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Effects of Reduced Periosteal Bone Formation and Endosteal Bone Loss

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DESCRIPTION

The challenges regarding identifying the existence of periosteal apposition during adulthood, its site specificity magnitude differs. In cross sectional studies secular changes in bone size may obscure or exaggerate periosteal apposition. These problems are not necessarily resolved by adjusting for height. Secular increases in stature occur male or female individuals. In some races but not all. These secular trends can produce misleading inferences when increments or lack of increments in diameters are used as surrogates of periosteal apposition. If there is no increase in periosteal diameter does not mean periosteal apposition exists there might be either no apposition or extreme condition of periosteal apposition. In some earlier born individuals the bone diameter may be shorter and had more slender bones than later born individuals. When periosteal apposition occurs, earlier born with more slender bones have an increase in bone diameter that comes to equal that in later born group leading to the weak inference that there was no periosteal apposition in the cross sectional sample.

When comparisons are made between races in cross sectional studies, the periosteal apposition is greater in men than women but men have a secular increase in bone size and women do not then the secular increase in men will blunt the increment in bone width across age in men and make it appear that the age related increase in vertebral and femoral neck diameters is similar to women and men. Longitudinal studies are also problematic because changes in periosteal apposition during aging are small. The precision of methods to determine bone diameter usually bone densitometry and problems with edge detection when bone mineral density is changing limit the credibility of these measurements. Periosteal apposition is believed to increase as an adaptive response to compensate for the loss of the strength produced by endocortical bone loss so there will be no net loss of bone, no cortical thinning and no loss of bone strength. In a prospective study it is reported that endocortical bone loss occurred in premenopausal women with concurrent periosteal apposition. As periosteal apposition was less than endocortical resorption, the cortices thinned but there was not net bone loss because the thinner cortex was now distributed around a larger perimeter conserving total bone mass. Moreover resistance to bending increased despite bone loss and cortical thinning because this same amount of bone was now distributed further from the neutral axis. So bone mass alone is a poor predictor of strength because resistance to bending is determined by the spatial distribution of the bone.

Thus, endocortical resorption increases during the perimenopausal period, yet periosteal apposition decreased it did not increase as predicted if the notion that periosteal apposition is a compensatory mechanism is correct. The cortices thinned as periosteal apposition declined further. Nevertheless, bending strength remained unchanged despite bone less and cortical thinning because periosteal apposition was still sufficient to shift the thinning cortex outwards. Bone fragility emerged only after menopause when accelerated in endocortical bone resorption and deceleration in periosteal apposition produce further cortical thinning.