Low Back Pain

Discussion paper prepared for
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Prepared by:
Dr. Albert Yee, M.Sc., M.D., FRCSC
Professor, Orthopaedic Division, University of Toronto
Orthopaedic Surgeon, Sunnybrook Health Science Centre

Dr. Safraz Mohammed, M.D.
Chief Neurosurgical Resident, University of Toronto.

Dr. Barry Malcolm M.D. FRCSC, M.B.A.
Assistant Professor, Orthopaedic Division, University of Toronto
Orthopaedic Surgeon, Sunnybrook Health Science Centre

Contributing Editor:
Dr. Marvin Tile C.M., M.D., B.SC (Med), F.R.C.S. (C)
Professor Emeritus, Department of Surgery, University of Toronto
Orthopaedic Surgeon, Sunnybrook Health Science Centre

Note: This discussion paper was first published in 1997 and was prepared by the late Dr. W.R. Harris, Professor Emeritus, Division of Orthopaedic Surgery and the late Dr. J.F.R. Fleming, Professor Emeritus, Division of Neurosurgery, University of Toronto. It was revised in 2003 by Dr. Stanley D. Gertzbein, Professor, Orthopaedic Surgery, University of Toronto.
Dr. Albert J.M. Yee is a Professor of Surgery in the Division of Orthopaedics, Department of Surgery, University of Toronto. He is the Co-Director of the University of Toronto Department of Surgery Spine Program. He is the Vice-Chair of Research, Division of Orthopaedics, University of Toronto. He graduated from the University of Toronto, Faculty of Medicine in 1992. He completed his orthopaedic surgical residency at the University of Toronto in 1999. During his surgical residency, he enrolled in the Surgeon-Scientist Program, Institute of Medical Science, Faculty of Medicine and received his Masters of Science degree at the University of Toronto in 1996. He completed his clinical spine surgery fellowship with Dr. Henry Bohlman at Case Western Reserve University, Cleveland, Ohio in 2000. Supported by a University of Toronto Samuel McLauglin Foundation Scholarship in Medicine, he spent an additional postdoctoral research fellowship year in Cleveland with Drs. Brian Johnstone and Jung Yoo.

Since 2001, he is a practicing Orthopaedic Surgeon at Sunnybrook Health Sciences Centre (SHSC). His clinical interests include adult spinal disorders and orthopaedic trauma. He is a consultant in surgical oncology at the Odette Cancer Centre (SHSC) and Bone Metastasis Clinic. He is a full member of the Institute of Medical Sciences (IMS), Faculty of Medicine and Cross-Appointed to the Institute of Biomaterials and Biomechanical Engineering (IBBME), School of Graduate Studies, University of Toronto. He is a translational researcher with interest in bone and vertebral metastases as well as degenerative diseases of the spine / intervertebral disc. He has received funding from numerous agencies including the North American Spine Society, Canadian Breast Cancer Foundation and the Canadian Institutes of Health Research. He is currently the Scientific Officer of the Biomedical Engineering Committee, Open Operating Grants Program (OOGP), Canadian Institutes of Health Research (CIHR). In 2011, he received the J. Edouard Samson Award from the Canadian Orthopaedic Foundation (COF), the highest award for sustained orthopaedic research in Canada. In 2013, he was also selected for the American-British-Canadian (ABC) Orthopaedic Traveling fellowship where 7 North American orthopaedic surgeons visited academic centres throughout the United Kingdom and South Africa.

Dr. Safraz Mohammed is Chief Neurosurgical Resident at the Division of Neurosurgery, University of Toronto, and is in his final year of training. He completed a one year Infolded Spine Fellowship at the University of Toronto in 2013. Dr. Mohammed is a medical graduate of the University of the West Indies, St. Augustine, Trinidad and Tobago. He has won several teaching awards during medical school and during his surgical training. During residency he has published 25 abstracts, 8 journal articles, one book chapter on Spine and Spinal cord injury in Critical Care (in progress), given three research oral presentations at a national level, and one invited lecture to the Canada National Neuroscience Symposium in 2013. Dr. Mohammed was recently recommended at a national level to the Canadian Association of Internes and Residents (CAIR) Resident Spotlight by the 2013-14 CAIR Education & Professionalism Committee.

Dr. Barry Malcolm graduated in Medicine from the University of Toronto in 1971. Following completion of his Orthopaedic Surgical Training at the University of Toronto, he did post graduate spinal surgery training in Toronto and in Minneapolis, Minnesota.
He received his Royal College Fellowship in Orthopaedic Surgery in 1977, and began active practice in Orthopaedic Surgery in 1978 at the Toronto East General and Orthopaedic Hospital where his practice comprised trauma and general orthopaedics with subspecialization in spinal surgery.

He moved to the Toronto Hospital in 1995 whilst doing an Executive MBA at the University of Toronto. There, he was Medical Director of Rehabilitation Solutions.

He is currently on the active staff of Sunnybrook Health Sciences Centre, was Part-Time Medical Director of the Working Condition Program and Sunnybrook Centre for Independent Living (SCIL), and he is on the consulting staff of the new Women’s College Hospital. He is an Assistant Professor, in the Department of Surgery at the University of Toronto. His current clinical practice is confined to the evaluation of people with spine conditions and their associated disabilities; and, teaching at the University of Toronto’s undergraduate and postgraduate levels in spine-related problems.

**Dr. Marvin Tile** graduated from the University of Toronto Medical School in 1957. He did post-graduate training in Orthopaedic Surgery at the University of Toronto from 1958 to 1963, and was awarded the Royal College Fellowship in Surgery (Orthopaedics) in 1963. He was granted the Detweiler Fellowship in 1963 and travelled extensively in Europe, visiting leading orthopaedic centres. He joined the faculty at the University of Toronto in 1966 and holds the rank of Professor (Emeritus) in the Department of Surgery (Orthopaedics).

His clinical and research interests have been in orthopaedic trauma care, and also in the management of arthritis, including hip and knee arthroplasty. He also has a major interest in low back pain.

He has published widely, especially in orthopaedic trauma. He has authored two texts: Fractures of Pelvis and Acetabulum, AOTrauma and Thieme, Fourth Edition, 2015 and Rationale of Operative Fracture Care with Dr. Joseph Schatzker, Springer-Verlag, 3rd Edition, 2005, now in 6 languages. Since 1966, he has been on the Active Staff in orthopaedic surgery at Sunnybrook Health Sciences Centre, a University of Toronto, fully affiliated hospital. He was Chief of Orthopaedic Surgery at that institution from 1971 to 1985 and Chief Surgeon from 1985 to 1996. He has been elected to many prestigious positions. He was the founding president of the Ontario Orthopaedic Association (1978-1980), Past President of the International Society for the Study of Lumbar Spine (1986-1987), Past President of the Canadian Orthopaedic Association (1991-1992), and in 1992-1994, Past President of the AO Foundation, Switzerland (devoted to research and education in fracture care, world wide). As well, he was Chair of the Sunnybrook Foundation (1996-2001). An endowed Chair in Orthopaedic surgery has been established in his name at Sunnybrook HSC and the University of Toronto. Dr. Marvin Tile has been a medical counselor in orthopaedics for the Tribunal since 2004. He is a Member of the Order of Canada.
This medical discussion paper will be useful to those seeking general information about the medical issue involved. It is intended to provide a broad and general overview of a medical topic that is frequently considered in Tribunal appeals.

