Supplemental Material Unit 6: Vertebrae and Intervertebral Disc Anatomy and Function

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We are going to take a closer look at the anatomy and function of the vertebrae and intervertebral disc (IVD). A majority of this information is from Dr. Stuart McGill's text, "*Low Back Disorders: Evidence-Based Prevention and Rehabilitation*". I believe anyone treating low back pain should have this book. I had the great opportunity to attend Dr. McGill's two day seminar in Saco, Maine. To give you some perspective on how much Dr. McGill is admired. I can tell you it is the only time I've seen rehabilitation and fitness experts alike ask an author to sign a copy of their book. You can find some of Dr. McGill's work at his website http://www. Backfitpro.com.

Our first look will be the vertebral body (*Figure 1*). The vertebral body is somewhat analogous to a barrel. Imagine a barrel with a hard outer layer with most of its volume inside consisting of a cork-like material. This depiction represents the cortical and trabeculae bone, respectively. The trabeculae bone forms an elaborate set of scaffolding. The architecture consists of vertical columns with cross beams running in oblique patterns. This pattern of scaffolding is a product of the dominant trajectories of stress[1]. The vertebral body also contains a network of veins. The venous structures of the vertebral body have no valves, which is uncharacteristic of most veins in our body. This serves a purpose in order to establish a hydraulic outlet from the vertebral body[1]. The vertebral body exchanges nutrients and waste products to-and-from the IVD.

The IVD under normal conditions is avascular. It receives oxygen and glucose from the venous system housed within the vertebral body. There is a key structure involved in this interface between the vertebrae and IVD. This is the vertebral end plate (*Figure 1*). The vertebral end plate is cartilaginous with a thick periphery and thin center. The movement of fluid to-and-from the IVD is driven by mechanical loading. For instance, we are normally a bit taller upon first getting up in the morning. You have probably heard that before. But why is it so? This is due to the spine being in a decompressed (non-weight bearing) state for several hours. Water and nutrients travel from the vertebral vasculature and across the end plate via diffusion. This causes the disc to rehydrate and replenish nutrients. During the course of the day gravity is steadily compressing our spine. The fluid begins to gradually diffuse from the disc through the end plate. Waste materials and spent nutrients are received by venous structures in the vertebrae. Waste products are then taken up and cycled out through the venous system.

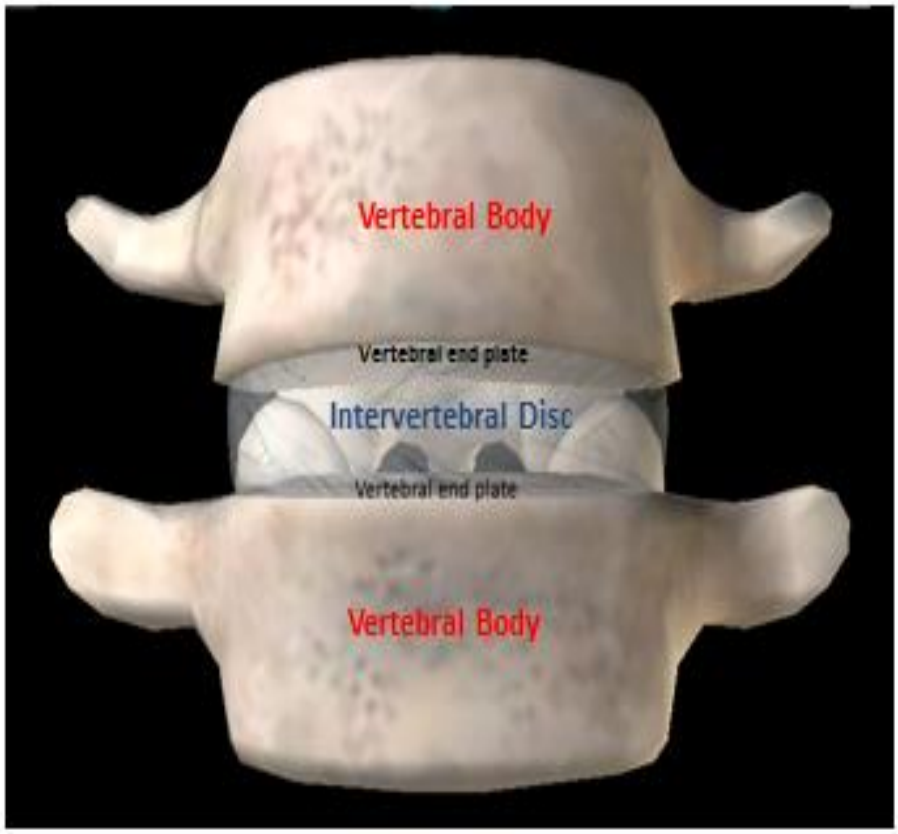

Figure 1: Basic Anatomy of the two vertebral segments (red) and intervertebral discs (blue). The locations of vertebral end plates (black).

