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*.

BRYAN WILLIAMS

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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?

ANDREW WRIGHT

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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.

T STUART-BLACK KELLY

Bath, Avon

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

capsid antigen. The IgM titre was screened routinely at 1/8 but no further dilutions were assayed. The discrepancy in the titres reported from Manchester and Colindale on roughly contemporaneous samples can only be attributed to methodological differences.

Antibodies to cytomegalovirus were detected in the laboratory of Professor Harold Stern, St George's Hospital Medical School, using complement fixation as stated in our article. Numerous attempts were made to isolate this virus from the patient's urine but without success.

Because of limitations of space, we could only summarise the serological data in our article and we could not fully discuss the diagnostic possibilities. We agree that one cannot be dogmatic about the nature of this patient's illness. It is our belief that the most likely explanation is that she had a primary infection with EB virus to which she was unable to make an adequate immunological response. Indeed, the antibody titres to cytomegalovirus may be attributable to cross-reaction of antibody between this virus and EB virus.¹ However, we agree that we cannot exclude the possibility that this patient had a double infection with both viruses and that the impaired response to EB virus reflects the immunosuppression induced by cytomegalovirus.

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¹ Horwitz, C A, *American Journal of Clinical Pathology*, 1977, **67**, 210.

1918 influenza epidemic

SIR,—Minerva (16 December, p 1722) says that there are few surviving medical witnesses of the 1918 influenza epidemic to remember any peculiarities of the disease.

I was serving in the RAMC at that time and was temporarily seconded to be medical officer to the First Reserve Brigade of the South African Infantry as two of its MOs had succumbed to the disease. I do not remember the "unique stench" mentioned in the American paper quoted, but I do recall vividly the curious violet cyanosis which often occurred quite soon after the onset of the attack. This was quite different from the bluish-grey of ordinary cyanosis and also from the pinkish hue of carbon monoxide poisoning. The phenomenon was so remarkable and widespread that a medical journal (I think the *BMJ*) sent an artist to make a watercolour sketch of one of our patients (colour-photography not having been invented). The picture was duly reproduced but the patient died the same evening and the artist a few days later.

The course of the disease could be extremely rapid. For example, it was quite common for two or three men to collapse during a morning route-march, to be carried back to barracks on stretchers, and to die before nightfall. I believe that every soldier who contracted influenza before he had been in Europe for three weeks died and that the South African expeditionary force lost more men from the disease than from wounds and gas poisoning combined. The tragedy was made worse by the fact that no available treatment appeared to have any beneficial effect, and also because most of the patients were of Boer descent and did not understand English.

Indeed, by a cruel irony, their fathers may well have fought *against* the British in the Boer War some 18 years before.

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Postoperative deep vein thrombosis in Nigerians on high-fibre diets

SIR,—It was with great interest that I read Mr U Osime's article (9 December, p 1607) and I would like to make a few comments.

Several years ago I visited Equatorial Africa and India with Mr Denis Burkitt and cross-questioned several hundred surgeons and doctors, and it is quite obvious that pulmonary embolism in these areas is very rare. Impressed by this fact, over five years ago we commenced giving bran routinely to our patients in one surgical ward—mainly urological. It was given preoperatively and post-operatively if the patients were able to take it. Since then we have had no case of pulmonary embolism and only one case of clinical deep vein thrombosis. Three other surgeons in different parts of the UK have had similar experience.

We conducted tests on 50 patients—the majority having had open prostatectomies—using ¹²⁵I-labelled fibrinogen. We found positive calf signs in 22%, but in the vast majority these were transient and reverted to normal in 48 hours. There was no case of clinical deep vein thrombosis. I would be interested to know how many of Mr Osime's were also transient in appearance.

Might it be that high-fibre diets alter fibrinolytic activity? If so, this could explain why the Nigerians rarely develop pulmonary embolism.

CONRAD LATTO

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Dealing with residual bile duct stones

SIR,—Mr R Mason (23-30 December, p 1788) rightly points out the difficulties in wire basket extraction of gallstones via the T-tube track when the track is narrow or tortuous, and recommends a 12 French gauge or larger T-tube brought out directly lateral to the common bile duct.

A large T-tube should certainly be used and there is usually no difficulty in placing such a tube in the dilated common bile duct which has contained stones. The track closes equally promptly after removal of the tube whether 14 or 16 French gauge or smaller T-tubes are used. The largest steerable catheter for stone extraction (Medi-Tech Company, Watertown, Massachusetts 02172) is 13 French gauge and Burhenne recommends a minimum T-tube size of 14 French gauge.¹ If an 8, 10, or 12 French gauge T-tube has been used and stones are subsequently found then the T-tube may be removed and a guide wire inserted, over which successively larger catheters are passed to dilate the track up to 14 French gauge. This may be done immediately before a first attempt at stone extraction six weeks after operation.

I would caution surgeons against bringing the T-tube out too directly. Many patients undergoing cholecystectomy are very obese and the T-tube exit wound may move downwards several inches when the patient stands up a few days after operation. If the T-tube takes too direct a route to the skin it can be

pulled out of the common bile duct. I have heard of a case in California that had fatal results. The T-tube should therefore describe a smooth curve between the common duct and the skin wound.

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¹ Burhenne, H J, in *Advances in Surgery*, vol 10, ed W P Longmire, p 121. Chicago, Year Book Medical Publishers, 1976.

Breathing and control of heart rate

SIR,—Your leading article (16 December, p 1663) entitled "Breathing and control of heart rate" comments on the physiological good sense of swallowing water to relieve faintness. Although this manoeuvre may stimulate respiration and thus relieve bradycardia it may also directly cause slowing of the heart.

The majority of reported cases of "deglutition syncope" appear to be associated with oesophageal abnormalities,¹ but even in people without oesophageal disease syncope may be so severe and repeated as to require insertion of a demand pacemaker.² In susceptible individuals a drink of cold water might therefore be an appropriate treatment for supraventricular tachyarrhythmias rather than for bradycardiac syncope.

Your leading article also draws attention to the complex interaction between lung inflation and bradycardia initiated by face immersion. This interaction has recently been clarified.³ Our results confirm that breath holding at inspiration leads to bradycardia, and show that face immersion has a marked effect only if performed at expiration. These findings explain why other works have not been able to demonstrate a consistent effect of lung volume on the diving response in man, since bradycardia is seen irrespective of face immersion at high lung volumes.

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¹ Tomlinson, I W, and Fox, K M, *British Medical Journal*, 1975, **2**, 315.

² Wik, B, and Hillestad, L, *British Medical Journal*, 1975, **3**, 747.

³ Openshaw, P J M, and Woodroof, G M F, *Journal of Applied Physiology*, 1978, **45**, 783.

Polyunsaturated fatty acids and colchicine in multiple sclerosis

SIR,—We were interested to read the report by Dr D Bates and others (18 November, p 1390) on the use of polyunsaturated fatty acids (PUFA) supplementation in multiple sclerosis (MS). We think that it is important to point out that the evening primrose oil (Naudicelle) capsules used in this trial contained dyes including tartrazine and ponceau R. In unpublished studies we have shown that both these dyes block the conversion of PUFA to prostaglandins (PGs) and this effect of tartrazine has also been reported by others.¹ The importance of this lies in the fact that the major biological function of the PUFAs is to give rise to PGs.² The effect of PUFAs in suppressing experimental allergic encephalomyelitis in rats is completely abolished by inhibition of their conversion to PGs.³

In preparation for a double-blind placebo controlled trial we have for the past six months