The lumbar spine of the human is made up of five lumbar vertebrae that are separated by five intervertebral discs (blue structures in fig. #1). The discs may be thought of as spinal shock absorbers, for they absorb the load of the body. They also allow for movement at the waist as they act as a pivot point and allow the lumbar spine to bend, rotate, and twist.

There are 23 discs in the human spine: 6 in the neck (cervical region), 12 in the middle back (thoracic region), and 5 in the lower back (lumbar region). This page shall focus on the lumbar spine; however, the thoracic and cervical spines are similar in make-up.

The disc is made up of three basic structures: the nucleus pulposus, the annulus fibrosus (aka annulus fibrosus) and the vertebral end-plates. Although their percent composition differs, the latter three structures are made of three basic components: proteoglycan (protein), collagen (cartilage), and water. We will learn all about these structures below.

Figure #1 depicts a Front view (AP) of the lumbar spine. Here we can see how the discs (blue) lie in between every vertebrae. Spinal nerves (yellow) have emerged from between every two vertebrae and travel down the lower limbs to innervate (give life to) the skin and muscle. Note how the sciatica nerve is formed within the pelvis by branches from the last three lumbar spinal
nerves. It is this giant nerve--i.e., the sciatic nerve--that causes so much trouble in many of us chronic pain sufferers. (more detail here)

**Figure #2** Shows a cut-away posterior view (PA view) of the lumbar spine. Now we can better visualize how the sciatic nerve is formed and see just how close the spinal nerve roots come to the back of the intervertebral discs. Note that the L5 root exits through the L5 IVF and the S1 root traverses downward and exits below the L5 disc through the sacral foramen. Any herniation of the posterior disc may compress and/or chemically irritate EITHER the exiting L5 root traversing S1 spinal nerve root and result in severe lower back pain and/or lower limb pain (i.e., sciatica). For more information on sciatica please visit my [Sciatica Page](#).

**Axial Disc Anatomy:**

The human disc has two basic parts: an inner jell o-like center called the Nucleus Pulposus and the Annulus Fibrosis. Let learn more about each:
The Nucleus Pulposus (#1 fig. #9-pink) is the water-rich, gelatinous center of the disc, which is under very high pressure when the human is upright--especially in the seated position. It has two main functions: to bear or carry the downward weight (i.e., axial load) of the human body and to act as a 'pivot point' from which all movement of the lower trunk occurs. Its third function is to act as a ligament and bind the vertebrae together. The Annulus Fibrosus (#2 fig. #9-green) is much more fibrous (tougher) than the nucleus. It also has a much higher collagen content and lower water content when compared to the nucleus. Its main job is to corral or hold in place or contain the highly pressurized nucleus (the nucleus is pressurized for hold up the weight of the body), which is constantly trying to escape its central prison. The annulus is made of 15 to 25 concentric sheets of collagen (a tough cartilage-like substance) that are called Lamellae (#9). The lamellae are arranged in a special configuration that makes them extremely strong and assists in their job of containing that pressurized nucleus pulposus.

Neurology:

I’m going to try to keep this section as simple as possible, but it will end up being quite a complex subject notwithstanding my effort. So hang in there!
The first thing to realize is that the ultimate reason why humans feel pain is because of nerves. Nerves carry pain messages / signals from the periphery (i.e., anything outside of the brain and spinal cord [CNS]) to the sensory cortex of the brain where they get interpreted into the feeling or perception of pain.

There are two types of nerves pertinent to our discussion: motor nerves (aka efferent nerves) and sensory nerves (afferent nerves). **Motor nerves** carry messages away from the brain and spinal cord (i.e., the CNS) outward to the muscles of the body, and **sensory nerves** carry messages (including temperature, touch, **pain**, and pressure messages) from the periphery into the CNS.

These nerves are sensitive to compression and/or chemical irritation, which can occur if they get pinched and/or bathed from an adjacent disc that is ripped open (see the Disc Herniation page). Such a syndrome can and does occur near the back (posterior) part of the disc where the nerve roots are in very close proximity (like the L5 roots in fig. #9). The **traversing nerve roots** (L5 roots in fig. #9 for example) in the lumbar spine are unusual in that they are made up of individually pia-mater-wrapped sensory and motor roots that do not separate until they reach the actual spinal cord, which is located at about the level of the L2 disc (there is some variation of this). Above the level of L2, the motor and sensory roots immediately branch into a ventral and dorsal root and enter the cord. (see figure #26)
The nerve roots exit the spine through bony holes called the **Intervertebral Foramen** (Red Zone in fig. #9). Once within the IVF the two nerve roots merge into one mixed nerve call the **Spinal Nerve**. The Spinal nerves are called "mixed nerves" for they contains both sensory nerve fiber (aka: afferent) and motor nerve fiber (aka: efferent). After leaving the spine through the IVF, the nerve splits into a posterior division (Dorsal Ramus) and an anterior division (Ventral Ramus). The **Dorsal Ramus** connects to the muscle and skin over the lower back, butt and Facet Joint (#5 in fig.9). The **Ventral Ramus** combine in the pelvis and form the giant Sciatic Nerve and Lateral Femoral Cutaneous Nerve that in turn connect to all the skin and muscle of the lower limbs (See my 'Sciatica Page' for more information). As we will learn below, the ventral ramus has a "recurrant branch" that connects to the back of the disc, as well as laterally to the sympathetic nervous system.
(Grey Ramus Communicans); this special nerve is called the Sinuvertebral nerve (SN) (see below).

As noted above, in the lumbar spine there is no spinal cord. Instead the nerve roots hang like a "horses tail" in an enclosed, CSF-filled sac called the Thecal Sac (red stars in Fig.#9). The thecal sac, is made of a mixture of pia-mater-wrapped lumbar, sacral and coccygeal nerve roots [both motor and sensory] and serves to protects these all-important roots. The nerve roots of the thecal sac also float freely in cerebral spinal fluid (CSF) that offers both protection and nutrition for the roots. Its covering is made up of two distinct but tightly bound layers called the dura mater and arachnoid mater. Note how the nerve roots within the thecal sac (fig.# 9 labeled S1, S2, S3, S4), which are collectively called the Cauda Equina (#4), are often in a highly organized configuration. Because of this arrangement, which usually puts the lower level traversing nerve root in front, it is possible for a large disc herniation to irritate/compress more than one root! This may explain why disc herniations do NOT always match their exact dermatomal distribution; i.e., a large disc herniation at L4 may clinically present as nerve root pain (aka: radicular pain, sciatica) and dysfunction of both the L4, L5 and even S1 nerve roots!!! The Epidural Space (#8 in fig. #9) is the space between the bony neural canal and the thecal sac; or the space outside of the thecal sac. In reality, this space is filled with blood vessels and fat and is grossly oversimplified in figure #9. Noteworthy is the fact that this is the region where 'epidural steroid injections' are placed. The Facet Joints (#5 in Fig. #9) (aka: zygapophyseal joints) of the spine are where the vertebrae articulate (join) with each other. Actually, the gap between the inferior and superior articular processes is the true facet joint (white region). Collectively the inferior and superior articular processes and the facet joint are called the Zygapophyseal Joints or articular pillars. These joints help carry the axial load of the body and limit the range of motion of the spine. They also make up the back border of the intervertebral foramen and may physically compress and trap the exiting nerves secondary to degenerative thickening (sclerosis); this condition is called lateral canal stenosis. The Ring Apophysis (#6) is the 'naked bone' of the outer periphery of the vertebral bodies. The very outer fibers of the disc (Sharpey's Fibers) anchor themselves into this region. Bone spurs (aka: Osteophytes) may arise from the ring apophysis as the result of the later stages of Degenerative Disc Disease (DDD) and/or Osteoarthritis (Spondylosis). Specifically, osteophytes arise from the prolonged 'pulling and tugging' of 'Sharpey's Fibers' at their anchor points. The Posterior
**Longitudinal Ligament** (PLL) (#7) is a strong ligamentous tissue which courses down the anterior aspect of the vertebral canal and is attached to the outer fibers of the annulus fibrosus. This highly innervated (supplied with pain carrying nerve fiber) tissue is the last line-of-defense the posterior neural tissue has against the irritating and inflammatory effects of nucleus pulposus.

**MRI AXIAL ANATOMY QUIZ:**

Okay, let's see if you have learned anything: Figure #8 is a real over-head view (aka: Axial View) of an L4 vertebra. Name the numbered structures.

**Here are the answers:**

Remember, this is a T2-Weighted Axial View, which allows you to see the nerve roots (e.g. 3) hanging freely within the cauda equina. On the T1-Weighted or Proton Density view, you can't see these roots.
NERVE ANATOMY IN AND AROUND THE DISC:

In reality things don't look so 'nice and neat' within the human body. The below picture demonstrates what real nerve roots look like:

**Figure. #15** is a back view (posterior to anterior) of a real human cadaver lumbar spine. The back part of the vertebrae (lamina & spinous processes) have been removed in order to see the dural sac (aka: thecal sac); the dural sac has been sliced open in order to see the dangling nerve roots of the cauda equina. Note: the cauda equina is only seen below the level of L2. Above L2, we have the
more familiar spinal cord.

**#1:** This ball-like structure is the ultra-sensitive Dorsal Root Ganglion (DRG) that contains the sensory nerve cell bodies. (the motor nerve cell bodies live out of harms way and are found in the dorsal horn of the spinal cord.) The DRG is found within the protective bony intervertebral foramen (cut away in this photo) and can be pinched/irritated from 'far lateral disc herniations' and/or lateral canal stenosis.