Clinical Perspective: A hydrated IVD provides a stiffer vertebral structure due to tautness of the surrounding ligaments. However, if someone has a disc herniation it can be problematic when first arising in the morning. There is evidence to show that patients limiting forward bending postures during the morning hours significantly reduce daytime pain intensity compared to those that do not. Also, patients may intuitively stretch out their lower backs first thing in the morning. This is likely due to a perceived sense of stiffness secondary to tautness of ligaments and change in the IVD hydrodynamics. This is not a good idea especially if their chosen method is knee-to-chest or toe touches. In fact, it could contribute to further disc injury.

Night time sleeping position is important. A good piece of advice to patients regarding sleeping position is to be supine with legs supported under pillows. This unloads the spine and allows for fluid exchange from the vertebrae, through the end plate, and into the IVD. Most patients will tell you "I can't sleep all night like that"! It is actually more important that you start there. In fact, about 80% of the nutrient exchange into the IVD occurs within the first couple of hours the spine is unloaded. It may be of less consequence which position you assume the remainder of the evening.

A closer look at the IVD shows us three major components: *Nucleus pulposus (1)*, *Annulus fibrosis (2), and End plates (3)* (*Figure 2*). Let us examine each component in more detail. The nucleus pulposus is a gel-like material with collagen fibrils contained within water and mucopolysaccharides[1]. It is essentially a sugary-snot that is incompressible. It possesses viscosity and a little bit of elasticity. The nucleus pulposus is somewhat analogous to a ball bearing (*Figure 3*). The annulus fibrosis is made up of concentric rings with multiple layers called lamellae. Collagen fibers making up the lamellae are oriented in an oblique fashion, and therefore each layer runs in opposing direction relative to adjacent layers. The outer most layer of the annulus fibrosis is anchored to the vertebral body through Sharpey's fibers. The inner fibers of the annulus fibrosis are anchored to the end plate (*Figure 2*).

