interpretation of a positive correlation between energy intake and height. Since height is a measure of the size of the subject, this interpretation is not implausible. In order to rule it out, it would be necessary to show that in the large sample studied there is no significant correlation between BMI and height.

E S COOPER

Department of Child Health,
University of the West Indies,
Kingston, Jamaica

J M GURNEY
P J JUTSUM

Caribbean Food and Nutrition Institute,
University of the West Indies,
Kingston, Jamaica

[1] Womersley, J, Durnin, J V G A, *British Journal of Nutrition*, 1977, **38**, 271.
[2] Benn, R T, *British Journal of Preventive and Social Medicine*, 1971, **25**, 42.
[3] Newens, E M, and Goldstein, M, *British Journal of Preventive and Social Medicine*, 1972, **26**, 33.

**We sent copies of these letters to the authors, whose reply is printed below.—ED, *BMJ*.

SIR,—Miss Pamela Mumford and Dr Elizabeth Evans are right in pointing to the relative imprecision of the methods we used for collecting dietary information from the 3500 subjects in our studies. The difficulties and inaccuracies inherent in all methods of collecting such data probably account for the absence of relationships between food intake, body weight, and metabolic characteristics in some studies. However, even using these imprecise methods we *did* find relationships, the key ones in our argument being statistically most unlikely to have arisen by chance. The question, then, is whether the imprecision of the methods we used might have spuriously created the correlations we found or affected their order of magnitude. We could only consider both these aspects briefly in our condensed report.

However inaccurate a method of measurement may be, so long as those errors in estimate (be they erroneous overestimates, underestimates, or both) are randomly distributed, they will not, other than by chance, introduce a systematic trend and so generate artefactual correlations. There is no reason for supposing that the method used for the assessment of food intake systematically underestimated for the heavier or overestimated for the lighter subjects. We accept that obese people sometimes fail to recall the whole of their food consumption but so do non-obese subjects[1 2]; lapse of memory is not necessarily proof of self-deception. A few obese subjects may consciously mislead their inquisitors but a "deception factor" is hardly likely to operate throughout the leanness-adiposity spectrum; the inverse relationship which we reported extended right across the range of BMI, not just between the obese and the remainder. While we think it very unlikely that the method of data collection actually created the relationship we agree that its imprecision may well have led to an underestimate of its true strength.

Other, apparently more accurate, methods of dietary evaluation might increase the value of the correlations we found; but they are not without their own quite serious problems of application. In theory, weighing and recording every item of food consumed is ideal but, in practice, it is so tedious, messy, socially embarrassing, and intellectually demanding that many subjects alter their eating habits or make guesses to ease the task. Many "drop out" or decline to participate, introducing biases in the population sampled, particularly at the lower end of the social scale[3]; whereas our method is acceptable to most. We have been able to compare our assessment of food intake with actual food consumption in a sample of subjects and have found a high degree of correlation. Repeated estimates weeks or months apart on other samples of our populations show very comparable mean intakes and significant preservation of rank order within the groups. We shall be describing our methods and these validating studies elsewhere in detail.

Finally, we would enter a mild protest at the slightly distorted way in which our conclusions have been put. We did not really "claim that the less people eat the heavier they are," a statement which might be thought to carry cause-and-effect implications that we certainly did not draw. We recorded data which suggested that the amount of food people ate was inversely related to their degree of adiposity, leaving the interpretation of this correlation open, though we made a few suggestions.

The comments of Drs Cooper, Gurney, and Jutsum are well founded. Keys *et al*[1] noted that, of all the indices of obesity, the BMI was least correlated with height, but some residual relationship remained. We did find a small but statistically highly significant negative correlation between height and BMI in our populations and consequently there was a positive correlation between caloric intake and height, although this only achieved significance in the Beecham and Whitehall men.

Multiple regression analysis, however, showed that there was no significant independent relationship between caloric intake and height when the BMI was introduced into the regression equation other than in the Whitehall men, while the BMI remained significantly negatively correlated with food intake independently of height in all population groups. Thus part of the inverse correlations we reported may be due to the confounding variable of height, but even after making statistical allowance for this the effect remains.

H KEEN
BRIONY THOMAS

Unit for Metabolic Medicine,
Guy's Hospital Medical School,
London SE1 9RT

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[1] Keys, A, *et al*, *Journal of Chronic Diseases*, 1972, **25**, 329.

Doctors and children's teeth

SIR,—Your recent leading article (12 May, p 1231) on children's teeth made many good points but unfortunately omitted to mention any means of ingesting fluoride other than in water. There are still many unenlightened parts of the country where this safe and simple procedure has not been instituted and the main alternative should be tablets; these ought to be stocked by all child health clinics and prescribed from infancy onwards in non-fluoridated areas. An earlier leading article[1] recommended a dose of 1 mg daily for children over 2 years, and advised the user to suck the tablet slowly rather than swallowing it to give a topical as well as a systemic effect.

Unfortunately drops for infants are not readily available and the tablets need to be crushed and dissolved in water—a task which will limit their use to the most dedicated of parents. What about a new preparation: Fluorabidec? .

TONY WATERSTON

Department of Child Health,
Ninewells Hospital,
Dundee DD2 1UB

[1] *British Medical Journal*, 1975, **1**, 535.

Trauma and Paget's disease of bone

SIR,—Further to Dr L R Solomon's article (7 April, p 931) and Dr T M Gasper's letter (5 May, p 1217), it might be appropriate to mention that in the French medical literature several cases of localised[1 2] and even generalised[3 4] Paget's disease of the bone have been reported to develop after trauma.

In the cases described the latent period between trauma and diagnosis varies considerably.[5] The relative frequency of involvement of the different bones—and particularly the fact that the right femur is twice as frequently affected as the left one—points to the possibility that trauma is at least a factor in the development of Paget's disease.[6] Trauma may precipitate a breakdown in the normal mechanism of gradual continuous replacement to which bone is constantly subjected and result in the development of Paget's disease.[6] The relative immunity of the bones of the foot and the mandible is difficult to explain.

Trauma, however, cannot be solely responsible for Paget's disease, as the microscopic ultrastructure, and especially the discovery of intranuclear inclusion bodies in the osteoclasts of bone specimens affected by Paget's disease and not in other normal or pathological bone specimens, provides indirect evidence that the condition may be due to a slow viral infection.[7 8] This hypothesis would also explain the geographical distribution of the disease and its tendency to run in families; but the virus has not yet been identified.

At present the aetiology of Paget's disease seems to be multifactorial: it is probably due to a slow virus infection, with trauma acting—in some cases—as a localising factor and heredity as a predisposing factor.

RONNIE HAMDY

Department of Clinical Gerontology,
St John's Hospital,
London SW11 1SP

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[6] Jaffe, H L, *Clinical Orthopaedics and Related Research*, 1977, **127**, 4.
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Differences between Leeds fractures and London fractures?

SIR,—We were surprised at the assertion of Dr M R Baker and others that their control data on plasma 25-hydroxy vitamin D (25-OHD) concentration are almost normally distributed (5 May, p 1218), and this led us to calculate the coefficients of skewness ($\sqrt{\beta_1}s$) for their series. The coefficients were 1·9