the Central Public Health Laboratory, Colindale. The IgM titre in the same sample was measured in the macroglobulin fraction of a sucrose density gradient, also using immunofluorescence against viral