seem to be completely unaware that the caravans they tow are wider than their car. I certainly have missed catastrophe by inches from towed caravans.

I would make a special plea for the separation of cyclists from motor traffic by special cycle routes or tracks in towns. Your correspondent says that the entrance and exit of cycle tracks may be awkward, but these could be controlled by traffic lights.

I do not think that a cycling proficiency test for any adult cycling on a main road should be mandatory, but all children who cycle should certainly take a school or similar cycling test. The wearing of helmets by cyclists should be encouraged, but it could be enforced legally only with difficulty. It must be remembered that the beauty of cycling, apart from exploring the byways that many motorists have forgotten, is the convenience for short journeys—no getting the car out of the garage, no difficulty with starting, no parking problem at the shops, etc.

May I suggest that your special correspondent might have got in touch with the Cyclists' Touring Club (69 Meadow, Godalming, Surrey), the cyclists' national body which runs an insurance scheme for cyclists and has the widest knowledge of the hazards of cycling, especially for adults. May I further suggest that anyone in later life taking up cycling for exercise and enjoyment would find the CTC most helpful in many ways quite apart from accidents, as I have done.

NOEL F KIRKMAN

Sale, Cheshire

No driving licences for alcoholics?

SIR,—With reference to your recent articles on road accidents and the unfit driver (25 November, p 1471) and on road accidents and driving behaviour (2 December, p 1551), I wonder how many doctors realise that all alcoholics are expected to notify their "disability" to the Driver and Vehicle Licensing Centre, Swansea, and are liable to a fine not exceeding £100 if they do not do so. Under present interpretation of the regulations it appears that they are likely to have their licences withdrawn unless or until they can claim or prove two years' total abstinence. I was astonished to learn that this was so when I was recently approached by the DVLC about a patient treated in this unit who had applied for routine renewal of his licence. (No offence had been committed, nor had the previous licence lapsed.) Only after I had reluctantly undertaken to notify the DVLC if this man took any drink, or failed to keep in touch with me every week, was a new (temporary) licence issued.

I am wholeheartedly in favour of road safety and I unreservedly condemn driving under the influence of alcohol; but it seems to me that the regulation is invidious, ineffectual, impractical, and likely to bring the law into disrepute. Alcoholics, when sober, are arguably no worse drivers than the rest of us, and many alcoholics are careful not to drive when drinking. Furthermore, very few alcoholics are likely to notify the centre and it is the more irresponsible and less insightful who will fail to do so.

How is the DVLC to be satisfied that two years' abstinence has in fact been reached? Are doctors to become big brothers for Swansea and lose the trust and co-operation of their patients? And what about the

(admittedly few) alcoholics who attempt to revert to social drinking or who have only an occasional lapse? The DVLC claims to have expert advice that "one year of complete abstinence should give rise to optimism, and two years to confidence in the provision of a driving licence." I wonder if the expert anticipated the results of his advice?

W A ELLIOTT

Tayside Area Alcoholism Unit
Sunnyside Royal Hospital
Montrose, Tayside

Erythromycin in legionnaire's disease

SIR,—Your leading article on legionnaire's disease (11 November, p 1319) comments that "American enthusiasm for erythromycin was not justified by the British experience." We saw three patients with a confirmed diagnosis of legionnaire's disease last summer who were all treated with erythromycin. They all recovered, though one developed renal failure requiring temporary peritoneal dialysis.

A particularly dramatic clinical response to erythromycin was shown in the third case, that of a 34-year-old woman admitted five days after returning from holiday in Spain. She had been unwell for three days with cough, headache, and vomiting with some diarrhoea. She was ill with a temperature of 40.6°C and a dry cough but had no abnormal signs in the chest. Splenomegaly was present. Chest x-ray demonstrated pneumonic consolidation at the base of the right lung and the clinical diagnosis of legionnaire's disease was confirmed by a titre of 1/32 rising to 1/1024 after a week. Erythromycin was started on admission, resulting in defervescence and rapid improvement within 24 h. The response was directly associated with the use of erythromycin and this was the only antibiotic used. In a disease which carries an appreciable mortality erythromycin should be administered promptly in any case of suspected legionnaire's disease. Response may be unsatisfactory in some patients but may be dramatic and life-saving in others.

