Bone densitometry

Is a valuable investigation

EDITOR,—Susan M Ott draws attention to some of the potential pitfalls associated with the use of bone densitometry in clinical practice.¹ Though I acknowledge that caution is needed in interpreting measurements of bone density, their value should not be understated. In particular, distinction should be made between the assessment of risk in an individual person and that in population screening. Because of the gaussian distribution of bone density and the gradient of the risk of fracture over its whole range, the proportional contribution to the total incidence of fracture of those with the lowest bone density is small; a major impact on the incidence can be achieved only by increasing bone density across the whole population. In clinical practice, however, the risk in an individual person is an important factor in decisions about treatment, based on analysis of the risk of disease on the one hand and the ratio of risk to benefit associated with treatment on the other. The present lack of strict densitometric criteria for treatment does not invalidate this approach, particularly for those at high risk.

The figure of around 6% quoted for the precision of bone densitometry is misleading since it applies only to Ward's triangle; for other regions of the femur, such as the femoral neck and trochanter, precision of 3% or less can be obtained with careful positioning and quality control. In the spine, precision is even better, values of around 1% being reported in young and middle aged subjects.² This compares favourably with reported annual rates of bone loss at this site in menopausal women of 1-5%³ and indicates that the ability of serial measurements to show effects of treatment in menopausal women is better than that implied by Ott.

Finally, the finding in prospective studies of a relation between areal bone density and risk of fracture⁴ justifies the clinical use of techniques that measure areal density. There is no evidence that volumetric density measurements are superior in predicting the risk of fracture, and the ability of areal bone density to capture an element of skeletal size may indeed be an advantage since bone size, independent of density, probably contributes to bone strength and hence risk of fracture. Bone densitometry represents a real advance in the diagnosis and clinical management of patients with osteoporosis; the cautions expressed by Ott should not be interpreted as indicating otherwise.

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Precision error is low

EDITOR,—We believe that Susan M Ott's statement that "a walk around the room causes the measurement [of bone mass] to change by up to 6% (at the hip)" is misleading and needs to be put into perspective.¹ The precision error of bone densitometers is usually expressed as the percentage coefficient of variation. We determined the coefficient of variation of duplicate measurements of bone mass at the hip in 19 subjects, removing the subjects from the bone densitometer between the scans. The subjects had bone mineral densities

ranging from normal to severely osteopenic. The coefficient of variation for our dual energy absorptiometer (Hologic QDR-2000) is 1.39% for the femoral neck, 0.92% for the trochanteric region, 0.98% for the intertrochanteric region, 0.94% for the total hip, and 2.42% for Ward's coefficients of variations reported by other investigators.^{2,3}

The change of 6% in bone mass mentioned in the editorial is equivalent to a coefficient of variation of 4.12%, which is much higher than the precision error found at our institution or in earlier reports. From our data we predict that a 6% error in measurements of bone mass of the total hip or at any region of the hip would occur less than once in 1000 times. Thus a 6% change in bone mass or repeat measurement is unlikely to occur when a dual photon x ray absorptiometer is used. Any short term change in bone mass of this magnitude suggests either malfunction of equipment or deficient technique in obtaining reproducible measurements of bone mass.

These opinions and assertions are the private views of the authors and are not to be construed as reflecting the views of the Department of the Army or the Department of Defense.

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Can predict those at high risk of fractures

EDITOR,—Many of the conclusions in Susan M Ott's editorial on measurements of bone mass are misleading if taken at face value.¹ The subheading—"Bone mass and strength not necessarily correlated"—suggests that some studies have shown a lack of correlation between bone strength and bone density. Bone mass and bone strength in vitro are strongly correlated,² but this does not undermine the caution needed in interpreting changes in bone mineral density as indicative of changes in bone strength.

Interpretation of a single measurement of bone mineral density relies on the concept of relative risk, where by some people who subsequently sustain a fracture have a normal bone density at baseline. The same is true for the measurement of blood pressure: most sustaining a stroke have a normal blood pressure. Bone mineral density, however, is as good a risk factor for predicting fracture as blood pressure is for predicting stroke.³ A recent study of over 9000 women followed up for a mean of 1.6 years shows that the risk of sustaining a hip fracture increases by up to 2.9-fold for each standard deviation decrease in bone mineral density⁴—rather more than the 1.5-fold to twofold stated by Ott.

Changes in bone mineral density in a particular patient with time need to be interpreted with caution, especially when measurements are made at the hip. Apparently dramatic changes can be taken as indicating improvement or dramatic bone loss but may simply be due to the precision of the measurement and poor repositioning technique. Dual energy x ray absorptiometry scanners have

precision of 1.5% or better for the femoral neck.⁵

Assessing changes in areal bone density in children is difficult, but Ott's conclusions are ambiguous. Bone area increases with growth, as does bone mineral content, but if bone area outstrips growth, bone mineral density will apparently decrease; this argues for care in interpretation.

Ott's editorial could be used by purchasers as an excuse for not supporting the development of bone density services whereas it should be used to ensure that services are purchased only from providers who run services with adequate quality control, have clinical experience in interpreting results, and offer additional clinical follow up of difficult cases. The indications for assessment of bone mineral density have been fully examined and referenced in a recent publication for purchasers produced by the scientific coordinating committee of the National Osteoporosis Society.³

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Depot medroxyprogesterone and bone density

EDITOR,—It is gratifying when publications arouse a vigorous debate, but the comments of correspondents on our paper¹ are disappointing. Either our writing is not clear or the correspondents have not read the paper properly.

Anne Szarewski and colleagues say that we described the theoretical risk of oestrogen deficiency and osteoporosis in long term users of depot medroxyprogesterone.² Our study in fact showed that spinal bone density increased in women who stopped using depot medroxyprogesterone and became oestrogen replete, whereas it did not either in women who continued to use this contraceptive or in women who had never used it. We deduced this from sequential measurements of bone density. To us this ranks as a prospective study, not a cross sectional one. We are also taken to task for not having given details of plasma oestradiol concentrations. We reported the values in women using depot medroxyprogesterone in our previous study.³ In the recent study 12 of the 14 women who stopped taking depot medroxyprogesterone resumed menstruation, and we took this as evidence of their being oestrogen replete. As described, we measured oestradiol concentrations in the women who remained amenorrhoeic.