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DYNAMIC LOADING, VERTEBRAL BODY FLUID AND ENDPLATE DEFORMATION

Abstract

Introduction: The intervertebral disc (IVD) is dependent upon movement of molecules into the vertebral body (VB) and across the vertebral end-plate for nutrient supply. Recent work showed that loading of the spine can impact diffusion and convection into the IVD. Despite the role the VB plays in supplying nutrients to the IVD, the impact of spinal loading on fluid movement in the vertebral body (VB) is not well understood. Methods: 24 male subjects underwent a step-wise load-sustained load-load release protocol using the Dynawell L-Spine device. A rapid (1.8 minutes) T2-weighted turbo-spin echo acquisition was implemented. Two scans were taken before loading and the scan repeated after 10kg, 14kg and 50%of body mass was applied. Load was then sustained at 50% of body mass for 14 minutes with five further repeated scans at this loading level. After load release, three follow-up scans at rest were performed. The IVD was segmented into 5 subregions anterior to posterior. The VB was segmented into 5 rows and 5 columns. Geometry and T2-weighted image intensity were measured. The lower lumbar IVDs (L2/3 to L5/S1) and VBs (L3 to L5) were measured and average data from all IVDs and VBs calculated. Results: The height of the VB reduced by 0.40mm (p<0.0001) at the end of sustained loading and did not return to pre-loading levels within 9 minutes after load release (0.14mm p=0.002). Average lumbar VB T2-weighted intensity reduced progressively with load (10kg: +0.06% p=ns, 14kg: -0.56% p=ns, 50% body weight: -3.26% p=0.001) and was reduced by 4.3% (p=0.0002) by the end of the sustained loading protocol. VB signal intensity returned to pre-loading levels at the first scan upon load release (-0.11% p=ns). Whilst IVD compression (height reduction) did recoil after load-release, IVD T2-weighted intensity did not (-4.0% p<0.0001 at final 50% scan versus 3.5% p=0.001 at final scan 9min after load release). <u>Discussion</u>: Loading of the spine at low loading levels results in inward bulging of the vertebral endplate under compression and reduces T2-weighted signal intensity in the vertebral body. We interpret these signal intensity changes to reflect reductions of fluid (blood) movement into the VB with load, and with a return upon load release. Deformation of the endplate and volume changes of the vertebral body with loading may be one mechanism for impacting nutrient supply to the IVD. The findings support an influence of loading/movement on nutrient supply to the VB and hence IVD.