**#2** This is the famous 'spinal nerve root' and is the number one target of disc herniation. A good sized paracentral disc herniation often will compress this adjacent structure and 'might', if coupled with an inflammatory reaction, ignite the nerve root into 'anger' and sciatica.

**#5** As the spinal nerves leave the spine and head-out into the body to do their respective jobs, they temporarily join into a mixed spinal nerve (#5). After this brief marriage, they split and become the smaller **dorsal primary rami** (#7) (which supplies the skin and muscle of the back) and the **ventral primary rami** (#6). The ventral primary rami (aka: anterior primary rami) of the bottom three nerve roots (L4, L5, and S1), merge within the pelvis to form the giant 'sciatic nerve', which not only causes so many of us grief when irritated but, importantly, gives life to the skin and muscle below the knees.

Inside the dural sac, you can see the free hanging **motor nerve root** (#4) and the **sensory nerve root** (#3). These nerve roots connect into the real spinal cord about at the L1 level. Pain signals travel along the sensory nerve root and register 'PAIN' within our brains in the sensory cortex, among other places.

The **Sinuvertebral Nerve: A nerve of mystery**

The **Sinuvertebral Nerves (SN)**, is a mixed nerve as well as it carries both autonomic fiber (sympathetic) and sensory (afferent) fiber. [note: the Autonomic Nervous System (ANS) is beyond the scope of this site.] The sensory portion of the sinuvertebral nerve, which has the capability to carry the feeling of PAIN to the brain, arises from the outer 1/3 of the posterior annulus fibrosus (yellow balls) and PLL (#7). It then splits and attaches to both the dorsal ramus and the grey ramus communicans, although this nerves anatomy and course seems to be quite anomalous. Of importance is the fact that if irritated, the nerve ending within the disc have the potential to generate both back pain and/or lower limb pain (**Discogenic Pain**). This lower limb
pain-referral has been greatly studied by Ohnmeiss et al. and is quite an interesting phenomenon. Discogenic Sciatica is the term I have given this referred discogenic pain. It is believed that the sinuvertebral nerve-endings are 'sensitive' to the irritating effects of degenerated nucleus pulposus, which may be introduced into the outer region from a grade three annular tear. (see may pages on Annular Tears for more information.) Amazingly, the sinuvertebral nerve also innervates (connects to) the disc above and below! So, the sinuvertebral nerve of the L4 disc also innervates the L5 and L3 disc. This may help explain why a L4 disc herniation/annular tear may clinically present with some signs of L5 and/or L3 involvement/overlap as well. It also carries autonomic nerve fiber to the blood vessels (not shown) of the epidural space. Sympathetic nerves control how the blood vessels function (vasomotor & vaso-sensory). Although rare, injury to these sympathetic nerves may cause RSD symptoms (now called CRPS) in the patients lower limbs; this usually would occur following surgery. (This may explain why I had a very slight case of RSD in my left foot following surgery - since my doc spent an hour cutting his way through a 'nest' of epidural vessels during my microdiscectomy.)

The exact pain-pathway (how pain travels from the disc to the spinal cord) of discogenic pain is another fascinating and controversial subject. It seems that the sensory pathway from the sinuvertebral nerves into the spinal cord, does NOT take the 'expected' route in every patient. Some research (101) has demonstrated that pain-signals travel from the disc, re-enter the IVF (via sinuvertebral nerve) and DRG at the SAME level. Other, more recent research has indicated that pain-signals travel from the disc, through the sinuvertebral nerve, through the Gray Rami Communicans, into the Sympathetic Trunk (ST), up the sympathetic chain to the L2 vertebral level (yes I said L2), through the gray rami communicans, into the L2 dorsal rami, into the L2 IVF, and into the L2 DRG (80, 81). The latter pain pathway is why some investigators believe that lower level disc herniations may present as L2 dermatomal pain (groin region) in some patients!

To drive-home my point that the pain path from the disc does NOT always re-enter at the same vertebral level, I present the 2004 randomized controlled investigation by Oh and shim (26). In an incredibly well designed outcome study, the latter investigators demonstrated that by 'cutting' (RF neurotomy) the Gray Ramus Communicans, the majority of chronic discogenic pain sufferers achieved substantial relief of their pain and avoid fusion surgery. (26) This proves that at least some of the incoming pain signals were traveling
toward the sympathetic trunk (which is on the anterior side of the vertebra) and NOT re-entering the spinal nerves at the same level.

A Committee Error?

Another interesting oddity about the design of the nervous system is the fact that 'the committee' decided to put the delicate sensory nerve cell bodies within the IVF and NOT the within spinal cord, which is where the motor nerve cell bodies are located. The Dorsal Root Ganglion (DRG), which houses these sensory nerve cell bodies, is seen as a tiny bulging structure within the IVF. This structure is 'super-sensitive' to compression (because it houses all these sensory nerve cell bodies) and can cause extreme back and leg pain if compressed and irritated by discal material and/or bony outgrowth (stenosis). The placing of these sensory nerve cell bodies in such close proximity to the disc and within such a narrow bony tunnel (the IVF) was not 'the committees brightest idea! You see if you damage the axon (nerve fiber) of a nerve, the chances are quite good for recovery, but if you damage the 'brain of the nerve fiber' (nerve cell body) the nerve's chances of recovery are much less. This explains why patients often never recover completely from that tingling burning, and numbness following a major attack of sciatica (disc herniation-induced radicular pain and dysfunction).

The View from the Side: (the Sagittal view)

Fig. #2 Is a sagittal view (aka: lateral view) of the 'motion segment'; Two vertebrae which are 'sandwiching' the intervertebral disc.

The disc [which is made of a annulus fibrosis (blue) and the nucleus pulposus (green)] is made up of three distinct areas: 1) The nucleus pulposus (green), which is a water rich (due to proteoglycan aggrecan & aggrecate molecules which trap and hold water within the disc) gel in the center of the disc; 2) The annulus fibrosis (blue), which is the fibrous outer portions of the disc that is made up of type I collagen; and 3) The vertebral end-plates (yellow), which are cartilaginous plates that attach the discs to the vertebrae and supply food (nutrients) to the inner 2/3 of the annulus and entire nucleus pulposus.
To further increase the strength of the annulus fibrosus, individual sheets of collagen are layered throughout the annulus. There sheets of collagen are called **lamellae** (black curved lines within blue). The very outer lamellae (Sharpey's fibers), unlike the inner lamellae, are anchored into the solid bony periphery (Ring Apophysis) of each vertebral body. This is the region that 'osteophytes' or bone spurs typically like to form. (Click [here](#) to see a real axial view of a 'motion segment'.)

**Disc Physiology 101:**

The normal human intervertebral disc, which is considered the largest avascular structure in the human body, is made up of two main components, proteoglycan and collagen (type I and type II). The annulus is mostly made of collagen, which is a tough fibrous tissue similar to the cartilage that is found in the knee, and the nucleus is made mostly of proteoglycan. Proteoglycans, which are produced by disc cells that resemble chondrocytes, are extremely important for disc function (see next paragraph) and are what 'trap' and hold water molecules (H2O) within the tissue of the disc. In fact both the disc and annulus are comprised mainly of water, i.e., the nucleus is 80% water, and the annulus is 65% water. Proteoglycans are the building blocks of the aggrecan molecule which is the true 'water trap' of the disc. Aggrecans combine within the disc on strands of hyaluronan acid to form huge structures called 'Aggregates'. These super water-filled proteoglycan aggregates are what give the healthy young disc its amazing strengths and pliability, in fact a well hydrated disc is often even stronger than the bony vertebral body. **Fig. #3:** Here we have the healthy disc of a teenager (cadaver). The water content is extremely high as you can even see by the 'glistening' appearance of the nucleus (which is the gray center of the white disc).

**Disc Function:**
In order for a disc to function properly, it MUST have high water content; this is especially true of the nucleus. A well hydrated (with water) disc is both strong and pliable. The nucleus pulposus needs to be strong and well hydrated to do its job (axial load), for it is this structure that supports or carries the lion’s share of the axial load (downward weight of body) of the body. With an undamaged annulus, strongly corralling a fully hydrated nucleus, the disc can easily support even the heaviest of bodies! As the disc dehydrates (loses water) the disc loses ability to support the axial load of the body (loses hydrostatic pressure); this causes a 'weight bearing shift' from the nucleus, outward, onto the annulus, outer vertebral body, and zygapophyseal joints (facets). Now, we have an 'over-load' on the annulus (which may trigger other destructive biochemical reactions) which, if severe and/or is imposed upon a genetically inferior annulus, will result in pathological DDD. (see below)

Hydration also is important with respect to disc nutrition. As we have already mentioned, nutrients (which all living tissue needs in order to survive) must diffuse (soak) through the discal tissue in order to reach the hungry disc cells. This diffusion process is much faster and easier IF the diffusing tissue has a high water content. We may use 'swimming' as an analogy: It's easier to swim through the water, than through the sands of a desert. The sands of the desert would be a dehydrated disc, and the water would be a hydrated disc. So, water and disc hydration are one of the key factors for a normally functioning spine and well feed disc.

So, we've learned WHY disc hydration is so important. Now it time to learn HOW this disc hydration is accomplished:

Water is held within the disc by tiny sponge-like molecules called proteoglycan aggrecans. These 'super sponges' have an amazing ability to attract and hold water molecules (324), and can in fact hold over 500 times their own weight in water; this gives the non-dehydrated disc the tremendous 'hydrostatic pressure' which is needed to bear the axial load of the body. Amazingly, the aggrecans water absorption is so powerful that over night (non-axial loading) the height of the disc and the body will actually measurably increase due to the discs engorgement with water. This phenomenon is called 'Diurnal Change' and is only present in non-degenerated discs.

