LOAD DISTRIBUTION IN VERTEBRAL BODIES IS AN INDICATION OF OSTEOPOROSIS

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Introduction:
Osteoporosis is characterized by bone loss and diminished architectural integrity, leading to an increased fracture risk. As a result of this the bone is not strong enough to carry the normal daily loads. In clinical practice, the best estimate of bone strength can be derived from BMD measurements. However, there is a large overlap in BMD levels between groups with and without spontaneous fractures. As a result of this overlap, the percentage of false negatives is 80% of the actual population at risk for fracture[1].

Another important aspect related to bone strength is the load transfer. In vertebrae, the load distribution between the trabecular core and the cortex is of particular relevance. It not only depends on the vertebral body, but also on the properties of the intervertebral discs. Kurowski and Kubo[2] have demonstrated that a healthy disc, with a load bearing nucleus, places more load on the trabecular core. Whereas a degenerated disc, with no load bearing of the nucleus, places most of the load on the cortex.

Different types of vertebrae may respond differently to changes in the intervertebral disc. Normal vertebrae may respond differently than osteopenic and osteoporotic ones. The aim of this study was to determine how a change in the mechanical behavior of the nucleus affects the load distributions in the different groups of vertebrae.

Methods:
In this study we used 49 vertebral bodies, which were categorized based on BMD. Ten were diagnosed as normal (BMD > 100 [mg/cm^3]), fourteen as osteopenic (80 < BMD < 100 [mg/cm^3]) and twenty-five as osteoporotic (BMD < 80 [mg/cm^3]). Three-dimensional computer reconstructions of the vertebrae were made using a CT scanner (Somatom Plus S, Siemens AG: in plane resolution 182x182 micron, slice thickness 1mm). The computer reconstructions were converted to FE-models with elements of approximately 910x910x1000 micron. The densities of the bone elements were linearly related to the Houndsfield units from the CT-data. The Young's modulus of a bone element was related to its density by a relationship taken from literature[3]. The Poisson's ratio was taken uniformly at 0.30. An artificial disc was placed at both endplates. The annulus elements had a Young's modulus of 10 MPa and a Poisson's ratio of 0.45[2]. The nucleus elements had a Poisson's ratio of 0.49. The Young's modulus was varied to represent either a degenerated nucleus (1 MPa) or a healthy nucleus (100 MPa). Altogether every FE-model contained about 91 000 isotropic eight node brick elements (Figure 1). A total load of 1000 N (about twice the load of normal standing[4]) was applied by means of a longitudinal displacement of the top plane relative to the bottom plane. The posterior side was compressed less than the anterior side, representing 2 degrees flexion (corresponding to *in vivo* movements of the spine[5]). The stress distributions throughout the vertebrae were evaluated. For every vertebra the load through the trabecular core and the cortex were evaluated. A segmentation program was used to determine whether an element belonged to the trabecular core or to the cortex (Figure 2)

Results:
Near the endplates the load was nearly equally shared between the trabecular core and the cortex (40% vs. 60% respectively). Moving away from the endplate, the load was gradually transferred to the cortex. In the middle the trabecular core carried only 25% of the load, the rest of the load (75%) was carried by the cortex (Figure 3). There was no significant difference in this load distribution between the three groups of vertebrae. When the stiffness of the nucleus was increased from 1 to 100 MPa the amount of load through the trabecular core increased as well. This effect was more pronounced in normal vertebrae than in osteopenic and osteoporotic ones. The load through the trabecular core increased by 3.6%, in the normal vertebrae; in the osteopenic and osteoporotic vertebrae the increase was 1.8% and 0.8% respectively. These differences were highly significant: normal vs. osteopenic p=0.000121, normal vs. osteoporotic p=0.000002 and osteopenic vs. osteoporotic p=0.000089 (student t-test).

Discussion:
The sensitivity of the load distribution to the characteristics of the nucleus was shown to be significantly different between the three groups (normal, osteopenic and osteoporotic: high, middle and low density). Rockoff et al.[6] have shown that the trabecular core carries 29-58% (mean ± SD) of the load applied to a vertebra. In the vertebrae in this study the trabecular core carried 22-49% (mean ± SD). These percentages are somewhat lower then those reported by Rockoff, which is most likely due to the difference in age groups (this study: 53-83, Rockoff: 27-69 (mean ± SD)). Rockoff found that the percentage of load carried by the trabecular core decreases with age. In the region near the endplates the trabecular bone carried 28-54% (mean ± SD) of the load but in the middle region these percentages dropped to 17-34% (mean ± SD). This difference is due to the transfer of the load from the trabecular bone near the endplates to the cortex toward the middle of the vertebra. These findings show that in normal vertebrae the load distribution is sensitive to the behavior of the disc, whereas in osteopenic and osteoporotic vertebrae the load distribution is not sensitive to the properties of the disc. The lower sensitivity of the load distribution in the osteopenic and osteoporotic vertebrae could also play a role in the adaptation processes. We speculate that the load distribution as found here enhances further bone loss in the trabecular core. Load distribution in vertebral bodies can thus be considered as an early indication of osteoporosis.
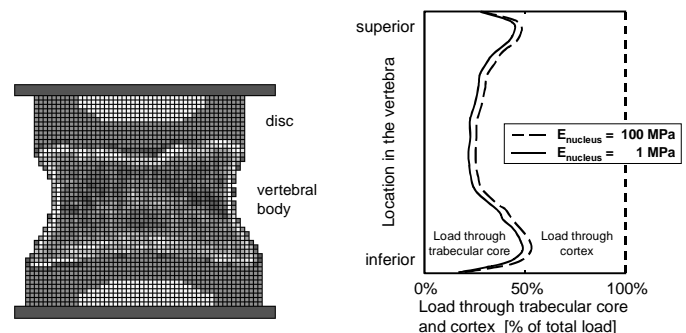

Figure 1: Mid coronal section of one of the FE-models

Figure 3: Load distribution in a typical vertebra.

Figure 2: Segmentation into cortex and trabecular core. Left the original vertebra, middle the cortex and right the trabecular core.

References:
[1] Melton et al., 1989, *Am.J.Epidemiol.*, **129**: 1000, [2] Kurowski and Kubo, 1986, *Spine*, **11**: 726, [3] Carter and Hayes, 1977, *J.B.J.S.*, **59A**: 954, [4] Nachemson, 1981, *Spine*, **6**: 93, [5] Adams and Dolan, 1991, *J.Biomech.*, **24**: 117, [6] Rockoff et al., 1969, *Calc.Tiss.Res.*, **3**: 163