Workplace Safety and Insurance
Appeals Tribunal

Tribunal d'appel de la sécurité professionnelle
et de l'assurance contre les accidents du travail

Low Back

Discussion paper prepared for

The Workplace Safety and Insurance Appeals Tribunal

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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. It was further revised in 2019 by Dr. Albert Yee, Professor of Surgery, (Orthopaedics) University of Toronto, Dr. Safraz Mohammed, Assistant Professor, Neurosurgery Division, University of Ottawa, Dr. Barry Malcolm FRCSC, M.B.A. Assistant Professor, Orthopaedic Division, University of Toronto and Dr. Marvin Tile, Professor Emeritus, Surgery (Orthopaedics), 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 Master 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 McLaughlin 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 the hospital's Holland Bone and Joint Program Chief and Marvin Tile Chair, Division Chief of Orthopaedic Surgery. He is also a consultant in surgical oncology at the Odette Cancer Centre (SHSC). 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 has been 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 seven North American orthopaedic surgeons visited academic centres throughout the United Kingdom and South Africa. In June 2019, he was awarded the distinction of Fellow of International Orthopaedic Research (FIOR) by the International Combined Orthopaedic Research Society (ICORS).

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 six 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, worldwide). 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). For more information about these papers, please consult the *WSIAT Guide to Medical Information and Medical Assessors*.

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1.0 Introduction

1.1 Historical Insights

Low back pain is an ancient affliction, known in old civilizations.

It was well known to the father of modern medicine, the Greek physician Hippocrates (460-377 B.C.E.). He wrote about low back pain and the recommended nonoperative care at that time. He also noted that the pain was often episodic and improved with time in most patients.

1.2 Clinical Epidemiology with Respect to Low Back Pain

A. Definitions

When he coined the term “clinical epidemiology” in 1938, John R. Paul defined it as “a marriage between quantitative concepts used by epidemiologists to study disease in populations and decision-making in the individual case which is the daily fare of clinical medicine”.[1,2]

Prevalence is the term used to study disease in populations and is defined as “the measurement of a condition over a period of time”

The formula is: # existing cases*/ population at risk* (*during a specific time period)

Incidence is defined as “the number of new occurrences of a condition (or disease) in a population over a period of time.”

The formula is: # new cases*/ population at risk* (*during specific time period)

Understanding the meaning or these terms, prevalence and incidence with respect to the copious literature on low back pain is essential as we describe the sequelae of both acute and chronic low back pain syndromes later in this paper.

B. Prevalence of Aging Changes in the Lumbar Spine (degenerative disc disease)

All parts of the musculoskeletal system (MSK) are affected by the aging process. This is true in all joints (e.g. hip, knee, shoulder), and equally true in the motion segments of the spine, especially in the lower lumbar and lower cervical segments.

This has led the discussion of back pain to be 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 related changes. These changes occur in all areas of the motion segment including the intervertebral disc, the annulus and also the facet joints.

In the general population, patients with age-related degeneration of the intervertebral disc or facet joints may or may not have any symptoms of pain.

The very extensive literature on this subject are very clear; these aging changes are almost universal occurring in 50% of people at age 50 and 80% at age 80. They are most often seen in the lumbar spine at the lower L4-5 and L5-S1 motion segments (or motion levels), and in the cervical spine at the mid C5-6 and C6-7 segments.

The normal aging changes can typically be seen on plain radiographs, CT and MRI and 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. In medical reports by radiologists on imaging, these structural changes are often detailed, with the clinical challenge being to determine if clinical symptoms correlate with and/or are caused by structural changes that may be present.

These aging degenerative changes, therefore, are present irrespective of any symptoms of low back pain, i.e. they may or may not be the cause of the patient's symptoms at that moment in time. These anatomical changes will be described in detail later in this paper (see 4.0)

The incidence of these aging changes is affected by heredity; some families are predisposed to develop marked changes at an early age. Aging change is unlikely affected by race, studies on this subject are confusing and often reflect the population in the particular study.

It is important to realize that age related degenerative change is seen in almost every individual over the age of 70 and in some individuals as early as the 20-30s.[3]

Therefore, it is unlikely that other factors influencing these age related degenerative changes in the lower back, such as gender and occupation have an effect on causation, but may act as aggravating factors of this pre-existing condition.

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.

C. Can injury or vocation influence the aging process; Causation vs Aggravation?

i) Causation:

Given that degenerative change is so pervasive in our society, it is unlikely that injury or one's occupation causes early disc degeneration. There are few credible scientific papers supporting that theory (see Andersson, Epidemiology, Chapter 1.2, D).[4]

These aging (degenerative) changes are an incremental process which occurs gradually over time. Degenerative change apparent on imaging studies such as X-Ray, CT or MRI shortly after the accident, was not the result of the accident.

ii) Aggravation:

Age related degenerative change results in a biomechanical and biological abnormal motion segment in close proximity to the exiting nerve root in the intervertebral foramen.

Although the individuals with these changes do not necessarily have any symptoms, they are at risk of becoming symptomatic often with no injury. On occasion, individuals with pre-existing degenerative changes may be vulnerable to symptoms arising from work injury – for example, the development of lumbar radiculopathy following injury in a situation where there was significant pre-existing degenerative changes that had already resulted in nerve root compression. This would be considered an aggravation with symptoms arising from a pre-existing condition with structural changes that were previously asymptomatic or minimally symptomatic. Such symptoms (eg. lumbar radiculopathy in the example provided) would typically arise within a short period of time (ie. within days up to ~1-2 weeks). Such injury may arise by repetitive bending and twisting movements, usually in the flexed position and/or associated with heavy lifting.

Some occupations, such as construction work and other manual employment, e.g. garbage collectors, warehouse workers, nurses, Personal Support Workers (PSWs) which involve repetitive bending, heavy lifting and twisting, may put the workers at risk as they age, thereby acting as aggravating factors for this pre-existing condition. Sitting for long periods may also contribute to symptoms of low back pain.

D. Incidence of Discogenic (Mechanical) Back Pain

As noted previously, this discussion paper will review the concepts of Discogenic or as it is often referred to, Mechanical low back pain, arising from the Motion Segments in the Lumbar Spine. The paper will also discuss other causes of low back pain, such as inflammatory disease.

Low back pain is a very common reason to seek medical attention, and accounts for more than 15% of sick leave. Epidemiology studies have indicated a lifetime prevalence of 90%, most resolving within four weeks of onset, with or without treatment.

The prevalence of mechanical (Discogenic) LBP has been well described in the classic paper by Dr. M.L.Rowe; “Low Back Pain in Industry, a Position Paper”[5] as well as other authors, including Alf Nachemson[6] and many others.[7]

In 1998, in a review of the Epidemiology of Low Back Pain, Gunnar Andersson wrote the following, still true today as the last decade of copious literature on this subject shows.

> "**Background-where we stand**
>
> Frequency
>
> 75-85% of all people will experience back pain (BP) in some form during their life. National statistics from the United States indicate a yearly prevalence in the 15-20% range. Back pain is the most frequent cause of activity limitation in people below age 45, the second most frequent reason for physician visits, the fifth most frequent for hospitalization, and the third ranking reason for surgical procedures. About 1% of the U.S. population is chronically disabled because of BP and another percent is temporarily disabled. About 2% of the U.S. work force have compensable back in juries each year, for a total of over 500,000 injuries."[4,]
>
> "National statistics from European countries reveal that 10-15% of all sickness absence is due to back pain, a percentage that has remained constant in spite of rising absolute numbers of lost work days per worker. The 1-year prevalence of back pain in European countries varies from 25-45%. Chronic BP is present in 3-7% of the adult population.
>
> Cross-sectional studies from several countries provide support for the national prevalence numbers. Although there are large differences in study design and methodological quality, there are remarkable similarities in the estimates of lifetime incidences and point prevalences (Table 1). The lifetime incidences vary from 50 to over 80% with average incidence of 60%."[8]

Conclusions of paper: LBP causes more global disability than any other condition. With the aging population, there is an urgent need for further research to better understand LBP across different settings.

1.3. Other Causes of Pain Arising in the Lower Back:

A. Red Flags:

Because of the high prevalence of discogenic low back pain in society, many clinics have a triaging process for determining which patients require consultation with a back surgeon. Health professionals screening patients must be aware of the ***Red Flags*** that may signal more sinister causes of low back pain, especially in older patients.

These include:

- The older patient with continuous pain, especially night pain or pain at rest.
- Constitutional signs, fever, weight loss.

- Risk factors, such as diabetes, drug use (steroids)
- Rapid onset neurologic loss including bowel and/or bladder control issues.

These patients require careful and urgent investigation.[9]

B. Other non discogenic sources of back pain include:

- Diseases of the abdominal viscera, especially retroperitoneal organs such as kidney and pancreas, including malignancy.
- Conditions of the retroperitoneal blood vessels, such as Aortic aneurysm
- Neurologic structures, e.g. spinal cord tumours
- Bone, the vertebra are a common site for metastatic cancer; most from breast, kidney, lung, lymphoma, myeloma, prostate, thyroid.
- Psyche

2.0 Anatomy of the Vertebral Column and the Lumbar Motion Segments

2.1. Motion Segment Anatomy

Understanding mechanical back pain requires an understanding of the complex anatomy of the spine, especially that of the Spinal Motion Segment (also known as the Functional Spinal Unit, FSU) Figure 1a.

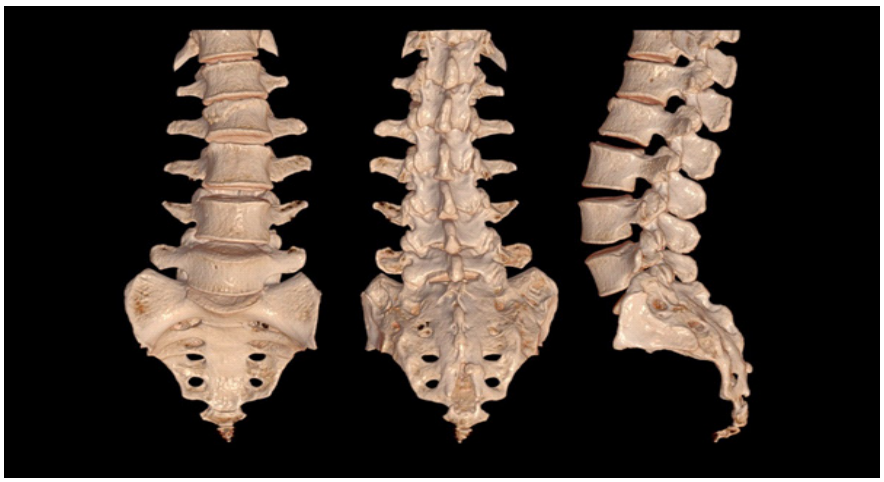


Figure 1a. The Lumbosacral Spine from the front (anterior), the back (posterior) and the side (lateral). Image courtesy of Radiological Imaging/Shutterstock.com.

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 sometimes called the neural arch (Figure 1b). 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.

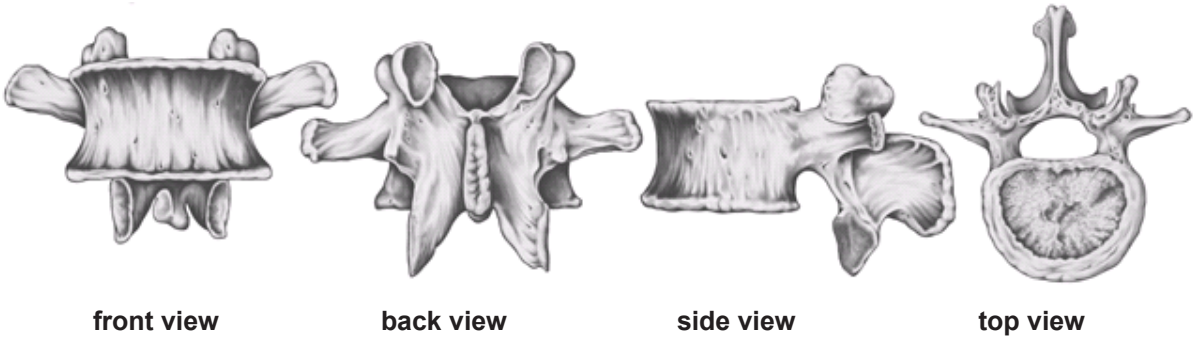


Figure 1b. The Normal Lumbar Vertebra from the front (anterior), from the back (posterior), from the side (lateral), and from the top. Image courtesy of stihii/Shutterstock.com.

A pair of vertebrae with its intervening tissues is called a ***spinal motion segment (aka spinal motion level);*** 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 facet joints are small synovial joints (like the little knuckle joints of the hand) at each spinal motion segment. The superior and inferior facets are connected by a section of lamina or neural arch called the pars Interarticularis (i.e. between facet joints).