Each medical discussion paper is written by a recognized expert in the field, who has been recommended by the Tribunal’s medical counsellors. Each author is asked to present a balanced view of the current medical knowledge on the topic. Discussion papers are not peer reviewed. They are written to be understood by lay individuals.

Discussion papers do not necessarily represent the views of the Tribunal. A vice-chair or panel may consider and rely on the medical information provided in the discussion paper, but the Tribunal is not bound by an opinion expressed in a discussion paper in any particular case. Every Tribunal decision must be based on the facts of the particular appeal. Tribunal adjudicators recognize that it is always open to the parties to an appeal to rely on or to distinguish a medical discussion paper, and to challenge it with alternative evidence: see Kamara v. Ontario (Workplace Safety and Insurance Appeals Tribunal) [2009] O.J. No. 2080 (Ont Div Court).
LOW BACK PAIN

Anatomy

A vertebra consists of a ‘vertebral’ body that is a block of bone in front of the spinal canal, which contains the nerve tissues (i.e. spinal cord, nerve roots), and which is protected at the back by a bony arch (i.e. the lamina). The laminae are attached to the vertebral body by paired pedicles. The bony arch at the back of the spine consists of right and left laminae joined together with a prolonged bony ‘spinous process’ (i.e. the bumps you can feel when you run your hand along the middle of your back).

The joined laminae and pedicles are sometime called the neural arch (Figure 1). Each spinal building block (i.e. vertebra) is joined to the next by the intervertebral disc in the front and by the facet joints (right and left superior [upper] and right and left inferior [lower]) at the back. A pair of vertebrae is called a spinal motion segment; and the motion segments together comprise the spinal column. The intervertebral disc consists of an inner part, the nucleus pulposus (NP; the soft gelatinous shock absorber-like core) and an outer part, the annulus fibrosis (AF; the fibrous ring). The nucleus is gelatinous and the annulus is tough and sinewy (Figure 2). The superior and inferior facets are connected by a section of lamina or neural arch called the pars interarticularis. The spinal cord typically ends behind the first or second lumbar vertebra (L1 -2; lumbar means ‘low back’). Below L1-2, the lumbar and sacral nerve roots leave the spinal cord and travel downwards through the spinal canal until they exit from the spinal column at their respective levels (Figure 3). The spinal cord and nerve roots are bathed in cerebrospinal fluid (CSF) and covered by an inner thin membrane (the arachnoid mater) and an outer thick membrane (the dura mater).

Between each pair of vertebrae or motion segment, 2 spinal nerve roots, one on each side, emerge through an opening called the intervertebral foramen (Figure 4), formed at the back or posteriorly by the overlapping upper facet of the vertebra below, and the lower facet of the vertebra above. The upper and lower margins are defined by the pedicles of the respective vertebrae, and the front or anterior margin is defined by the vertebral body and lateral aspect of the intervertebral disc.

Vertebrae are also connected to each other by a complex of ligaments (Figures 4, 5), with overlying muscles.
Figure 1 - Top image shows the normal lumbar vertebra as seen from above; bottom image shows a side view of the normal lumbar vertebra

Image illustrated by Liane Friesen
Figure 2 - Cross-section through normal intervertebral disc showing various ligaments
Lumbar and sacral spine from behind showing nerve roots

*Figure 3 - Lumbar and sacral spine from behind showing nerve roots*
Figure 4 - Side view of normal vertebrae showing ligaments and intervertebral disc
Muscle anatomy

A number of muscles are attached to the vertebral column. The most important is the sacrospinalis (erector spinae) which is the name of a group of muscles that originate from the pelvis and are attached to the vertebrae from behind (Figure 6). It brings the back to the vertical from the bent position as well as controlling side-to-side motion.
Where does back pain come from?

Pain sensitive nerve endings are located in the ligaments that join the vertebrae together, in the muscles alongside the spinal column, in the facet joints (similar to other small moving joints in the body and contain cartilage, subchondral bone, and a joint capsule), in the annulus portion of each intervertebral disc, in the periosteum covering certain bone surfaces and on the dural membrane that covers the nerve roots. Stimulation from irritation of these nerve endings is transmitted to the brain through a branch of each nerve called the ‘posterior primary ramus’. Pain usually felt by individuals in or across the low back region and might sometimes spread to the buttocks and groins. Occasionally the pain travels down the back or front of the thigh(s) towards the knee. This is sometimes called “referred pain”; and when present, has no associated signs of nerve root conduction loss (power, reflex, or sensor change), or nerve root irritation (see discussion below). Conceptually, pain from the facet joints or posterior elements is aggravated by arching (extending) the back i.e. extension-induced symptoms, but not by bending forward (flexing). Conceptually, pain
from bending forward might be mediated through nerve endings in the annulus fibrosis of the disc i.e. flexion-induced symptoms. An individual’s “directional preference” in terms of pain provocation (for example, pain worse with forward bending) and easing (for example, pain better with forward bending) can be used to assist in pain relief, and active forms of treatment.

In common back strain, injured soft tissues become painful. It is not possible to determine precisely which structure is affected; hence the notion of “concept” of pain generation based on flexion – extension movements. The reported symptoms are back dominant. There may be muscular spasm that is observed on physical examination, with or without muscular tenderness (soreness immediately beside the bony projects (i.e. spinous processes) in the middle of the back. Most heal in a few days or at most a few weeks and are not likely to be a source of continuing pain.

Another type of pain is that caused by compression of a spinal nerve root. Such pain usually travels down the extremity in a nerve root distribution, and is the dominant pain. It is called sciatica when it is in the posterior thigh and calf and involves the L5 and/or S1 nerve roots; and is called femoral nerve pain, when it is in the anterior thigh and/or antero-medial shin when it involves (L2), L3 and/or L4. Nerve root compression may be accompanied by neurological findings including numbness in a specific area of skin (dermatome) supplied by that nerve root; and/or weakness of specific muscles (myotome) supplied by that nerve root; and/or deep, gnawing aching in the ligaments, bone and periosteum (sclerotome) supplied by the nerve root; decreased or absent knee (L4) or ankle reflex(S1) (depending on which nerve is affected); and a positive nerve root irritative stretch test (straight leg raising = SLR; or hip extension + knee flexion with patient prone, lying on their side, or standing = positive femoral stretch). Straight Leg Raising or Femoral stretch test is positive if it reproduces a patient’s typical leg dominant pain. Back pain with this manoeuvre is a negative test. This nerve root or radicular pain (i.e. neuropathic pain) is different from and must be distinguished from the local or “referred” pain from low back muscles, ligaments, annulus, periosteum, and facet joints. It is caused by very specific pathology, e.g. nerve root compression from a disc fragment that also causes chemical irritation between the fragment and nerve root – this results in neurogenic pain.

