Slipped a Disc, Did You?

by Daniel J. Schneck

"All I was doing was tying my shoelaces—a very benign activity—and suddenly the pain was excruciating! I froze. I couldn't move." "Honest, I was just brushing my teeth and the next thing I knew I couldn't get up! The pain was unbearable. I thought I would die." "Would you believe, I just bent over to pick up a pencil that I had dropped? That's the last thing I remember before I wound up in the hospital emergency room! I passed out from the agony."

These and many similar comments are representative of the general public's (and medicine's) misconception of how easy it is to herniate intervertebral spinal discs (in the vernacular, to "slip a disc"). Spinal discs are the 24 (at least) sponge-like spacers located in the gaps between articulating (via posterior facet joints) adjacent vertebrae (the bones of the spine). Lying in front of (anterior to) the facet joints, the discs start in the intervertebral space between the atlas (the first vertebra) and the axis (the second vertebra) and extend all the way down to the intervertebral space between the sacrum (five fused vertebrae forming one sacrificial bone) and the coccyx (four fused vertebrae forming one "cuckoo's-bill-like" tailbone). Collectively, they account for about 25% of the roughly 61-cm (24-in. in the typical adult female) to 71-cm (28-in. in the typical adult male) vertebral column.

Spinal discs have several functions, among them: 1) to form strong joints, thus coupling contiguous vertebrae into a sturdy, yet flexible column; 2) to give the spine a double-S-shaped, characteristic curvature, when viewed from the side, thus giving it an inherent resistance to buckling by allowing it to "give" under compression loading; 3) to allow for various bending, twisting, and complicated movements of the vertebral column; 4) to absorb impact/shock/vibration loading of this structure; and 5) to act, along with the bony vertebrae, as the main compression load-bearing members of the spine—in parallel with ligaments and muscle tendons acting as the primary tensile load-bearing members and stabilizing components to prevent laxity of this flexible anatomic structure.

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In order to fulfill their load-bearing function, spinal discs are endowed with highly hydrophilic (water-loving) mucopolysaccharide gels (mainly chondroitin-4- and 6-sulfate, with lesser amounts of keratan sulfate) that can bind water in quantities up to 100 times their own mass, the latter being in a molecular weight range from 0.5 to 4.0×10^6. These gels are laid down along and enmeshed within a complicated, three-dimensional lattice framework of type II collagen fibers (modulus of elasticity $\approx 5 \times 10^9$ dynes/cm^2). To allow for ample extrafibrillar space in which to confine large quantities of water, the fibers of the collagen matrix are spaced rather far apart in the innermost region of the disc, forming a soft, pulpy, highly elastic structure called the nucleus pulposus. Moving out toward the periphery of the disc, these fibers coalesce into a somewhat dense region of more tightly packed strands, forming a ringlike band called the annulus fibrosus.

Contrary to the way this anatomical configuration tends to be described, i.e., as being analogous to a jelly-filled donut, there is no clear dividing line between the nucleus pulposus and the annulus fibrosus—the former simply merges smoothly into the latter, there being no two, uniquely separate, self-contained regions of the intervertebral disc material. Rather, this material is composed of integrated tissues that are eventually attached through 1-mm-thick hyaline fibrocartilage end plates to the vertebrae on each side of the intervertebral space, forming a disc-filled vertebral sandwich.

As anyone who has ever taken a bellyflop into a swimming pool can attest, water is very resistant to compression loading and distributes pressure quite uniformly throughout its substance. It is thus not surprising to learn that the compression strength of a healthy spinal disc in the lumbar (lower-back) region is on the order of some 1500 pounds or more (about three-quarters of a ton!). Furthermore, as the proteoglycans gel absorbs water, it swells, which also prestresses the surrounding collagen fibers, biasing them to the point where the annulus fibrosus can tolerate tensile stresses ranging anywhere from 200 to 750 pounds per square inch (psi) without failing. Even in shear, the strength of spinal discs is quite impressive; they are able to tolerate without consequence a torsional exposure as high as 550–600 psi.

