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.

PER BJÖRNTÖRP
LARS SJÖSTRÖM

University Department of Medicine I,
Sahlgrenska Sjukhuset,
Göteborg, Sweden

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

Sussex Geriomedical Society,
Boxgrove Temperance Inn

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.

V BALASUBRAMANIAN
EDWARD B RAFTERY
F D STOTT

Department of Cardiology and
Division of Bioengineering,
Northwick Park Hospital and
Clinical Research Centre,
Harrow, Middx

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

Worcester Royal Infirmary,
Worcester

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.

D G WILSON
Senior Medical Officer

Dounreay Nuclear Power
Development Establishment,
United Kingdom Atomic Energy
Authority,
Thurso, Caithness

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