Aging Change Lumbar Spine

Any discussion of back pain is often dominated by the term “Degenerative Disc Disease”. This is an inappropriate phrase because what is being described is usually not a ‘disease’ rather it more typically reflects normal aging change, with a genetic predisposition that we are now starting to better understand. A better description would be "age or genetic related" physiologic changes. It involves not only the disc, but also the facet joints – recall the anatomy of the motion segment. In the general population, patients with age-related degeneration of the intervertebral disc or facet joints usually do not have any symptoms of pain. The normal aging changes typically seen on
plain radiographs, CT and MRI may be misinterpreted by physicians, and patients as being evidence of something abnormal, which in turn might lead to unnecessary investigation, worry/anxiety and sometimes surgery. Epidemiological studies have shown that most people have one or two episodes of back pain during their lifetime, but it is important to note that there is no proportional relationship between the presence and severity of degenerative changes and the presence and severity of symptoms. Normal age related lumbar changes must be distinguished from work-related or traumatic injury through clinical and radiographic evaluation.

With gradual aging, there is loss of water (i.e. dehydration) from the nucleus pulposus with resulting thinning of the disc space between the adjacent vertebrae, which can be seen on plain x-rays. The narrowing of the disc space causes the annulus fibrosis to “bulge” and this can be seen on CT or MR scans – bulging is frequently charted in investigation reports. It does not usually cause symptoms but if the bulging is excessive one or more nerve roots may be compressed with resultant symptoms. Compression symptoms eg. radicular nerve root pain cannot be implied by the appearance on a test such as a CT scan or MRI scan – the individual’s history and physical examination must support symptoms that would be consistent with and correlate to the area of nerve compression that may be observed on an imaging scan. When the bulge is centrally located and there is plenty of room in the spinal canal, nerve roots are rarely compressed. A lateral bulge, if very large, may sometimes compress a nerve root resulting in unilateral radicular symptoms. Over time, along with disc bulging, aging changes may also result in the formation of a bony outgrowth, often called a spur or syndesmophyte, at the periphery of disc. Another result of the disc height loss is that the facet joint relationships are distorted. This can lead to wear and tear changes in them described as “facet osteoarthritis”, which is described on investigations as spurs (osteophytes) or osteochondrophytes – they are typical of osteoarthritis in any synovial joint in the body.”

The incidence of these aging changes is affected by heredity and race. Some families are predisposed to develop marked changes at an early age. Aging change is commoner for example in Caucasians than in Asians.

Current scientific evidence is evolving that supports a significant genetic component to the age related degenerative process – estimated to be as high as 80%. These age-related changes seen on all imaging studies may be as high as 50% by age 50, and 80% by age 80. Recall, there is no convincing evidence that these structural changes that are so obvious on the x-ray or scans cause pain. Despite advances in science, we still do not fully understand the association of chronic mechanical low back pain in patients with imaging evidence of age related disc degeneration. Some individual with advanced degenerative changes evident on radiologic imaging have very little or no back pain, whereas other individuals with less advanced degenerative changes, or none, have more clinically significant back pain. A common teaching point is that in the vast majority of individuals, medical investigations do not confirm the source of back pain, which is not to say that the investigations are “negative” for structural
changes. In a study of 484 individuals, regular x-rays performed for neck or back pain only had direct therapeutic relevance in 0.4% of individuals (Leichtle UG et al., J Back Musculoskeletal Rehabil, 2014 Jun 24. Epub ahead of print)

**Spinal Stenosis**

This age-related process in the neck or low back can lead to a condition called ‘spinal stenosis’ which means “narrowed spinal canal.” The commonest cause is age-related degenerative change. It is important to realize that degenerative change is seen in almost every individual over the age of 60-70 and in some individuals as early as the 20-30’s (Boden SD et al. J Bone Joint Surg Am, 1990 Mar; 72(3):403-8). It is important to recognize too that spinal stenosis is usually asymptomatic; recall it is a descriptor of anatomy. It is an imaging finding, not a clinical syndrome. The associated degenerative changes can be the source of back/buttock pain. Occasionally, the combination of disc and facet degeneration is associated with intermittent radiating leg dominant symptoms = neurogenic claudication (i.e. activity related leg pain resulting from neurologic compression such as in spinal stenosis). Neurogenic claudication is the *sine qua non* of symptomatic spinal stenosis. Clearly, clinical judgement derived from the history and physical examination is critical since ‘spinal stenosis’ is commonly seen and documented in CT scan and/or MRI scan reports. Understandably many individuals and their physicians are concerned about the diagnoses listed on these radiology reports; diagnoses that require careful clinical correlation to symptoms that may be present.

**Retro-Spondylolisthesis**

Sometimes radiographs/investigations show that disc narrowing/degeneration is accompanied by backward displacement of the upper vertebra on the lower =retro-spondylolisthesis (Figure 7). It takes years to develop, and its presence does not automatically imply symptoms. Translated from the Greek it means spondylos=vertebra, listhesis=slip, retro=backwards.

**Spondylolisthesis**

In common usage, spondylolisthesis, sometimes described as anterolisthesis, refers to forward slip of the upper vertebra on the lower. There are several types. Most common are “degenerative” or pseudospondylolisthesis (Figure 8), and “lytic” spondylolisthesis (Figure 9). The latter is the result of a bony defect in the pars interarticularis of the vertebra and will be discussed later. There is also a ‘congenital’ form of spondylolisthesis.

Degenerative spondylolisthesis (Figure 8) usually occurs at L4-L5 and is up to 3-6 times more frequent in women than men (Kalichman L et al., Spine 2009,
Jan 15:34(2):199-205). It may be discovered on plain radiographs done in the
evaluation of low back pain that can radiate to the buttocks, lateral pelvis, greater
trochanteric areas, and occasionally into the lower extremities. Several nerve roots
may be compressed based upon imaging (most commonly L5 and L4 in the case of
degenerative L4-5 spondylolisthesis). Compression may occur in the central portion
of the spinal canal (i.e. central spinal stenosis), at the sides of the spinal canal (i.e.
lateral recess stenosis), or where the individual nerve roots exit the spinal canal in the
foramen (i.e. foraminal stenosis). Again, it is important to emphasize that the presence
of these imaging changes may or may not be associated with either back or leg pain
symptoms.

Symptomatic Age-Related Changes

There are some situations where aging without injury may cause symptoms: 1) a disc
problem (bulge, protrusion, herniation, sequestration); 2) spinal stenosis (narrowing
of the spinal canal); 3) facet joint arthritis; and 4) thoracic age-related (degenerative)
changes.

1. Disc Problems

The intervertebral disc is comprised of a ‘gelatinous’ inner core surrounded by a
thicker outer fibrous annulus structure (not dis-similar to a ‘jelly donut’!). Age related
degeneration of the disc results in stiffening or a ‘less gelatinous’ inner ‘nucleus
pulposus core’. Age related degeneration can also result in tearing of the annulus
fibrosus which often is not related to specific injury. An MRI scan can demonstrate
a ‘High Intensity Zone (i.e. HIZ lesion) that signifies annular tears that have been
suggested to be associated with a poorer clinical outcome. Low back pain symptoms
may be more common in individuals with HIZ changes versus no HIZ changes.
Significant acute traumatic injury often results in imaging findings apart from an
isolated HIZ lesion (for example spinal fracture, facet joint dislocation, spinal
ligamentous disruption). The presence of an isolated HIZ lesion may not be a reliable
marker of traumatic history to disc disruption. Annular tears seen on MRI scans are
also present in asymptomatic individuals.