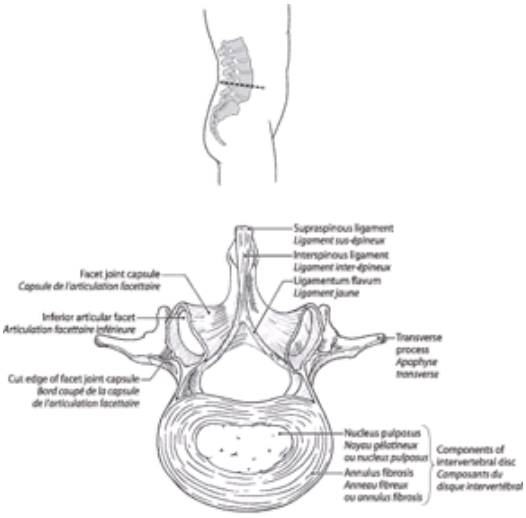


Figure 2a.

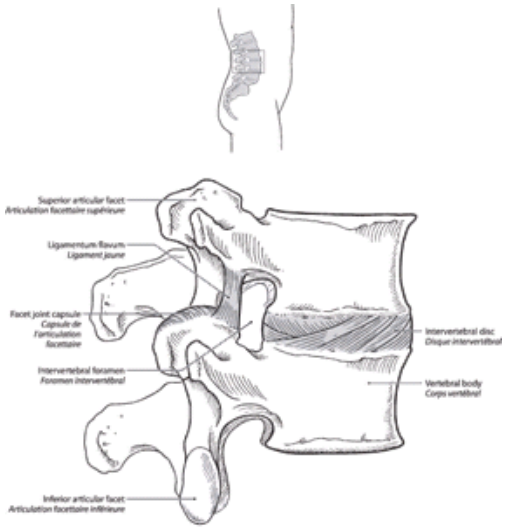


Figure 2b.

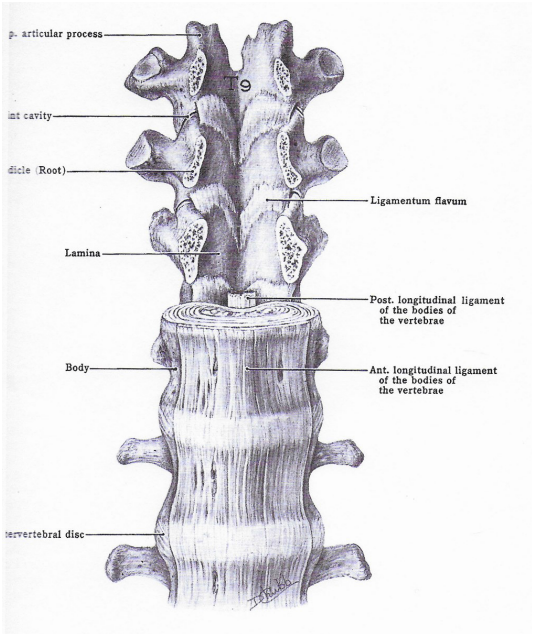
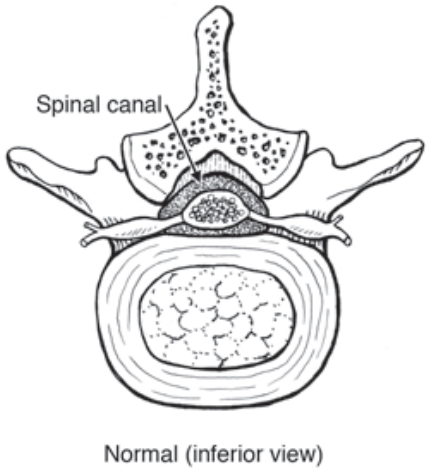


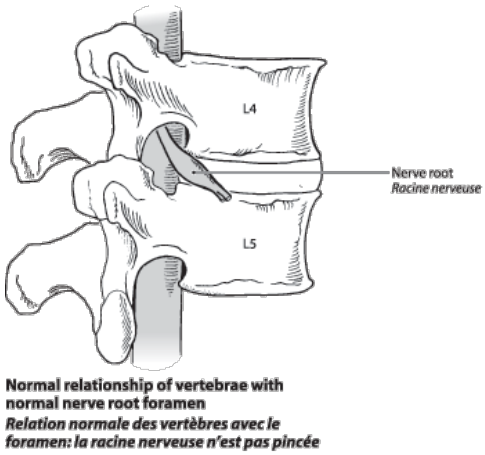
Figure 2c.

Figure 2. Spinal Motion Segment showing the intervertebral disc with the nucleus pulposis and annulus fibrosis and the ligaments. Figure 2a. from above, Figure 2b. from the side, lateral, Figure 2c. from the front, anterior, also with cut out to show the posterior structures surrounding the cauda equina (neural) (from Grant's Atlas of Anatomy, Williams and Wilkins, 1978, 7th edition).

Between each pair of vertebrae or motion segment, two 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 (Figure 3).





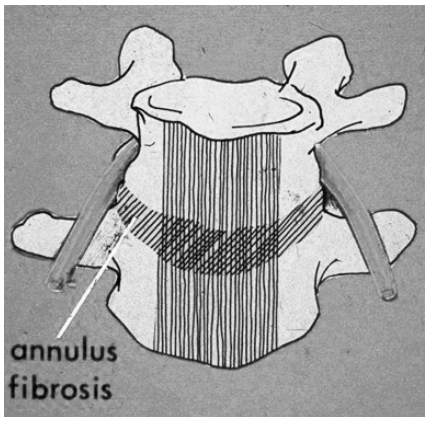


Figure 3. Spinal Motion Segment showing the intervertebral disc with the nucleus pulposis and annulus fibrosis and the ligaments: (clockwise) from above; from the side; from the front with the neural elements, caudal equina and exiting nerve roots.

The spinal cord typically ends behind the first or second lumbar vertebra (L1 -2; lumbar). 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) (Figure 4).

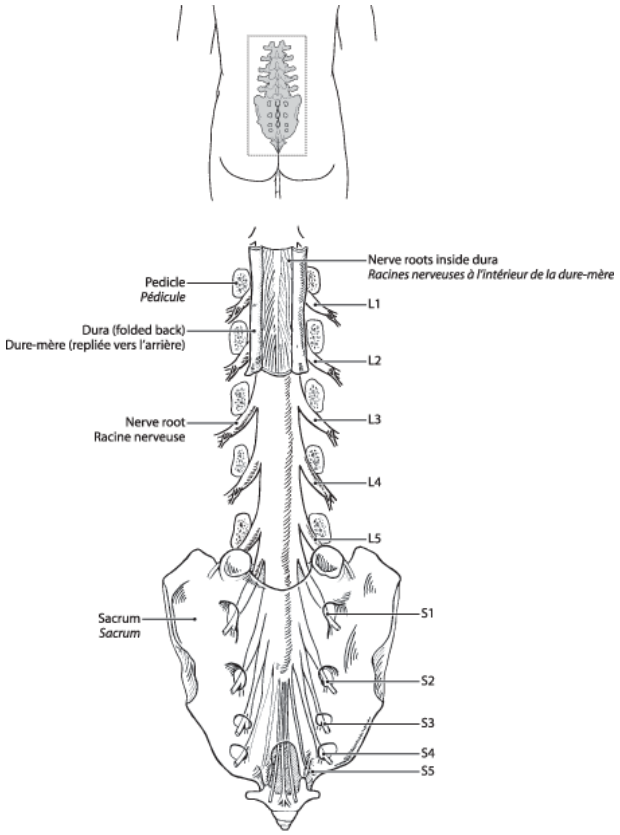


Figure 4a. Lumbar and sacral spine from behind showing the cauda equina and exiting nerve roots (illustration by Liane Friesen)

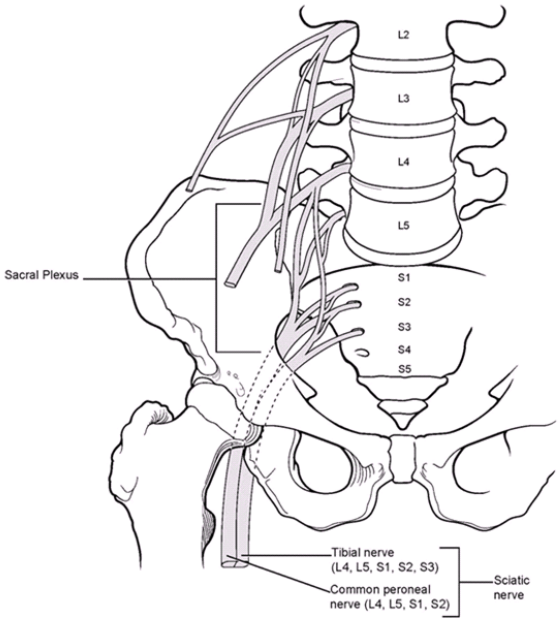


Figure 4b. Lumbar and sacral spine from the front including pelvis

Vertebrae are also connected to each other by a complex of ligaments with overlying muscles (Figure 5).

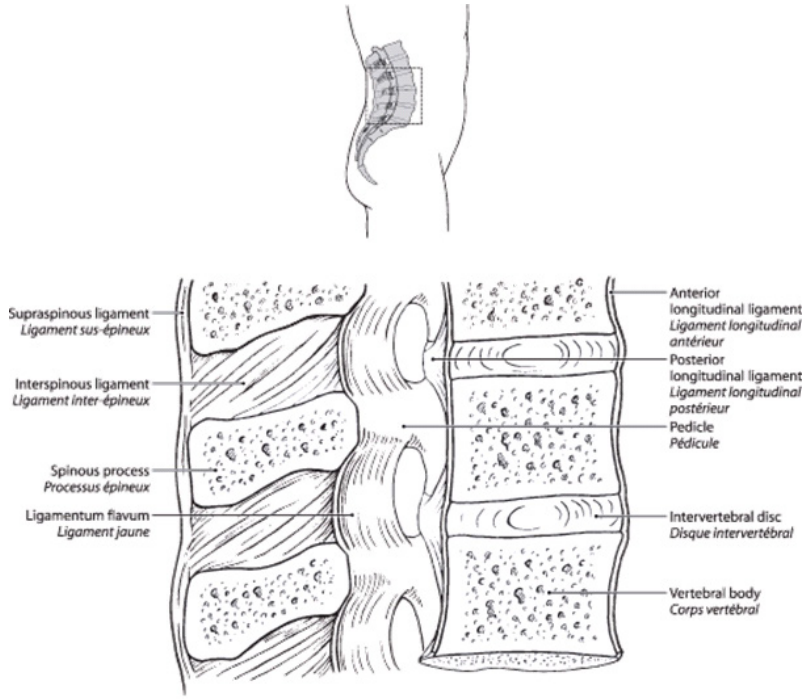


Figure 5. Midline area through vertebral column and principal ligaments. Image illustrated by Liane Friesen

This anteroposterior view of the lumbosacral spine motion segments shows the nerve roots exiting the intervertebral foramina and forming the major nerves to the lower extremity, the Femoral nerve (L3 and 4) and the Sciatic (Mostly L5, S1, some L4).

2.2 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 and rotatory motion.

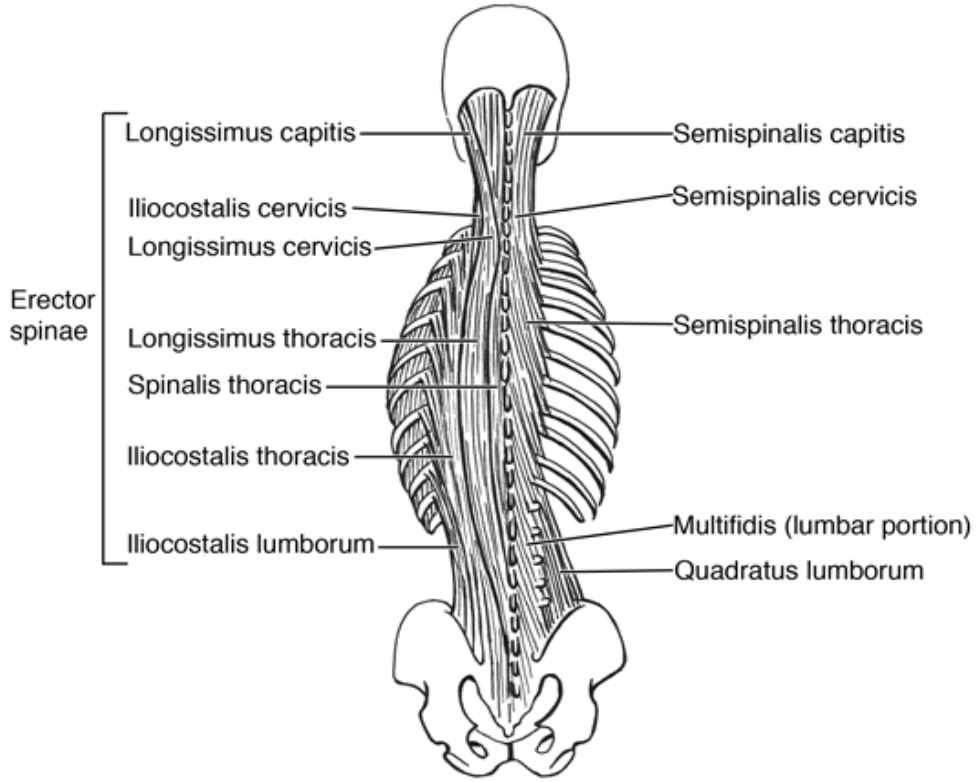


Figure 6. View of the dorsal spine demonstrating paraspinal musculature

3.0 Aging Changes in the Lumbar Spine Motion Segment

3.1. Prevalence

In summary of section 1.2, as noted above, these aging changes are almost universal occurring in 50% of people at age 50 and 80% at age 80. They are most often seen in the lumbar spine at the L4-5 and L5-S1 motion segments, and in the cervical spine at C5-6 and C6-7.