Disc cells, particularly the chondrocyte-like cells of the nucleus and inner annulus, manfacture proteoglycan aggrecan molecules. Like little factories, they create, replace and rebuild aggrecan molecules. As long as the disc cells have food (glucose), building material (amino acids) and oxygen all is
well in disc-land. It is also important for them of have a non-acidic working environment, which is taken care of, since wastes are diffused out of the disc the same way nutrients diffuse in. In the living disc up to 100 aggrecans combine on a long piece of 'hyaluronan acid' to form giant proteoglycan aggregate molecules. It's these aggregates that are found within the disc in the real world.

Disc Nutrition:

The intervertebral disc is the largest avascular structure in the human body. The reason for this is because it has no direct blood supply like most other body tissue. Nutrients (food) for the disc are found within tiny capillary beds (black arrows) that are in the subchondral bone, just above the vertebral end-plates. This subchondral vascular network 'feeds' the disc cells of the all important nucleus and inner annulus through the diffusion process. The Figure on the left shows the 'disc feeding setup' for disc. Note that the outer annulus has its own blood supply that is embedded within the very outer annulus. This is a much more efficient system and nutrients don't have to diffuse very far to find their hungry disc cells. The 'more direct' blood supply of the outer annulus is why tears of the outer 1/3 of the annulus will heal/scar shut with the passage of time, which unfortunately is not true of the rest of the disc. Research has indicated that disc tears will not heal in the inner zones of the disc - probably because of the avascular nature of the inner two thirds of the disc. Note the nutrients (pink balls) diffuse directly into the tissue of the outer annulus, where as the nucleus and inner annulus has a much longer diffusion route that is block by the vertebral end-plates. Note how the nutrients (pink balls) are released from the blood vessels (red) in the subchondral bone just under the vertebral end-plates. These nutrients must 'diffuse' or soak their
way through the vertebral end-plates and into the disc. This 'diffusion method' is how the cells of the disc get the nutrients oxygen, glucose, and amino acids which are required for normal disc function and repair. This poor blood/nutrient supply to the disc is one of the main reasons that the disc ages and degenerates so early in life. (Read my Disc Degeneration page for more information.)

The 'diffusion feeding process' is enhanced somewhat by a phenomena called 'Diurnal Change'. Our discs have the ability to expand and compress over the course of a day. As we start the day our discs, like squeezing out a sponge, will compress and dehydrate because of the gravity and physical activity which place axial loads upon the discs. In fact a healthy disc will shrink down some 20% (104), which in turn decreases our height by 15 to 25mm (194, 441, 815). As we sleep and decompress our spines, our discs swell with water plus nutrients and expand back to their fully hydrated state. This tide-like movement of fluids in and out of the disc will help with the movement of nutrients into the avascular center of the disc. (Click here to learn more on Diurnal Change).

Super Advanced Anatomy & Physiology:

The Nucleus Pulposus:

The nucleus pulposus is a hydrated gelatinous structure located in the center of each intervertebral disc that has the consistency of toothpaste. Its main make-up is water (80%). Its solid/dry component make-up are proteoglycan (65%), type II collagen fiber (17%) and a small amount of elastin fibers. Collectively the proteoglycans and the collagen are called the 'nuclear matrix'. The cells of the disc, which produce the water holding proteoglycan molecules are very similar to chondrocytes seen in articular cartilage and are also held within the matrix.

Proteoglycans are found in several structural forms within the disc but the most important 'arrangement' is called a proteoglycan aggrecan. These aggrecans main function is to trap and hold water, which is what gives the nucleus its strength and resiliency. Like a 'super sponge', aggrecans can trap and hold over 500 times their weight in water!

The nucleus has two functions. The first is to bear most of the tremendous axial load coming from the weight of the body above and second to 'stand-up' the lamellae of the annulus - so that the annulus can reach its full weight
baring potential. In order for proper weight bearing the nucleus and the annulus MUST work hand in hand.

**The Annulus Fibrosis:**

The annulus is the outer portion of the disc that surrounds the nucleus. It is made up of 15 to 25 collagen sheets which are called the 'lamellae'. The lamellae are 'glued' together with a proteoglycans. These sheets encircle the disc and, in concert with the nucleus, give the disc tremendous axial load strength.

The posterior portion of the annulus if further strengthened by the 'posterior longitudinal ligament'. This structure is the final barrier between the disc and the delicate spinal cord, and nerve roots.

The biochemical make up is similar to that of the disc only in different proportions. The annulus is 65% water, with the collagen, both type I and II making up 55% of the dry weight, and proteoglycans (mostly the larger aggregate type - 60%) making up 20% of the dry weight. 10% of the annulus also contain 'elastic fiber' that are seen near where the annulus attaches into the vertebral end-plate.

The lamellae are made up of both Type I (very strong type) and Type II collagen fiber. The very outer lamellae are almost all Type I. As we move inward toward the nucleus the more Type II is seen and less Type I. The very inner layers are very hard to distinguish from the nucleus. There is not a clear boundary between the nucleus and the annulus.

A simply amazing fact about the lamellae design is that the collagen fibers that make-up each lamellae all run parallel at a 65 degree angle to the sagittal plane. Even more amazing is the fact that the each lamellae are flipped so that the 65 degree angle alternates between every lamellae, one to the right then one to the left. This design greatly increases the shear strength of the annulus and makes it had for cracks to develop through the layers of the annulus. This is just amazing if you think about it!! Brilliant design!

The function of the annulus is to help the nucleus support the axial weight from the body. The annulus does need some help from the nucleus in order to achieve its strongest configuration. It relies on the nucleus to push it outward which keeps the lamellae from collapsing inward. The nucleus must keep a
very high hydrostatic pressure to achieve this. We saw what happens when the nucleus losses hydrostatic pressure under the 'Disc Degeneration' page. Bogduk used the analogy of a rolled up telephone book standing on end, to describe how strong the annulus could be when the nucleus holds the inner lamellae or phonebook in a rolled up position. If you unroll the phone book our 'on end phone book' it would not longer be able to support much axial loading.

The Vertebral End-Plates:

Both the top and the bottom of each vertebrae (spinal bones) are capped with a thin ¾ millimeter cartilaginous pad called the 'Vertebral End-Plate' (Figure #1). Despite their name, these end-plates are NOT attached to the subchondral bone of the vertebrae but are instead strongly interwoven into the annulus of the disc (156, 388). It is for this reason, as well as strong morphological similarities, that the vertebral end-plates are considered part of the disc and NOT part of the vertebral body.

The biochemical morphology of the end-plates is extremely similar to that of the disc: Water, proteoglycans, collagen and cartilage cells (chondrocytes). The concentration scheme of these components also mirrors that of the disc: The center of the end-plate is mostly water and proteoglycan. As we move outward toward the periphery, more and more collagen is seen with less and less proteoglycans. This similar biochemical makeup and distribution scheme helps the diffusion of nutrients between the subchondral bone of the vertebra and the depths of the disc.

The very outer rim of the vertebrae is NOT covered by the end-plate, which leaves a ring of exposed bone on the periphery of the top and bottom of each vertebra. This exposed peripheral area is called the 'Ring Apophysis' and is often a site for the development of spur formation associated with the degeneration process.

References:


**General Information on DDD and Disc Aging:**

Unlike other tissues of the human body, the poorly vascularized intervertebral disc tends to undergo degeneration of its internal structure at a surprisingly early age. In fact, award-winning research has demonstrated such degeneration usually begins within the first decade of life! (6,15) Thankfully, most of the time this degeneration is harmless and considered just part of the natural aging process. However, in some folks, the degeneration process runs amuck and spells the proverbial "beginning of the end" for the disc as it leads to destruction of the disc and
chronic pain. Why some people’s discs succumb to such severe disc failure (above right) and pain and others don't continues to be a mystery, although heredity certainly plays a significant role in this process. It is this abnormal accelerated form of pathological disc aging and degeneration that I called **Degenerative Disc Disease** (DDD). The figures above demonstrate the difference between normal (left) and severely degenerated (right) lumbar discs. Note the moderate to severe loss of disc height (right); a conditions that is called discopathy or (when in combination with arthritic change) discogenic spondylosis. The loss of disc height often causes the secondary problem of stenosis: the closing of the holes where the spinal nerves, nerve roots and spinal cord reside.

One physical cause of DDD, and natural disc aging for that matter, is **poor disc nutrition**. As you may remember from the disc anatomy page, the majority of disc tissue has no blood supply (it's avascular). The disc gets its food (nutrients) via diffusion from blood within the upper and lower vertebral endplates. Normally, the nutrients pass from the endplates through tiny pores the into the hungry discs. Arthritis of the endplate (sclerosis) has the negative effect of decreasing the diameter of these pores, which in turn decreases the rate of food-flow to the disc, which in turn causes the disc to die, i.e., DDD. In fact, recent award winning scientific investigations (700, 5) have confirmed the foregoing explanation of disc degeneration.

Therefore, any attempts at injecting new disc material into a degenerated disc (such as live disc cells)--in hope of healing the damaged disc--will only lead to failure, for the new disc material will soon meet the same fate of the original disc tissue: it will starve to death.

Research has also demonstrated that DDD is associated with pain-producing conditions of the spine such as annular tears, disc protrusions and spinal stenosis (201,206,219,227). In fact, in 10% of the population, DDD will result in permanent chronic pain and life-long disability (250-253). Technically it's not the actual process of DDD that results in chronic pain, it's the evil "end-phases" of the disease that cripples these unfortunate few. These end-phases, as mentioned above, include annular tears (aka: Internal Disc Disruption or IDD) (203,209,216,231); disc protrusions (227); nerve in-growth (900,904,905,906); and stenosis.