A defect or tear in the annulus might allow some of the nucleus pulposus to protrude
into the annulus, (protrusion), through the annulus (herniation), or migrate up or down
behind the vertebral body (sequestration). The disc or nucleus fragment might be
asymptomatic or might compress one or more nerve roots (Figs. 10, 11) resulting in a
leg dominant radicular pain pattern, and neurological signs and symptoms. The great
majority of patients with such a protrusion or rupture get better in a few weeks with
eventual resolution of the pain (Baldwin NG, Neurosug Focus, 2002 Aug 15;13(2):E2).
A few such patients (~10%) fail to get better and may require surgery. Some may get
better but are vulnerable to recurrent pain in the future.
2. **Spinal stenosis**

In spinal stenosis (Figure 12), the gradual formation of bony outgrowths with ligamentous thickening narrows the spinal canal and the openings (intervertebral foramen) through which the spinal nerves emerge. Again, the observation of spinal stenosis on a CT scan and/or MRI scan does not imply symptoms, and in most cases is not associated with nerve related symptoms. Spinal stenosis is not caused by trauma except in very rare circumstances, where post-fracture healing with deformity results in stenosis. Mechanical back pain presumed to be on the basis of the degenerating spine is common.

When compared to the results in alleviating low back pain from age-related degeneration, spinal surgery, in general, is more predictive in improving leg dominant or radicular pain symptoms and signs arising from a) nerve root compression as discussed in the section immediately above, and b) neurogenic claudication secondary to spinal stenosis. Narrowing of the spinal canal may be associated with leg pain, numbness/tingling and/or weakness ("my legs feel rubbery") typically brought on by walking and disappearing slowly with rest, particularly in the sitting flexed forward position. Prolonged standing can also be aggravating. If the radicular symptoms are severe and disabling, surgery to decompress the affected nerve roots may be required (i.e. laminectomy, or a ‘surgical’ spinal decompression procedure).

As previously stated degenerative changes in investigations, including spinal stenosis, disc herniation, and nerve root compression may be observed/reported in up to 57% of asymptomatic individuals 60 years or older (Boden et al., J Bone Joint Surg Am, 1990 Mar;72(3):403-8). In asymptomatic individuals, these changes are called ‘false positives’, and increase with advancing age on investigations. Correlation to clinical symptoms and signs by trained physician experts in spinal history and physical examination are critical to determine if any medical/surgical intervention is warranted. In people who have a small diameter spinal canal to begin with (i.e. they are born with it, i.e. ‘congenital spinal stenosis’), the nerve roots are even more vulnerable to the age related degenerative wear and tear process that further narrows the spinal canal. It will be important for the radiologic report to indicate that this patient has a congenital component to their spinal stenosis.

In summary, given the general prevalence of aging changes in the spine, it is not uncommon for work related injuries to be superimposed upon a pre-existing condition, such as age-related degenerative change, spinal stenosis, lumbar spondylosis or degenerative spondylolisthesis; frequently reported as asymptomatic and non-disabling. Most common work related injuries are a soft tissue sprain/strain. Rupture of a disc in a normal motion segment is uncommon without severe trauma, but in an abnormal motion segment, relatively trivial or minor rotation may precipitate the onset of a disc herniation.
3. Facet Joint Arthritis

As discussed above (Page 9), with disc height loss or narrowing, the relationship between facet joints and an intervertebral disc changes, resulting in altered facet joint mechanics/loading, which often leads to facet joint osteoarthritis. The osteoarthritic process is typical of any synovial joint in the body and can include joint swelling (effusion) with capsular hypertrophy, articular cartilage loss resulting in “bone-on-bone”, subchondral bony sclerosis, and formation of chondrophytes (cartilage spurs) that become osteophytes (bony spurs) over time. These changes can be seen on investigations including plain radiographs, CT and MRI scans. Although rare, isolated facet joint arthritis can occur. Radiographically evident facet joint osteoarthritis can be asymptomatic. When symptomatic, facet joint or posterior element pain is conceptually induced or aggravated by arching (extending) backwards, which loads the affected joint(s), but not by bending forward (flexion) which unloads the facet joints. The typical pain is back dominant, and frequently radiates to the area of the posterior superior iliac spine/PSIS. On occasion with extension, it can be associated with short-lived radiating symptoms into the lower extremity in a nerve-root distribution, which subsides with return to the neutral/upright position or lumbar flexion. Facet arthritis is one of the anatomical components of spinal stenosis affecting the foramen, and subarticular spaces (Figure 12).

4. Thoracic Age-related (degenerative) Changes

Thoracic spine motion segments are identical in basic structure to the rest of the spine, but differ somewhat in morphology because of the attachment of ribs at each segment through the costo-transverse and costo-spinal joints (synovial joints). The presence of the rib cage limits thoracic spine movement i.e. it is stiffer, when compared to the cervical and lumbar spines.

Intervertebral disc degeneration is commonly seen in the thoracic spine on plain radiographs with advancing age. Degenerative changes are usually asymptomatic; but when symptomatic, can be associated with a mechanical thoracic dominant pain pattern i.e. related to loading, positioning and movement, including thoracic rotation. Working concepts for pain generation i.e. posterior element and/or anterior (discogenic) depend on direction of provocative movement.
Thoracic radicular pain occurs, and can be referred from degenerative changes; or rarely, secondary to thoracic disc herniation. The radicular pain can radiate around the chest wall to varying degree from paraspinal to around the rib as far as the anterior chest wall (dermatome maps). It can be sharp, lancinating, and very debilitating, and may be associated with sensory symptoms (tingling, numbness) in the same radiating location. Intermittent suggests referred mechanical symptoms; constant suggests more acute disc pathology. The level of radicular distribution can sometimes be helpful in determining a level(s) of involvement within two or three motion segments.

A large thoracic disc herniation is rare, but a potential surgical emergency because the spinal cord ends at L1-2. A large disc can compress the spinal cord resulting in cord compression, and paraparesis. In addition to pain, physical examination will demonstrate upper motor neuron changes in the trunk and lower extremities. Recall, cauda equina compression (i.e. Lumbar spine below ~ L1-2 level) is associated with lower motor neuron findings.
Figure 7 - Top image shows the normal relationship of vertebrae with normal nerve root foramen; bottom image shows backward slip (retropondylolisthesis) of L4 upon L5 caused by degenerative weakening of ligaments and facet joints.
Figure 8 - Degenerative spondylolisthesis - forward slip of L4 upon L5 vertebra due to degenerative changes in ligaments and facet joints. Lower picture shows how nerve root may be compressed.
Figure 9 - Top image shows Spondylolysis (bone defect in ‘pars interarticularis’) with no slip (i.e. without spondylolisthesis); bottom image shows Spondylolisthesis (forward slip of one vertebra in relationship to its neighbour below) due to spondylolysis
Figure 10 - A ruptured (herniated) nucleus pulposus compressing the nerve root, as seen on cross-section.

Image illustrated by Liane Friesen
Figure 11 - Side view showing ruptured nucleus pulposus compressing the nerve root in the intervertebral foramen
Figure 12 - Top image shows the normal spinal canal; middle image shows the developmental (congenital) spinal stenosis (narrowing of spinal canal) due to very short, thick, bony pedicles; bottom image shows degenerative spinal stenosis; cross-section of lumbar vertebra showing bony overgrowths (hypertrophy) of facets causing narrowing of spinal canal. This condition is the result of degenerative changes which are part of the aging process.
Other Abnormalities of the Lumbar Spine

1) Spondylolysis and Spondylolisthesis

In spondylolysis (Figures 9, 13), the pars interarticularis has a defect or gap, where the bone is replaced by gristle. As the gristle is not calcified, it appears as a defect in the x-ray. This is called spondylolysis (see ‘break in bony arch’ in Figure 13). Spondylolysis occurs most commonly in the 4th and 5th lumbar vertebrae, and can be unilateral. While the gristle is very strong it is not as strong as bone. Over time it may stretch permitting the upper vertebra to slip forward on the lower one (Figure 13). This slip is called a “lytic”, isthmic, or spondylolytic spondylolisthesis.

