showed that loss of muscular action on bone is a potent cause of osteoporosis. Clinical, demographic, and postmortem studies have confirmed the importance of the relationship between bone and muscle. Thus N. Westlin and B. Nilsson at the orthopaedic clinic, Malmö Hospital, have shown that bone density increases with physical activity and is also related to the quadriceps power. Geographical variation in the incidence of hip fracture indicates that a lifetime of hard physical activity is the best prophylaxis against osteoporosis.² Measurements taken at routine necropsies show a linear relationship between spinal bone mass and the weight of the psoas muscles.³

Professor Nordin is clearly unable to explain the selective osteoporosis of the spine on a hormonal or biochemical basis. But if osteoporosis is a wasting disease of bone due to lack of use then there is a simple explanation for the crush fracture syndrome. It is only too easy to go through life without moving one's back but even the life of a dowager requires some movement of the limbs. The growth of techniques of passive spinal movement (osteopathy and chiropractic) is a byproduct of the tendency of civilized man to avoid exercising his back. You rightly stress the need for prevention, and suggest that the acquisition of a large bone mass in childhood with the help of diet will be of value. But surely the lesson that we must learn is that a daily routine of physical activity is as beneficial to the skeleton as it is to the heart and lungs.

As a profession we appear to be no more enlightened concerning the value of regular exercise than our patients. We drive everywhere in cars and imagine that a weekly game of golf will keep us fit. What Professor Nordin and all of us should be preaching is a daily regimen of running and spinal exercises. If everyone adopted this we would not only be able to close down our coronary care units but would also be able to halve the number of beds on our orthopaedic and geriatric wards.—I am, etc.,

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¹ Geiser, M., and Trueta, J., *Journal of Bone and Joint Surgery*, 1958, 40B, 282.

² Chalmers, J., and Ho, K. C., *Journal of Bone and Joint Surgery*, 1970, 52B, 667.

³ Doyle, F., Brown, J., and Lachance, C., *Lancet*, 1970, 1, 391.

Halothane Hepatitis

SIR,—Halothane-induced hepatitis may or may not be an entity, but the evidence presented by Dr. P. Sharpstone and others (20 February, p. 448) that it is preventable is not convincing.

I have personal knowledge of their Case 11 and feel that a few further details may be of interest. This 66-year-old man, who was admitted for investigation of haematuria and frequency, had received a course of tetracycline from his general practitioner one month before. He had been losing weight and this was attributed to a reducing diet which he had been following for some time. After his cystoscopy he developed a severe urinary infection for which he was prescribed ampicillin in doses up to 500 mg q.d.s. for the next twelve days, continuing beyond the day of his laparotomy. Thus the pyrexia after these two operations was by no means inexplicable and

certainly gave no clue to an underlying hepatic cause.

At necropsy the liver weighed 1,280 g and the histological report (for which I am indebted to Dr. I. M. Tuck) was as follows: "The liver shows advanced biliary cirrhosis. Liver tissue is reduced to small disorganized lobules separated by wide bands of fibrous tissue in which there is a complexity of proliferating bile ducts."

Considering all the aspects of this case it seems unreasonable to argue that the terminal hepatic failure was due to halothane per se, let alone that it was preventable.—I am, etc.,

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Abdominal Aortic Aneurysm and Peptic Ulcer

SIR,—Your leading article (16 January, p. 129) discusses the association between peptic ulcer and abdominal aortic aneurysm, and points out the increased incidence of peptic ulcer in patients with abdominal aortic aneurysm. I have also been impressed by this association and in my experience the frequency of peptic ulcer in patients who have attended this hospital with abdominal aortic aneurysm has been higher than might reasonably have been expected. (The general necropsy figure of 7.2% mentioned in your article bears this out.)

Analysis of 30 patients with abdominal aortic aneurysm seen in this hospital in an eight-year period shows that six cases (20%) have been associated with definite symptoms of duodenal ulcer, and it has been possible to substantiate this diagnosis in each case.

A 46-year-old woman found to have a gastric ulcer was treated by Bilroth-I partial gastrectomy and an abdominal aortic aneurysm was seen at operation. Two patients had a laparotomy and were found to have active duodenal ulcers. Abdominal aortic aneurysms were found to be present in both these cases and resection and Teflon graft was performed at a subsequent operation. Two patients with haematemesis had abdominal aortic aneurysms. Barium meals showed duodenal ulcer craters and scarring was present in the duodenum at the time of elective surgery for aneurysm. A 64-year-old man presented with a perforated duodenal ulcer four days after the diagnosis of abdominal aortic aneurysm. Plain x-ray of the abdomen showed calcification but no free gas. This case particularly illustrated the difficulty in diagnosis which can exist when these two conditions are present in one patient.—I am, etc.,

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Streptococcal Meningitis

SIR,—Today, in the 1970's, streptococcal infections are decreasing in both incidence and severity. We have come to feel secure with antibiotics, but when a fulminating virulent streptococcal infection does occur we are as impotent as we were 30 years ago. Particularly rare is meningitis due to beta-haemolytic streptococci. It is still occasionally

seen in babies and infants, but over the age of three or four years it is extremely unusual today.

We report here a recent case which was marked by an apparently insidious onset of illness, with sudden deterioration in the patient's condition and a rapidly fatal outcome.

The patient was a 31-year-old woman. She had a one week's history of cough and a cold, but she had been out and about on the day prior to her emergency admission to hospital. On the day of admission, she vomited several times, and drowsiness and slight confusion were first noted at about 8 p.m. She had neck stiffness and a temperature of 97°F (36.5°C) when seen by her general practitioner at 10.30 p.m. Over the ensuing half hour she lost consciousness, and on arrival in hospital at about midnight she was in deep coma with signs of decerebrate rigidity. There was no rash or signs of focal infection.

Lumbar puncture produced a turbid cerebrospinal fluid with 2,000 W.B.C./mm³, predominantly polymorphonuclear, a protein concentration greater than 150 mg/100 ml, and a sugar content of 0.5 mg/100 ml. Gram-positive cocci could be seen on the stained film of the cerebrospinal fluid.

Progress was relentlessly downhill despite treatment with large intravenous doses of benzyl penicillin and cloxacillin, and the patient died seven hours after admission. Postmortem examination showed the presence of purulent exudate in the meninges, without other significant findings. The adrenals were healthy. Culture of blood and cerebrospinal fluid subsequently grew group A beta-haemolytic streptococci, phage type M-R-T3/13/B3264, sensitive to both penicillin and cloxacillin.

This case provides us with a sinister reminder that serious streptococcal infection, particularly when it results in meningitis, is still very much a potential killer despite modern advances in medicine.

The patient was admitted under the care of Dr. Hillas Smith.—We are, etc.,

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Nature's Transplant

SIR,—The immunological inertia of viviparity¹ indicates that an exchange of information often occurs between the mother and the developing offspring, concerning each other's profiles of transplantation antigens.² This information traffic may be in cellular or subcellular form. In humans erythrocytes are known to pass from fetus to mother and, it has been claimed,³ from mother to fetus. A traffic in leucocytes has been inferred from the detection of 46, XX cells in the cord blood of male infants^{4,5} and from the detection of 46, XY cells in the peripheral blood of pregnant women bearing male children.^{6,7} Jacobs *et al.*⁸ offered other explanations, apart from transplacental traffic, for the findings of Walknowska *et al.*,⁶ and reported that 17 "46, XY" karyotypes were detected among 24,484 cells from 1,285 apparently normal non-pregnant females—a much lower incidence however than the 34 "46 XY" cells found in 13,210 metaphase figures by Walknowska *et al.*