**The MRI Appearance:**
The diagnosis of DDD is best made on T2-weighted MRI imaging (Fig. #1) (27), although some of the late appearances of DDD (disc collapse, osteophytosis, and sclerosis) may also be seen on CT scan and X-ray. Such MRI appearances are easy to spot and are characterized by a loss of signal intensity (loss of whiteness) of discal tissue, which makes the disc appear black instead of bright white. Technically, this blackening of the disc(s) occurs because the disc has dehydrated (lost water content) and is dying. This 'blackening' is called disc Desiccation. Since the MRI signal intensity (whiteness) is directly related the disc's water content (215,226), any loss of discal water will proportionally decrease the 'whiteness' of that disc on T2-weighted MRI. So, in layman's terms, the dryer the disc, the blacker and more degenerated it will look on MRI. Figure #1: Here is the classic presentation of DDD as seen in this T2-weighted sagittal (lateral) image. Note the bright white and healthy L3 disc (above the L4 vertebra) in comparison to the 'black' and desiccated L4 and L5 disc. Also note a 4mm herniation at the L4 disc (between L4 and L5 vertebrae) and a 9mm herniation at the L5 disc. Also note there is a loss of disc height at both L4 and L5, in comparison to the thicker L3 disc.

Why some discs prematurely degenerate (DDD) and cause chronic pain and others don't is still somewhat controversial, however, it is becoming clearer that poor genetics (397-399,403,413a); a past history of moderate to severe spinal trauma; or have an occupation that is heavy, and labor-intensive are the main risk factors (201,16). These factors will be discussed in depth below.

Warning:

In order to really understand DDD and disc aging, you must understand a few basic principles of disc physiology. I'm going to assume that you understand normal disc physiology and anatomy. If you don't please go (here) and learn your basic structures, and more importantly, learn the basics physiology of the disc. You will need to know why water (hydrostatic pressure) is so very important for normal disc function (allows the nucleus to support the axial load of the body), and how the cells of the disc maintain discal water content (via proteoglycan aggrecan production).
In order to understand DDD, we must first understand the natural disc aging process, or the 'normal pathway' of degeneration, which occurs in all humans to varying degrees and does NOT lead to pain.

**NATURAL DISC AGING: (aka: NDA)**

The most common and striking feature of disc aging and degeneration is the loss of the proteoglycan molecule from the nucleus of the disc (333, 26). Other findings of aging include a progressive dehydration (18), a progressive thickening (via cross-linking, glycation and CML formation), brown pigmentation formation (66) and increased 'brittleness' of the tissues of the disc (62).

**The Two Main Factors of Disc Aging:**

There are two main factors that are involved in the aging process of the disc and both of these factors are amplified because of the already poor vascular supply of the disc:

1) **Idiopathic blood vessel/nutrient loss and dehydration:**

**The short version:** For unknown reasons the nucleus of the disc losses much of its vital blood supply during the first decade of life (6). Without sufficient nutrients (which are contained in the blood) the cells of the disc begin to die (500) and the disc (especially the nucleus) becomes depleted of water. The drop in water/proteoglycan content is one or the classic signs of disc aging (333). Because of this dehydration of the nucleus, there is ultimately a 'weight-bearing shift' that occurs from the nucleus onto the outer annulus, ring apophysis, and the zygapophyseal joints. This increase stress upon the preceding posterior structures may lead to further more severe forms of aging, i.e., DDD.

**The long version:** Under the physiology section of the 'Disc Anatomy' page, we have learned how important disc nutrition is in maintaining a normally functioning disc. To recap: as long as the cells of the disc receive an adequate nutrient supply (which is obtain from the diffusion of oxygen, glucose, and amino acids [pink balls] from capillary beds just above the end-plates, into the disc), they will happily manufacture the proteoglycan molecule, which combines within the disc to form the larger aggrecan and aggregate molecules. It is these aggrecan molecules that trap and hold water within the disc. A fully hydrated disc will have a very high hydrostatic pressure (osmotic pressure) which makes the nucleus pulposus (which is 80% water in a normal disc) incredibly strong and able support the lion’s share of the axial load from the body.
Remember, the nutrients in the inner annulus and nucleus have a 'crummy feeding system' to being with. As you can see on the model on the left, the nutrients (pink balls) have a long way to 'diffuse' in order to reach all the disc cells.

Without an adequate supply of nutrients, the cells of the disc will die. The preceding fact was substantiated by the 2001 Volvo Award winning study of Horner and Urban (500), who studied the viability of living human disc cells under different conditions. They concluded that if the cells of the disc failed to get proper nutrients - such as oxygen, or glucose - or if the pH level of the disc rose (because waste is not being diffused out of the disc), disc cells would die and stop producing the vital proteoglycan molecule; without proteoglycans, the disc loses its water content (dehydrates) and losses its hydrostatic pressure (osmotic pressure) (241). This lost proteoglycan content is the most striking feature of disc aging and degeneration (333). Other research has confirmed this cell death as well. In 1982, Trout and Buckwalter discovered that by adulthood over 50% of the cells of the disc were dead (321).

So what's killing the disc cells and resulting in this loss of proteoglycan content?

Starvation! It seems that the human disc becomes 'nutritionally compromised' from the moment we begin to stand and walk. In 2002, Boos et al. observed an idiopathic "obliteration" of portions of the nutrient-providing capillary beds, which lie just above the vertebral end-plates. (Remember that these capillary beds are the ONLY source of nutrients for the cells of the inner annulus and nucleus.) Amazingly, this 'auto-destruction' begins within the first two years of life, and worsens over the next 8 years. Specifically, they stated that between the ages of 3 and 10 there was "a dramatic decrease of physiologic vessels in the end-plate..and an abundance of areas with obliterated vessels. and a substantial increase in (disc) cell death." (6) THESE FINDINGS WERE THE 'SMOKING GUN' that scientists had been waiting for and suggested that the initial causation of disc aging and degeneration was 'nutritional compromise', secondary to an idiopathic loss of the discal blood supply above the vertebral end-plates. Needless to say, Boos and company won the '2002 Volvo Award in Basic Science' for this most shocking discovery.
Other factors affecting disc nutrition via diffusion rates of nutrients through the vertebral end-plates include end-plate calcification (506, 537, 538, 552,), the effects of changes in blood flow patterns secondary to arterial stenosis (522, 524-527), smoking, diabetes, and exposure to vibration (500, 517).

The Vicious Cycle of Disc Aging:

This progressive loss of proteoglycan and dehydration begins to 'snowball' out of control. Not only because of the progressive loss of nutrients, but also because of the fact that decreased hydrostatic pressure also slows the production of proteoglycan by the disc cell (11). Here's what this vicious cycle looks like:

As the nutrient supply within the disc drops (because of blood vessel obliteration and later end-plate mineralization), the disc cells start to die. Because there are fewer available disc cells around to make proteoglycan, there is a drop in the amount of circulating proteoglycan aggrecan molecules. This decrease in the aggrecan molecule, (which is what holds water within the disc) results in both dehydration, and a decrease in hydrostatic pressure within the nucleus. The loss of hydrostatic pressure has two negative effects on the disc: a) it will cause a further decrease in the amount of circulating proteoglycan aggrecan molecules, for we know from the work of Handa et al. that disc cells need a constant hydrostatic pressure level of 3 atm to function normally (11). Any increase or decrease in hydrostatic pressure caused a reduction proteoglycan production, which in turn decreases hydrostatic pressure even more - hence the vicious cycle. b) Now, these biochemical changes begin to change the biomechanics of the disc: With the decrease of hydrostatic pressure the nucleus, like a deflating beach-ball, can no longer carry the full axial-load (weight) of the body. A 'shift' in the axial-load distribution begins to occur, with the periphery of the disc (outer anulus, ring apophysis, and zygapophyseal joints) taking on more and more of the load and stress. Experimentally, the anulus of a degenerated disc shows a very high 'stress-load' on the anulus and NOT the nucleus (17, 12). We will later learn that this 'load-shift' can be greatly accelerated if the volume of the nucleus is increased by trauma-induced structural damage to either the end-plate (compression fracture) and/or tearing of the inner anulus.

2.) Non-Enzymatic Glycation & the aging process: Glycosylation (aka: Glycation)

Glycation (aka: Glycosylation , or non-enzymatic glycation) is a biochemical reaction which occurs when reduced sugars (like glucose) come in contact with proteins (like disc collagen) in an avascular environment. The more avascular the tissue, the more severe this reaction occurs. Since the disc is the largest avascular tissue in the body, the glycation process thrives within its substance and results in a slow but steady transformation of disc collagen into a thicker and more brittle substance. Specifically, this reaction occurs between the protein molecules within the collagen, and free floating glucose (reduced sugar). This reaction is called 'posttranslational protein modification' or simple Glycation. Here's how it works: In the absents of oxygen, reduced sugars start to 'rub against' (bind) the proteins within the collagen. The proteins can only take
so much 'rubbing', and soon are transformed into what is called an 'Advanced Glycation End-Product' or AGE. These converted discal collagen strands (AGEs) become much more brittle and also much more 'sticky', i.e., they love to combine with their glycated neighbors in a process called 'cross-linking'. This 'cross-linking' phenomenon makes the disc thicker, more fibrous and more susceptible to the development of DDD (62). It also stains the discal tissue a distinct shade of brown (66) as noted in figure #2.

Fig. #2: Here we have early disc aging. The tissue of the disc has turned brown, secondary to the glycation process, and the disc has become much 'drier and still that the disc seen in adolescents. For comparison sake,

Fig. #3 shows us what the disc of a teenage looks like. Note the well demarcated, wet looking, nucleus (gray center) and NO ugly brown tissue. Ah to be young again!