In the general population, patients with age-related degeneration of the intervertebral disc or facet joints may or may not have any symptoms of pain.

The normal aging changes can typically be seen on plain radiographs, CT and MRI and 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 (incidence) 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.

3.2. Aging Changes in the Motion Segment

The age related pathoanatomical changes alter the normal biological and biomechanical environment of the spinal motion segment. These aging (degenerative) changes are an incremental process which occurs gradually over time (see 2.1, 3.1). Degenerative change apparent on imaging shortly after the accident, was not the result of the accident.[10]

Understanding the anatomy of the normal motion segment is the key to understanding these aging changes and are seen in Figures 1-5 above and in Figures 7-8 below.

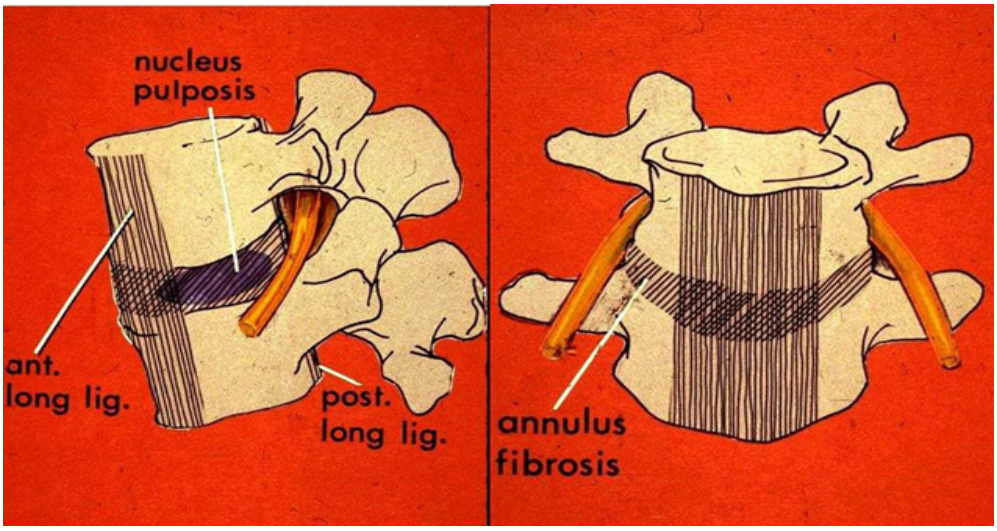
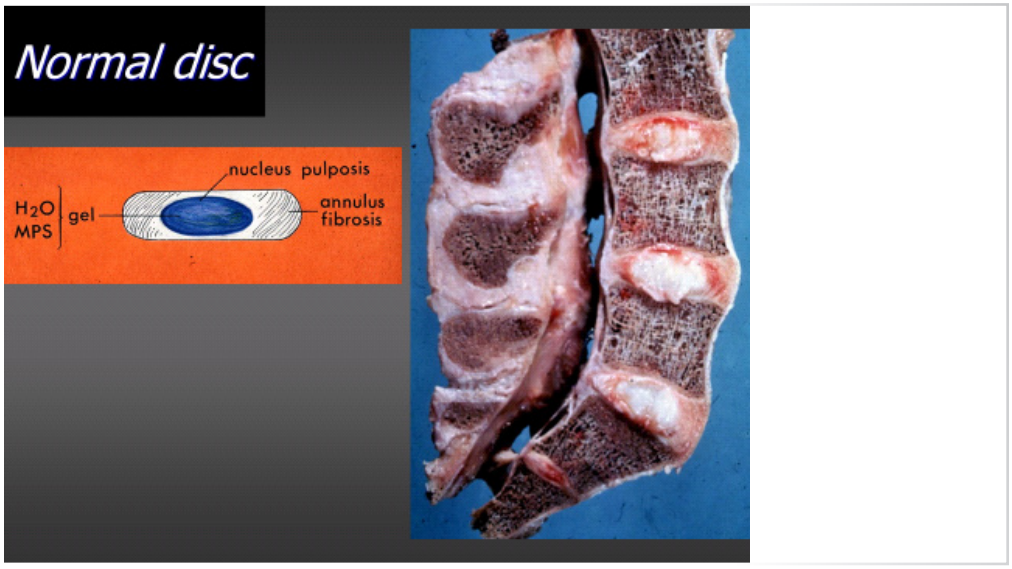


Figure 7. Anatomy of a Normal Spinal Motion Segment, 2 vertebra, posterior arch and facet joints, intervertebral disc, neural elements.



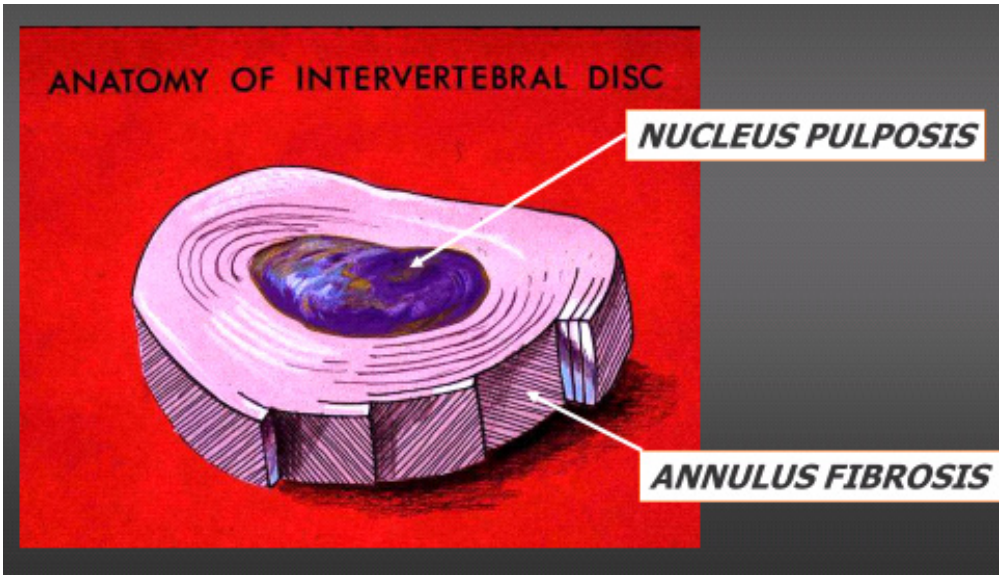


Figure 8. Anatomy of the normal Intervertebral disc showing the properties of the normal nucleus pulposis (above) and the complex anatomy of the annulus fibrosis (below). Proteoglycans are mucopolysaccharides (MPS) bound to protein are commonly found in connective tissues and abundant in the nucleus pulposus (NP) of the intervertebral disc (IVD). Proteoglycans are comprised of glycosaminoglycans (GAGs) which have strong water (H2O) binding abilities. Negatively charged chrondroitin sulphate and keratin sulphate GAG complexes attached to core proteins exert osmotic swelling pressure to draw water to support the hydrostatic properties of the IVD under mechanical loading.

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 in mid to advanced stages on plain x- rays.

There are also changes to the chemical elements in the nucleus and annulus. In general, the collagen content in the nucleus pulposis increase with aging, whereas the Chondroitin sulfate and polyanion may decrease. The notochordal cell population of the disc also decreases as we transition into adulthood and this cell population may be important to disc health which has been an area of translational research focus.

The narrowing of the disc space causes the annulus fibrosis to “bulge” circumferentially in the transverse plane and this can be seen on CT or MR scans; bulging is frequently charted in radiologic investigation reports. The bulging disc may show impingement on the traversing nerve roots crossing the intervertebral disc at that motion segment.

Given the loss of water content and the biochemical changes in the disc, the disc loses its resilience. As a result, microscopic and/or macroscopic tears of the annulus fibrosis also occur with normal activity during the aging process and can be seen on advanced imaging studies.

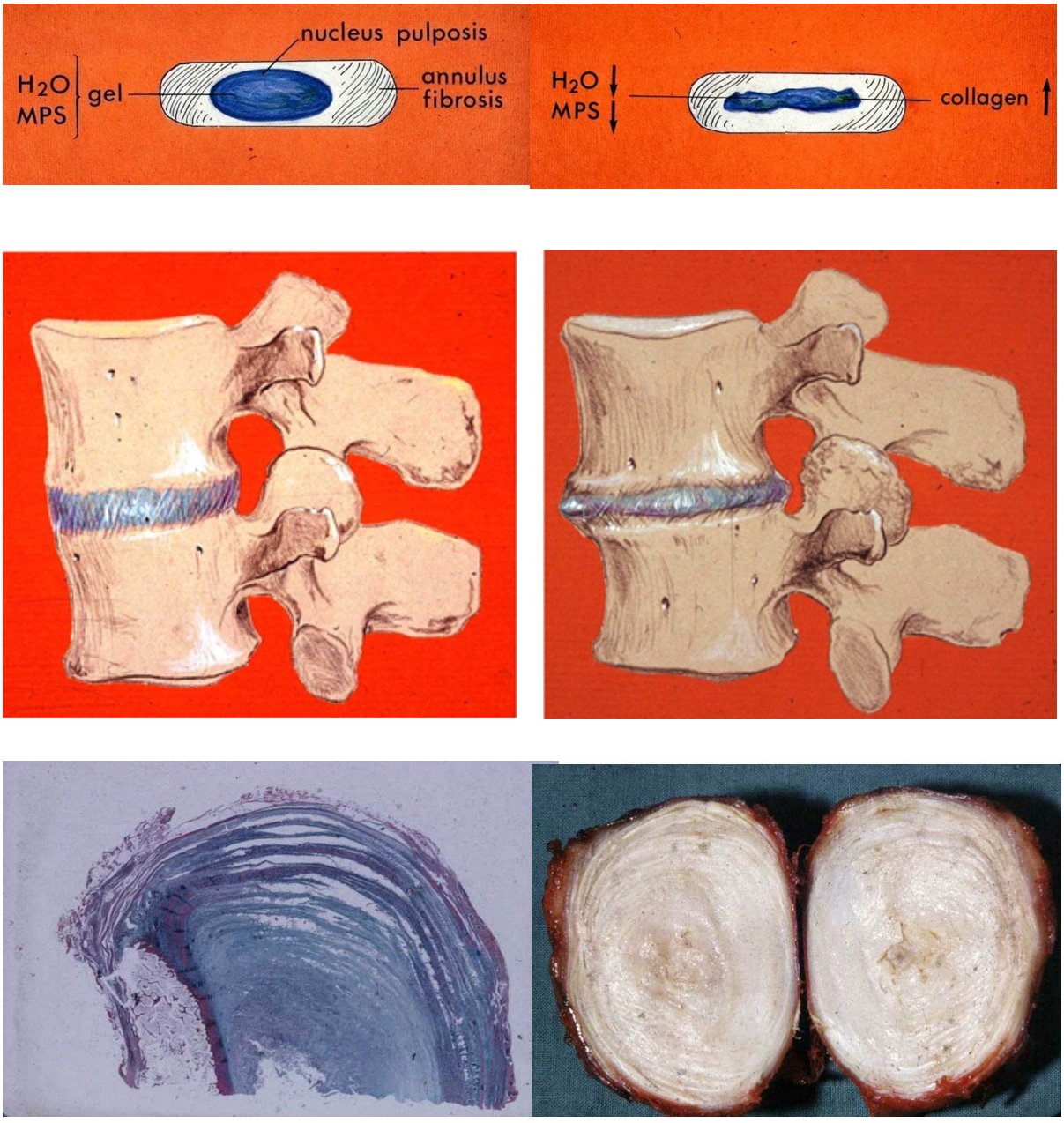


Figure 9. Aging changes in the nucleus pulposis, including loss of water content (wrinkled skin analogy) and change in chemical content (see Figure 8) as well as degenerative changes in the annulus fibrosis, causes loss in intervertebral disc height and facet arthrosis (osteoarthritis) which further leads to narrowing of the intervertebral foramen (i.e. foraminal stenosis) which may impinge upon the existing nerve root at that motion segment

The weakening of the annulus allows the nucleus, now under increased pressure from these changes to take a path of least resistance. With normal activities of bending, lifting and twisting, the hardened nucleus may protrude or bulge through these annular defects. Again, all these changes occur with aging and do not necessarily reflect trauma or work related activity.