There are different causes of spondylolytic spondylolisthesis. In the most common type (isthmic) the spondylolysis defect is thought to occur most commonly during the early adolescent growing years (Figure 13). It occurs in 5% of Caucasians and in almost 20% of Inuit. It is commoner in ballet dancers and acrobats/gymnasts who arch their backs a lot. Many people with it have no symptoms for a long period of time, and some never have any complaints. But symptoms (back ache) can occur in a person with pre-existing and painless spondylolisthesis, either spontaneously or as the result of a strain or repetitive lifting. Once symptoms commence, they tend to recur.
2) Sacralisation of the 5\textsuperscript{th} lumbar vertebra

In this congenital condition, the lowest (5\textsuperscript{th}) lumbar vertebra is fused to the sacrum, reducing the number of movable motion segments in the lumbar spine from 5 to 4. It does not cause symptoms. There may be more than usual wear and tear of the next disc up (between L4 and L5) causing premature aging change in many patients. Often the transverse process of the 5\textsuperscript{th} lumbar vertebra articulates with the ala of the sacrum (pelvis) by means of a false joint (pseudoarthrosis) but this does not typically cause pain. In some instances, the transverse process of the 5\textsuperscript{th} lumbar vertebrae is fused to the sacrum (Figure 14).

\textbf{Figure 13} - A ‘break in the bony ring of vertebrae’ (i.e. spondylolysis) may cause a ‘forward slippage’ (i.e., spondylolisthesis) of lumbar vertebra L5 on top of the sacrum (S1). This type of spondylolisthesis is known as ‘isthmic spondylolisthesis’.
Figure 14 - This demonstrates normal lumbosacral anatomy on the left image. Anatomical variations (which may or may not be associated with back or sacroiliac joint related symptoms) such as a transition segment is depicted on the right image – on one side (see black arrow), the transverse process appears fused to the sacrum.

3) Lumbarisation of the 1\textsuperscript{st} sacral segment (Figure 15)

In this congenital condition the first sacral segment is separated from the second by a true intervertebral joint, increasing the number of joints in the lumbar spine from 5 to 6. It does not cause symptoms.
Figure 15 - This diagram illustrates where the first sacral segment (S1) behaves like a lumbar vertebra.

4) Scoliosis (Figure 16)

This is sideways curvature of the spine. It can be congenital, secondary to paralysis (such as poliomyelitis) or idiopathic (i.e. no known cause). As the curve increases, the ribs on the concave side are jammed together forcing the vertebrae to rotate. In turn this makes the ribs on the convex side more prominent causing a "hump back". Any type of scoliosis is often associated with premature aging changes in the discs at the apex of the curve. It can cause back pain. Asymptomatic scoliosis may be observed in patients who have radiographs performed for other non-spinal conditions. Thus in patients who have scoliosis and claim work related back symptoms, the facts must be interpreted with caution. ‘Sciatic’ scoliosis is sometimes seen in acute disc protrusions. It is not a structural deformity of the back but the result of muscle spasm.
5) Ankylosing Spondylitis

This is an inflammatory arthritis that affects the spinal column, sacro iliac joints and sometimes the hips. It occurs almost exclusively in young males. It is one of a family of inflammatory arthritis called sero-negative arthropathies, and includes lupus, Reiter’s disease, arthritis associated with inflammatory bowel disease (Crohn’s) and psoriatic arthritis. The cause of ankylosing spondylitis is unknown although associated with a white blood cell surface marker human leukocyte antigen B27 (i.e. HLA-B27).

The condition produces fusion of the spinal column, sometimes in a flexed position so that victims of it have trouble seeing where they are going. It is characterised by intermittent flare-up(s) of back pain often with leg radiation so that it can mimic a herniated disc. Eventually the process “burns out” leaving the patient with a stiff but painless spine (Figure 17). Although historically some authorities believed that trauma plays a role in its onset, the evidence is that it is not caused by trauma. However, these individual are susceptible to trauma as their bones are often weaker (i.e. lower bone density, osteopenia, osteoporosis) and they may develop unstable fractures (i.e. broken bone, broken spine) as a result of injury. Increasing back pain in a patient with a history of ankylosing spondylitis merits close clinical and radiologic evaluation to rule out a fracture.
6) Conditions that physicians may erroneously consider to be the cause of pain.

a) Schuermann’s Disease aka Adolescent Round Back; Hyperkyphosis

This is an abnormality of the growth plates on the upper and lower surfaces of the vertebral body during puberty, which ceases at skeletal maturation. It can result in a marked increase in the normal rounding (kyphosis) of the thoracic spine in adolescents that persists through adulthood. It is seldom a cause of back pain: its principle effect is cosmetic. It is mentioned here because some physicians are puzzled by it. Radiographs show wedging of the vertebral bodies at the apex of the kyphosis that are frequently mis-interpreted as compression fractures. Clinical correlation is required.

b) Schmorl’s Nodules

These are indentations of the nucleus pulposus into the body of the vertebra above. They are normal and are never a source of pain.

c) Separate Apophyseal Ring; Limbus Vertebra

Sometimes the growth plate (see Schuermann’s disease above) fails to fuse completely to the vertebral body at the cessation of growth and appears in the x-ray as triangular piece of bone separated from the upper outer edge of the body. They are a variation of normal anatomy and do not cause symptoms.
d) Diffuse Idiopathic Skeletal Hyperostosis (DISH, or Forestier’s Disease)

DISH is an idiopathic form of degenerative arthritis diagnosed and characterized radiographically (plain radiographs, CT scan) by “flowing” calcification along the anterior and anterolateral aspects of four contiguous vertebra (only on right side in thoracic spine), and calcification/spurring at tendon and ligament insertion sites e.g. pelvis, olecranon, patella/knee, os calcis. The sacroiliac joints are not involved. Thoracic spine is most frequently involved. It is more common in men (65%) and with advancing age. Symptoms include back pain and stiffness, and tendon/ligament insertional pain.

7) Tumors and infections – Spinal tuberculosis or tumours, both primary and metastatic, and inflammatory processes such as osteomyelitis or discitis all cause back pain. They are readily diagnosed by appropriate imaging and not caused by trauma.

Back pain may also be caused by an abnormality of organs in or behind the abdominal cavity (eg. aorta, kidneys, pancreas, gall bladder, etc.). There are a great many causes of back/leg pain and it is important that a careful diagnosis be made in each case before jumping to the conclusion the pain is necessarily due to work related activity or injury.

Problem Areas in Appeals Related to Back Symptoms

1) Can aging changes cause backache?