Finally, the unstable AGEs molecules, which produce another evil biochemical called the 'free radical', oxidize into a much more stable structure called a CML (N-Carboxymethyl -lysine). CML formation has been found to be an excellent indication of discal aging (15). In fact Andreas and Boos won the 1997 Volvo Award for their work in using the presents of CML-modified discal protein as an indicator for the various stages of aging (15). I'm not going to review this study for it's out of our scope, but for those of you who need-to-know, his paper is an excellent read.

Well, that about does it for the natural aging process of the disc. Let's now take a look at the more serious form of aging, DDD.
CURRENT RESEARCH:


**Internal Disc Disruption (AKA - IDD): A General Overview**

Just because that MRI of yours was deemed "normal" by your doctor does NOT always mean that your back and leg pains aren't coming from problems within that very disc. This is especially true if your supposedly normal MRI demonstrates a bulging and/or 'Black' appearing disc on T2 Weighted MRI images (132).

In his 1986 presidential address, Dr. H. V. Crock told members of the international spine society that 'internal disruptions' within the architecture of the disc could result in back pain and even lower limb pain without the presence of spinal nerve root compression (9). He termed this condition "Internal Disc Disruption," herein IDD. IDD occurs when the disc develops a rip or tear (or in medical language, a full thickness radial annular tear) that bisects the disc from inside to outside and allows communication between the jello-like center [nucleus pulposus] and the nerve-infested periphery of the disc [annulus fibrosis].

Fig.#5 demonstrates such an annular tear within a real human cadaver disc. The white arrows demonstrate a full thickness radial anular tear within the L4/5 disc that completely bisects it. Note also that the disc above (the L3/4 disc) has a small tear in the outer fibers of the annulus (black arrow) that has not made it (yet) into the middle of the disc. This type of tear is called a 'rim lesion'.
To understand how IDD may cause 'pain', you will need to know some basic disc anatomy. I've covered anatomy ad nauseam 'here', but if you're too lazy to go there, I'll give you a quick refresher here:

**Fig.#4** demonstrates the normal lumbar disc anatomy: Here, in this over-head view, we have the nucleus pulposus (pink) surrounded by the stronger anulus fibrosus (green). Normally, the anulus fibrosus is strong enough to corral the pressurized nucleus pulposus and keep it from escaping outward. Of particular interest to upcoming discussion is the tiny 'Sinuvertebral Nerves Endings' (yellow poke-a-dots) that are embedded within the substance of the outer 1/3 of the anulus fibrosus. We know through scientific investigation that these nerve fibers have the ability to both "initiate" and "carry" pain messages into the spinal cord and up to the brain if they become irritated (386-388, 439).

ADVANCED: The pain pathways (how the pain gets from the disc to the brain) for discogenic pain are still very controversial and may not function as traditional anatomy has taught us. Traditionally, pain signals that originate in the nerve roots adjacent to the disc or in the disc move from that root, into the corresponding DRG and into the spinal cord. However, some new research suggests that pain signals from the lower lumbar discs (L5 and L4) are (at least in part) detoured up the sympathetic nerves (i.e., gray ramus communicans) and into the upper lumbar DRGs - especially at the L2 level. (11, 259, 260) Clinically, in some patients it then would be possible for patients with L4 and L5 disc problems to have L1 or L2 dermatomal pain (groin and anterior thigh pain).

**IDD: In a Nut Shell:**
When a Radial Annular Tear enters the outer 1/3 of the anulus, (Fig.#3) and exposes the sinuvertebral nerve-endings to degenerated nuclear material (cytokines), pain may well occur secondary to chemical irritation of these pain-carrying fibers. This type of pain is called 'Discogenic Pain,' which means that the pain arises from within the disc and not the adjacent neural tissue. In Fig. #3 the disc has ripped through or "disrupted" and has allowed nuclear material (pink) to escape into the outer and sensitive 1/3 of disc. The sinuvertebral nerves (yellow dots) in contact with this degenerated nuclear material have become inflamed (red dots) and irritated, which in turn causes pain signals to 'firing' off pain signals to the dorsal horn of the cord and then to the brain. Some patients even suffer a referred type pain (discogenic sciatica) down the lower limb(s) from this condition, yet they have no traditional compression of the adjacent nerve roots. [jump to tutorial]

**HISTORY OF INTERNAL DISC DISRUPTION: (IDD)**

IDD was first described by Crock in 1970 (8) and again in 1986 (9). It was then described as a 'disruption' of the internal architecture of the disc without signs of disc protrusions or without positive signs for nerve root compression'.

In his 1986 presidential address, Dr. H. V. Crock told members of the international spine society that internal disruptions within the architecture of the disc could result in back pain and even lower limb pain without the presence of spinal nerve root compression (9). He termed this entity 'Internal Disc Disruption' or IDD.

In 1995, a 'Dream Team' of well respected and Volvo Winning researchers (Schwarzer, Aprill, Derby, Bogduk) set out to test and further develop Crock's theory of IDD and convincingly calculated the prevalence (frequency) of IDD in patients with chronic low back pain. (2) The study also attempted to determine if traditional examination findings and/or specific patient symptoms could be predictive of the diagnosis of IDD. By following the strict criteria specified by the 'International Society for
the Study of Pain in its taxonomy' (21), these investigators calculated the **prevalence of IDD to be between 30% and 50% with a 95% confidence limit**. They also concluded that neither traditional examination findings nor patient symptoms could predict whether or not a patient had IDD. Unfortunately, it looks like provocation discography remains the only way to confirm the diagnosis of IDD.

The theory of IDD as a source of chronic back pain is not without its critics. In 2003, Lee et al. reviewed the research on IDD from 1985 through 2000, (10) although the papers review were mostly on radial tears, and HIZ. He summarized that of the 13 research papers on IDD and similar topics, there was not much agreement on what made the diagnosis of IDD. There was, however, some general agreement between the groups on what constituted the diagnosis of IDD: lower back pain, reproduced on provocative discography (concordant pain was a strong indicator), and a normal neurological examination, i.e., no loss of reflexes, no loss of muscle strength or atrophy, and no sensory loss. That's it, only two factors! Other criteria for the diagnosis of IDD were not so universally agreed upon were: the presents of an HIZ (high intensity zone) within the posterior outermost region of the disc on the T2-weighted MRI, disc degeneration, and a history of trauma.

Based on their review of '15 years worth of research', Lee et al. boldly concluded that "**IDD is not real, but a hypothetical disease**". This Korean group further stated the following; "**Our personal view is that IDD is a doctor-made disease, that is, an iatrogenic disc disorder, which may lead to an unconventional invasive operations (referring to the IDET procedure and Lumbar Fusion).**" (10) Lee felt that because the diagnosis was so dependent upon the 'subjective input' from the patient, during discography, that the diagnosis should be thrown out!

**IMHO:** Lee, who is **way out of his usual area of research** on this subject, is going to get 'blasted' for making such aggressive statements against the theory of IDD, which has been accepted by the **'North American Spine Society'** (21) and **'International Society for the Study of Pain in its Taxonomy'** (21)! For the typical non-mentally compromised chronic pain patient, the diagnosis of IDD can be made with a reasonably degree of medical certainty by using the criteria that the International Society for the Study of Pain in its Taxonomy have adopted (Here) for the criteria.

**THE RESEARCH:**

Although controversial (436), discogenic pain secondary to IDD is thought to be responsible for a substantial number of chronic back and leg pain cases where obvious nerve root compression is absent/lacking (132,2). In fact the famous multi-time Volvo Award Winning author, professor Nikolai Bogduk MD, believes IDD is the "**most common cause of chronic low back pain**" (1,2) and may be often over-looked by the treating physician." (132).
Quality scientific research has demonstrated that 40% of all chronic back pain is caused by the radial annular tears of IDD (2), and often presents (62% of the time according to Ohnmeiss et al.) as back pain and/or pain down the lower extremity, i.e., sciatica (6).

The exact mechanisms of discogenic pain are still controversial; however, the development of a full thickness Radial Annular Tear that leaks nuclear material (cytokines) into the outer annulus is most certainly involved in this syndrome. This annular disc leak theory has been confirmed scientifically via numerous quality peer-review investigations (105,115,116,123,124,131).

Recently, it has been demonstrated that IDD was the causative factor in about three-quarters of severely acute nonspecific low back pain patients. More explicitly, in 2005 Hyodo et al (16) performed MRIs and discography on 55 patients who all suffered severe, immobilizing, non-specific low back pain without sign of neurological deficit. In 73% of these patients, a full thickness non-epidurally leaking annular tear was identified on discography that responded to fluoroscopic lidocaine irrigation (a powerful anesthetic) by instantly 'stopping' the patient's perception of severe pain. (16) The aforementioned experiment strongly advocates that full thickness annular tears – or IDD – are a major cause of severe acute non-specific low back pain.

THE DIAGNOSIS: Discography & Gadolinium-DTPA Enhanced MRI

The 'International Society for the Study of Pain in its taxonomy' (21) has adapted the following set of criteria for diagnosing IDD: ► no visible disc herniations may be seen on MRI or CT; ► during provocation discography injection of the suspect disc with contrast, a recreation of patients 'exact' back and/or leg pain must occur (353,9); ► injection the disc above or below the suspect disc must be non-painful; this acts acts as a 'control disc' or normal disc; and ► a grade 3 or 4 radial anular fissure must be demonstrated on CT discography (2,351,352,355).

