

office, does not require direct blood pressure measurement in the cerebral arteries.

Lest the reader conclude that there is no common ground in this debate, we do agree with Dr. Mazess that spinal BMC cannot be sufficiently predicted from measurements at other sites, such as radius and that hip BMC cannot be sufficiently predicted from spine BMC, and vice versa. We do not, however, believe that all of the answers are in; rather we suspect that some important questions have not yet been asked. Will it be necessary to measure every skeletal site at potential fracture risk in order to screen the population and select those individuals who most need preventive therapy? In the case of hypertension and stroke risk, it has not proved necessary to measure cerebral artery blood pressure; peripheral artery measurements are sufficiently predictive for clinical screening. However, it took years of longitudinal stroke incidence data to establish this relationship. Therefore, it will also require additional data to confirm the relationship now suspected between BMC and fracture risk.

We continue to test the hypothesis that peripheral BMC measurements can predict future fracture risk, taking care to include all possible risk factors and BMC measurement sites, without presupposing the results. Despite the incomplete data and our imperfect knowledge, both the nihilistic and the "shotgun" approaches to osteoporosis prevention should be abandoned. For the present, rational osteoporosis preventive choices for individual patients depend upon objective measurements, and their acceptance into clinical practice are strongly influenced by cost considerations.

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Correction: Three Phase White Blood Cell: Diagnostic Validity in Abdominal Inflammatory Diseases

In an article by Becker et al. in *J Nucl Med* 27:1109-1115, 1986, please note the following corrections.

Page 1110, left column, lines 1 and 6 should read 90 g/7 min.

Page 1110, left column, lines 3 and 33 should read 18.5 MBq.