INTRODUCTION:
In many elderly people, one or more vertebrae lose height, especially anteriorly. Such anterior “wedge” deformities lead to senile kyphosis a condition that can cause pain, disability and loss of self-esteem. Metabolic processes weaken elderly bone, and altered load-sharing in the elderly spine causes mechanical damage to be concentrated in the anterior vertebral body (1). The resulting deformities are often referred to as “fractures”, but the insidious onset of many vertebral deformities suggests that gradual time-dependent “creep” processes can also contribute. Bone is known to have viscoelastic properties (2), but creep deformity of whole vertebrae has not previously been investigated. We hypothesise that creep processes can contribute to anterior wedge deformity in elderly human vertebrae.

METHODS:
17 cadaveric thoraco-lumbar “motion segments”, consisting of two vertebrae and the intervening disc and ligaments, were obtained from 11 human cadavers aged 42-89 yrs (mean 66 yrs). Each specimen was subjected to a constant compressive load of 1.0 kN for 30 minutes. Vertebral deformations in the sagittal plane were monitored at 1 Hz using an optical MacReflex system, which located the centres of 6 reflective markers glued to pins inserted into the lateral cortex of each vertebral body. In-plane accuracy was better than 10 µm. Two pins each defined the anterior, middle and posterior vertebral body height, and deformations were expressed as a % of vertebral height at zero load.

RESULTS:
Figure 2 shows typical vertebral creep during a 2 hour test. The smoothness of the curves suggests a quasi-continuous process. Creep deformations were comparable to the initial elastic deformations, but only the latter appeared to be recovered after 2 hours of unloading. Creep and elastic deformations were greatest for the anterior vertebral body. Table 1 summarises average results for the 30 minute creep tests. ANOVA showed that creep was greater anteriorly than posteriorly (P<0.01), giving rise to an average anterior wedging of the vertebral body of 0.2° (intact vertebrae) and 1.8° (fractured vertebrae). Creep also increased markedly after fracture (P<0.001) especially in the anterior vertebral body which was damaged most by the overload event.

DISCUSSION:
These results support our hypothesis: they show that gradual creep processes arising from sustained compressive loading can contribute to anterior wedge deformity of the human vertebral body. Creep is greatest anteriorly because this is where bone loss is greatest in elderly vertebrae (1). The underlying mechanism is unclear, but the fact that creep increases when the bone is damaged suggests that some continuous or repeated yielding processes are involved. Lack of creep recovery (during 2 hours) suggests that bone creep is not primarily attributable to microscopic fluid displacements (as in cartilage) because such fluid flow is reversible.

In these experiments, vertebral wedge deformity after 30 minutes of physiological loading averaged 0.2° and 1.8° in intact and damaged vertebrae respectively. In living people, body temperature would probably increase creep rate, but vertebral deformity over long periods would largely depend on how rapidly creep decreased with time (Fig. 2) and on how much it could be recovered. Creep deformity could possibly be reduced in-vivo by postural advice, or by wearing orthoses.

REFERENCES: