Introduction
The presence of an osteoporotic vertebral compression fracture increases the risk for subsequent vertebral fractures. Residual kyphotic deformity is considered a risk factor for subsequent fractures; however, it cannot explain the increased fracture risk after biconcave osteoporotic fractures in the lumbar spine, and cannot always predict the risk for adjacent fractures after a wedge fracture in the thoracic and thoracolumbar spine. We hypothesized that endplate fracture will alter the pressure profile of the intervertebral disc and result in more concentration of load at the anterior part of the adjacent vertebra when the spine is flexed, increasing the risk for adjacent fractures in osteoporotic spines.

Materials and Methods
Six fresh-frozen, human lower thoracic (T8-T12) or thoracolumbar (T10-L2) specimens (age 61 to 82), each consisting of 5 adjacent vertebrae, were used. A novel technique was utilized to selectively fracture only one of the endplates of the middle VB of each specimen. Through a small anterior opening a void was created selectively under the upper or the lower endplate and was extended to one-third of the VB trabecular content. The void was randomly assigned to the upper third of VB in three specimens and at the lower third in the other three. The specimen was then placed in flexion and compressed using bilateral loading cables passing through guides attached at the sides of the VBs, until a fracture was observed by fluoroscopy. The fracture was reduced with spinal extension under 150 N preload, until complete correction of vertebral kyphosis was achieved. After cementation of the fracture, the rest of the VB's trabecular content was evacuated through a separate small anterior opening, and the VB was completely filled with cement.

Specimens were tested in flexion-extension (±6Nm) under 400N preload, first intact and then after the index fracture and cementation of the middle VB. Pressure was recorded at the discs above and below the fractured VB and strain was recorded at the anterior wall of the adjacent VBs before and after the fracture. Disc pressure was normalized so that pressure in neutral position under 400N preload was taken to zero, to compensate for thermal-drifting of sensors after cement curing. Range of motion (ROM) was measured using biaxial inclinometers attached to the top VB of the specimen. Disc pressure and strain values before and after the index fracture were compared using repeated measures ANOVA.

As a final step, the specimen was placed in 2/3 of maximum flexion and was loaded using the bilateral loading cables that were attached to actuators. The loading was gradually increased from 0 to 3000N or until a subsequent fracture was observed on fluoroscopy.

Results
In the intact specimens, nucleus pressure gradually increased during flexion. This can more evenly distribute the load during flexion to the entire surface of the endplate and avoid excessive load concentration to the anterior portion. After an endplate fracture, the nucleus is no longer capable of increasing its hydrostatic pressure in flexion, which in turn forces the anterior annulus to bear more weight, and transfer it "unevenly" to the anterior part of the VB. The strain was nearly doubled at the VB adjacent to the fractured end plate, but changed only by about 20% at the other adjacent VB. This small strain increase in the VB adjacent to the intact endplate could be explained by the increased flexion ROM after the fracture. The altered mechanical behavior of the nucleus can be ascribed to the increased available space after the endplate depression. A paradoxical observation from this study was that after the index fracture, the pressure in the nucleus gradually decreased during flexion. It can be assumed that the anterior annulus, being the only portion of the disc capable of bearing axial load, functioned as a fulcrum in flexion causing distraction at the mid intravertebral area during flexion.

After loading the specimens to failure, all adjacent fractures were located at the VB next to the damaged endplate. The fractures started as a depression of the anterior portion of the endplate close to the anterior wall, which subsequently led to anterior wall collapse when loading was continued.

Discussion
This experiment was designed to isolate the effects of endplate fracture on the load bearing ability of the adjacent disc and its role as a risk factor for subsequent adjacent VB fracture by eliminating other previously proposed factors. The vertebral kyphosis was completely reduced by spinal extension. Cement was similarly distributed under both endplates that were carefully abraded, all attached trabeculae were curetted away and the VB was evenly filled with cement.

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Figure: Example of disc pressure and anterior wall strain changes after upper endplate fracture of the middle vertebra.

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