From a biomechanical point of view, the weakest link in the spinal disc chain is actually in the cervical (neck) region, where, if subjected to tensile forces on the order of 200 pounds or less, disc end plates can either fail themselves or break loose from their attachment to the vertebrae. Such loading can result, for example, from an accident wherein a flailing, unsupported head causes the neck to hyperextend/hyperflex or snap back and forth very rapidly, in whiplash fashion—hence the popular term for one such type of injury.

The bottom line, however, as I stated in a previous editorial ("Hermeneutics: Things Are Not Always As They Seem," *Am Lab* 2003; 35[12]:6–8) is that absent a traumatic incident, such as a serious motor vehicle accident, getting thrown from a horse and landing on your head, or falling down a flight of stairs, spinal discs do not fail as a result of an acute, one-time, benign event like tying your shoelaces. In fact, if a disc does rupture as a result of a traumatic injury, such failure is invariably accompanied by the presence of microscopic cracks in the bony vertebrae. In other words, the discs are not the first structural components of the spine to fail when it is subjected to excessive mechanical loading. Rather, in the hierarchy of such components, the first to give are the delicate spinal ligaments and muscle tendons—experiencing microfailure at around 500 Newtons (112.4 pounds) and total failure at about twice that amount. Next are the vulnerable weak spots in the vertebrae, cracking at compressive forces on the order of 3000 Newtons (674.4 pounds) in the cervical region, increasing to 5000 Newtons (1124 pounds) in the lumbar region; only then do we get to spinal disc failure, followed last by failure of the spinal cord itself when its protection breaks down.

So why does it appear that such a minor event as brushing one's teeth can seriously damage a spinal disc? The answer is simple: The event in question is not the cause of the problem! In fact, the problem might not even be the disc, per se, but rather any one or a combination of factors from a laundry list of confounding variables, including: 1) an instability resulting from weakened ligaments (the first to fail) in the vertebral column, 2) a paravertebral muscle spasm, 3) an abnormal lateral curvature of the spine (scoliosis), 4) a vascular insufficiency (as is the case in a related syndrome called tension myositis), 5) a misalignment of the spinal column (which can often be repaired by chiropractic intervention), 6) osteoarthritis (a joint disease), 7) spinal stenosis (a narrowing of the vertebral canal through which the cord passes), and/or several other contributing factors that do not involve the discs at all. Even if they do, one needs to distinguish between the actual cause of an affliction and an event that merely serves to make an already-existing affliction symptomatic. Truth be told, spinal discs fail far less com-

monly from biomechanical damage than they do from progressive biochemical degeneration. That is to say, as early as the teenage years (and even younger, in rare cases), one begins to experience a patchy loss and disappearance of the proteoglycan gel in the nucleus pulposus.

Spotty loss of the hydrophilic gel leads to an uneven drying out of the spinal disc tissue. As parts of it become desiccated, the ability of the thus-weakened tissue to tolerate compressive loading is seriously compromised. Perhaps more important (or at least as important), the ability of the disc to distribute compressive loads uniformly throughout its substance is also seriously impaired. Thus, when squeezed top to bottom, the disc, rather than withstanding such a force without consequence, instead bulges sideways in all directions. As a consequence, the intervertebral disc space narrows (visible on a radiograph), but it does so unevenly. The key, then, to understanding whether or not you will experience the symptoms of a degenerated disc condition is to realize that it all depends on its state of dehydration, and where a desiccated, weakened disc will bulge under compression loading (have you ever bounced up and down on a water bed and had fun noticing—when you pushed down at one point—how the fluid-filled mattress responded by bulging at another point?). If the bulge extends toward the back and side (postero-laterally) of the vertebral column, where the spinal nerves emanate from the spinal cord itself, the protruding disc (perhaps helped along by your bending over to brush your teeth, tie your shoelaces, or do anything of an equally benign nature) can exert pressure on these nerves, causing considerable pain and discomfort that seems to have resulted from such an innocent activity! In the extreme, the pressure generated in a significantly degenerated disc, bulging under compressive load (which can even be relatively light), might be great enough to rupture the surrounding fibrocartilage (annulus fibrosus), causing the disc to herniate ("slip"), and its innards to be extruded into an adjacent vertebral body or into the surrounding spaces and cavities of the spine. This is the so-called "straw-that-broke-the-camel's-back" explanation for a disc herniation, although, in reality, it does not quite happen that way. The eventual rupture involves the final stages of a degenerative process that progresses more like the fraying of a couch pillow.