Over time, along with disc bulging, aging changes may also result in the formation of a bony out growth, often called a spur or syndesmophyte, at the periphery of the disc.

Aging changes also affects the facet joint which leads to wear and tear changes; a secondary facet osteoarthritis typical of osteoarthritis in any synovial joint in the body.

Just as your finger knuckle joints can enlarge over time due to osteophytes with aging as well as with osteoarthritis, your facet joints also may develop these changes which include loss of articular cartilage and is the formation of spurs (osteophytes) or osteochondrophytes;

To review, the intervertebral disc is comprised of a ‘gelatinous’ inner core surrounded by a thicker outer fibrous annulus structure (not dissimilar 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 fibrosis 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 and even disc protrusions/herniations 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 (extruded with the term sequestration referring to a disc fragment that is no longer in continuity with its native intervertebral disc). The disc or nucleus fragment might be asymptomatic or might compress one or more nerve roots (Figures 10, 11) resulting in a leg dominant radicular pain pattern, and neurological signs and symptoms.

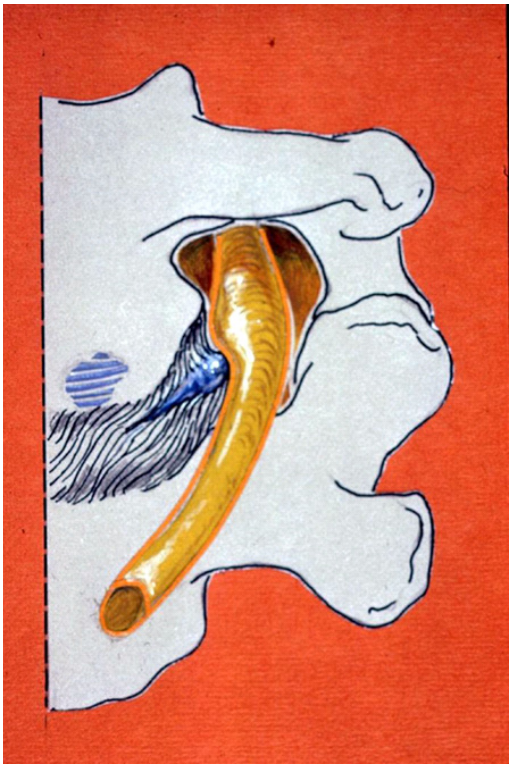


Figure 10. Biomechanical and Biological abnormal motion segment in close proximity to the exiting nerve root in the intervertebral foramen is the result of these aging changes.

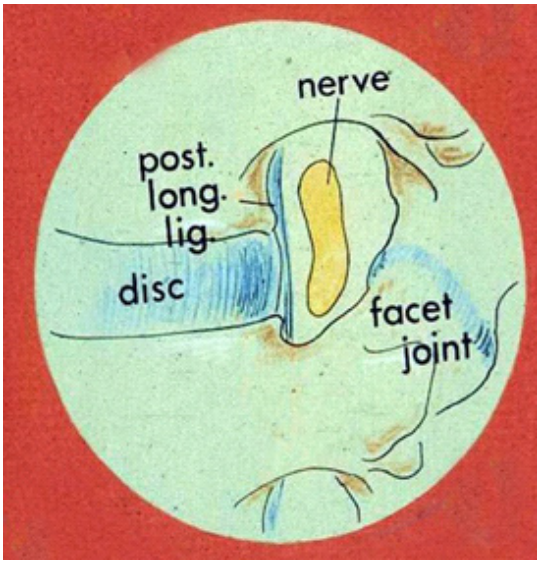


Figure 11a. Intervertebral Foramen
[blue: musculoskeletal; yellow: neurological]

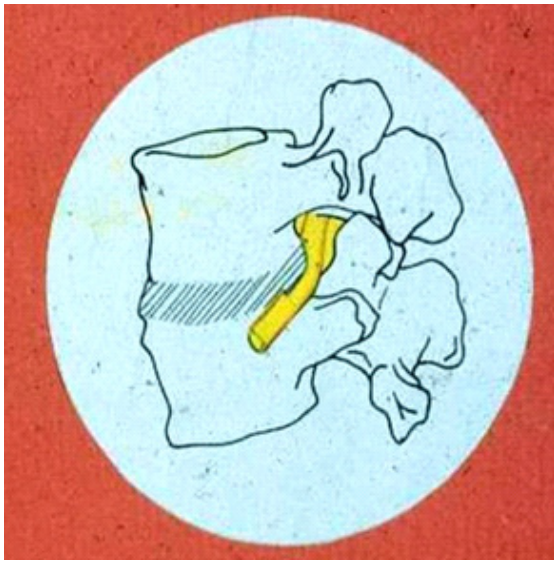


Figure 11b. Nerve root exiting foramen

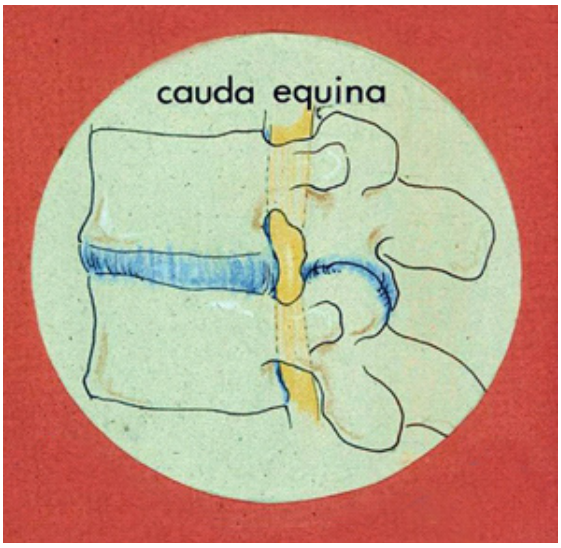


Figure 11c. Lateral view showing musculoskeletal (disc, facet joints) and neurological (cauda equina) site of pain receptors [blue: musculoskeletal, yellow: neurological]

Figure 11. Discogenic pain arising in a Spinal motion segment may arise in the pain receptors located in the musculoskeletal system and/or from the neurological system. Pain receptors in the musculoskeletal system are found in the posterior longitudinal ligament and in the outer fibres of the annulus fibrosis, in the neurologic system, they are related to the nerve root in or beyond the intervertebral canal or to the cauda equina in the spinal canal. The pain may be triggered by trauma, inflammation (e.g. infection, chemical, diabetes).

The majority of patients with such a protrusion or 'disc rupture' get better in a few weeks with eventual resolution of the pain.[11] 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.

In summary, the net effect of these aging changes is alteration to the normal biological and biomechanical environment of the spinal motion segment. These changes occur in close proximity to the nervous system, especially the exiting nerve roots at the intervertebral foramina.

Given the prevalence of these changes as we age; in most patients, medical investigations do not confirm the source of back pain; that can only happen with a careful correlation of the findings with the clinical findings of the patient (see 5.0, 8.0).[12]

3.3 Premature (Juvenile) Degenerative Aging Changes

Although these changes are labelled Aging or Degenerative, in some people they may occur prematurely, even in teens. The reasons this may occur in a very small percentage of the population is not known, but it is well documented. Disc

degeneration progressing to disc protrusion even leading to nerve compression and need for surgical disc removal does occur and is well documented. The changes are usually unrelated to trauma.[13]

3.4. 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 costotransverse and costovertebral 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, it 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 provocation of trunk movements, flexion (anterior bending) or extension (posterior or backward motion).

Thoracic radicular pain occurs, and can be referred from degenerative changes with foraminal stenosis; or occasionally, secondary to thoracic disc herniation. The radicular pain can radiate around the chest wall to a 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 symptoms suggest referred mechanical symptoms; constant symptoms may suggest 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 distally 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 (UMN) changes in the trunk and lower extremities. This clinical presentation is called thoracic myelopathy. Recall that with cauda equina compression (i.e. Lumbar spine below ~ L1-2 level) is associated with lower motor neuron (LMN) findings.

3.5 Summary:

Given the frequency of these age related changes in the lumbar spine, which can be seen on imaging studies in the lumbar spine and in people with or without pain, rational treatment of those individuals with pain can only proceed when a careful clinical assessment matches the changes seen on imaging.[14]

4.0 Where Does Low Back Pain Come From?

Given the almost universal nature of these aging changes in people with and without back pain, where does the pain come from?

The origin of pain requires a knowledge of the location of pain receptors.

In the lower back or lumbar area, these receptors may be found in the musculoskeletal system and the neurological system.

4.1. Musculoskeletal Pain

A. Spinal Motion Segment (Discogenic, facet joints)

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 behind or the groin in front. Occasionally the pain travels down the back or front of the thigh(s) towards the knee, however, pain symptoms are typically more dominantly felt in the low back region (i.e. axial based mechanical low back pain) versus distally in the legs (see also section below on neurogenic pain regarding leg dominant pain symptoms).

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); 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 to guide active forms of treatment.

It is also important to realize that not all individuals may describe ‘directional preference” in their presenting condition.

B. Back Strain (ligament, muscle, fascia)

Back strain is an overused diagnosis and may be inaccurately used. It is used so frequently in reporting back injury, that it has lost any practical meaning in true diagnosis of the pain source.

Soft tissue injury does occur, and the injured soft tissues become painful.

The injury may be a direct blow to the back causing contusion and bruising (ecchymosis), even hemorrhage into the muscle (hematoma).

Some lifting and twisting injuries, especially in younger people may have a true muscle strain caused by a stretch to the muscle fibres or a tear from the fascia.

The reported symptoms are back dominant. There may be associated muscular spasm and adaptive deformity that is observed on physical examination, with or without muscular tenderness over the spinal muscles. Most heal in a few days or at most a few weeks and are not likely to be a source of continuing pain.

4.2. Neurogenic Pain

A. Radicular; Nerve Irritation and/or Compression.

Another type of pain is that caused by compression of a spinal nerve root, called radicular pain. 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. This is sometimes called “referred pain”, and this distinction between radiating (i.e. radicular) leg dominant pain versus low back dominant (i.e. axial pain) helps clinicians differentiate whether there is a significant neurogenic pain component due to structural nerve root compression. If present, there may be associated signs of nerve root conduction loss (power, reflex, or sensor change), or nerve root irritation (i.e. positive straight leg raise test).

What can be challenging, on occasions, is the concurrent presence of axial back with radicular leg pain symptoms. An individual, for example, with a symptomatic lumbar disc herniation at L4-5 or L5-S1 may present with axial back pain as well as referred pain into the buttock and radiating distally into the leg below the knee towards the foot. The leg and distal extremity referred radiation is radicular or neuropathic in nature, however the referred pain felt in the buttock (versus the axial low back) can either be referred axial back pain and/or it could be part of the radicular neuropathic nerve root pain that overlaps the buttock region into the leg.

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).

If the Straight Leg Raising or Femoral stretch test reproduces a patient's typical leg dominant pain, it is considered a positive test; that is, the pain is very likely of neurogenic origin. However if those tests reproduce only back pain with this maneuver, the test is considered a negative test; that is, the pain is unlikely to be of neurogenic origin.

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 inflammatory irritation between the fragment and nerve root – this results in neurogenic pain.

Radicular pain may be caused by a herniated intervertebral disc (protrusion, extrusion, sequestrated), by nerve compression in spondylolisthesis, and by a combination of boney osteophytes arising from the vertebral body and/or the arthritic facet joint and soft tissue disc material or annular tears in the intervertebral foramen; so called root (or foraminal) stenosis (see Figures 11, 14).

B. Neurogenic Claudication: 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 70 and in some individuals as early as the 20-30s.[3]

It is important to recognize 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, namely neurogenic claudication (i.e. activity related leg pain resulting from neurologic compression such as in spinal stenosis).

Neurogenic claudication is the distinguishing feature 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 require careful clinical correlation to symptoms that may be present.

5.0 Pain Syndromes Arising from the Lower Back Motion Segment (Discogenic)

Low back pain may arise without injury, or with relatively minor trauma, such as bending over and tying one's shoe laces. People may present to a health professional with acute low back pain which we can arbitrarily define as pain arising acutely and lasting no more than 8-12 weeks before resolution; or more chronic pain which may be recurrent or chronic unremitting pain.

This pain is often referred to in medical notes as mechanical back pain, meaning pain with movement of the spine. It is a grab bag term, and has no meaning with respect to causation. Pain with movement may occur with discogenic pain, but also muscular strain and other causes.