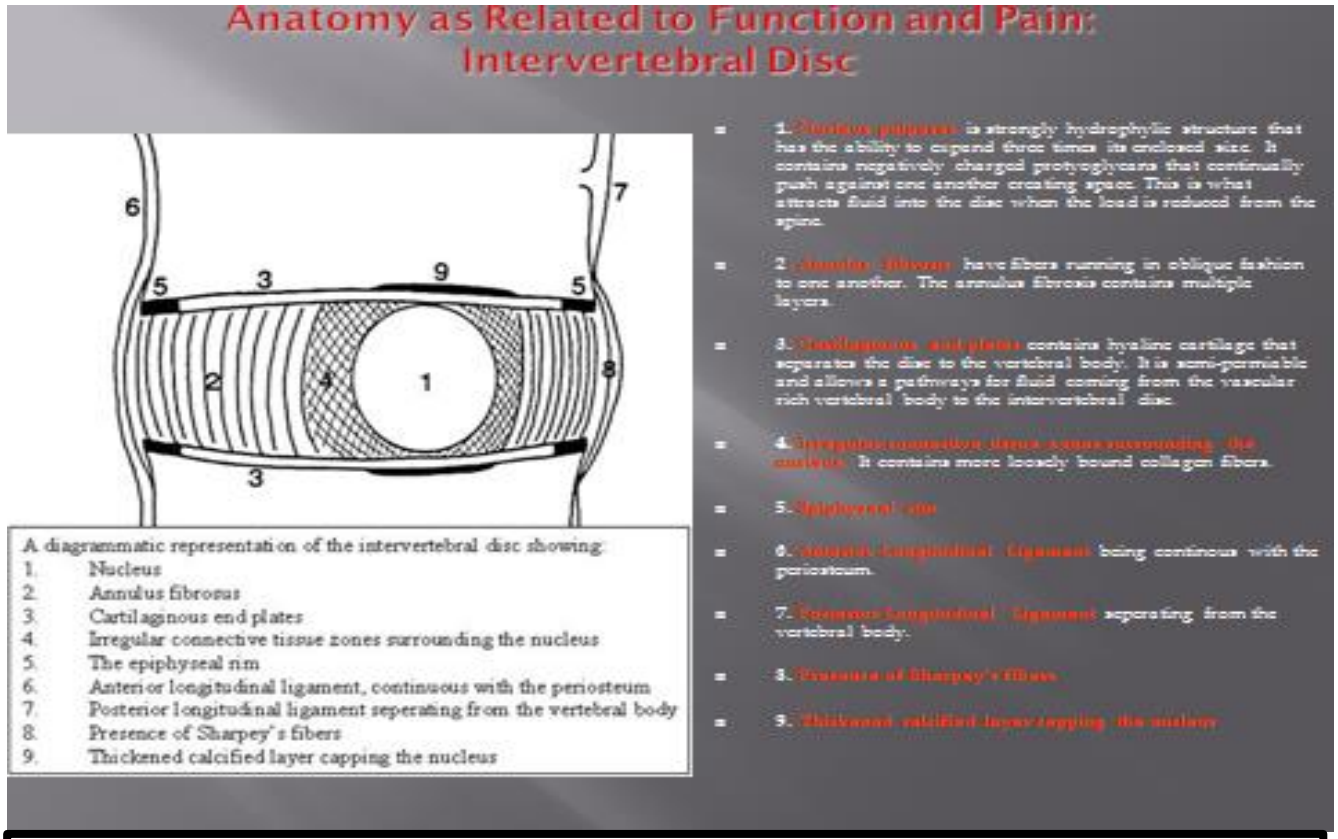

Figure 2: Components of the Intervertebral Disc.

Clinical Note: A basic knowledge of vertebral-disc anatomy should debunk the "slipped disc" myth. An IVD does not literally slip. If so, we would have many individuals with spinal cord injury. You can imagine how simplified analogies, by well intention medical providers, may in fact place misconceptions in the minds of their patients. Analogies are powerful things – good or bad.

Under compressive load the nucleus pulposus is pressurized and applies hydraulic forces both vertically and outward into the vertebral end plate and inner lamellae, respectively (*Figure 4*). The nucleus pulposus under normal conditions is an incompressible structure. The hydraulic forces directed vertically into the vertebral end plate causes it to bulge into the vertebral body. The vertebrae resist

compressive load through fluid expulsion of the venous structures and trabeculae architecture. It is now strongly suggestive that this disc-end plate-vertebrae assembly is the shock absorber, versus the IVD exclusively. A loss of normal IVD hydrodynamics leads to the outer layers of the annulus to bulge outward, but the inner layer bulges inward under compressive load. Over time progressive demyelination of the lamellae allows the nucleus pulposus to leak and breach in between these layers. If this migration continues to progress the nucleus pulposus extrudes through the outer annulus and becomes what is known as a herniation.