M W MCKENDRICK
A M GEDDES

Department of Communicable and
Tropical Diseases,
East Birmingham Hospital,
Birmingham

Smear campaign

SIR,—I read with interest the letters from Mr T R Cullinan and Dr Leslie Ellis (16 December, p 1711) and am in full agreement with their thoughts on this subject. As one who has had more than 25 years' experience in vaginal cytology, may I explain some of the points they raise?

Firstly, the form of which Dr Cullinan so aptly comments. Having myself been many years in advance of the Department of Health and Social Security I had to design my own form and I soon learnt that the more items you put on it the less likely you are to get any worthwhile answers. My form required 11 pieces of information which I considered necessary to the correct interpretation of the smear. Over many years I found I was lucky if I got 75% of these facts filled in. I was concerned at national level over the setting up of the present form. My role was to prevent it ever seeing the light of day, but, alas, in this object I was singularly unsuccessful. Among

its main disadvantages are its length and complexity and the fourth carbon copy never seems to come out. It does not fit easily into any known filing cabinet and in its present form it is unsuitable for computerisation. These things I discovered by using it in conjunction with my colleagues both in hospital and in general practice.

Both Mr Cullinan and Dr Ellis ask for information on the timing of resmearing, and the answer to this is easy. In the first instance, the resmear should be in one year (a small percentage of first smears are false-negatives) and thereafter at five-year intervals until the age of 60, when it can be discontinued.

Dr Ellis quotes the mystical figure of age 35 as the time of taking the first smear and I can tell him how this was arrived at. Many years ago the late Dr Joe Bamforth was asked by the Department at what age smearing should commence. Not being a clinician he consulted a senior gynaecological colleague (who, incidentally, was not known for his interest in cervical cancer), who replied that he had never seen the disease before the age of 35. Thus this figure stuck and of course anything which sticks at the Department can only be removed by revolution or explosion. It is many years since Dr Bamforth told me these facts, but, despite the arguments of many of us, this mystical and mythical figure is largely adhered to. The fact is that ideally the first smear should be taken as soon as possible after the first act of intercourse, however young that may be.

STANLEY WAY

Gosforth,
Newcastle upon Tyne

Comparison between communities

SIR,—The series "Epidemiology for the Uninitiated" by Professor Geoffrey Rose and Dr D J P Barker makes fascinating reading to those interested in the epidemiology of diseases in developing populations.

In their recent article on "Aetiology-comparison between communities" (25 November, p 1483), we were taken aback by the view that the reported low incidences or prevalences of certain diseases in developing populations—for example, in Africa—may be more apparent than real, depending on how hard one looks. However, not all observations are made at small, isolated, rural mission hospitals: some are made at large, well-equipped city hospitals, of which there are several on this continent.

The authors refer to appendicitis. Locally (1) from records of operations in hospital, (2) from prevalences of operations in school pupils, and (3) from prevalences of operations in the huge number of labourers in the mines (whose medical facilities are excellent) we know that the low prevalence of appendicectomies in blacks is entirely real and not apparent. In large centres of population patients are assuredly not "more likely to die before they reach hospital."

Uncertainties due to variations in diagnostic practice are mentioned. In numerous fields there are differences in standards or in diagnostic criteria—growth, blood pressure, rickets, diabetes, coronary heart disease, etc. Accordingly the problem of determining whether ulcerative colitis (cited by the writers) is or is not rare is appreciated. However, the very much lower occurrence of the disease in urban South African blacks is undoubted.

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.

A R P WALKER
ANNETTE DUVENHAGE

South African Institute for Medical
Research,
Johannesburg

I SEGAL

Gastroenterology Department,
Baragwanath Hospital,
Johannesburg, S Africa

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.

S G BROWNE

Leprosy Study Centre,
London W1

- 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.

T J BASSLER

American Medical Joggers Association,
Centinela Hospital,
Inglewood, California

- 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