in one or two approved centres. The history of previous treatments for multiple sclerosis makes the need for caution all the more obvious.

At the moment, the best treatment a clinician can offer a patient with multiple sclerosis is sympathetic understanding, treatment and control of infection, advice about how to avoid stresses, treatment of spasticity by antispasmolytic agents such as diazepam and baclofen, and the judicious use of good physiotherapy. Help with spasticity and the control of infections and other stresses must continue to be the cornerstone of current treatment.

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Bones in space

Maintenance of the integrity of bone and of a skeleton able to resist the physical stresses of everyday life depends on several factors.[1] Four predominate: the force exerted on bone by its muscular attachments, the hydrostatic forces responsible for an adequate flow of blood, piezoelectric forces, and the effect of gravity. This complex of stimuli continuously influences bone, a living organ always remodelling itself.

Space flight, with its absence of gravity, has provided a unique opportunity to study the effect of this force and its relative importance in physiological mechanisms. In the zero gravity environment of space man's structure has been described as becoming as awkward as a gothic cathedral launched into orbit—picturesque but inappropriate. Certainly creatures which float in quasi-weightlessness have no need for appendages, and analysis of man's behaviour in these circumstances suggests a remorseless drive to reduce the size of his limbs. Radiographic studies[2] during Project Gemini showed loss of bone in astronauts exposed to short periods of weightlessness, findings similar to Russian results from the Soyuz 9 flight.[3]

The loss of calcium in zero gravity is related to bone atrophy from disuse and parallels the response that occurs with immobilisation of a limb or absolute bed rest. Metabolic balance studies and direct measurements of bone density have shown a loss of about 4 g of calcium per month, equivalent to 0·3-0·4% of total body calcium. Mineral is lost from bone unevenly, however, with greater total losses from trabecular bone, so that rarefaction visible radiographically may occur in parts of long bones after four to eight months in space, and the strength of critical bones may be endangered.

For example, decrease in the density of the os calcis was shown in two of the three crew of Skylab 4, an 84-day flight. No changes have been found in the hormones that control the activity of the osteoclasts and osteoblasts, and dietary manipulation in flight has had no effect upon calcium excretion.[4] Nor did a programme of rigorous exercises have any effect on the rate of calcium loss. The current view is that tropic innervation of the muscles is disturbed in zero gravity, reducing the muscular force applied to bone with a resultant relentless loss of minerals. This persisting loss of bone calcium increases the risk of renal damage, including the formation of calculi, and of hypertension in those undertaking extended space missions.[5] Though thin bone trabeculae can return to normal thickness, trabeculae that have been lost completely probably cannot be restored.[4] Informed speculation suggests that critical demineralisation may occur after nine to 18 months in orbit unless measures can be undertaken to halt or slow the process.

Among the many biological changes that will be intensively studied in further spacelab experiments investigations into the mechanisms underlying skeletal integrity will have priority. Possibly this line of research may also be of some benefit to the earth-bound clinician managing patients with osteoporosis. The bone changes that occur in space are mostly reversible on return to normal gravity, a result which should remind clinicians of the importance of direct longitudinal stress on the integrity of bone; and by implication it reinforces the rationale of graduated increasing weight bearing in the management of patients with osteoporosis.

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Diet and bowel cancer

Adenocarcinomas of the colon and rectum are two of the most common cancers in industrialised communities. Migrants coming to the United States from Japan, where the incidence is low, adopt the higher rates of their new country within one generation.[1] This points to the action of a potent environmental influence, and the search has concentrated on four constituents of the diet: meat, fat, fibre, and alcohol.

International comparisons have shown a correlation between the intake of meat and both incidence and mortality rates from cancer of the large intestine.[2] Nevertheless, the epidemiological evidence for this association remains equivocal,

with negative findings from—for example—an analysis of secular trends and geographical patterns of beef consumption within the United States[3] and from case-control studies. A positive association was found, however, between consumption of meat and cancer of the large intestine in Hawaiian Japanese.[4] Possibly that positive result may have been obtained while the findings in similar studies have been negative because the diet of the Hawaiian Japanese population is appreciably heterogeneous.[2]

