LOCAL BIOMECHANICAL INFLUENCES IN OSTEOPOROTIC VERTEBRAL “WEDGE” FRACTURES

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INTRODUCTION:
Hormonal changes and inactivity lead to systemic bone loss and osteoporotic fractures in many elderly people. However, it is not clear why the vertebral body should be affected so often, or why its anterior region should characteristically sustain a “wedge” deformity. These features may be caused by local biomechanical factors, because local alterations in vertebral architecture are associated with age-related degenerative changes in the adjacent intervertebral discs (1).

We have shown that severe disc degeneration reduces disc height, and this in turn causes the neural arch to become substantially load-bearing in erect postures (2). In effect it “stress-shields” the vertebral body to such an extent that, in many old specimens, more than 50% of the spinal compressive force is resisted by the neural arch, and loading of the anterior vertebral body is greatly reduced (3). We hypothesise that this alteration in load-sharing in old spines leads to a redistribution of bone mass, so that the anterior vertebral body becomes vulnerable to fracture when subjected to high compressive loading in stooped postures.

METHODS:
Forty thoraco-lumbar motion segments (two vertebrae and the intervening disc and ligaments) were obtained from cadaver spines aged 62-94 yrs (mean 79 yrs). All spinal levels between T9-10 and L4-5 were represented. Bone mineral density (BMD) was measured for various regions of each vertebra using a Lunar Piximus DXA scanner, and water immersion to calculate volume. Specimens were mounted in cups of plaster for loading on a Dartec computer-controlled materials testing machine. The distribution of the applied compressive force (1.5 kN) on each vertebra was measured by pulling a needle-mounted pressure transducer along the sagittal midline diameter of the adjacent disc. In most regions of the disc, transducer output is approximately equal to the compressive stress acting perpendicular to the transducer membrane (4).

“Stress” measurements were integrated over area to give the force acting on the anterior and posterior halves of the vertebral body (2). These forces were subtracted from the applied 1.5 kN to indicate the compressive force resisted by the neural arch. Measurements were repeated with the specimens positioned to simulate various postures in life. The strength of each motion segment was determined by compressing it to failure while positioned in 4-6° of flexion to simulate a stooped posture. Strength was calculated as the force resisted at the elastic limit. Disc and vertebral morphology were assessed from radiographs, and from digital photographs of tissue sections.

RESULTS:
Load-bearing by the neural arch in erect posture increased in the presence of intervertebral disc degeneration, and was inversely proportional to the average height of the disc (P<0.01). High neural arch load-bearing was associated with low BMD in the anterior vertebral body (P<0.01). The posterior shift in bone density in specimens with high neural arch load-bearing was apparent from radiographs (Figure 1)

Compressive damage resulted in a 1.3% reduction in motion segment height, and was usually confined to one of the two vertebrae. Compressive strength was inversely related to neural arch load-bearing as shown in Figure 2.

Measurements on the damaged and undamaged vertebrae in each motion segment were very similar. BMD in the anterior half of the vertebral body was the best univariate predictor of compressive strength (R² = 0.78). BMD of the whole vertebra, or of other vertebral regions, were relatively poor predictors of strength. Stepwise multiple linear regression yielded the following model of motion segment compressive strength (R² = 86%): Strength = 1.26*10⁴*BMDa + 97.5*Area – 14*NA, where BMDa = BMD of anterior half of vertebral body (g/cm²), Area = minimum X-sectional area of vertebral body (cm²), NA = neural arch load-bearing (% of applied load).

Forcing age, gender and spinal level into the model did little to improve the prediction of strength.

DISCUSSION:
The results clearly support the hypothesis: they show that altered load-sharing in elderly spines can influence the distribution of bone mass, and hence the spine’s compressive strength. Intervertebral disc degeneration and narrowing cause the neural arch to resist much of the compressive force acting on the spine whenever it is held erect. The anterior vertebral body is then effectively “stress shielded” and loses BMD, weakening the spine when it is loaded in moderate flexion. In life, the spine would be flexed during activities such as stooping to pick something up. The small age-dependence of results probably reflects the relatively narrow age range of old specimens tested. If these results are applicable to living people, then measurements of BMD in the anterior half of the vertebral body may prove to be a better indicator of fracture risk in elderly people than measurements of overall vertebral BMD.

REFERENCES:

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Figure 1. Radiograph of motion segment that exhibited high load-bearing by the neural arch. When image density was optimised for the apophysial joints, the spinous processes became invisible.