September 3 November 1990

Bone density screening for osteoporosis

SIR,—We are afraid that Dr van Hemert's reply (Sept 29, p 818) to our views (Aug 25, p 502) will add further confusion to what should be a straightforward approach to bone density screening for osteoporosis. Osteoporosis is a condition that is identified by the presence of a low bone mineral density (BMD), a consequence of the decline in bone mass per unit volume. Whether that disease expresses itself as a fracture in the future depends on several other factors such as a continuing adverse lifestyle and absence of sex hormone replacement therapy (contributing to further bone loss), a fall, or a lack of muscle and fat which may protect against the consequences of a fall.

Van Hemert confuses the diagnosis of the disease by BMD scanning with risk evaluation for osteoporosis. We are agreed that clinical risk factors have poor diagnostic value but to use, as he does, metacarpal cortical bone loss measured by a magnifying glass from X-ray films of the hands as a predictive test is mistaken.[1,2] Trabecular bone loss is the major feature of postmenopausal osteoporosis due to oestrogen deficiency and is the main reason for the greatly increased prevalence in women. Dual photon bone densitometry screening identifies this trabecular loss in the spine or hip, the critical areas of fracture. His work did not test this predictive performance. The important epidemiological question is not the overlap in BMD between fracture and non-fracture groups but whether fracture rates in postmenopausal women who have preserved their BMD are equivalent to those in young normal women. Epidemiological evidence suggests that this is likely to be so.[3]

To assert that osteoporosis "seldom leads to death" is to ignore UK Department of Health figures for 1985: there were 44 000 fractures of the hip and almost 9000 of those patients died and 22 000 became invalids, dependent on relatives or the State.[4] Van Hemert himself really makes the point for bone density screening when he emphasises that all menopausal women have bone loss which is likely to be very difficult to restore later. The point of screening is to identify the problem early, when sex hormone replacement will prevent further bone loss for as long as it is taken.[5] Where he is mistaken is in assuming that all women lose bone at 1% per year, taking 30 years to lose 30%—losses which he says cannot be restored. He seems to feel that this is the critical level of loss. In our osteoporosis and menopause clinic we have seen patients who have lost up to 18% of bone in 6 months, while others on treatment have increased BMD by up to 15% in one year, thereby significantly diminishing their risk of fracture. We agree that a 1% loss of bone will have little influence on the risk of fracture, but 10% will, by a factor of 2–3-fold.[6] Van Hemert does not say how he would discover the "rapid bone losers" without dual photon bone density screening and follow-up scans.

We replied in detail previously to the difficulty of offering oestrogen therapy to all menopausal women, concentrating instead on those with osteoporosis as an identifiable group at risk. In such patients the logic of fracture prevention is more likely to prevail over personal, although not necessarily well informed, preference to do nothing and hope for the best. The continuing great reluctance of doctors and patients to consider osteoporosis screening and sex hormone replacement will most effectively be answered by the growing realisation of the prevalence of this condition in women, with rising mortality and morbidity. We hope that this will not take 30 years nor be considered a matter unworthy of attention by a caring profession, as van Hemert's words might imply: "osteoporosis, though a common disease, occurs almost exclusively among old to very old people".

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