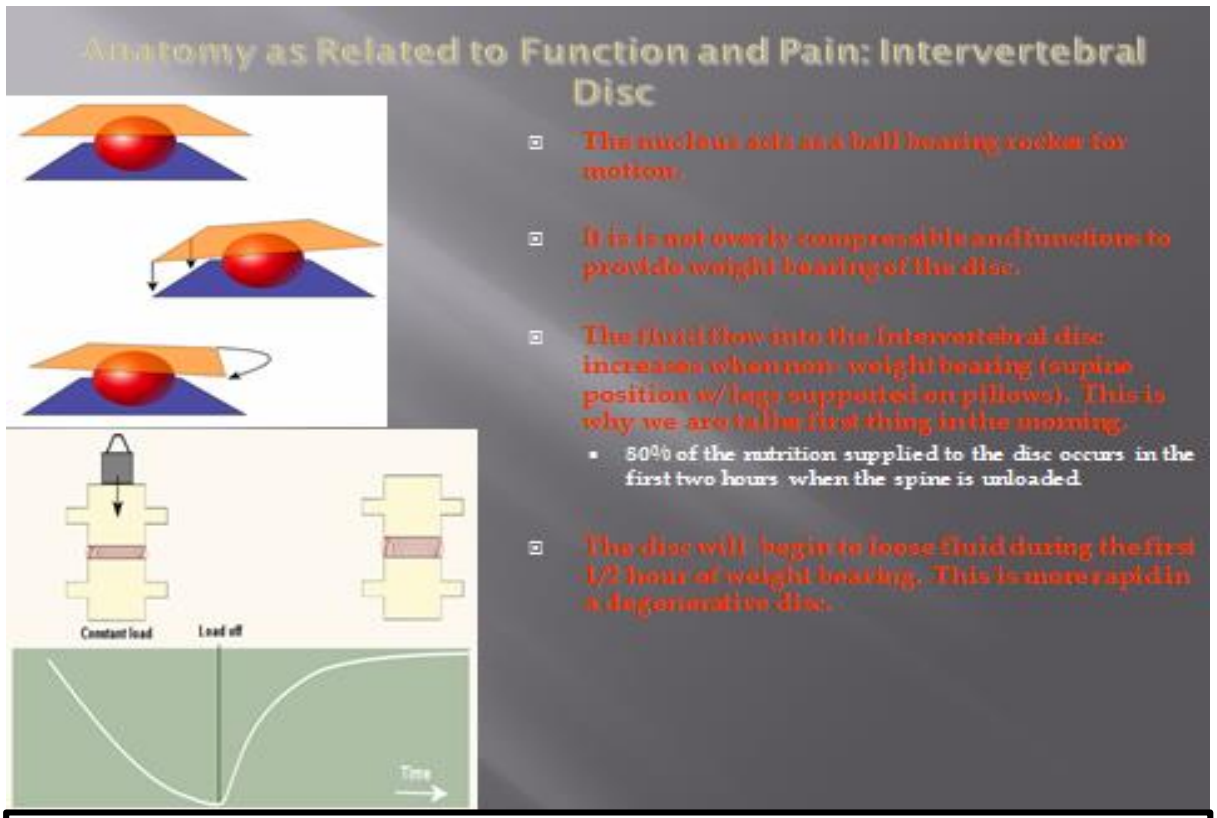

Figure 3: Nucleus pulposus structure and function based on analogy to a ball bearing. Movements between vertebrae occur over an incompressible nucleus pulposus.

