Nucleus pulposus fluid pressure decreases after the degenerative loss of incompressibility and area
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Introduction: Intervertebral disc (IVD) degeneration and rupture are common etiology of low back pain [1]. Nucleus pulposus (NP), the central part of IVD, is filled with fluid that changes volume depending on the load and maintains the hydrostatic pressure in the disc [2]. During disk degeneration, the NP loses fluid and hydrostatic pressure is reduced or absent [3]. Instead of uniform stress distribution across the disc, a degenerated disc stress profile shows multiple peaks in outer layers of annulus fibrosus (AF) and low stresses in NP [2, 4]. Measuring the fluid flow changes within healthy and degenerated IVD in vivo or ex vivo is a difficult task. An alternative approach is finite element (FE) analysis which provides estimate of stress and fluid flow inside IVD in normal and degenerative case. It is hypothesized that the loss of NP incompressibility and surface area decrease pore fluid pressure and fluid velocity. The study objective was to predict fluid flow changes in IVD after loss of NP incompressibility and surface area, using a nonlinear FE model.

Materials and Methods: A poro-viscoelastic FE model of L3-L4 spine functional unit was previously developed (Fig. 1) and validated against cadaver range of motion data. Cortical bone (E=12GPa, v=0.3) and posterior bone (E=2.5GPa, v=0.3) were considered non-porous. Cancellous bone, cartilage endplate, NP, and AF were assigned solid and fluid bulk modulus, along with porosity and permeability values. Ligaments were modelled as hypoelastic truss elements with strain dependent stiffness. IVD degeneration was simulated by changing the surface area and Poisson’s ratio (compressibility) of the NP. Healthy L3-4 model was modified to create 6 degenerated IVD cases: 1) NP Poisson’s ratio, v = 0.45; 2) NP v=0.40; 3) NP v= 0.45 and ANP= 15% AIVD; 4) NP v= 0.45 and ANP= 25% AIVD; 5) NP v= 0.40 and ANP= 25% AIVD; and 6) NP v= 0.40 and ANP= 15% AIVD. L4 lower endplate was fixed in all directions. Outer ring of annulus fibrosus could exchange fluid with the environment. A compressive preload of 400N (Comp) was applied followed by 10Nm pure moment in extension (Ext), flexion (Flex), lateral bending (LB), and axial rotation (AR) motions were simulated. Pore pressure, and pore fluid velocity were estimated after simulation.

Results and Discussion: Pore fluid pressure were estimated as decreasing in NP and increasing in AF with increase in NP compressibility and decrease in surface area (Fig. 2). However, loss in change in area of NP matrix cause pressure and velocity to drop more compared to increase in compressibility only. Similarly, pore fluid velocity decreased up to 80% and increased up to 20% in NP and AF, respectively. Loss of fluid flow increases the degradation of the NP over time while increase of fluid pressure leads to bulging out of AF and nerve impingement.

Translational Impact: Loss of incompressibility and stiffening of matrix in NP together cause large alternations in fluid flow. Loss of fluid flow increases the degradation of the NP over time while increase of fluid pressure leads to bulging out of AF and nerve impingement.

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