The epidemiological evidence of an association between fat consumption and large intestinal cancer is similarly equivocal. International comparisons show a correlation between fat intake and cancer rates[5]; but analyses within countries and case-control studies generally do not confirm the association.[6] Other data, however, suggest that meat and fat may play a part in carcinogenesis. Differences can be shown in the faecal flora in populations with high and low rates of cancer of the large intestine.[7] Anaerobic bacteria such as *Bacteroides* are more abundant in areas of high incidence. There are several possible mechanisms through which bacterial activity on substances derived from meat or fat could be carcinogenic. For example, a high intake of dietary fat might increase the colonic concentration of bile acids, whose subsequent metabolism by bacterial flora might produce carcinogens.[8]

The close geographical and secular association between bowel cancers and other non-infective diseases of the bowel such as appendicitis and diverticulosis has suggested a link with a refined carbohydrate diet, low in fibre.[9] A recent comparison between a rural area of Finland with low rates of colorectal cancer and Copenhagen, where the rates are high, showed that the intake of dietary fibre was higher in the country area.[2] A high intake of unabsorbable fibre could protect against bowel cancer in several ways: bulky stools might dilute carcinogens; the reduced bowel transit times might lessen the contact between carcinogens and the mucosa; and fibre might alter the faecal flora.

Separate from the interrelated hypotheses on meat, fat, and fibre are observations on alcohol intake and bowel cancer. A survey in the United States showed a striking correlation between beer consumption in 47 States and mortality from colorectal carcinoma[10] that was greater for rectal than colonic cancer (though the distinction between these two sites in epidemiological studies may be of doubtful value since so many of the lesions occur around the junction of the sigmoid colon and rectum). The evidence for an association between beer and colorectal cancer is inconclusive, but a recent study of mortality among blue-collar workers in a Dublin brewery has provided further support.[11] Over 20 years there were 32 deaths from cancer of the rectum compared with an expected 18·2 as estimated from the rates among all inhabitants of Dublin. There was also evidence of an association for carcinoma of the colon though the relative risk was lower: 32 deaths observed compared with 27·3 expected. How beer drinking could lead to large bowel carcinoma is not clear.

At present, therefore, the evidence against any particular dietary factor in causing bowel cancer is inconclusive. As is so often the case, further epidemiological surveys are needed.

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Welcome award: belated justice

Doctors in the NHS will welcome their 1980 pay award,[1] while seeing it as belated justice. But at a time when the Government is trying to keep public sector pay rises within prescribed cash limits it must have swallowed hard before approving the Review Body's recommendations that give the profession average rises exceeding 30% (p 1327), backdated to 1 April 1980. The size of the increases, which range from over £1200 for house officers to nearly £4000 for GPs, and over £4000 for senior consultants, is in part a consequence of the restraints imposed by previous Government pay policies, which, according to the BMA, have cost the medical profession £340 million —not allowing for inflation—in irretrievably lost income since 1975. This is a substantial loss which other groups should recognise when contrasting their 1980 pay settlements with those of doctors. For the first time in five years doctors' pay has caught up with others in comparable walks of life and the Review Body's 1978 commitment to restore this parity was seen "as essential to the maintenance of an effective and efficient NHS for the benefit of the community as a whole."[2]

The public reception accorded to this award—10·7% of it is the final part of the three-stage 30% "catching-up" recommendations made in 1978—should not make doctors feel guilty. But criticisms from Whitehall and Fleet Street about the alleged inflationary effect of independent pay reviews[3] will cause some uneasiness in the profession, who may fear loss of the Review Body. Even the juniors, who for the past two years have eschewed the Review Body system, may now recognise its worth. The medical and dental professions have the oldest independent review machinery and despite occasional malfunctions and two near breakdowns it has served the professions reasonably well and should be preserved. Like all machinery, however, it requires regular overhaul and the clash with the professions over how far it can legitimately go in criticising agreements negotiated with the DHSS (p 1330) and the extent to which it operates a pool system of payment must be quickly resolved. On the latter point, not all doctors will be convinced by the Review Body's protestations (p 1330) that it operates no pool. In this *Tenth Report*, for example, the incremental pattern of the medical assistants' scales is "improved" at the expense of a downward adjustment in their extra-duty payments, surely an illustration of the pool principle. While the experienced people who serve on the Review Body are bound to form definite views about any agreements