a sexual contact by gonorrhoea patients is a valid candidate for the procedure known as "epidemiologic treatment" even when cultures (which may miss 20% or more of such gonococcal infections) are negative. If chemotherapy is adequate, a link in a chain of infection may thus be broken. A patient who would refuse a diagnosis of venereal disease may willingly accept this precautionary measure, just as chemoprophylaxis is accepted for ophthalmia neonatorum.—I am, etc.,

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Perforated Duodenal Ulcer

SIR,—May I write very shortly on this subject (19 October, p. 155) simply to point out that patients suffering from perforated duodenal ulcer, although under the care of the surgeon, benefit greatly by an avoidance of operative treatment and the institution of a routine conservative regimen, already well described in the literature? No early case (within the first six hours) would be treated otherwise in my ward. The benefits include reduced incidence of subphrenic abscess; no possible complications associated with abdominal wounds—that is, infection, stitch abscess, disruption, herniation; no possible complications associated with general anaesthesia—that is, cardiac arrest, respiratory arrest, atelectasis, bronchopneumonia; and, of course, a perfectly clean abdominal field for elective surgery if it should be indicated in due course.

These patients with perforation do extremely well on properly applied and supervised conservative regimen, and are usually better in a matter of three to four days, and can be very thoroughly investigated shortly after this. The diagnosis, of course, must be accurate and we must always be prepared to change course and operate if the response is not prompt within two or three hours of commencement of treatment.

Finally, I do not think that these days a partial gastrectomy should ever be contemplated for treatment of perforated duodenal ulcer, and seldom for duodenal ulcer as an elective operation. In my view the operation of choice is undoubtedly selective vagotomy and Finney pyloroplasty, but as an elective procedure, except in cases of haemorrhage.—I am, etc.,

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Chronic Lead Intoxication Mimicking Motor Neurone Disease

SIR,—We were particularly interested in the letter from Dr. B. Livesley and Dr. C. E. Sissons (9 November, p. 387) which described lead-poisoning mimicking motor neurone disease.

In a recent survey carried out by one of us on cases of motor neurone disease throughout Britain 17% gave a history of known contact with lead in the past. A point of some interest is that motor neurone system disease shows itself long after actual exposure to the lead.

In one case published in 1954[1] a schoolmistress who had handled lead 20 years previously presented with atypical motor neurone weakness and showed considerable improvement after treatment with calcium versenate. Her improvement has continued although she still has some motor disability at the present time. Another severe case of motor neurone disturbance with marked weakness, wasting, and fasciculation was recently seen in a farmer who 25 years previously had been treated for acute lead poisoning contracted through drinking cider contaminated with lead. Another case of motor neurone disease seen recently in a clergyman who has since died showed an unexpected contact with lead, in that he had painted his church with white lead ten years previously. We have also had a patient like the case described by Drs. Livesley and Sissons, of acute motor neurone disease who in the past had had long contact with red lead application to ships.

We are at the moment trying to investigate the various aetiological factors in motor neurone disease, and undoubtedly lead is an important factor to bear in mind, and, though it is probably only the cause of a group of such cases and various factors may play a part in the final damage to the motor neurone, it may be a clue to an understanding of other causes of this unfortunate disease. We agree with Drs. Livesley and Sissons that lead can turn up in the most unexpected places, and of course poisoning has been described from nipple shields, ice-cream containers, and theatrical paint. In every case of motor neurone disease a detailed occupational history is of great importance.

The final cause of motor neurone disease remains unknown, but obviously genetic, viral, deficiency, and toxic factors may all play a part in disturbing enzyme function at a cellular level.—We are, etc.,

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Studies on Osteoporosis

SIR,—Professor C. E. Dent and others (12 October, p. 76) in their studies on osteoporosis made the incidental observation that 24% of the white women showed severe calcified abdominal aortas while none were found in the two groups of Bantu women.

I have used lateral radiographs of the lumbar spine for the detection of calcified atherosclerosis in abdominal aortas in a series of investigations.[1,2] In a comparative radiological study on the incidence and the severity of calcified atheroma of the abdominal aorta in 1,252 unselected patients between the ages of 50 and 80+ (680 males, 572 females) I found that women over the age of 65 have a distinctly higher incidence of severe calcified aortas than men of the same age groups. Moreover, the most extensive calcified lesions were found in women with senile osteoporosis.[3] The consistency of this observation led to the concept of a relation between senile osteoporosis and calcified aortas. My concept was investigated by Anderson *et al.*[4] They confirmed the existence of a highly significant association between osteoporosis and aortic calcification, but assumed this association to be mainly due to the increasing incidence of both conditions with age. However, if age alone were the determining factor, we would expect that one or both of these conditions would show a similar incidence in both sexes. Instead, we find a female predominance of osteoporosis and likewise a female predominance of severe calcified abdominal aortas. Professor Dent and his colleagues found no positive correlation between the patients with severe aortic calcification and those with severe osteoporotic spines. However, their number of patients in the highest age groups, in whom this correlation is almost a constant feature, is rather small. Their finding is not in conformity with my observations nor with those recently reported by Bernstein *et al.*[5]

Senile osteoporosis is only an advanced stage of a physiological ageing process, and there are large variations in the rate of progression between the sexes and from individual to individual. When the results of the investigation by Professor Dent and his colleagues are seen in the context of my concept of a relation between osteoporosis and atherosclerosis, it seems likely that sustained physical activities may be one of the factors delaying the progression of osteoporosis and of atherosclerosis, as shown in their two groups of Bantu women.—I am, etc.,

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SIR,—I was very interested in the article by Professor C. E. Dent and his collaborators (12 October, p. 76), providing further evidence of the rarity of the elderly type of osteoporosis among the South African Bantu. This phenomenon is well known to experienced clinicians in Southern Africa,[1,2] as also the related rarity of fractures of the femoral neck,[3] but true prevalence data have not, to my knowledge, been previously presented.

What has particularly intrigued some of us is the fact that the Bantu bones appear particularly "good," physically and biochemically,[4,5] despite a lifetime of low calcium intake averaging under 400 mg. daily in several surveys.[6-8] Presumably the Bantu today are particularly good intestinal absorbers of calcium ("adapters" to low intake).[2]—I am, etc.,

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