On the other hand, if discs bulge away from the posterior regions of the spine, people can have this condition for years and years, even their entire lives, without ever becoming symptomatic. Indeed, if one were to perform a CT scan or MRI exam on just about any person over the age of 30—with or without back pain—the results would invariably show age-related disc degeneration (i.e., desiccation, discoloration that results from altered blood perfusion, the appearance of osteophytes, and related bone spurring, etc.), often involving consequent bulging. One is likely to find such bulging discs four out of five times when examining individuals over age 60, and one in five of them, even when pain-free, will actually have ruptured (herniated or slipped) a disc without ever having experienced symptoms. In other words, without our necessarily being aware of it (as is also true of cardiovascular diseases), most of us are walking time bombs.

Whether or not that bomb will go off, and how serious it will be, depends also on two important additional considerations. The first is weight. Since the compressive strength of a degenerated disc has been significantly compromised as a result of aging, it can obviously not tolerate being squeezed as much as it did when it was healthy. Thus, the greater one's body mass index (weight, in kilograms, divided by the square of one's height, in meters), the more the disc problem is exacerbated. A body mass index between 19 and 24.9 is considered to be normal, 25–29.9 is overweight, 30–39.9 is obese, and greater than 40 is considered to be morbidly obese. It is desirable, for the sake of the health of one's discs, to have less weight hanging off of the spinal column and to help support that weight by developing and maintaining good abdominal muscle tone (i.e., a strong "sixpack of abs," including oblique and transverse muscles).

The second contributing variable to disc disease (and physiologic ill health in general) is smoking. The scientific literature is replete with evidence of a strong association between smoking and peripheral arterial disease. Peripheral arterial disease leads to inadequate perfusion of body tissues. In turn, these tissues suffer from nutritional deprivation, and worse, the waste products of metabolism accumulate, leading to toxic and inflammatory responses, and possibly necrobiosis (a gradual, degenerative death of tissue). Thus, discs that are not properly perfused with an adequate supply of blood will degenerate a lot faster than normal (not to mention being seriously impaired from healing well). Smoking is a prime contributor to peripheral ischemia.

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In limited space, I have, of course, greatly oversimplified the etiology of disc disease. In addition to the primary trifecta of causes—age-related degeneration, exacerbated by excessive weight, aggravated by smoking—there are many additional confounding factors, not the least of which are: 1) an anatomical predisposition (perhaps inherited) to substandard disc performance, 2) systemic pathologies with disc complications (such as arthritis, diabetes, and various musculoskeletal afflictions), 3) underlying malignancies and/or neurologic deficits, 4) psychological anxiety and emotional stress (with the consequent proliferation of toxic stress hormones), 5) dietary deficiencies, 6) poor body mechanics (especially when lifting), 7) allergies, 8) sedentary lifestyle (especially sitting for long periods of time and getting little exercise), 9) history of repeated low back insults, 10) gender (chronic low back pain tends to be more prevalent in women), and 11) athletic participation (subjecting one to possible sports injuries), to name a few. The point I want to make, however, is that you don't slip a disc as a result of an acute, one-time, benign event like getting up out of a chair, coughing, or sneezing. You might, perhaps, make an already slipped disc symptomatic by doing something that is apparently innocuous, but you don't cause it to rupture by that activity! Discs are a great deal stronger than we give them credit for.

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