Acute back pain arising from a lumbar motion segment (discogenic) may arise spontaneously without trauma or may arise following injury. The resultant pain may be back pain only, back pain associated with referred pain into the proximal lower extremities, or neurogenic (radicular) lower extremity pain.

There are some situations where pain without injury may cause symptoms: 1) a disc problem (bulge, protrusion, herniation, sequestration); 2) spinal stenosis (narrowing of the spinal canal); and 3) facet joint arthritis.[15, 16, 17]

5.1. Acute Low Back Pain; Spontaneous Onset; the Role of Inflammation, Enthesopathy

Spontaneous onset pain without injury is common throughout the musculoskeletal (MSK) system. This occurs where tendons, ligaments or fascia attach to bone in the region of a joint and they are collectively called enthesopathies or tendon into bone syndromes. Pathological inflammation (enthesitis) as in conditions such as ankylosing spondylitis, psoriatic arthritis, and rheumatoid arthritis can also occur with the umbrella term of enthesopathy.

Common examples of enthesopathy include shoulder pain (rotator cuff,) elbow pain (tennis elbow), hip pain (trochanteric tendinitis), heel pain (Achilles tendonitis), foot pain (plantar fasciitis) and in all other anatomical areas where tendon, ligament or fascia attach to bone (e.g. wrist, hand, etc.).

This is also true in acute low back pain; most patients who seek help from health care providers for low back pain have no history of injury. As in most enthesopathies, the pain arises in regions of aging or degenerative changes, or as we have described previously, in a biological and biomechanical abnormal motion segment (see Figures 10,11).

5.2. Acute Low Back Pain; Associated with a Traumatic Event

A. Can Injury or Vocation Influence the Aging Process; Causation vs Aggravation?

i) Causation:

Given that degenerative change is so pervasive in our society, it is unlikely that one's occupation causes early disc degeneration. There are few credible scientific papers supporting that theory (see Andersson, Epidemiology, chapter 1.2, D).

These aging (degenerative) changes are an incremental structural process which occurs gradually over time and may or may not be symptomatic. Degenerative change apparent on imaging studies such as x-ray, CT or MRI shortly after the accident, was not the result of the accident.

ii) Aggravation:

Age related degenerative change results in a biomechanical and biological abnormal motion segment in close proximity to the exiting nerve root in the intervertebral foramen.

Although the individuals with these changes do not necessarily have any symptoms, they are at risk of becoming symptomatic often with no injury. On occasion, individuals with pre-existing degenerative changes may be vulnerable to symptoms arising from work injury – for example, the development of lumbar radiculopathy following injury in a situation where there was significant pre-existing degenerative changes that had already resulted in nerve root compression. This would be considered an aggravation with symptoms arising from a pre-existing condition with structural changes that were previously asymptomatic or minimally symptomatic. Such symptoms (eg. lumbar radiculopathy in the example provided) would typically arise within a short period of time (ie. within days up to ~1-2 weeks). Such injury may arise by repetitive bending and twisting movements, usually in the flexed position and/or associated with heavy lifting.

Some occupations, such as construction work and other manual employment, e.g. garbage collectors, warehouse workers, nurses, Personal Support Workers (PSWs) which involve repetitive bending, heavy lifting and twisting, may put the workers at risk as they age, thereby acting as aggravating factors for this pre-existing condition. Sitting for long periods may also contribute to symptoms of low back pain.[19]

B. Biomechanics. Type of Forces Associated with Injury to the Lumbar Spine

As just noted above in A.ii) most patients presenting with low back pain have either had no injury, or relatively low energy injury from rotation of the trunk in flexion, often lifting an object.

Compression (i.e. a slip and fall from standing height versus a fall from height) and directional forces (axial loading, flexion-distraction, shear/rotational) along with patient factors (i.e. bone density) determine the severity of injury to soft tissue or bony structures in the spine.

In biomechanical testing and in clinical events, injury to the intervertebral disc complex is rare in a normal motion segment but may occur in a biologically and biomechanically abnormal motion segment secondary to aging changes (see Figures 10,11,12). Injuries can be purely soft tissue, bony, or both.

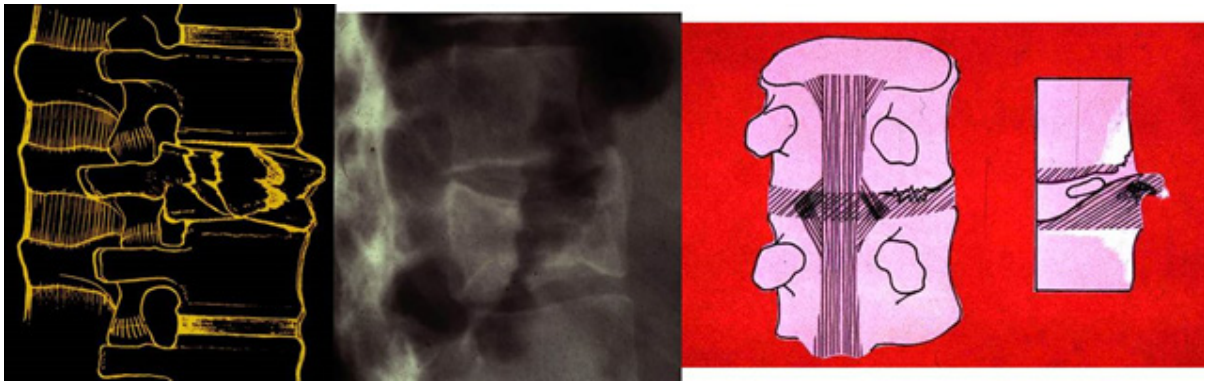


Figure 12a. **Figure 12b.**

Figure 12a. Compression force causes a burst fracture, or with flexion, a compression fracture with low or high energy forces. **Figure 12b.** With low energy angular or rotational forces, injury to the intervertebral disc can only occur in a biologically and biomechanically abnormal motion segment (see Figure 10).

a. Low energy forces. The low energy forces which cause the majority of discogenic low back pain are usually of a twisting, rotation nature, often associated with lifting with the trunk in the flexed position.

Therefore, with l*ow energy forces*, injury to the intervertebral disc in a motion segment with normal bone density, only occurs in a so called degenerative disc, that is, a motion segment with significant aging changes as described above. Biology can never be framed in absolute terms, but any exceptions to that statement would be rare.

As noted in 3.3, this is also true in teenagers; when premature aging changes are noted in juvenile disc degeneration.

In patients with significant vertebral osteoporosis, these forces may cause compression fractures in the lumbar spine with little or no trauma, and is a source of back pain, especially in the elderly female population (see 6.2).

b. High energy forces. However, with high energy forces as occur in high speed motor vehicle accidents or falls from a height, injury to the intervertebral disc does occur. These are often distraction or shearing injuries as seen in lap belt injuries. The body is thrown forward, the lap belt holds and a mid to upper lumbar disc may tear at the annulus vertebral junction. This force may also cause intraabdominal bleeding from rupture of bowel, liver or spleen. These injuries are caused by external forces which may be massive.

The universal introduction of shoulder straps into all new vehicles has greatly reduced these injuries (see Figure 13a).

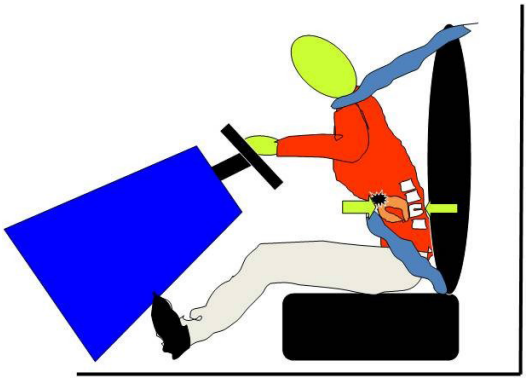


Figure 13a.

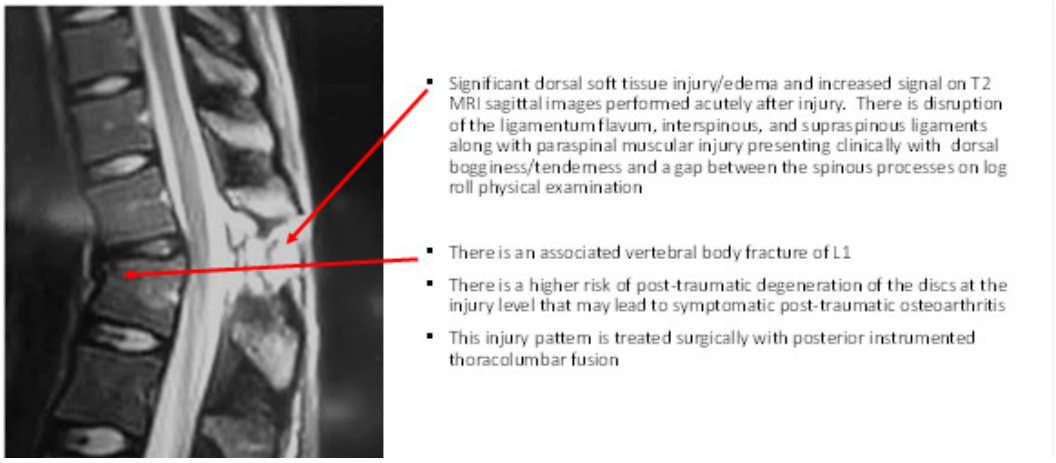
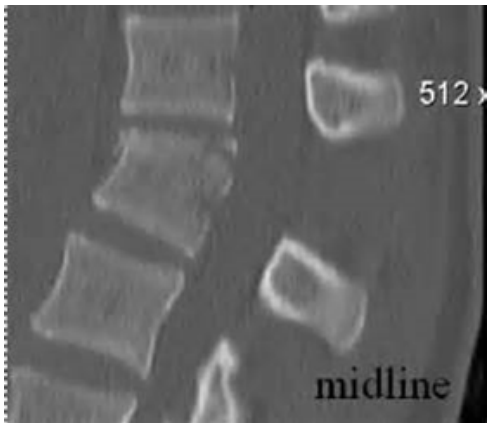


Figure 13b.



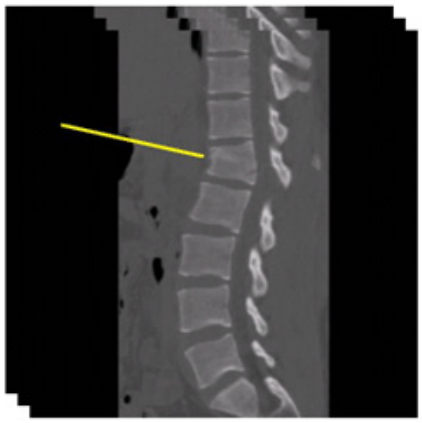


Figure 13c. **Figure 13d.**

Figure 13b. High energy flexion-distraction injury to the T12-L1, thoracolumbar spine causing a posterior soft tissue disruption as noted. As well, as seen in **13c.** and **13d.**, the L1 vertebra sustained a compression fracture as well as an injury to the T12-L1 intervertebral disc.

C. How Does Injury Aggravate or Accelerate Symptoms in People with Age-Related Changes?

In pain arising from an injury to an abnormal motion segment, either work related or not, the forces are usually low energy, even relatively minor, and usually involve flexion and rotation (twist). Common scenarios include lifting and rotating, often with the legs extended. Even the motion of bending over to tie one's shoe laces may have the same effect.

Since all epidemiologic studies have shown the prevalence of the abnormal degenerative disc at 50% of the population at age 50, many of these injuries occur in the 40-60 age groups, and are not gender specific.

In non-work related instances, the injuries are often sports related, such as hitting a golf ball or related to activities of daily living around the house involving lifting and rotation.

Work related injuries are also usually minor compared to high energy motor vehicle crashes or falls from heights and often involve lifting and rotation.

Common examples of such injuries include the following:

- Construction worker lifting a heavy board from the ground in the flexed position and twisting to install it
- Landscape worker planting a tree; pain occurs as the worker lifts the root ball, twists to put the ball into the prepared hole in the ground

- Nurse or Personal Support Worker (PSW), lifting a heavy patient from bed while in a flexed position and twisting
- Bending and pulling to put on a work boot.

Since the motion segment is biologically and biomechanically abnormal in this situation, minor trauma usually involving flexion and rotation with or without a lift, may further injure the disc and/or spinal soft tissues and cause pain (see 3.2, and Figures 10, 11b.).

The injury to the preexisting degenerate intervertebral disc may be a further tear to that already present in the annulus fibrosis. It may cause the nucleus pulposis to herniate through the annulus fibrosis occasionally leading to nerve root irritation and/or compression.

This injury is often called by the public a “slipped disc”, more formally, a “herniated intervertebral disc” (see 5.3 below).

This pain is often referred to in medical notes as mechanical back pain, meaning pain with movement of the spine. It is a grab bag term, and has no meaning with respect to causation. Pain with movement may occur with discogenic pain, but also muscular strain and other causes.

D. Timing of the symptoms.

Most patients with an injury to the disc complex will have immediate pain at the time. The pain may be minor, often shrugged off, but may often become worse, especially in the first 24-48 hours after injury.

If the pain is noted at a later time, for example 2-4 weeks later, only a careful history of all the facts, especially documentation, will determine any relationship of pain to alleged injury. Also important would be a review of a detailed physical examination and the results of any investigations that may have been done and recorded. The pain may be low back dominant if there is no concomitant nerve root irritation or it may be associated with referred radicular leg (neurogenic) pain in some cases where there is nerve root compression by discal changes. The neurogenic leg pain component can follow the onset of low back pain from injury, and if related to injury the neuropathic component would typically arise within 2-4 weeks from injury if not already present from the immediate time of injury. Most patients’ symptoms with a disc injury following trauma will resolve over a period of several weeks and by three months at least 90 percent of patients will be significantly better (see 1.2, Epidemiology).

E. In Summary,

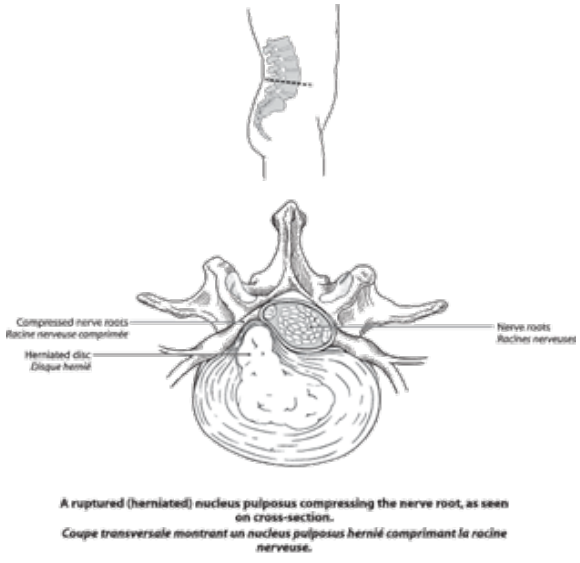
Injury to a pre-existing abnormal motion segment (degenerative) caused by the aging process may occur in a work related activity. The sequela of such an injury are well described in the spine literature, and are irrespective of non-work related or work related factors. As noted, these injuries are usually caused by low energy forces.

Adjudicators involved in these appeals have to look carefully at each case. Prognostic factors include whether this is a first episode, a recurrent episode, or a chronic ongoing occurrence. The timing and history of symptoms, physical examination findings, and any correlative imaging results guide medical opinion.

5.3. Herniated Intervertebral Disc.

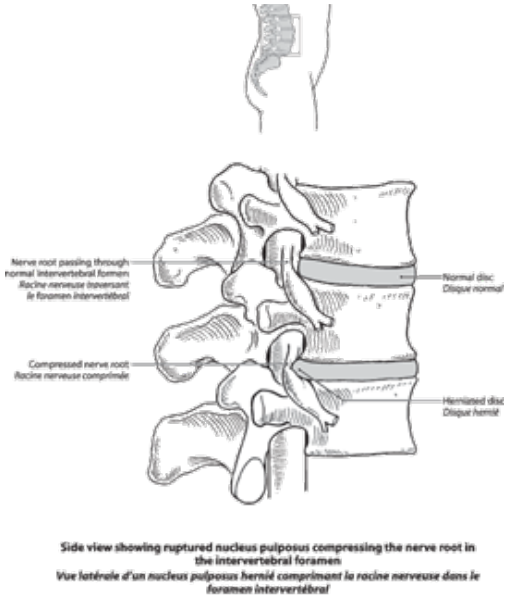
A. Pathoanatomy

Prior to the classic paper by Mixter and Barr,[19] most cases of acute low back pain were thought to arise in the sacroiliac joints, or in the ligaments and muscles of the lower back. Sporadic reports in the literature referred to the small fragment in the spinal and nerve canal as enchondromas or other benign tumours; we now know they were referring to lumbar disc herniations (Figure 14).



A ruptured (herniated) nucleus pulposus compressing the nerve root, as seen on cross-section.
Coupe transversale montrant un nucleus pulposus hernié comprimant la racine nerveuse.

Figure 14 a.



Side view showing ruptured nucleus pulposus compressing the nerve root in the intervertebral foramen
Vue latérale d'un nucleus pulposus hernié comprimant la racine nerveuse dans le foramen intervertébral

Figure 14b.

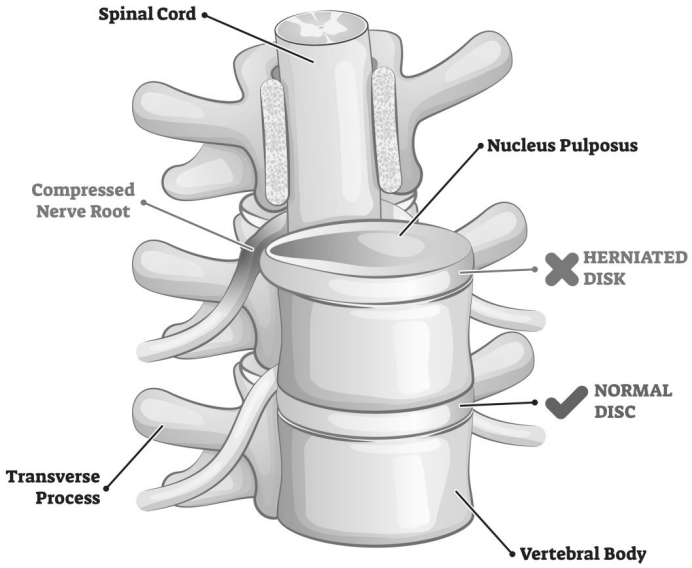


Figure 14c. Frontal view, showing a disc herniation in proximity to the nerve root. Image courtesy of VectorMine: Shutterstock.com

As we have stated above, we now know that the majority of cases of episodic acute low back pain are discogenic and usually arise with no or minimal trauma.

Lumbar disc herniations form a continuum, hence many different names are used to describe them (Figure 15).

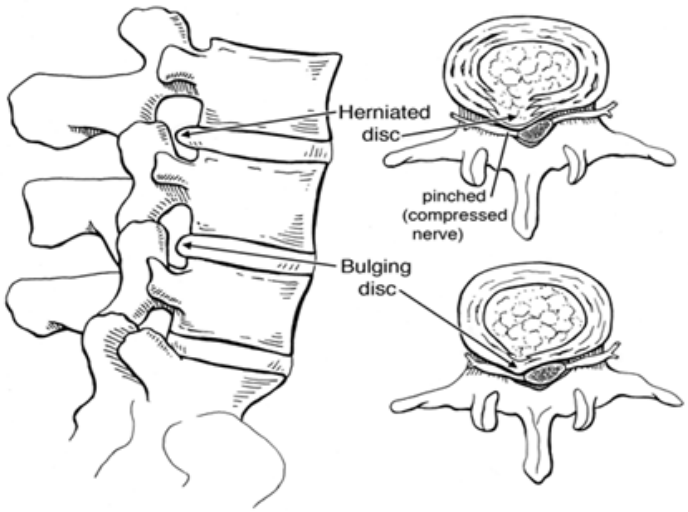


Figure 15a. Diagram showing a disc bulge (i.e. 'generalized' and more broad based as depicted on the lower right image) that does not compress neural elements as compared to a 'focal' herniated disc (top right image) that tend to compress neural elements and may cause symptoms of radiating leg pain, numbness and/or weakness.

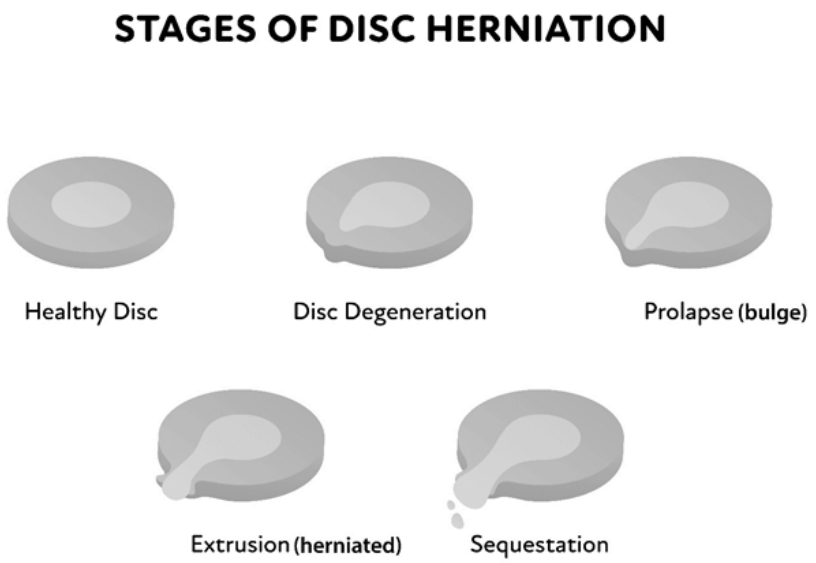


Figure 15b. The stages or continuum of disc herniation. The term prolapsed or bulging disc is used when the outer fibres of the annulus remain intact, the nucleus may bulge but is retained within the annulus.

The term bulging (prolapsed) disc is used when the outer fibres of the annulus remain intact, the nucleus and annulus may bulge into the spinal canal but the nucleus is retained within the annulus.

If the nucleus pulposis extends through the torn fibres of the annulus, we use the term herniated (extruded) disc. The disc has 'ruptured or slipped', and there is a defect in a portion of the annulus fibrosis that has allowed nucleus pulposus extrusion. In this situation, the nuclear material remains in continuity with the remainder of the intervertebral disc space.

If the nuclear material herniates and also separates completely from its native intervertebral disc and is lying free within the spinal canal or nerve root region, we call that a sequestration (the fragment, a sequestered disc herniation).

All the changes noted in the disc (bulge, extruded herniation, or sequestrated herniation) can be seen on spine CT or MRI and can also been observed in patients with no symptoms (Figure 15, see also Figures 29 and 30).

B. Stages of Disc Herniation

If the nucleus pulposis extends through the torn fibres of the annulus, we use the term herniated or extruded disc. The disc has 'ruptured or slipped', and there is a defect in a portion of the annulus fibrosis that has allowed nucleus pulposus extrusion. In this situation, the nuclear material remains in continuity with the remainder of the intervertebral disc space.

If the nuclear material herniates and also separates completely from its native intervertebral disc and is lying free within the spinal canal or nerve root region, we call that a sequestration (the fragment, a sequestered disc herniation) (Figure 15c).

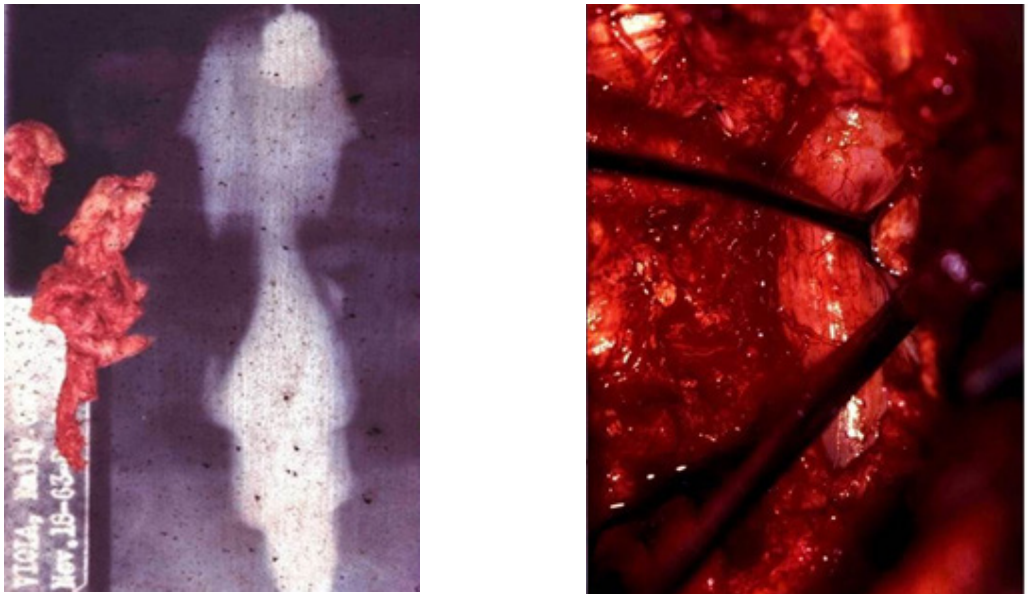


Figure 15c. Nerve root compression; caused by the nucleus pulposis extruded into the spinal canal, called a sequestered disc herniation.(bottom left shows myelogram and fragment removed at surgery, bottom right intraoperative photo showing the disc fragment compressing the neural tissue)

C. Clinical Symptoms, Neurogenic Pain

Lumbar disc herniation may become symptomatic involving the neural elements (neurogenic, or neuropathic pain, (see 4.2)); the result may be nerve root irritation and/or nerve root compression (Figure 16).