Provocation discography, which may actually further damage the disc, should ONLY be attempted if the chronic pain patient can no longer live with their pain syndrome and is contemplating IDET, SED, DiscTRODE, interbody spinal fusion or ADR.
Furthermore, their Oswestry better be at least a 50! (Oswestry)

The 'Gold Standard' in making the diagnosis of IDD is a very painful and invasive test called 'Provocation Discography' with follow-up CT discogram. There are two components to provocation discography: the first is an attempt by the doctor to 'provoke' or 'cause' the patient to feel their 'usual' pain (concordant pain) by pressurizing the disc with a contrast material. Note: in Fig.#6 the center of the disc is being filled with contrast material (white). If you look closely, you can see the fine 'white' needle (black arrows) entering the posterior of the disc. This (fig. #6) represents a normal disc that 'holds' the dye within the nucleus and does not demonstrate any anular tearing.

**Fig.#7,** on the other hand, demonstrates two completely disrupted discs: The contrast material (black in this photo) has NOT been contained within the center (nucleus) of the disc. This time, it has clearly leaked through internal 'disruptions' within the posterior anului of the L4/5 and L5/S1 disc. In fact, the L4/5 disc has been completely disrupted and it leaking contrast material directly into the epidural space (black arrow). The latter situation is called a Grade 5 anular tear or Grade 5 IDD. (learn about the dallas discogram naming system and the different degrees of disc disruption [here](#) about half way down the page.) This situation may indicate big trouble, especially if you are one of unfortunates who are 'sensitive' (allergic) to those leaking biochemicals (cytokines), for the delicate posterior neural structures 'dwell' adjacent to the posterior of the disc and may become inflamed and/or damaged from this leakage. More explicitly, substance like TNF-alpha, IL-1, IL-6, NO, Phospholipase A1 may stimulate some form of 'attack' within the nerve root and ultimately lead to permanent nerve (axon) death. Neuropathic pain may be spawned.
MRI Identification: Gadolinium-DTPA Enhanced MRI

Although provocation discography with CT discography is the "gold standard" when it comes to making the diagnosis of symptomatic IDD, the procedure itself can inflict damage upon the disc and "spawn" degenerative disc disease (30-34,530). As an alternative, the use of gadolinium (contrast) enhancement may be considered. Gadolinium-DTPA, which is injected into the blood stream during the MRI, will "light-up" the granulation tissue that forms within a healing/healed full thickness annular disc tear.

![Fig. #9: The MR images to the left demonstrate how gadolinium will "light-up" a healed annular tear. Note the L4 disc shows no sign of posterior disc tearing (black arrow); however, after the administration of gadolinium during the MRI, the same T1 image demonstrates the remains of the massive annular tear (red arrow) I suffered back in 2002.](image)

The gadolinium also high-lights continued swelling/granulation tissue within my L5 disc over 1 year post micro-discectomy. No disc herniations are noted.

The HIZ phenomenon also give us a clue that Internal Disc Disruption might be involved in the patients pain syndrome although this T2-
weighted MRI finding is highly controversial. For more information, visit my [HIZ page](#).

**The IDD Tutorial:**

To begin this tutorial, let's look at what a normal disc looks like from the over-head view (axial):

In Figure #1, the basic anatomy of the disc is shown: First note the gelatinous and hydrated *nucleus pulposus* (#1 pink) that is corralled (held in place) by a tough and fibrous *anulus fibrosus* (#2 green). To give the anulus fibrosis, which is like the tread of a tire, extra support posteriorly, the posterior longitudinal ligament or *PLL* (#7 blue) exists and is tightly bound to the outer fibers of the anulus. Also note the posterior neural structures: #10 (motor & sensory nerve roots), #3 (mixed spinal nerve roots), and red star (free-hanging nerve roots within the cauda equina). It is these delicate neural structures that often become damaged and perpetually generate pain. To learn more about spinal anatomy, go 'here'.

It is extremely important to understand that, unlike the rest of the avascular disc, the outer 1/3 of the anulus fibrosus, the cauda equina (red star) and the PLL (blue #7) are innervated with (full of) tiny nociceptive C-fibers (pain carrying nerve fiber) that, if irritated, have the potential to cause severe PAIN and DISABILITY within the patient (4,55,56).
THE BIRTH OF INTERNAL DISC DISRUPTION:

The first step in the IDD process is for the disc to first degenerate (lose water content and become brittle) and then (usually because of trauma to the back or neck) tear open from the inside out, or, sometimes, from the outside in (30-34,530). Ironically, however, it seems that IDD can both 'cause' disc degeneration or result from its presents. (5) The disc in figure #2 shows what is commonly called Degenerative Disc Disease (DDD). DDD, which can only be seen on T2-weighted MRI that will affect the disc by causing a loss of water content, which in turn causes the disc to become brittle and prone to tearing. In Figure #2, which represents a Grade IV Radial Anular Tear, our disc has obviously changed in appearance when compared to Figure #1 and now demonstrates disc desiccation (dark green appearance), bulging (note how the posterior of the disc is no longer concave and has bulged into the nerve roots), and a full-thickness radial anular tear (red arrow) that has allowed nuclear material (pink) to come in contact with the ultra-sensitive sinuvertebral nerve-endings (yellow poke-a-dots). For some of us, this situation is truly disastrous! Full thickness radial anular tears, however, (red arrow) are not the only anular sign of the degeneration process: concentric anular tears (white arrows) and rim lesions - which also may result in severe back pain - are also often
present in the pathologically degenerated discs and may also eventually spawn the deadly, disc-extrusion-producing, full-thickness radial tear (30-34). Note in Figure #2, the sinuvertebral nerve-endings adjacent to the anular tear have become inflamed (red), pissed-off, and are sending pain signals up to the brain through both the sympathetics (gray ramus) and the same-level afferent nerve roots.

In figure #7, the situation has worsened into the grade V radial anular tear (ship's anchor). This massive disruption, which may or may not be 'leaking' nuclear material upon the adjacent nerve roots, is irritating even more sinuvertebral nerve-endings and is probably resulting in much more patient pain and suffering and even may cause referred pain down the back of the leg (fake sciatica, or discogenic sciatica) that mimics sciatica (6,7).

The pain mechanism of IDD not only comes from irritation of the now-exposed sinuvertebral nerve endings: a the second mechanism of pain may occur from mechanically pressure upon these nerve endings. To make a long and complex explanation short: because of the massive anular disruption (red arrow), the inside of the disc (nucleus pulposus - pink) can no longer support the weight of the body and 'shifts' this axial load outward onto the already irritated and pissed-off posterior anulus. This added mechanical pressure, like squeezing a cut finger, further irritates the sinuvertebral nerve roots and create
even more back and possible leg pain. Remember, this condition, which may or may not show up on MRI, will affect about 40% of all chronic back pain sufferers and often requires surgical decompression via fusion.

As if things aren't bad enough, let's meet the dreaded **Grade V Full Thickness Anular Tear:**

**Figure #8**, depicts such a condition: Not only is the disrupted disc generating back (discogenic pain) and possibly leg pain (discogenic sciatica), but now we have the potential for the posterior neural structures (traversing nerve roots, exiting nerve roots, and dura of the thecal sac) to become irritated, inflamed, and even killed! This condition, which I'm probably the proud owner of, may baffle even the most astute doctor, by causing a full blown, EMG-confirmed, radiculopathy WITHOUT the presence of any sign of the classic nerve root compression! Here's how it may work: The leakage of degenerated nuclear material from crack(s) that have sprung in the final layers of the anulus fibrosis of the disc will **soak** the delicate nerve roots (which make up the sciatic nerve) with degenerated nuclear material that is filled with all sorts of potentially inflammatory and irritating biochemicals (TNF-alpha, NO, PLA-2, Metalloproteinases, IL-6, PGE-2, Substance-P, Calcitonin gene-related peptide). Although the exact mechanism is of this **Chemical**
Radiculopathy is unknown, it undoubtedly involves the formation of nerve root inflammation, intraneural edema formation, and ultimately intraneural fibrosis (287). OR, in layman's terms: stuff leaks out from the back of the disc and damages (often permanently) the nerve roots that make up the giant sciatic nerve that courses from the low back down the back/side of the lower limb. We experience this nerve damage as PAIN in the back and down the leg - sciatica.

TREATMENT OPTIONS:

Warning: These recommendations are for Educational Purposes ONLY and should never be substituted nor take the place of an examination or treatment plan by your own medical doctor! Do NOT implement any of these courses of treatment without the approval of your medical doctor.

IDD is a very, very tough condition to treat, especially since the diagnosis is fairly controversial to begin with and many primary doctors have never even heard of it. Conservative care is ALWAYS the first form of treatment! If this fails then provocation discography is indicated before proceeding to the more aggressive treatment options but your Oswestry should be in the 50s. Here are the current (7-28-04) treatment options available of IDD:

<table>
<thead>
<tr>
<th>TREATMENT OPTIONS FOR IDD (in order)</th>
</tr>
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<tbody>
<tr>
<td>Conservative Care, Mediation &amp; Mother Care:</td>
</tr>
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</table>

Around 90% of all IDD sufferers will obtain satisfactory relief from their pains by just hanging in there and using conservative measures. However, it's very easy for the patient to become frustrated by the fact that IDD often takes many months (18 months on average) to heal. I would NEVER recommend a patient rushing into a decompressive fusion (or even SED) until they have waited at least 18 months (preferably 24 month) unless serious medical complication occur (loss of bowl & bladder control, progressive neurological deficit, or severe intractable pain). Conservative treatment option include the following: Medication, Gentle Traction treatments (via RPT or Chiropractor), VAX-D (if you can afford it), and non-dynamic spinal stabilization training/exercise (maybe swimming). The worst thing to do is just around and do nothing! Try and stay as active as possible without severely flaring yourself up. Figure out a way to get that heart rate up in an aerobic zone to enhance blood flow to the disc, which should help with the healing process.
Although this is still considered a 'fringe', there is now some anecdotal evidence that injecting a 'chemical soup' into the disc (and facets) may have some benefit for pain relief in the chronically disabled (13). This chemical soup includes the following: Chondroitin Sulfate, Glucosamine Hydrochloride, DMSO, Marcaine, dextrose (50% of the mix!). This chemical soup is injected under fluoroscopy directly into the disc and facet joints. A pilot study demonstrated that 57% of a group of long-time chronic pain sufferers got about a 74% decrease in both their disability scores and their pain levels. However, PLEASE remember that this was a very small pilot study that needs to be followed up upon, so the results although promising must be taken with a 'grain of salt'. There were lots of 'problems' with the study which I've commented upon in my review of this paper. (Here: Klein, Mooney, Derby et al.)

