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Osteoporosis

In the past radiologists and clinicians have used the term osteoporosis to mean merely reduced bone density regardless of cause and underlying pathology. But some years ago the German morbid anatomists Gerth[1] and E. Rutishauser and A. Maulbetsch[2] described the pathology as being distinct and in particular distinguishable from that of osteomalacia. Later F. Albright and E. C. Reifenstein,[3] in their classic book published in 1948, made the distinction on clinical and biochemical grounds. Their definition of the condition as a disorder of too little bone of normal composition still stands.

Recently C. E. Dent and L. Watson[4] have published an excellent comprehensive review of our present knowledge of the subject. According to D. S. Howell,[5] osteoporosis is the commonest metabolic bone disorder encountered in medical practice, 1 in 4 females and 1 in 5 to 6 males over the age of 70 being afflicted in population studies reported by M. R. Urist and colleagues.[6] In this study about four-fifths of the patients had osteoporosis of the senile or postmenopausal type. But as ageing bone shows much the same changes when it atrophies it is difficult to say at what point physiological ageing becomes early osteoporosis.

M. Trotter and his colleagues,[7] measuring apparent densities of whole bones obtained at necropsy, found that they steadily lost density from the age of 20 onwards in both sexes, and in negro and white races alike. P. J. Atkinson and his colleagues[8] found from biopsy studies that bone density decreased after 50 years of age. In a radiological survey R. A. Caldwell[9] found that bone density decreased steadily and at increasing speed after the age of 20 in both sexes, while J. S. Arnold,[10] studying lumbar vertebrae taken at necropsy, reported a steady decrease from the age of 30 onwards. G. A. Rose[11] concludes from the collected evidence that in most cases osteoporosis is not a disorder which comes on suddenly in late adult life, but is a condition towards which all people gradually progress when they pass their twentieth birthday. With this conclusion Dent and Watson agree, and they see no real advantage in using the term "senile osteoporosis" if it is meant to imply that something new and fresh is happening in old age.

The classification of osteoporosis is difficult owing to dearth of knowledge about its aetiology. Dent and Watson[4] have only two categories: (1) unknown causes (idiopathic osteoporosis), which includes senile, postmenopausal, adult idiopathic, and juvenile idiopathic; and (2) causes known or postulated, a category which includes everything else —immobilization, hypogonadism, rheumatoid arthritis, hyperadrenocorticism, a total of 20 in all. Howell[5] has five categories: (1) hormonal lack—postmenopausal and congenital ovarian agenesis (oestrogen deficiency), eunuchoidism and senility (androgen deficiency), and diabetes mellitus; (2) hormonal excess—hyperthyroidism, acromegaly, and Cushing's syndrome; (3) nutritional lack—inadequate diet and chronic wasting diseases; (4) physical agents and poisons—excessive *x*-ray

therapy, overdosage with ultrasonic waves, and metal poisoning; and (5) disuse. In either classification it is clear that though causes may be postulated there is often considerable disagreement about the truth of the postulate. These categories are in fact no more than convenient trays into which temporarily to put the facts pending more knowledge of the subject.

Osteoporosis may be associated with no symptoms whatever or with slight, severe, or disabling bone pains. It is common to find evidence of collapse of a vertebral body when pain becomes acute, the pain settling, whatever the therapy, to a dull ache with occasional exacerbations thereafter. This back pain may be well localized or may radiate round the trunk or into buttocks and occasionally legs. It is eased by complete immobilization and aggravated by sudden movements such as coughing or sneezing. Compression of a nerve root is uncommon and of the cord very rare. Though the bones are brittle and break easily, the fractures heal well, and the pains usually improve greatly or disappear within a month. Dent and Watson point out that the pain in osteoporosis is not generalized, nor is it nagging, persistent, and unremitting, as it is in severe osteomalacia. It is a suddenly severe pain, improving, then relapsing if further crush fractures occur, a pain due to fracture of brittle bone which is not tender rather than to strain on tender soft bone, as in osteomalacia.

