apparent fall in LAA concentration was simply due to the increasing proportion of granulocytes in the differential white blood cell count, and that a true fall in LAA concentration had not occurred. After the acute "stress" had subsided and the leucocytosis returned to normal, however, the LAA concentration and serum ascorbic acid concentration remained low, presumably because of tissue depletion of ascorbic acid. Both may remain low for up to 56 days.²

We also found that tissue damage is probably necessary to produce the long-term depletion of LAA and serum ascorbic acid concentrations since the acute changes in LAA only can be produced by tetracosactrin infusions in normal subjects.²

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¹ Irvin, T T, Chattopadhyay, D K, and Smythe, A, *Surgery, Gynecology and Obstetrics*, 1978, **147**, 49.

² Vallance, B D, Hume, R, and Weyers, E, *British Heart Journal*, 1978, **40**, 64.

³ Hume, R, *et al*, *British Heart Journal*, 1972, **34**, 238.

Back pain—what can we offer?

SIR,—It astonishes me to read the suggestion implicit in your leading article (17 March, p 706 that causes of "back-pain" sufficiently severe to warrant reference to a rheumatology clinic may safely be diagnosed without radiography of the relevant portion(s) of the spine, with no loss of accuracy and with a bonus saving to the NHS of "many thousands of pounds each week." This amounts, in my alarmed medicolegal view, to an advocacy of technical professional negligence on a massive scale.

May I, however, offer an observation (based in part on personal experience) on the significance of radiological changes and their relation to symptoms? The latter are commonly caused by pressure on nerves of radiolucent soft structures, such as, for example, a prolapsed nucleus. The development of convincing bony changes, which represent nature's attempt at a healing process, may take many years. Symptoms and radiological signs are usually asynchronous, the latter slowly progressive, the former variably intermittent.

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SIR,—Your leading article "Back pain—what can we offer" (17 March, p 706) rightly questions the routine use of radiographs. The radiologist with limited information about the patient has difficulty making a valid comment about degenerative change, and the general practitioner who does not see the x-rays cannot easily relate a report to the patient's symptoms.

Orthopaedic surgeons are increasingly aware that the size of the lumbar spinal canal can be an important factor in the aetiology of back pain. When canal measurements are available¹ radiographs can demonstrate additional compromising lesions that will compress the canal contents. This may be disc degeneration, posterior vertebral bar formation, shingling of two vertebrae with slight retro-

spondylolisthesis, facet-joint arthritis, spondylolysis, or spondylolisthesis. In themselves these radiological features may be insignificant, but in the presence of a narrow canal they can indicate a structural abnormality responsible for symptoms.

For a patient whose back pain is prolonged or recurrent a diagnosis of "non-specific back pain" is inadequate, and x-rays become an important investigation.

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¹ Porter, R W, Wicks, M, and Ottewill, D, *Journal of Bone and Joint Surgery*, 1978, **60-B**, 481.

Does adipocyte hypercellularity in obesity exist?

SIR,—We are glad that Drs P Björntörp and L Sjöström (20 January, p 197) agree that hyperplastic obesity is a rare condition. If hyperplastic obesity is likely only to be present in individuals with body weights of over 150 kg—that is, a weight range corresponding to that in our three individuals in whom such interest is expressed—then one is dealing with a very unusual and small group of patients. If Dr Björntörp and his colleagues¹ set the upper limit of normality for fat cell number only 1 SD above the mean then obviously one can begin to define a larger proportion of obese patients (and indeed of the normal-weight population) as hyperplastic. We are also glad to note that the Göteborg group now considers a value for total fat cell number as only an index of the true cell number—an index on which we consider one should place little reliance.

The other comments we found somewhat misleading. Our reference standards for obesity, the Metropolitan Life Tables, are those recommended by both United Kingdom and USA expert committees; they are widely used, readily obtained, and accepted by most investigators of obesity. Our three subjects whose body fat measurements worry the Swedish group had additional measurements, as indicated in the paper, with ⁴⁰K and ⁴²K; in two patients body fat had been marginally overestimated and in one underestimated. The number of controls (27) would need to be substantially increased if we wished to establish accurate normal limits for the index of fat cell number, but our values accord with those of other workers and our conclusions stand.

We agree that we did not have adipocyte measurements on omental tissue from grossly obese patients. When this has been done by other workers² the results have matched our predictions in that the internal cells have finally expanded to equal the size of the subcutaneous adipocytes—a development that will lead to a spurious increase in calculated fat cell number when the calculations are based on subcutaneous samples alone.

Odd conclusions can indeed be drawn by mixing our data on fat cell sizes (0.73 µg

triglyceride for subcutaneous and 0.29 µg for omental sites) with an erroneous estimate of 50 kg for body fat rather than the actual 30.5 kg quoted in table II of our paper. At 50 kg of body fat the mean subcutaneous cell size, estimated from Björntörp's own data,³ would be about 0.9 µg triglyceride and that of omental cells, estimated from the work of Salans *et al*,² would be 0.70 µg. If we retained the 2:1 ratio for subcutaneous and visceral fat then the "true" fat cell number would remain unchanged and therefore similar to that found in other groups of patients with body fat corresponding to 30.5 kg and 16 kg (see table).

We accept the need for improved methods for measuring subcutaneous and deep body fat. Our choice of the best estimates yet available for the proportions of body fat in the subcutaneous tissue indicates the potential magnitude of the errors associated with measuring the total number of fat cells in the body.

We do not agree with the explanation proposed for validating hyperplastic obesity: true hypercellularity of the adipose mass must exist because the limits of normality are defined to exclude the top 3% of the Gaussian range. The question is then whether hypercellularity specifically relates to the problem of obesity, and if so how often and to what effect.

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¹ Björntörp, P, *et al*, *American Journal of Clinical Nutrition*, 1975, **28**, 445.

² Salans, L B, Cushman, S W, and Wersmann, R E, *Journal of Clinical Investigation*, 1973, **52**, 929.

³ Sjöström, L, and Björntörp, P, *Acta Medica Scandinavica*, 1974, **195**, 201.

Health and safety at work

SIR,—I read with interest Dr B J Boucher's thoughtful article "Guidance on preparing local rules to help implement the Health and Safety at Work, etc, Act, 1974" (3 March, p 599). Dr Boucher stated that "we have found that direct guidance from the DHSS or NHS sources or from professional bodies on what is now required from NHS staff for safety at work has been slow to appear and inadequate in its coverage of the diversity of NHS activities; codes of practice are not yet available for much of our work."

Some parts of hospitals have been subject to the Factories Act and to the Offices, Shops and Railway Premises Act. Areas such as wards, operating theatres, and laboratories became subject to health and safety legislation in 1975, together with many other aspects of activity associated with hospitals. I was informed by the Health and Safety Executive some while ago that an area director has been nominated by the Executive to identify health and safety problems in the hospital service and to

Calculated total body fat cell number with increasing body fat

Total body fat (kg)	Fat cell sizes (µg of triglyceride)		Fat masses on a 2:1 ratio (kg)		Fat cell number (× 10 ¹⁰)		Total number (× 10 ¹⁰)
	SC	Omental	SC	Omental	SC	Omental	
16	0.43	0.16	10.6	5.4	2.5	3.4	5.9
30.5	0.73	0.29	20.4	10	2.8	3.4	6.2
50	0.9	0.70	33.3	16.7	3.7	2.4	6.1

SC = subcutaneous.