SED (selective endoscopic discectomy) was created by the innovative Dr. Anthony Yeung MD who uses an endoscope to enter the disc (transforaminally or intralaminally), look around, and repair anular tears. The beauty of this technique is that he is not bound by the limitations of fluoroscopy, which may cause improper placement of any tear-sealing device, for he can physically 'see' inside of the disc and anular tissue and this insures exact placement of the RF probe and/or laser.

You can think of this technique as an 'eyeball-guided' debridement of a damaged disc which is followed by an attempt to destroy granulation tissue and inflammatory tissue within and round the anular tear. He, thankfully, does NOT use IDET technology to perform the annulloplasty, but rather uses specifically directed RF energy to accomplish the task of ridding the disc of pain-producing tissue in and around the anular tear. Any nuclear fragment within the tear (which are the precursors to disc herniation) are removed.

WARNING: Although this procedure has a good 'self-proclaimed' track record, the doctor still refuses to put his procedure to the ultimate test: a double blind investigation where it's compared with traditional discectomy, IDET, and Sham treatment. Because of this, I CAN NOT INCLUDE ENDOSCOPIC SYLYE DISCECTOMY FOR THE TREATMENT OF COMPRESSIVE DISC HERNIATIONS AND EXTRUSIONS that result in radicular pain. However, I think the treatment makes a lot of sense for the treatment of IDD and eventually will be proven to be as effective for decompressing the disc as traditional discectomy. Another downfall is cost. Although I'm not 100% sure, I've heard that this procedure costs between $15,000 to $25,000.00 which insurance may or may not cover? I've recently reviewed both of Dr. Yeung's Endoscopic Procedures: SED and ENDOSCOPIC DISCECTOMY.

IDET, which 'indirectly' uses Radio Frequency (RF) to heat discal tissue, is NOT what I'm recommending here. Annulloplasty preformed with discrode technology (or SED technology) uses RF energy to 'directly' heat the target discal tissue. The cannulas...
(wires that are used to produce the heat) are more steerable (the doctor has more control of where he places the cannula) and can generate a more controlled form of heating in a more specific location. The goal of RF annuloplasty is to destroy pain producing tissue and nerve fiber within the annular tear, and encourage the annular to heal. The biggest disadvantage is that the doctor must use fluoroscopy to see where he's got the needle tip (which generates the heat which 'cooks' the evil IDD tissue). Although this method is better than nothing, it's not nearly as accurate as the SED procedure.

Again, there are no double blind studies out on this technology, but I expect they will be coming. IDET has had several negative investigations, and I have heard and seen many failures to recommend its use.

If all else fails, this is your last stop! ADR is now available in the US (one or two levels) and is probably one of your best bets. Dynesis is still in clinical trials but looks promising as well. Both of the aforementioned are probably better options than traditional interbody fusion since they not only decompress/remove the diseased disc, but they allow for the spine to retain some of its natural motion - which is thought to lessen the chance of 'over-loading' the disc above and below the fusion (the domino effect). I'll comment more on these three final options at a later date. However, your Oswestry score had better be in the 50s before you attempt this drastic of a procedure. The empirical success rates are only about 33% with another 33% getting worse and the final 33% staying the same.

References:


6) Ohnmeiss DD, et al "Degree of disc disruption and lower extremity pain" Spine - 1997; 22(14):1600-1665


A disc herniation is the term given to any uneven out-pouching or bulging of the posterior region (back region) of the intervertebral disc as seen on MRI (you can not see disc herniations on X-ray). The bigger the herniation, the more likely it is to cause "trouble," for the patient, i.e., back and/or leg pain—the latter of which is called sciatica (technically, radiculopathy). Although research has indicated that the size of the herniation has nothing to do with the amount of pain and dysfunction that the patient suffers (small, non-surgical herniations can hurt just as much as huge ones).
**Figure # 10**, which is a sagittal (from the side view) T2 Weighted MRI lumbar image, demonstrates two types of disc herniation: the L5/S1 disc has suffered a 9mm disc extrusion (red arrow) that is not contained by the PLL. The L4/5 disc has suffered a smaller 4mm disc protrusion (green arrow) that is contained by the PLL. The L3/4 (blue arrow) is completely normal and has no disc material projecting posteriorly into the epidural space. Also note that the L3/4 disc is white in color, which indicates it is non-degenerated (i.e., full of water and healthy proteoglycan). The two herniated discs (L4/5 & L5/S1) are "black" on this MRI image, which indicates disc desiccation (lack of water and proteoglycan) and is termed "degenerative disc disease" (DDD), which is usually a precursor to disc herniation for it weakens the annulus which contains the pressurized nuclear material.

In layman's terms, a disc herniation occurs when the jello-like center of the intervertebral disc (nucleus pulposus) tears its way through the back-outer portion of the disc (annulus fibrosus) and invades the space (anterior epidural space) where the delicate nerve structures live. And the presences of this nuclear material (which is filled with biochemical irritants called cytokines) in the anterior epidural space may severely irritate these neural structures (i.e., the dura of the thecal sac and nerve roots that make up the giant sciatic nerve), which in turn may cause severe back and/or leg pain.

In this tutorial we will explore just how a disc herniation occurs and discuss some of the more common classification of herniations.

The term **Disc Herniation** (or "disc prolapse" as they use in Europe) is a broad, general term that includes three specific types or sub-classes of disc abnormalities based upon the condition of the posterior longitudinal ligament ("PLL") that fortifies the back of the disc. The three main classifications of disc herniation are **Protrusion** (aka: contained herniation or sub-ligamentous herniation), **Extrusion** (aka: non-contained herniation, or trans-ligamentous herniation) and **Sequestration** (aka: free fragment). These terms will be discussed more below.

**General Information and Confusion:**

In 1934 the syndrome of "disc herniation" was born when Mixter and Barr first proclaimed that a posterior rupture of the intervertebral disc that allowed nuclear material to escape and compressed the adjacent spinal
nerve root(s) was a common cause of back and leg pain - sciatica (125). For nearly 70 years, this assertion has held true without much challenge (170).

However, modern research as demonstrated that the relationship between disc herniation and its often associated sciatica are a far more complex and bewildering phenomenon than once realized. For example, since the invent of MRI, we have learned that some patients have disc herniation on MRI, yet have no pain at! And, visa versa: some patients have terrible back and leg pain yet have no disc herniation or visible disc defect on MRI! (Click here for the false positive rates for MRI.) Even more confusing is the fact that some patients who completely recover from the back pain and sciatic, still have the same disc herniation appearance on follow-up MRI!

Other ironies of disc herniation have been discovered as well. For example, we have learned from the work of Karppinen et al. that the size and severity of disc herniation do NOT correlate with the degree of patient pain, disability, or suffering (170). That is, small disc herniations and even disc bulges may causes just as much pain and disability as massive disc herniations and even extrusion.

Another strange irony is the fact that smaller and innocent looking disc herniations (i.e., contained herniations, protrusions, subligamentous herniations and/or disc bulges) are often more difficult to treat and respond less favorably to decompressive surgery (discectomy) and conservative care than do the larger and more advanced disc extrusions and sequestrations. (50) Moreover, symptomatic contained herniations have a poorer prognosis for recovery than do the larger more complete disc extrusions and sequestrations do. (50) And, to further cloud the water, we now know that sciatica (a horrible burning lower limb pain associated with disc herniation) is not always causes by the direct compressive pressure from a herniated disc. That is, it can be caused from nuclear material "leaking" from the back of the disc onto the adjacent nerve roots, i.e., chemical radiculopathy (3,4) and/or from chemical and pressure irritation of the posterior intradiscal nerve fiber, i.e., the sinuvertebral nerves, which is called discogenic sciatica (1,2).
So, diagnosing a patient with complaints of back and lower limb pain is certainly not as easy as once believed.

**TREATMENT OPTIONS: SURGERY VERSUS CONSERVATIVE CARE**

One of the most surprising discoveries regarding treatment options for disc herniation-induced radiculopathy is that disc surgery and conservative non-surgical treatment work the same with respect to over-all long-term improvement. The only advantage disc surgery has is that it often get the injured person out of pain faster and back to work fast IF they have the surgery in time and they are a proper candidate. There is, however, an important exception to this rule for patients who are forced into disc surgery because (1) they simply can't stand the pain, (2) they have a progressive worsening of neurological symptom (i.e., worsening of muscle weakness in the upper or lower extremities) and (3) the development of the dangerous cauda equina syndrome. All of the latter three conditions are surgical musts in most cases.

