Vertebral Strength Gains From Small Rise in BMD

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NASHVILLE, TENN. — A little increase in bone mineral density associated with parathyroid hormone use appears to translate into greater vertebral strength, according to data presented at the annual meeting of the American Society for Bone and Mineral Research.

To assess changes in vertebral strength, the researchers used finite element analysis, a technique borrowed from engineering, where it is used to design bridges, skyscrapers, airplanes, and more, said Dennis M. Black, Ph.D., a professor of epidemiology at the University of California, San Francisco.

Starting with a quantitative CT (QCT) scan, the structure of interest is divided into many finite elements—in this case voxels—each of which is assigned material properties based on the bone mineral density (BMD). Computer simulation is then used to apply a set of forces—in this case compressive force—to the model to estimate the mechanical response. One of the responses of the most interest is strength, which in engineering terms means the applied force necessary for the structure to fail—in other words, the force necessary for the vertebra to fracture.

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The researchers used the technique in a pilot study to assess vertebral strength changes, using data for 19 randomly selected women enrolled in the Parathyroid Hormone and Alendronate for Osteoporosis study. The postmenopausal women had all been randomized to receive 100 mcg of parathyroid hormone (1-84) daily for 1 year. QCT and dual x-ray absorptiometry (DXA) measurements were performed at baseline and at 1 year. The QCT data was used to look at changes in estimated vertebral strength using finite element analysis.

The average estimated compressive strength was 4,522 N. At 1 year the average estimated compressive strength was 5,715 N—a statistically significant increase in overall vertebral strength of 29%. While overall vertebral strength increased by 29%, overall BMD increased by only 6% based on DXA measurements.

The researchers were also able to virtually "peel away" the outer 2 mm of each vertebra (assumed to be cortical bone), in order to assess changes in the strength of trabecular bone. The increase in trabecular BMD was 29%, as measured by QCT. Trabecular bone accounted for 70% of the increase in total strength over the course of 1 year. At baseline, trabecular bone strength accounted for about half of total bone strength. "From this we inferred that the majority of the increase in strength is attributable to increases in trabecular strength," Dr. Black explained.

The researchers also compared the increase in strength due to an average increase in bone density with the increase in strength due to a redistribution of bone density in the vertebra. To do this, the researchers assumed that the vertebra has a homogeneous density. So each element in the vertebra is assigned the same density—the average density for the vertebra. "So if we saw that the average density change leads to a strength change that is small, we would then infer that some of the overall increase in strength is due to a redistribution of density," said Dr. Black.

In fact, they found that the increase in average bone density accounted for most—but not all—of the increase in strength. "This suggests that not all of the increase in strength can be attributed to the average change in bone density," he said.

Researchers had previously validated the technique for assessing vertebral compressive strength by comparing the results of compression tests on cadaveric vertebra (Bone 2003;33:744-50). The finite element measures of strength correlated well with the test results.

The study was funded in part by NPS Pharmaceuticals Inc., maker of Preos (recombinantly produced, full-length human PTH), which is under development. Dr. Black also receives consulting fees from NPS Pharmaceuticals and is speaker for Merck & Co. Inc., maker of Fosamax (alendronate). ■