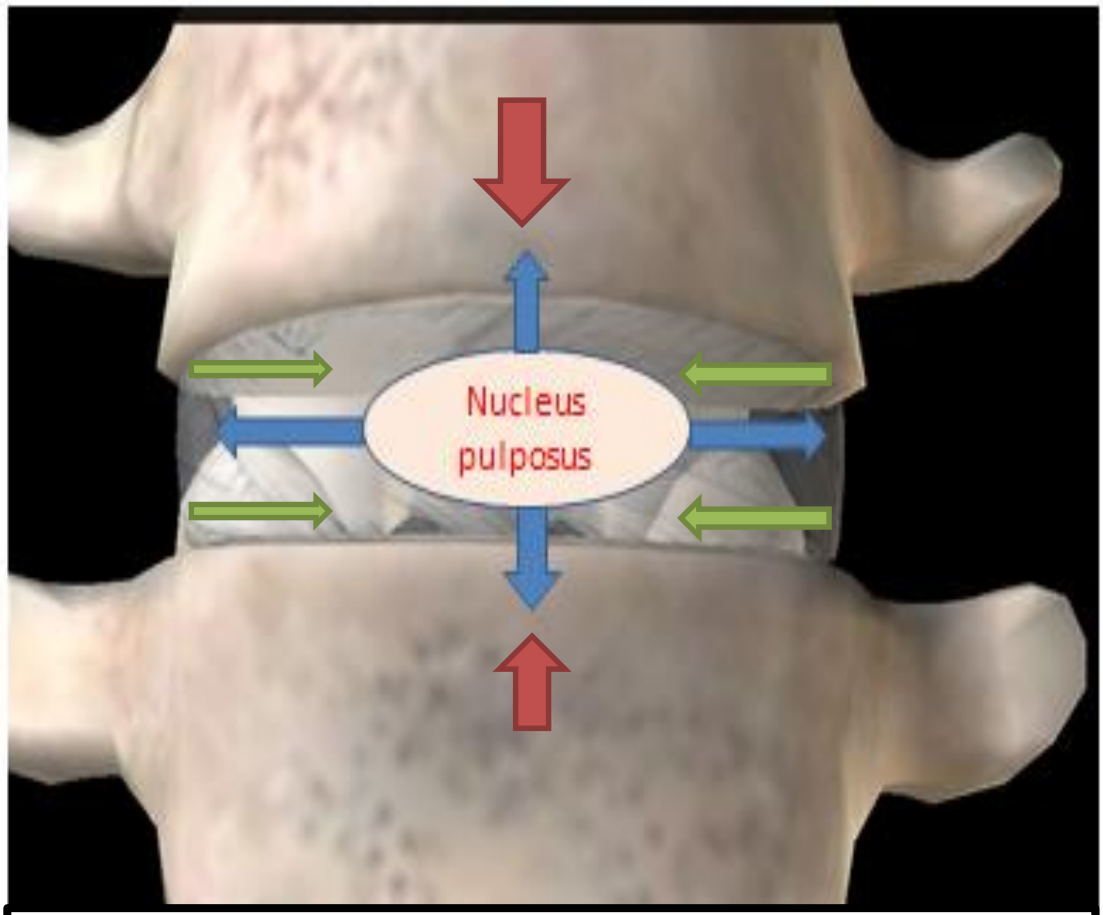

Figure 4: Under compressive load the nucleus pulposus is pressurized and applies hydraulic forces both vertically and outward into the vertebral end plate and inner lamellae (blue arrows). The venous supply housed within the vertebral body provides resistance (red arrow). The outer layers of the annulus fibrosis become taut and provide counter resistance as well (green arrows).

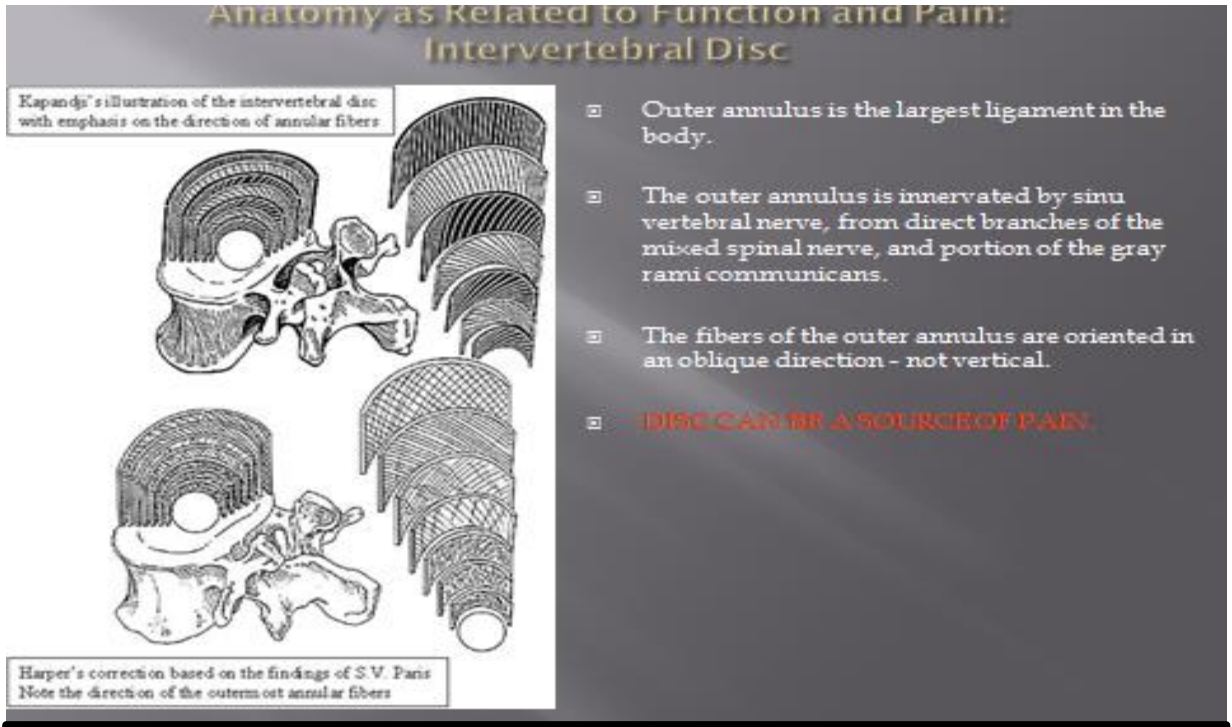

Figure 5: Layers of the annulus. Notice the configuration of the fibers for each layer. This structural architecture provides tensile strength to the disc.