**THE RESEARCH:**

Over the last thirty-years there have been several well-designed medical investigations to support the notion that surgery is no more effective than conservative care--in the long run--as a treatment for disc herniation related back pain / radiating lower limb pain or neck pain / radiating upper limb pain. The best in my humble opinion was the Volvo Award Winning Weber study. The bottom line of this randomized trial was what I just said: disc surgery may get you out of pain and back to work faster, but in the long run there is no real difference in treatment outcome.

In a more recent study, (2007) Peul et al published the results of their medical investigation into surgical outcome of sciatica in the prestigious New England Journal of Medicine. Like Weber, they randomized over 200 patients into either a disc surgery group or a conservative care (non-surgical) group and found the same result: the patients who had surgery, got rid of their leg pain faster; however, at the one year follow-up, the surgical patients were no better off than the ones who went the non-surgical route [An abstract of the study is here].
DISCECTOMY:

According to the medical research, if you must have surgery for disc herniation-related back pain / sciatica, open discectomy or microdiscectomy may be the way to go (99). However, surgery is indicated only if conservative treatment has failed and/or if you have the danger signs associated with disc herniation: loss of bowel and/or bladder control (cauda equina syndrome); progressive worsening of the neurological state (root-related atrophying muscles, progressive muscle weakness [foot drop]); absent reflexes and/or a worsening of pain.

When to have the surgery is also critical in order to increase the chance of success (not so with fusion type surgeries). That is, you certainly don't want to wait any longer than one year before having the surgery for disc-herniation related sciatica (50)--three or four months is about all you should wait. See the Surgery Timing Page for more information.

ENDOSCOPIC & LASER:

With regard to the non-invasive techniques, such as endoscopic discectomy, laser discectomy, etc. I'm not a believer and I do not recommend them at this time. With regard to Laser discectomy, neither does a 2007 meta-analysis (i.e., a study of all the research ever done on Laser discectomy) that was done by Goupille et al (26). The bottom line of the study was that the authors could not recommend the surgery. More explicitly, they stated, "This treatment cannot be considered validated for disc herniation-associated radiculopathy resistant to medical treatment." So, until the inventors and proponents of these procedures step-up to the plate and published some high quality medical investigations (like what Peul et al just did) to prove efficacy (effectiveness), then I'm not a believer.

THE TUTORIAL: THE BIRTH OF A DISC HERNIATION

Lets begin our tutorial with a quick review of the normal disc, and then proceed through each type of herniation. (For a full review of disc anatomy and physiology, please visit my Disc Anatomy Page.)
The Normal Disc:

**Figure #1**: The "Nucleus Pulposus" (pink #1), which is a water-rich gel-like mass of proteoglycan material, has the duty to support the tremendous 'Axial-Load' (weight) of the body. This nucleus is 'corralled' by the stronger 'Annulus Fibrosus' (green #2). The annulus is made out of concentric rings of a cartilage-like material called 'lamellae' (#9). It is this specially arranged collagen that gives the annulus the tremendous strength needed to hold that nucleus in place. **Key Concept**: The nucleus pulposus, because of the tremendous axial load upon it, is constantly trying to escape from the confines of the center of the disc. If it does manage to escape (tear) through the PLL (#7), the appearance on MRI is called a **disc extrusion**. The 'Posterior Longitudinal Ligament' (PLL #7) shields the delicate posterior neural structures and acts as a last line of defense against the potentially irritating nucleus pulposus. Note the posterior disc is 'concave' in shape, as outlined by the PLL. (It will not stay concaved for much longer!) The 'posterior neural structures', which are very sensitive to pressure and chemical irritation, include the following: 'Spinal Nerve Roots' (L4, L5, S1), 'Dura Mater or the Thecal Sac' (red star), and the 'Dorsal Root Ganglion' (DRG). To learn about the anatomy and physiology of the disc go to: [Disc Anatomy]. And finally we have the **Sinuvertebral Nerve** (# SN). The Sinuvertebral nerve innervates (connects to) the outer 1/3 of the annulus fibrosus. These tiny nerve ending have the ability to carry **PAIN** messages to the brain and are thought to be one of the causes of **discogenic pain**. (Read my [IDD page], for more information on discogenic pain.) Oh, one more thing; the **epidural space** (#8) contains the **traversing nerve roots** (L5) that are often the favorite target of the compressive disc herniation.

**THE DISC BULGE**: The First Step Toward Disc Herniation:
In order for a disc to herniate, its structural components must first 'weaken'. This weakening occurs as a result of Disc Degeneration. Disc degeneration occurs naturally, to some degree, in all disc, but in some people the process become especially severe and damaging. The 'bottom-line' of the degeneration process is that the annulus becomes dried (desiccation) and brittle, hence allowing for the development of Disc Bulging and full thickness posterior annular tearing, or Internal Disc Disruption.

Figure #2 demonstrates the 'pre-cursor' to a disc herniation. This type of disc lesion - that bulges into the anterior epidural space without any area of focal-ness or out-pouching - would be called a 'Disc Bulge' on MRI (only because the MRI can NOT show the condition within the disc), although in reality it is a 'Grade 3 Radial Anular Tear' (you would need CT discography to identify the tear) that has disrupted the posterior annulus and allowed irritating nucleus pulposus material to enter into the outer fibers of the disc. Again, this in of itself (IDD) may cause severe and disabling pain in some unfortunate people; however, the subject of Internal Disc Disruption is not the focus of this page. Also note that the PLL, although bulged, continues to be intact and has not ruptured. As well shall see later, the PLL is the 'key' to differentiating between a disc protrusion and a disc extrusion. Finally, note that the Sinuvertebral nerves are irritated (red) and are sending pain signals on to the brain through the sympathetic nervous system (gray ramus communicans). Also note that this IDD may cause some referred lower leg pain as well (spinal nerve has some orange in it to indicate referred pain.)

DISC PROTRUTION: Posterior Longitudinal Ligament is still Intact.
**Figure #3** demonstrates a 4 millimeter disc protrusion and represents a worsening of our disc bulge. The posterior of the disc is 'focally' or 'eccentrically' pushing backwards into the anterior epidural space and has contacted, and even somewhat compressed, the traversing nerve root (white star) and right front corner of the thecal sac. Note that the PLL (blue) still has NOT be disrupted and is still "containing" the near-herniated nuclear material.

The type of presentation in Figure #2. would be 'officially' classified as a 'Disc Herniation' or, more explicitly, a **Disc Protrusion** (aka: contained herniation or subligamentous disc herniation).

Although disc protrusions are seen in about 30% of the normal non-symptomatic population, nerve root compression is not, and if much more indicative of a 'problem. This patient may well be suffering right sided radicular pain (**sciatica**) and/or lower back pain as a result of compression/irritation of the traversing nerve root and/or irritation of the sinuvertebral nerves in the posterior of the disc.

**THE DISC EXTRUSION: The Posterior Longitudinal Ligament has ruptured.**
Figure #4. demonstrates a more serious progression of our pathologically degenerated disc: An 8 millimeter **Disc Extrusion** (aka: non-contained herniation, transligamentous herniation) is now present. The PLL (blue) has finally been defeated and has completely ruptured, hence allowing for further migration of the nucleus pulposus into the anterior epidural space. Note the marked displacement of the traversing nerve root (white star) AND the exiting nerve root (green star) (which has now turned completely red with inflammation and venous congestion - the precursors for Radiculopathy). This Disc Extrusion is NOT typically seen in the asymptomatic person and is often an indication for surgical decompression; the **sooner the better** IF you're NOT improving with conservative care. Another interesting phenomenon about extrusions are the fact that these larger disc lesions have a greater ability to be 'reabsorbed' by the body! This 'shrinkage phenomenon' has been demonstrated time and time again in the literature; in fact, you can expect that in 80% of large disc extrusions, there will be at least a 50% 'shrinkage' of size (5,6). Unfortunately, this doesn't always mean that the pain associated with the extrusion will fade! Some patients recover from disc extrusion yet demonstrate NO change in the size of their extrusion at all, where others fail to recover yet their extrusion has markedly decreased in size! That just goes to prove that we still have a lot to learn about the relationship between disc herniation and pain!

**DISC SEQUESTRATION:** The Final End-Phase of the Disc Herniation.
**Figure #5.** represents the end-of-the-line for the cycle of disc herniation. Now we can see that a big 'chunk' or 'fragment' of nuclear material has detached itself from the main body of the extrusion and loose in the epidural space. Note the resulting severe compression of the traversing nerve root (white star), the exiting nerve root (green star) and the lateral aspect of the Thecal Sac (blue star).

**Sequestration** (aka: sequester, free-fragment) may be excruciatingly painful (back and leg pain - sciatica) and, if centrally located, may occasionally cause the patient to lose control of their bowl and bladder function, i.e., **Cauda Equina Syndrome**, which is considered a 'Medical Emergency'!

As with the disc extrusion, the sequestration may also undergo a reduction in size from a combination of an immune attack (macrophage attack) and dehydration, although frequently the patient will need immediate decompressive surgery to beat this monster!

**MRI DISC HERNATIONS:** Some real pictures.
Figure #6 demonstrates a large 9mm disc extrusion (red star) as visualized on both the Axial (overhead) and Sagittal (side) views.

Note that this extrusion has completely blotted out (can't see) the right traversing S1 nerve roots (left side of image) and has pinched it against the lamina (tiny green arrow). Note the thecal sac is moderately to severely compressed by this large herniation, as noted on both the axial and sagittal images (between blue arrow and red star).

This young man (24 years) has avoid surgery and is doing fairly well, although his days of heavy work are probably over for good.

References:

2) Ohnmeiss DD, et al "Degree of disc disruption and lower extremity pain" Spine - 1997; 22(14):1600-1665 (also in several other journals in 1999)


