# EDITORIAL

## THE PROBLEM OF BONE LOSS

As if chronicity is not enough, osteoporosis pursues a fluctuating course, defies therapeutic efforts and relentlessly holds a great many in its hurtful clutches. It has been with us since prehistoric times, but has yielded very little to all our modern technology-based attempts to understand it.

Loss of bone mass is the pathological basis of the disease, but age alone brings about bone loss. It has been found that, beginning at the age of thirty-five years, bone attrition rate proceeds at 7 per cent per decade for males and 9 per cent for females; the process fortunately plateaus off at about sixty-five years. Patients with osteoporosis may therefore be regarded as having undergone exaggerated age-related bone loss. However the pathophysiology of age-related bone loss is itself shrouded in the enigma of ageing and impinges on the young complex science of gerontology. This approach to clinical osteoporosis is unlikely to help for yet some time to come, but it has the advantage of drawing attention to the need to consider age in assessing clinical osteoporosis.

Bone of course, far from being cold and lifeless, is really very much a live organ. It is continually undergoing resorption and accretion (bone formation). At equilibrium, bone resorption is nicely balanced by bone accretion and there is no change in bone mass. (The state of affairs presumably exists in adulthood, up to the age of about thirty-five). If resorption exceeds accretion, loss of bone mass, hence osteoporosis, results. In the smaller group of patients with certain clear-cut underlying conditions giving rise to an excess of bone resorption over bone accretion, osteoporosis is to be expected. Corticosteroid excess, thyrotoxicosis, acromegaly, hyperparathyroidism, immobilisation and myelomatosis number among such conditions. Thus in corticosteroid excess, not only does bone resorption increase but bone accretion also decreases, causing a marked excess of resorption over accretion. During prolonged immobilisation, bone resorption proceeds at the normal pace but bone accretion slows down, again resulting in resorption exceeding accretion. There are obviously many possible combinations of changes in bone resorption and accretion that culminate in bone resorption being greater than accretion and a net bone loss.

In idiopathic osteoporosis a definition of the precise nature of imbalance between bone resorption and bone accretion, causing bone loss, is the first essential step in our understanding of the disease. This unfortunately has proved to be the stumbling block. The disease process is slow, incurring "small" changes in bone resorption/accretion. These changes are "small" because they are close to the intrinsic error of all available methods of investigation and it is not surprising that reports of conflicting results abound. These results serve only to cloud the issue, and reflect the present state of our ignorance. Attempts at identifying aetiological factors by retrospective and statistical studies have not been helpful either. Thus lack of gonadal hormones, favoured by the observed post-menopausal spurt in the progress of the disease, cannot apply in the many cases that begin in middle age when gonads are still in normal function. It would be difficult to explain on this basis, the entity of idiopathic juvenile osteoporosis, an unusually acute and self-remitting form found only in people not older than 40 years of age. Dietary calcium deficiency appears important in experiments with rats, but is unlikely to be relevant to human subjects, there being no correlation between calcium intake and prevalence of osteoporosis. Thus locally, the calcium intake is about a quarter to a third of that in the West, but our problem of osteoporosis is certainly no worse. Lack of physical activity, known to cause reduction in bone accretion may be more important in aggravating the disease in those who are already afflicted and are restricted in activity by pain. Excessive parathyroid activity, postulated by

some, has not been supported by parathyroid hormone assay.

It would appear that no single aetiological factor plays a principal role. However, it is possible that osteoporosis is a manifestation of diverse aetiologies and it may be that a distinct aetiological factor is to be found in each individual case. Alternatively the aetiology may be multifactorial, a term that is conveniently used often to hide ignorance or lack of confidence.

Treatment of secondary osteoporosis is clearly that of the underlying cause. As for "idiopathic" osteoporosis, the only logical approach in treatment, given our present state of understanding of the diseases, is to concentrate on measures which enhance bone accretion and those which inhibit bone resorption. Factors known to stimulate bone formation include gonadal hormones, phosphate, physical activity and fluoride (although bone formed under the stimulus of fluoride therapy tends to be somewhat fragile). Vitamin D is essential to ensure formation of adequately mineralised bone. Factors suppressing bone resorption are calcium and calcitonin. In any patient, a combination therapy should be considered and given a fair trial. A useful starting combination could be fluoride, calcium and vitamin D.

The symptom of pain in osteoporosis characteristically fluctuates, while investigative techniques are generally not adequate to monitor the slow changes in bone itself. The only method of testing efficacy is long-term prospective assessment. For the doctor and his patient, the point to remember is that the therapeutic measures, if they are going to help, take effect clinically only after a long latent peroid that may run into months. There is no sure or quick answer to this common chronic problem.