Apart from the idiopathic variety affecting women after middle-age (senile and postmenopausal), perhaps the commonest variety seen in the last 12 years has been that in patients with rheumatoid arthritis receiving corticosteroid therapy. These years were an era of corticosteroid overdosage, overdosage now being defined as anything above 7 mg. daily of prednisone or its equivalent of other analogues of cortisone. The patients have usually been on continuous therapy at a higher dose level for several years, and most are women over 40 years of age. In a study of 61 rheumatoid patients treated with corticosteroids and 36 not so treated B. McConkey, G. M. Fraser, and A. S. Bligh[12] found that severe osteoporosis of the spine occurred equally in both groups, though all five crush fractures that occurred were in the treated group, with none in the untreated. While osteoporosis is undoubtedly a part of rheumatoid disease, immobility and disuse also have a role in its causation, and treatment with an antianabolic agent in addition makes these patients particularly vulnerable.

In systemic lupus erythematosus, polyarteritis nodosa, pemphigus, or any other killing disorder the risk of osteoporosis and crush fractures can be fairly weighed against the benefit of continued life, and the same is true of many cases of severe intractable asthma, but in less dangerous diseases the risks balance up differently. Fractures of ribs are common in these cases, of long bones rare, and the same is true of idiopathic Cushing's syndrome.

Few patients die because of their osteoporosis, though together with other features of the underlying disorder it may contribute to the patient's death. Danger signals, according to Dent and Watson, are continuing loss of height for more than five years and a vital capacity decreasing to less than 1,000 ml. owing to the slow collapse of the thoracic cage. Treatment remains unsatisfactory and largely empirical. Even when the cause is known, as with prolonged prednisone therapy, it is not easy, and often impossible and dangerous, to withdraw or even drastically to reduce the drug. Treatment with sex hormones, male or female or both, has been given, but even after almost 30 years' experience with one of the earliest, methyl testosterone, we are still uncertain of its worth in osteoporosis. Infusions of albumin[3] and of pooled plasma and plasma from cases of osteoporosis[13] have been tried.* Strontium[14] and sodium fluoride[15] have been advocated, but have later been found to be unhelpful.[11 16 17]

B. E. C. Nordin[18] and M. Harrison and his co-workers[19] reported that they had produced a strongly positive calcium balance in osteoporotic patients by means of a high intake of calcium, though G. A. Rose[20] was unable to confirm their results. Dent and Watson do not consider that calcium supplements by mouth have any part to play in the routine treatment of most patients with osteoporosis.

We are left, therefore, with a common condition whose aetiology is still imperfectly understood. The review by Dent and Watson,[4] which won the Maurice Davidson award of the Council of the Fellowship of Postgraduate Medicine, is worthy of detailed study. Some aspects of the treatment of the disease are also reviewed at page 295 of the *B.M.J.* in an article intended to complement this week's programme on osteoporosis in the B.B.C. series for doctors, "Medicine Today."

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Paterson/Brown Kelly Syndrome

Over sixty years ago D. R. Paterson described in this journal[1] the condition of inflammation, accompanied by spasm and stenosis, of the lower pharynx. Paterson pointed out that this may be a cause for dysphagia, and in 1919,[2] discussing dysphagia in women, he noted that cheilosis, glossitis, pharyngitis, and postcricoid carcinoma could be a sequel.

Anaemia was not mentioned in either of Paterson's papers, but, also in 1919, A. Brown Kelly[3] added this feature to the account given by Paterson, and thus the British eponym Paterson/Brown Kelly syndrome was introduced. In the United States H. S. Plummer had described (though not published) a series of patients with long-standing iron-deficiency anaemia, whom he said tended to develop hysterical dysphagia. It was not until 1922 that P. P. Vinson,[4] his pupil, published a full account which emphasized that the spasm was secondary to anaemia. Neither Plummer nor Vinson mentioned postcricoid carcinoma as a complication.