It is important to recognize that aging change may or may not be the source of back pain -- see also section above on ‘Aging changes in the spine’. Radiologic findings suggestive of aging changes include degeneration of discs, osteophytes, disc bulges / disc herniation, and spinal stenosis. These radiographic findings may or may not be associated with symptoms. In summary, all these structural changes are recognized to occur with aging and do not usually imply trauma or work-related injury. It is important to note that there is no proportional relationship between the presence and severity of degenerative changes and the presence and severity of symptoms. Age related degeneration of the discs typically appear ‘black’ on a T2-weighted MR scan, reflecting age related loss in disc hydration. Individuals who are symptomatic with chronic back pain as a result of degenerating discs may be diagnosed with ‘Degenerative Disc Disease’.

2) Can an injury precipitate aging change?

This is an important question. Injury does not cause aging changes and this question links well with question 3) below as to whether injury can aggravate and/or accelerate aging change. Even without injury, everyone’s spine structurally will ‘age’ over time as
evidenced by repeated radiographic imaging of the same individual over the years. Experts believe that high-energy injury (for example, those that result in traumatic spinal burst fracture, traumatic flexion-distraction bone/ligamentous injury, and traumatic spine dislocation) can result in regional structural changes that appear radiologically ‘accelerated’ when compared to normal aging related changes that may be observed in regions distant to the area of injury. We term these 'accelerated' local changes ‘post-traumatic osteo-arthrosis/arthritis’. Unfortunately, radiographic features of local post-traumatic arthrosis are also quite similar to what may be observed with aging changes (i.e. loss in discal height, osteophytes, facet arthrosis, etc.). As such, clinical correlation to reported radiographic findings is paramount in interpreting potential causality to injury.

3) Can an injury aggravate or accelerate pre-existing aging change?

This is a question whose answer is currently unknown. There is a lack of high-level consistent scientific evidence and natural history studies on this.

4) Does previous back surgery cause back pain?

In some patients it can. When surgery is performed for a herniated disc resulting from degenerative changes, it usually relieves the leg pain (sciatica) but patients commonly have grumbling intermittent back discomfort that persists for years. This is particularly true if the patient has had a prior history of intermittent back pain prior to their surgery. In this situation, back pain most typically relates to their pre-existing aging changes of the disc. There are, however, some situations where individual may develop back pain following back surgery. For example, an individual who undergoes a spinal fusion (eliminating motion between one or more spinal motion segments) will have increased mechanical loads placed on motions segments that remain above and below the fusion region. Over time, these individual may develop back pain as a result of this increased loads placed on adjacent motion segments (i.e. adjacent segment degeneration).

5) Do spondylolysis and spondylolisthesis cause back pain?

The answer is yes, it may. These structural conditions occur in approximately 6-8% of the population, more common in Inuits as well as adolescents with a history of repetitive hyper-extension activity (i.e. competitive level gymnasts, high-school football line-backers). There is also a likely genetic component to this condition as it is also associated with ‘spina bifida occulta’ (a defect in the posterior / dorsal bony arch of the spine in the low back/sacral area), and may more commonly be observed in twins. Some individuals have this pre-existing structural condition and may go through their entire life without significant symptoms arising from this condition – as such, the true prevalence in the general population is probably underestimated. Some individuals may start developing symptoms as a result of age related degenerative changes superimposed upon this pre-existing structural condition. Back pain, with or without radiating leg symptoms (that may follow the L5 nerve root distribution) are typical in
symptomatic patients. We do need to recognize that in some individuals, a traumatic episode may herald the onset of back and leg symptoms in what may have been a relatively asymptomatic pre-existing condition prior to acute injury. Judicious review of pre-injury medical records will be important in determining potential causality of injury in aggravating this type of pre-existing condition.

6) Can scoliosis cause back pain?

Yes, it can in some individuals. It is usually causally unrelated to the patient’s activity, but activity may cause back pain in patients who have scoliosis. In work related claims for back symptoms, the facts must be interpreted with caution.

Question and Answer Format

1. How is back injury diagnosed?

Back injury is diagnosed based upon clinical history and physical examination. Where available, radiologic imaging may support a clinical diagnosis, however, it is important to recognize that in the vast majority of cases of soft tissue injury to the back, radiologic imaging is non-contributory. The history of an injury, the location and onset of back-related symptoms following injury as well as the pattern of pain is important. Hall et al. (Spine J 2009, Aug 9(8):648-57) has described an effective method in classifying low back pain. The most common back injury is a soft tissue sprain/strain and does not necessarily relate to a disc or facet problem. Pain following this type of injury is commonly felt directly in the back. Other back dominant pain patterns (I or II) are thought to be mechanical in nature and may originate from a disc or facet problem. When symptoms of pain start to radiate away from the low back region (for example pain traveling into the legs) a physician may diagnose sciatica, claudication, or radiculopathy. Leg pain dominant patterns include sciatica (Pattern III) or claudication (Pattern IV).

Physical examination focuses on consistent reproducible findings. Information is obtained through observation of the patient, inspection of the area of injury, and specific examination tests. Tenderness and/or spasms (i.e. increased muscle tone causing splinting) in the lower back may suggest a sprain/strain; however this observation is least reliable because of its subjective nature. Following an injury, there may also be visible signs of acute trauma (bruising in the back region, abnormal swelling, gaps or step in the back). Neurological changes including objective evidence of motor weakness anatomically linked to specific nerve roots (i.e. myotomes, Figure 18); reflex changes, which are linked to nerve roots and numbness in a nerve root distribution (i.e. dermatomes, Figure 19) is more concerning if objectively reproducible. Loss of control in bowel/bladder function may prompt specific neurologic examination that includes determining the presence or absence of sensation in the regions around the anus (i.e. ‘saddle anaesthesia’).
Myotomes

Figure 18 - Diagram showing the myotomes (muscle groups and the supplying nerve roots)
Figure 19 - Diagram showing the dermatomes (regions of the skin and the respective nerve roots)
Low Back Pain

Figure 20 - T2-weighed sagittal MRI scan of the lumbar spine. Hydrated discs appear white on this image (see arrow pointing to a normal hydrated disc). Degenerating discs will appear more black (see Figure 21). Nerve roots can be seen within the spinal canal.

Figure 21 - An axial image (left) demonstrating a central disc herniation. A sagittal image (right) demonstrating the same central disc herniation - this has resulted in narrowing of the spinal canal. Note that on T2 weighted MRI image sequences, the lumbar discs appear ‘darker’ (black) in this figure when compared to healthy ‘white’ discs as shown in Figure 20. This is due to loss in disc hydration as a result of the degenerative disc process.
If physical examination findings are not conclusive, or if reported symptoms do not match available radiologic imaging (Figures 20 and 21), additional investigations may become available. For example, a neurologist may also perform Electromyography (EMG) and Nerve Conduction Studies (NCS) that may complement history, physical examination, and imaging results.

2. Are there any particular ergonomic risk factors in work related activities that would create an increased risk for back injuries?

There is a lack of consistent high-level scientific evidence on this topic although some associations have been reported. These include:

1. Prolonged static posture.
2. Frequent or repetitive stretching to the end limit of range of motion or to beyond physiologic range of motion positions. Such movements can involve lifting from the floor or lifting to overhead, or using rotational force (i.e. twisting from side to side) while handling bulky or heavy objects.
3. Lifting of heavy loads repeatedly.