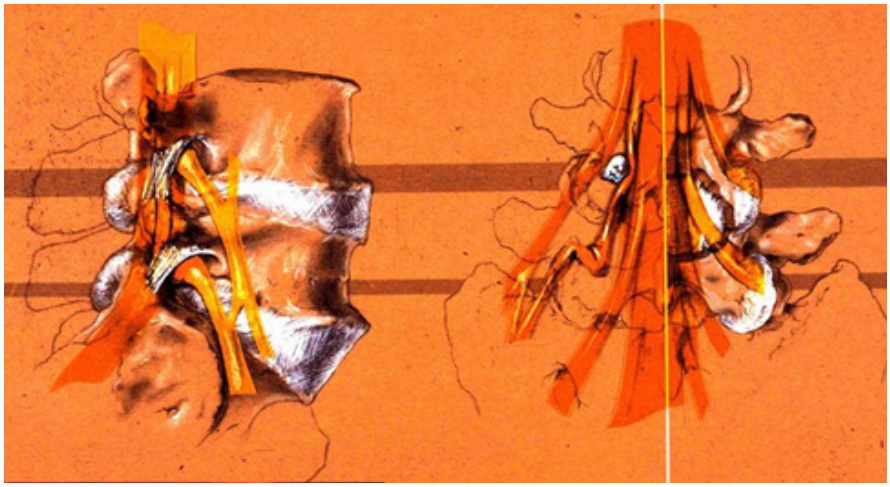


Figure 16a.

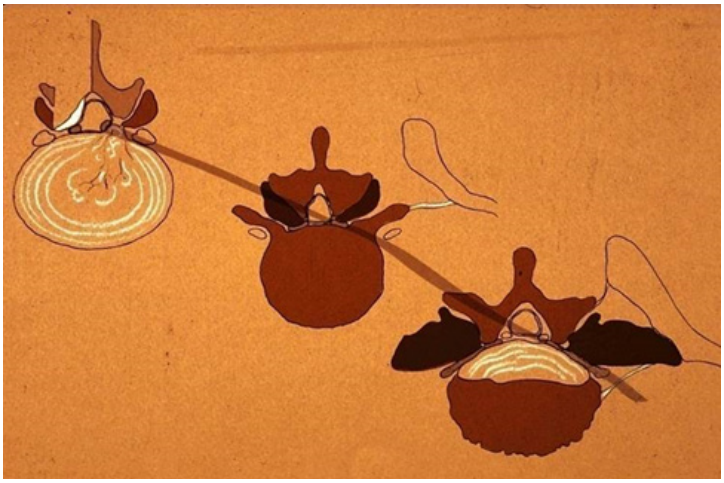


Figure 16b.

Figure 16. Nerve compression may occur anywhere along the path of the nerve root, within the spinal canal, within the root canal, or outside the canal. **16a.** Lateral view on left showing nerve roots exiting the intervertebral foramen; posterior view on the right showing the right L5 nerve root with compression by a disc herniation, (arrow); on the opposite side, a small laminectomy has exposed the compressed nerve, the disc herniation was removed, the L5 root decompressed allowing the root to return to normal, thereby relieving pain and nerve dysfunction in most cases (arrow). **16b.** Cross sectional views of the L5 nerve root compressed by a herniated disc (left), by the facet joint and osteophytes in the root canal, (centre) and outside the canal at S1(right).

Nerve root irritation if often seen in the acute phases of a lumbar disc herniation causing nerve root compression. Nerve root compression simply refers to the structural discal-vertebral changes that cause physical compression of the nerve(s). The presence of and extent of nerve root irritative symptoms or related physical signs depends on the acuity or chronicity of symptoms and also the degree of structural nerve compression.

a. Nerve root irritation: is characterized by referred pain along the distribution of the affected nerve, often associated with nerve root irritative physical signs (see straight leg raise, SLR below). Pain distribution along affected nerves include:

- L3-4, (4th nerve root, femoral nerve), the anterior thigh/shin,
- L4-5 the lateral calf to the big toe (5th nerve root, Sciatic nerve),
- L5-S1, (S1 nerve root, Sciatic nerve branch), the posterolateral calf to the heel and 5th toe.

These 3 nerve roots L4, L5, and S1 account for most of the cases of low back symptoms associated with nerve involvement as these are the most common vertebral levels for aging degenerative lumbar spine changes (L5-50%, S1-45%, L4-5%).

For the upper lumbar nerve roots, extending the hip with the knee flexed may cause severe anterior thigh pain (i.e. positive Femoral nerve stretch test); whereas patients with sciatic nerve pain have restriction of straight leg raising (SLR) because of pain – symptomatic leg elevation in either the supine or sitting position is limited and reproduces the individuals reported radiating neurogenic leg pain.

Many do not progress beyond the irritation phase, but ultimately neurological abnormalities may occur.

The nerve syndromes may develop slowly, or very rapidly, within a few hours.

b. Nerve root compression: is characterized by sensory, motor, and reflex changes (common nerve roots L4,L5,S1 as noted below); these changes are present on a careful neurological examination, and are diagnostic when the clinical findings match the imaging studies (CT, MRI) (Figure 16).

- L4; a history of anterior thigh pain and/or numbness that may radiate into the anterior shin below the knee, and the neurological examination showing anterior thigh/shin numbness, a weak quadriceps muscle (knee extension), and an absent or diminished knee reflex.
- L5, a history of posterior thigh and lateral calf pain that may radiate into the dorsal foot and big toe and/or numbness, and the neurological examination showing numbness on the dorsum of the foot to the big toe, weakness in the toe extensors to a complete inability to raise (i.e. dorsiflex) the foot at the ankle (eg. a drop foot), no reflex change.
- S1, a history of posterior thigh pain, lateral/posterior calf pain to the heel and 5th toe/sole of foot area and/or numbness, and the neurological examination showing numbness along the lateral calf and heel to the little toe/sole of foot, weakness in the calf muscles (inability to raise one's body weight standing on the tip toes), and a diminished or absent ankle reflex.

Often, the pain may lessen or even disappear without surgical removal of the disc herniation; the neurological abnormalities may improve, or not, usually the absent reflexes do not return.

Continuing radicular pain with nerve deficit and a large compressive disc herniation on MRI matching the clinical finding is the usual indication for surgical disc removal. This surgery is now done with minimally invasive techniques as an outpatient procedure with excellent results for pain relief and improvement of the neurological picture (see 9.2).

Saddle anal numbness (i.e. skin area overlying the saddle seating region around the anus) and/or loss of bowel and bladder function, the so-called cauda equina

syndrome requires urgent clinical and imaging assessment and possibly emergent spinal decompression.

6.0 Chronic Pain Syndromes:

6.1. Recurrent (Episodic) Discogenic Pain

Although this topic is not truly chronic LBP and therefore does not belong in this section, we have included it here to remind readers that Recurrent or episodic LBP does occur and is relatively common. In the literature, one may find various attempts to measure the frequency. In the Rowe study, the workers had one or two episodes of episodic acute pain throughout their working life; each attack was usually very similar to the first, as described in the previous chapter. This includes the demographics, the causation with or usually without significant trauma, and the usual favourable outcome. A small percentage, as in the primary episode will progress and develop neurological dysfunction, and some of those patients will require surgical disc removal

6.2. Facet Joint Syndromes:

The facet joints are small synovial joints located posteriorly at each spinal motion segment, as described in the anatomy chapter above. All synovial joints are susceptible to various forms of arthritis, the commonest, osteoarthritis, but also inflammatory arthritis. The facet joints are not exempted from the aging process affecting the spinal motion segments. As the disc narrows as a result of these changes, it no longer functions as the shock absorber of the segment. This puts abnormal biomechanical forces on the facet joints and they develop secondary osteoarthritis; loss of articular cartilage, spurs (osteophytes). (Figures 17a.,17b.)

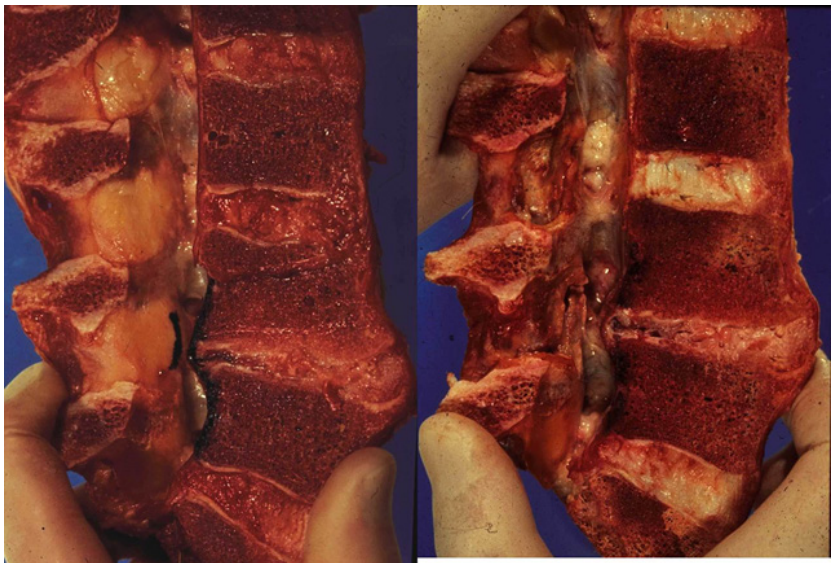


Figure 17a.

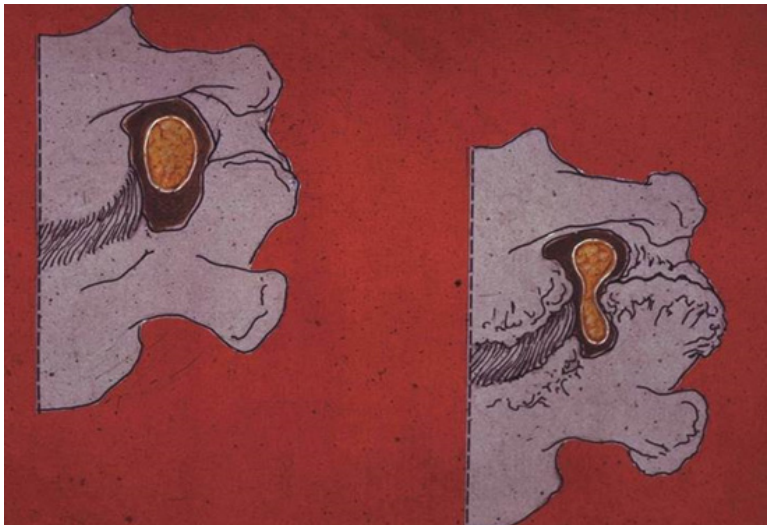


Figure 17b.

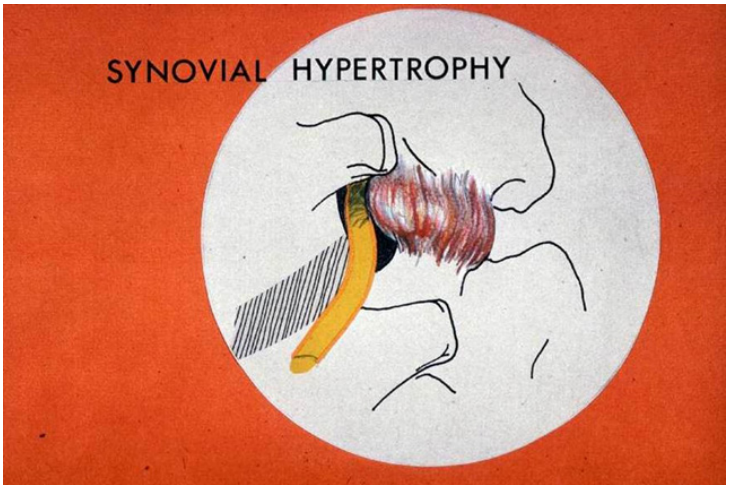


Figure 17c. Inflammation in the joint (synovitis) as a cause of pain

Figure 17. Facet joint pain caused by arthritic changes in the joint and inflammation. In Figure 17a., note the cadaver dissection showing the severe degenerative disc at L4-5, clearly different than the normal disc above at L3-4; on the left the ligamentum flavum (yellow ligament) covering the facet joint, on the right, the ligament removed showing a severe arthritic facet joint. Figure 17b. shows the narrowing of the root canal by osteophytes from the joint an vertebral margin with impingement of the nerve root.

