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A pioneer in menopause research, Dr. Utian founded the world's first menopause clinic in Cape Town, South Africa, in 1966 and established the Cleveland Menopause Clinic in 1983.

Recipient of many research grants and awards, he is the author of more than 150 scientific publications and five books. He is the Honorary Past-President of the International Menopause Society and Honorary Founding President and Executive Director of The North American Menopause Society. He is also Chairman of the Council of Affiliated Menopause Societies (CAMS) of the International Menopause Society.

Bone Mineral Density Testing: Storm Clouds Rising

The use and potential for abuse of bone mineral density (BMD) testing are escalating rapidly. BMD testing is increasingly being promoted as a universal requirement, with BMD being measured centrally or peripherally and the tests being advertised, publicized and charged for. In short, BMD testing has become big business. But much of this enthusiasm might well rest on a shaky foundation. My concern relates not to the use of BMD in research, but to its application to the individual patient.

Widespread confusion exists as to the utility and utilization of BMD testing in relation to osteoporotic bone disease. There are many uncertainties, but two key questions are in need of urgent answers. Firstly, can we really define, case by case, who is truly at risk for osteoporotic fracture by a single BMD measurement, and when and where that fracture might occur? Secondly, does serial testing give us a true indication of the success or failure of treatments we have provided and hope our patients are utilizing? An addendum to both questions would query the actual quality and consistency of the testing itself.

Permit me to briefly outline my concerns about these two questions in the hope that you will give the matter more intense scrutiny.

Question 1: BMD testing does not measure bone turnover, stability or strength, but bone turnover is probably the most important factor in selecting a treatment for preventing osteoporosis. Indeed, the three most recently published multicenter fracture prevention trials (for alendronate, risedronate and raloxifene) report similar rates of fracture prevention (40-50%) for very different gains in bone density (2.4-8.2%).¹ Indications are, moreover, that the risk of fracture might fall well before bone density peaks.

Questions also remain as to the value of extrapolating BMD at one site for risk assessment at another. In a recent attempt to answer such questions with respect to postmenopausal women over the age of 50, it was reported that nearly half the population had undetected low BMD—including 7% with osteoporosis—as determined by the arbitrary WHO criteria, which are based on *T*-scores comparing older to younger populations. Of course, fractures are not occurring at anywhere near this number. As a group, those with low BMD (-2.5) had four times the fracture rate of those with normal BMD. The authors concede that “no single measurement (peripheral or central) identifies all women with low BMD, nor does any measurement necessarily identify individuals who will experience fracture.”²

Little wonder, then, that attempts are being made to determine who *not* to measure rather than who to measure. Although none of the various guidelines developed for clinical indicators are yet perfect, they continue to merit further investigation.³

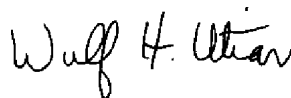
Question 2: My second question relates to serial testing. I have commented above about the variability of BMD response to different therapies. Crandall⁴ reported recently that “there have been no controlled studies showing that change in treatment based on serial BMD measurement results in improved patient outcomes. Whereas studies have shown changes in BMD during antiresorptive therapy to be predictive of fracture reduction in groups of patients, their utility in individual patients remains inconclusive.”

My concern with respect to treating patients is even more fundamental. Women often start with peripheral readings, usually taken in “shopping center screens,” followed by either central or peripheral measurements obtained elsewhere. Follow-up readings are invariably done on different densitometers. Under these circumstances, it is simply not possible to track the change in BMD in one individual patient. As the number of densitometers increases nationwide, competition for scans is likely to make this situation even more pervasive.

I worry even when the repeat scans are undertaken on the same densitometer. There appears to be a failure on the part of all too many clinicians to recognize or understand the issues of accuracy and precision. These are precise terms with major clinical implications.⁵ Densitometry is a qualitative measurement technique that mandates quality control. Most clinicians do not know the precision of the densitometer on which their clinical decision making is to be based.

What does this all imply for clinical practice? My current sense of the situation is that BMD testing is a tool with positive potential for diagnosis and follow-up. As currently utilized, in general, there is however also a wide scope for misuse and abuse. There is, therefore, an urgent need for high standards of accuracy and precision, and the understanding thereof. Clinicians certainly have no excuse for utilizing these instruments without a clear understanding of the strengths and limitations of the technology, as well as an intimate knowledge of the quality-control measures and precision levels of the equipment they are using for clinical decisions. Without such understanding and knowledge, BMD testing is of no greater value than arbitrary measurements of height and weight, or skin thickness, at serial visits—tests that are without cost.

There is considerable risk associated with bringing this entire technology into question. As a first step toward preventing that, and to enhance the value of BMD in clinical practice, consider reading some of the references below.

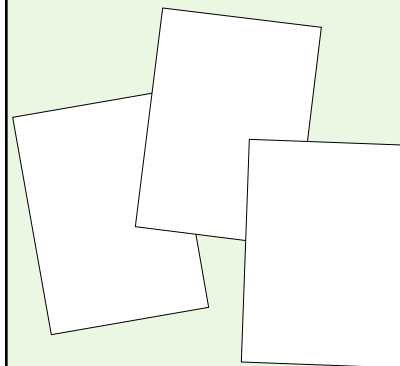


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