3. What is the relationship between degenerative disc disease or other back conditions and repetitive activities?

Degenerative Disc Disease (DDD) simply implies an individual whose back pain is thought to arise from age related degeneration of their discs, as often confirmed by radiologic imaging. As aforementioned, some individuals may have a degenerating disc without any symptoms of back pain. Causality and relationship to other back conditions and repetitive activities is often a topic of debate. Currently, age, obesity, smoking and genetic factors have been ‘associated’ with degenerating discs. There is no direct causal link currently between repetitive activities and the onset of DDD. Obesity has been linked to a poorer outcome for many medical conditions and these patients may be at higher risk than the general population for recurrent chronic back pain episodes. It is important to note that there is no proportional relationship between the presence and severity of degenerative changes and the presence and severity of symptoms.

4. How does one determine the degree to which an aggravation accelerates the progression of an underlying condition (DDD or OA)?

It is currently impossible to determine the degree to which an aggravation accelerates the progression of an underlying condition such as DDD or OA if indeed it does. There is a lack of high level consistent scientific evidence and natural history studies
supporting injury accelerating the progression of underlying DDD or OA. See also response to question 2 in above section of 'Problem Areas in Appeals Relating to Back Symptoms'.

5. What is the role of repetitive motions in the development of the underlying non-compensable condition?

Referring to the answer for number 3, repetitive motions is not proven to cause degenerative disc disease; additionally there is a lack of high level and consistent scientific evidence that it will worsen other non-compensable conditions.

6. What is the relationship between any particular back condition and any other specific mechanisms of injury?

See responses to above questions.

7. Can there be a delay in the onset of symptoms after back injury? If so to what extent?

This depends on the symptoms. Back pain presents acutely very shortly after injury. In some patients who develop a radiculopathy (radiating leg pain and leg symptoms arising from the nerves), the onset of leg symptoms may be more gradual typically presenting within 2 weeks from the onset of injury – however the onset of back pain from the injury is also immediate in these individuals.

8. Can back pain radiate to the hip (i.e. buttock region, groin), lower leg and foot? Under what circumstances would that occur? Can hip, lower leg or foot pain radiate to the back. Are there ways to distinguish when the pain is due to a back injury as distinct from other condition?

Radiation of pain from the back into the legs generally implies a nerve inflammatory and/or nerve compressive issue arising from the back. Nerve root irritation along the sciatic nerve distribution will typically present with leg symptoms radiating below the knee, whereas symptoms arising from nerves supplying the femoral nerve will present with symptoms radiating into the front of the thigh. We also recognize that there are also many other sources of leg pain that include vascular, localized lower extremity joint arthritis, and other conditions that may affect the legs. Leg symptoms radiating up towards the back is less common and may reflect more of a localized leg extremity condition. Again, careful history and physical examination will be necessary
to determine if a back injury is the source of pain as distinct from other conditions. As an example, it may be helpful to determine if range of motion (for example in flexion or in extension) of the low back reproduces the typical pain the individual experiences.

9. Are there other conditions/diseases that would predispose someone to back injury?

Yes. Recognizing that consistent high-level scientific evidence is currently lacking, most experts believe that some patients may be more vulnerable. Patients with degenerative disc disease, osteoporosis, inflammatory conditions (such as ankylosing spondylitis), and conditions affecting bone density and/or quality (i.e. osteoporosis, renal failure, etc.) may be at higher risk for back pain related injury. Relational causality needs to be individualized in these situations considering history, physical examination, as well as available radiologic imaging. Pregnancy has also been linked to an increased risk of back pain related symptoms, particularly in those individuals with a prior history of back pain.

10. If someone has back injury are there any particular restrictions in work activities that would be appropriate?

Mandated medical restrictions (i.e. a physician suggests that this physical activity is not medically recommended) need to be individualized. In general, in the acute phase after back injury, we advise patients on a ‘very brief (1-2 day) rest’ period followed by a graduated physical activity regime. Patient ‘functional tolerances’ may reflect what an individual may be able to perform physically, with those activities not ‘medically restricted’ by their treating physician. The patient, the type of back injury, and an understanding of a patient’s particular work environment and work related activities are all considered in determining any potential restrictions that may be required.

11. Can you please explain what is meant by mechanical back pain, definition, etiology etc?

Mechanical pain is caused by loads, positions and movements in terms of provocation, aggravation, and modification. In general, it is a term that is used to describe any type of back pain caused by placing either normal or abnormal stress or physical strain on structures of the vertebral column.
There may be several potential sources for this pain. A muscle strain may result in injury of the muscle - such as minor tears in the muscle fibres (Figure 22). Direct injury to muscle such as with a direct blow leading to a contusion or hematoma in the muscle may result in muscular pain with spinal range of motion. Ligamentous stretching or sprain/strain may cause activation of pain nerve fibres. Mechanical pain may also be caused by the disc (i.e. discogenic back pain) or the posterior elements including facet joints.

12. Can you please explain what is meant by the term “disc bulge” when used in diagnostic imaging reports?

The disc is like a jelly donut (it has a thicker more fibrous outer lining and a more ‘gelatinous’ interior). Mechanical loading or age related changes may result in more general dispersion (or ‘bulge’) of the disc on an axial (cross-sectional) CT scan or MRI scan. This ‘bulge’ is broad based and is different to that typically seen with a more focal disc herniation or disc protrusion (Figures 23 and 24). Disc bulges are common radiologic findings on an MRI or CT scan and are not caused by work injury.
Figure 23 - Diagram showing a disc bulge (i.e. ‘generalized’ and more broad based as depicted on the lower right image) that does not compress neural elements as compared to a ‘focal’ herniated disc (top right image) that tend to compress neural elements and may cause symptoms of radiating leg pain, numbness and/or weakness.
13. Can you explain when and how spontaneous back pain occurs that is not related to injury and that arises in the absence of an external injuring process?

This is the most common type of back pain and studies show that the majority of cases of spontaneous sudden-onset low back pain are classed as nonspecific. It is called nonspecific simply because the exact cause of the pain is not usually evident. In other words, there is no specific problem, disease or condition that can be clearly identified as the cause of the pain.

14. What is spinal stenosis, and how may it be related to low back pain from injury?

Spinal stenosis is a narrowing (stenosis = narrowing) of the spinal canal (Figure 25).
Low Back Pain

Figure 25 - Diagram showing stenosis of the spinal canal

Spinal stenosis is a narrowing of the spinal canal

It is caused by a number of factors as follows:

1. Aging and degeneration (most common)
   a. ligament hypertrophy (thickening)
   b. bulging discs
   c. Syndesmophytes/enesthmophytes facet joint hypertrophy – osteochondral spurs
   d. fractures of the vertebral bodies

2. Arthritis
   a. Osteoarthritis (i.e. aging and degeneration, most common)
   b. Rheumatoid arthritis

3. Congenital narrowing of the canal as in congenitally short spinal pedicles

4. Subluxation – abnormal movement of one vertebra forwards over another causing narrowing of the canal and/or nerve root foramen. See Figures 7 and 8.

5. Soft tissue masses - these include spinal tumors, infection, abscess, hematoma, cysts etc.

6. Trauma
Back injury rarely causes spinal stenosis and the term ‘spinal stenosis’ as observed on most radiologic imaging reflects a pre-existing condition that may or may not have been previously asymptomatic. A high-energy injury (for example a fall from significant height resulting in a ‘bursting fracture’ of the lumbar vertebrae) may cause acquired spinal canal stenosis with some of these individuals presenting with a neurologic deficit in the lower extremity.