Most disc herniation in the lumbar spine occurs in the posterior lateral portion of the disc. *Figure 6* shows a vertebral segment between L4 and L5. You can see the posterior longitudinal ligament provides reinforcement on posterior-central portion of the IVD. This is vitally important to protect the contents of the spinal canal. Under laboratory conditions, McGill et al[1] found that lumbar disc herniation followed a particular pattern. The cyclic loading of the disc under end range forward bending (flexion) with axial rotation. The nucleus pulposus followed a posterior lateral progression. What is interesting is that this portion of the IVD is less fortified by ligaments, and also receives direct innervation by the sinuvertebral nerve. A breach to this area of the disc will certainly be in close proximity to the nerve root (*Figure 6*).

Clinical Perspective: Partial herniation of nucleus pulposus may remain dormant for some time until later in life when repeated bending becomes sufficient to create migration outward to the posterior longitudinal ligament[1]. Also, the nucleus pulposus is an unexposed structure under normal conditions. During embryologic development it becomes completely encased by the developing vertebra. Once this "virgin" structure breaches the outer annulus, the body can not recognize it, and therefore the immune system attacks it. The result is the development of inflammation and changes in pH balance in the area of the herniation. Under normal conditions a nerve root is not sensitive to mechanical stimuli. In the presence of inflammation, a nerve root becomes very sensitive to mechanical stimuli and becomes a pain generator.

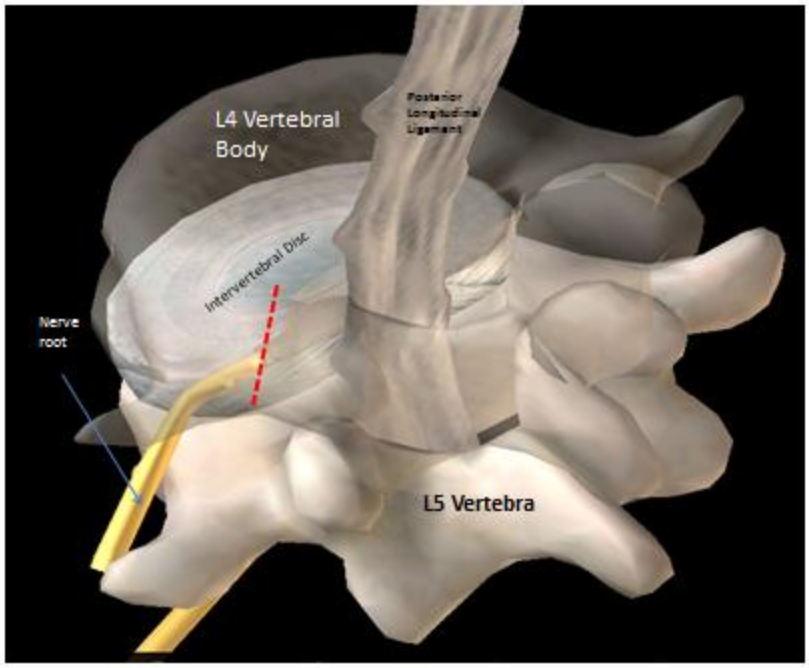

Figure 6: L4-L5 segment with location of posterior longitudinal ligament, IVD, and nerve root. Red dotted line represents posterior lateral track of a disc herniation.

Clinical Perspective: Mechanism of disc herniation from McGill[1]:

- Must be at full end range of forward bending
- Conditions of repeated cyclic loading under fully bent postures
- Full forward bending and rotation under cyclic loading
- Disc must be hydrated in order to herniate (greater prevalence in younger adults)
- Sedentary occupations

Here is some other food for thought! It is a fact of life that as we age so does our spine. No kidding? But a good many of our patients will tell us, "They saw disc bulges on my MRI!" or "My spine is degenerating!" It is akin to getting gray hairs in some respect. Our nucleus pulposis will dehydrate, the annulus will develop some fissures, and the facet joints develop some arthritic changes. However, imaging is not all that accurate. In fact, some sources indicate that less than 10% of LBP can be diagnosed anatomically on an MRI. How about this one…about 40% or more of the general population, without complaints of LBP, will have MRI evidence of disc bulges or herniation. What does it all mean? We have a ways to go in understanding LBP. Imaging is a great asset but it's not without its own limitations. In most cases, we are treating movement dysfunctions not a radiographic image.

References

1. McGill S. Low Back Disorders. Champaign, IL: Human Kinetics; 2007.