Patients who have symptoms from spinal stenosis may experience neurogenic claudication (pain in one or both lower limbs upon ambulation or standing for a period of time, leading to fatigue in both legs that is eased by sitting down), or nerve root compression/radiculopathy (leading to pain, numbness or weakness in a particular region of the lower limb).

Selected Glossary of Terms

aging disc: Disc demonstrating the features of normal aging.

annular fissure: Separations between annular fibers, separations of fibers from their vertebral body insertions, or separations of fibers that extend radially, transversely, or concentrically, involving 1 or many layers of the annular lamellae. Note that the terms “fissure” and “tear” have often been used synonymously in the past. The term “tear” is inappropriate for use in describing imaging findings and should not be used (tear: nonstandard). Neither term suggests injury or implies any knowledge of etiology, neither term implies any relationship to symptoms or that the disc is a likely pain generator, and neither term implies any need for treatment. Also, see annular gap, annular rupture, annular tear, concentric fissure, HIZ, radial fissure, transverse fissure.

anterior displacement: Displacement of disc tissues beyond the disc space into the anterior zone.

annulus, annulus (abbreviated form of annulus fibrosus): A multilaminated ligament surrounding the periphery of each disc space, attached superiorly and inferiorly, to end-plate cartilage and bone and blending centrally with nucleus pulposus.

asymmetric bulge: Presence of outer anulus beyond the plane of the disc space, more evident in one section of the periphery of the disc than another, but not sufficiently focal to be characterized as a protrusion.

broad-based protrusion: Herniation of disc material extending beyond the outer edges of the vertebral body over an area greater than 25% (90 degrees) and less than 50% (180 degrees) of the circumference of the disc.
bulging disc, bulge (n), bulge (v): 1. A disc in which the contour of the outer anulus extends, or appears to extend, in the horizontal (axial) plane beyond the edges of the disc space, over greater than 50% (180 degrees) of the circumference of the disc and usually less than 3mm beyond the edges of the vertebral.

capsule: Combined fibers of anulus and posterior longitudinal ligament. Note: The interface between outer anulus and posterior longitudinal ligament can be indistinguishable, making useful the term “capsule” and the derivative “sub-capsular,” which refers to disc tissue beneath the capsule.

chronic disc herniation: Disc herniation with presence of calcification, ossification, or gas accumulation within the displaced disc material, suggesting that the herniation is not of recent origin.

concentric tear: Tear or fissure of the anulus characterized by separation, or break, of anular fibers, in a plane roughly parallel to the curve of the periphery of the disc, creating fluid-filled spaces between adjacent anular lamellae.

degenerated disc, degeneration (n): Changes in a disc characterized by desiccation (drying), fibrosis and cleft formation in the nucleus, fissuring and mucinous degeneration of the anulus, defects and sclerosis of end-plates, and/or osteophytes at the vertebral apophyses.

disc space height: The distance between the planes of the end-plates of the vertebrae cranial (superior) and caudad (inferior) to the disc.

extra-foraminal zone: The zone beyond the sagittal plane of the lateral edges of the pedicles, having no well-defined lateral border.

extra-ligamentous: Posterior or lateral to the posterior longitudinal ligament.

extruded disc, extrusion (n), extrude (v): A herniated disc in which, in at least one plane, any one distance between the edges of the disc material beyond the disc space is greater than the distance between the edges of the base in the same plane, or when no continuity exists between the disc material beyond the disc space and that within the disc space.

focal protrusion: Protrusion of disc material so that the base of the displaced material is less than 25% (90 degrees) of the circumference of the disc.

foraminal zone: The zone between planes passing through the medial and lateral edges of the pedicles.

free fragment: A fragment of disc that has separated from the disc of origin and has no continuous bridge of disc tissue with disc tissue within the disc of origin. Syn: sequestrated disc.
**hard disc**: Disc displacement in which the displaced portion has undergone calcification or ossification and may be intimately associated with apophyseal osteophytes.

**herniated disc, herniation (n), herniate (v)**: 1. Localized displacement of disc material beyond the normal margins of the intervertebral disc space.

**intra-dural herniation**: A disc from which displaced tissue has penetrated, or become enclosed by, the dura so that it lies within the thecal sac.

**osteophytes**: Focal hypertrophy of bone surface and/or ossification of soft tissue attachments to the bone.

**paracentral**: In the right or left central zone of the vertebral canal.

**prolapsed disc, prolapse (n), prolapse (v)**: (Non-Standard) 1. A herniated disc in which disc tissue has protruded or extruded at the level of the disc and below into the supra-pedicular level.

**protruded disc, protrusion (n), protrude (v)**: 1. A herniated disc in which the greatest distance, in any plane, between the edges of the disc material beyond the disc space is less than the distance between the edges of the base in the same plane.

**ruptured anulus**: Disruption of the fibers of the anulus by sudden violent injury.

**Schmorl's node**: See intrervertebral herniation.

**sequestrated disc, sequestration (n), sequestrate (v)**; (var: sequestered disc): An extruded disc in which a portion of the disc tissue is displaced beyond the outer anulus and maintains no connection by disc tissue with the disc of origin.

**sequestrum**: Disc tissue that has become displaced from the disc space of origin and lacks any continuity with disc material within the disc space of origin.

**spondylitis**: Inflammatory disease of the spine, other than degenerative disease. Note: Spondylitis usually refers to non-infectious inflammatory spondyloarthopathies.

**spondylosis**: Spondylosis deformans, for which spondylosis is a shortened form. Any degenerative changes of the spine that include osteophytic enlargement of apophyseal bone.

**spondylosis deformans**: Degenerative process of the spine involving essentially the anulus fibrosus and characterized by anterior and lateral marginal osteophytes arising from the vertebral body apophyses, while the intervertebral disc height is normal or only slightly decreased.
**sub-articular zone:** The zone, within the vertebral canal, sagittally between the plane of the medial edges of the pedicles and the plane of the medial edges of the facets, and coronally between the planes of the posterior surfaces of the vertebral bodies and the under anterior surfaces of the superior facets. Syn: lateral recess.

**sub-capsular:** Beneath the composite of anulus and posterior longitudinal ligament

**syndesmophytes:** Thin and vertically oriented bony outgrowths extending from one vertebral body to the next and representing ossification within the outer portion of the anulus fibrosus.

**tear of anulus, torn anulus:** See fissure of anulus and rupture of anulus.

**undisplaced disc:** A disc in which all disc material is within the intervertebral disc space.

**vacuum disc:** A disc with imaging characteristics suggestive of gas in the center of the disc space, usually a manifestation of disc degeneration.

**vertebral body marrow changes (Modic’s classification):** Reactive vertebral body modifications associated with disc inflammation and degenerative disc disease, as seen on MR images. Type 1 refers to decreased signal intensity on T1-weighted spin-echo images and increased signal intensity on T2-weighted images, indicating bone marrow edema associated with acute or sub-acute inflammatory changes. Types 2 and 3 indicate chronic changes. Type 2 refers to increased signal intensity on T1-weighted images and isointense or increased signal intensity on T2-weighted images, indicating replacement of normal bone marrow by fat. Type 3 refers to decreased signal intensity on both T1 and T2-weighted images, indicating reactive osteosclerosis.