The incidence is as noted, ~50% at age 50, and ~80% at age 80, with almost all occurring at the clinically important levels of L4-5, and L5-S1.This arthritic picture can be seen on imaging studies, x-ray, CT, MRI, and are part of the disc aging syndrome.

The patients may be asymptomatic, suffer no pain or other disability. In patients with pain, the clinical assessment, history and physical examination, must correlate

with the imaging studies to be meaningful. When chronic pain does develop, it is usually episodic and may be associated with secondary synovitis in the facet joint (Figure 17c).

The pain is usually lower back pain but radiation to the buttocks and posterior thighs is common. Wearing high heels appears to alter lumbar lordosis and erector spinae activity. Direct causality with back pain is difficult to prove given the prevalence of back pain in the general population with some reports describing a potential association. One pain pattern described includes directional preference where extension causes increased pain, which may relate to osteoarthritic facet pain syndrome; this is usually treated non-operatively, lifestyle changes, joint injection, nerve ablation to joint, medication, and therapy (exercise).

Another effect of this facet arthritis results from the increasing size of the osteophytes causing narrowing of either the intervertebral canal and/or the spinal canal. The spurs may cause impingement of the neural elements, in the intervertebral canal, the nerve root, causing radiculopathy, in the spinal canal, the cauda equina causing spinal stenosis. Occasionally synovial cysts can arise directly from the facet joint and if located in the subarticular region of the joint within the lateral recess of the spine, it can cause symptomatic neurologic compression. The sequelae of these neurological syndromes are discussed in other sections.

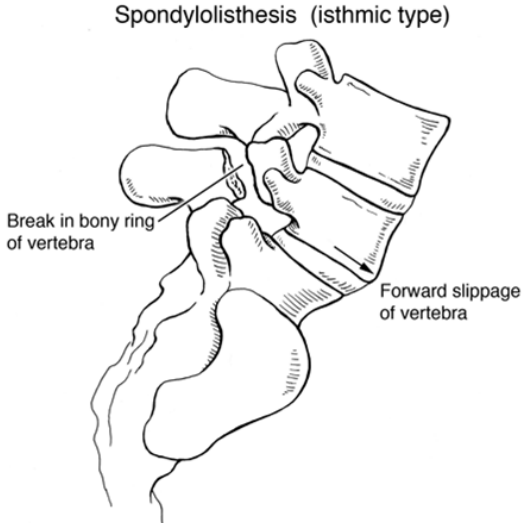


Figure 18. Isthmic Spondylolisthesis. A ‘break in the bony ring of vertebrae’ (i.e. spondylolysis) may cause a ‘forward slippage’ (i.e., spondylolisthesis) of lumbar vertebra L5 on top of the sacrum (S1). This type of spondylolisthesis is known as ‘isthmic spondylolisthesis’.

6.3. Spondylolysis and Spondylolisthesis

A. Spondylolysis

In spondylolysis (Figure 19), the pars Interarticularis has a defect or gap, where the bone is replaced by fibrous tissue (gristle). As the gristle is not calcified, it appears as a defect in the x- ray. This is called spondylolysis. Spondylolysis occurs most commonly in the 4th and 5th lumbar vertebrae, and can be unilateral.

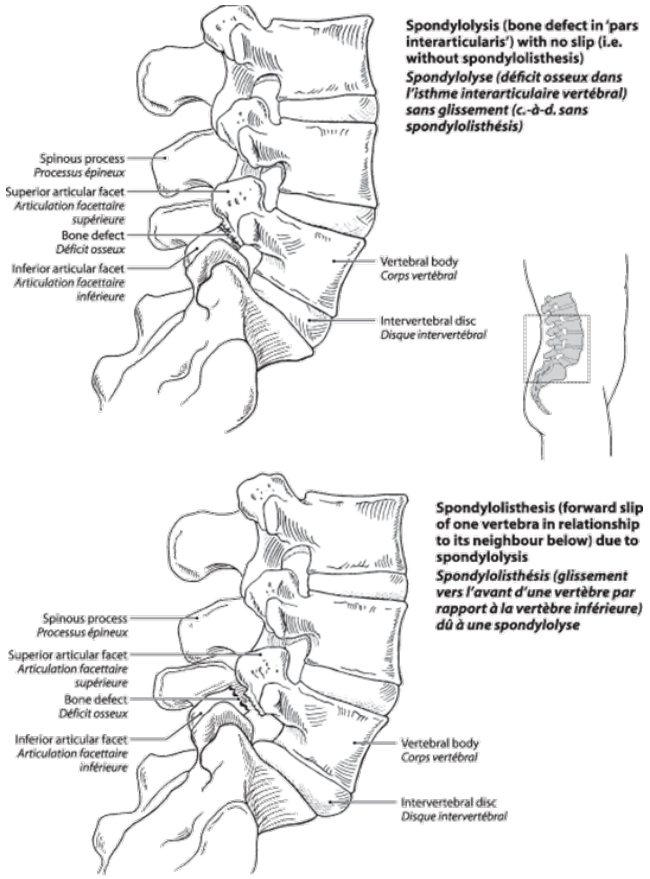


Figure 19. Top image shows Spondylolysis (bone defect in 'pars Interarticularis') with no slip (i.e. without spondylolisthesis); Bottom image shows Spondylolisthesis with forward slip of one vertebra in relationship to its neighbour below with bone defect in the pars Interarticularis.

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 18,19). This slip is called a "lytic", isthmic, or spondylolytic spondylolisthesis.

B.Spondylolisthesis

In common usage, spondylolisthesis, sometimes described as anterolisthesis, refers to a forward slip of the upper vertebra on the lower. There are several types.

Most common are “lytic” spondylolisthesis (Figure 18, 19b), the result of a bony defect in the pars Interarticularis of the vertebra. There is also a ‘congenital’ form of spondylolisthesis. Degenerative spondylolisthesis (Figure 20) is common secondary to age related changes at the L4-5 motion segment.

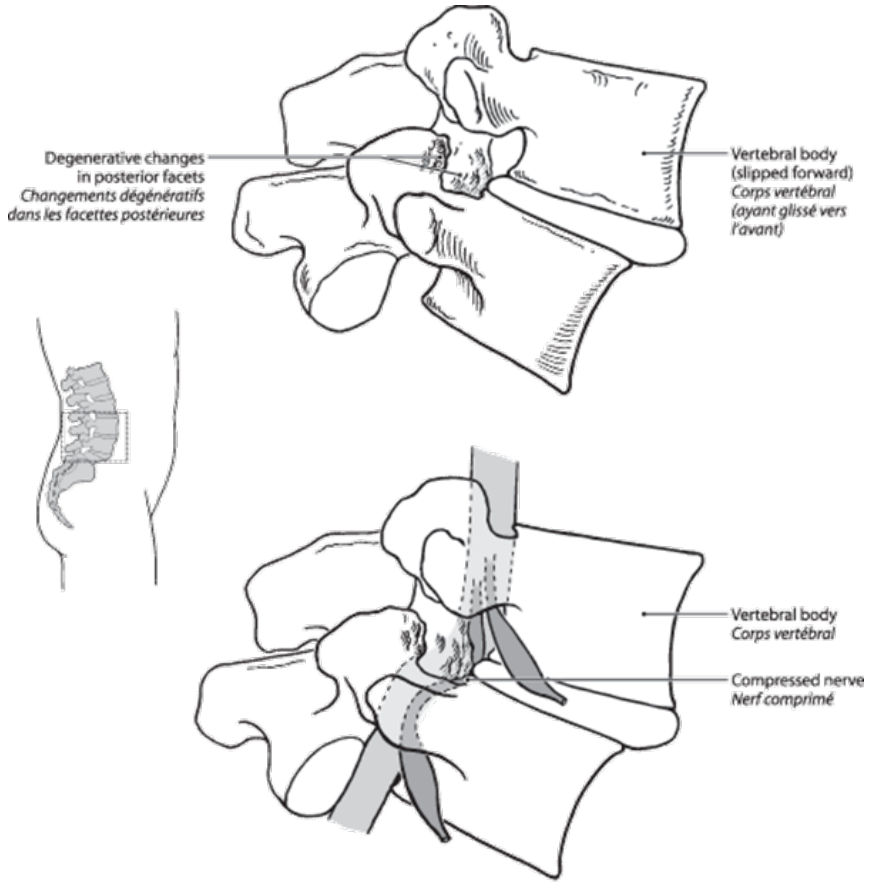


Figure 20. Degenerative spondylolisthesis - forward slip of L4 upon L5 vertebra due to degenerative changes in ligaments and facet joints. Lower picture shows how nerve root may be compressed.

i) Isthmic, (lytic) (Figure 18, 19b)

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. 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, repetitive lifting, or traumatic injury. Once symptoms commence, they tend to recur. Because the foramen is narrowed at the slip level, individuals may also present with referred radicular leg pain symptoms. These symptoms can be intermittent,

described as electric and/or shooting, and can be difficult to pinpoint to a specific myotome/dermatome distribution in the leg.

The presence of an isthmic spondylitic defect in young people, especially teenagers, who complain of significant low back pain and disability, is likely the cause of the disability; in older patients, the cause of the pain may be more likely from other multi-level aging causes (discogenic, inflammatory, etc.).

ii) Degenerative Spondylolisthesis

Degenerative spondylolisthesis is an important cause of low back disability in patients over age 50. The degenerative slip usually occurs at L4-L5 and is up to 3-6 times more frequent in women than men.[20]

It may be discovered on plain radiographs done in the evaluation of low back pain. These radiographs may show a narrowed disc complex, a forward slip of L4 on L5 (i.e. anterolisthesis); standing lateral x-rays in flexion and extension may show instability (i.e. anteroposterior translation and/or angulation) at this disc level. Sometimes radiographs/investigations show that disc narrowing/degeneration is accompanied by backward displacement of the upper vertebra on the lower called retro-spondylolisthesis. This may also be accentuated by flexion extension lateral films.

The back pain often radiates to the buttocks, lateral pelvis, greater trochanteric areas, and occasionally into the lower extremities.

Nerve roots may be compressed, most commonly L5 and L4 in the case of degenerative L4-5 spondylolisthesis, occasionally causing a neurological deficit.

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).

The site of nerve compression and its severity can be seen on imaging studies such as CT and MRI, and the clinical relevance, radiculopathy and/or cauda compression requires a careful history and physical examination (see 5.1B).

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.

6.4. Spinal Stenosis

Spinal stenosis simply refers to anatomical structural narrowing of the spinal canal and the nerve elements without reference to the cause. (Figure 21) There are many causes broadly grouped into congenital (i.e. born with) or acquired (e.g. aging changes, infection, tumour). The most common cause of spinal stenosis is 'degenerative' associated with aging changes (Figure 22, see Figure 9).

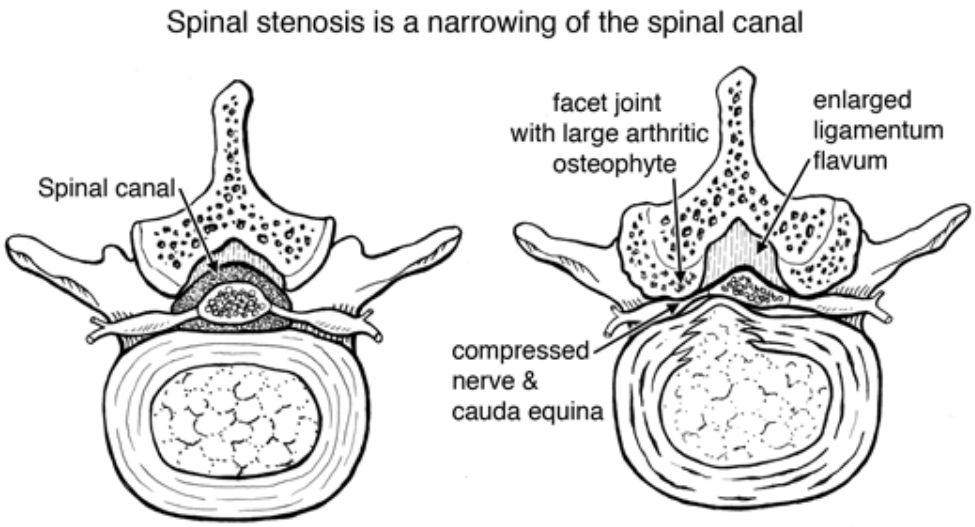


Figure 21. Diagram showing stenosis (narrowing) of the spinal canal; the narrowing causes may be caused by many factors as noted in the drawing on the right.

In spinal stenosis, the gradual formation of bony outgrowths (osteophytes) with ligamentous thickening narrows the spinal canal and the openings (intervertebral foramen) through which the spinal nerves emerge also narrow. 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 or in situations where 'burst-type' fractures occur that narrow the central spinal canal (more typically from higher energy trauma unless there is underlying low bone density situations such as osteoporosis or tumours).

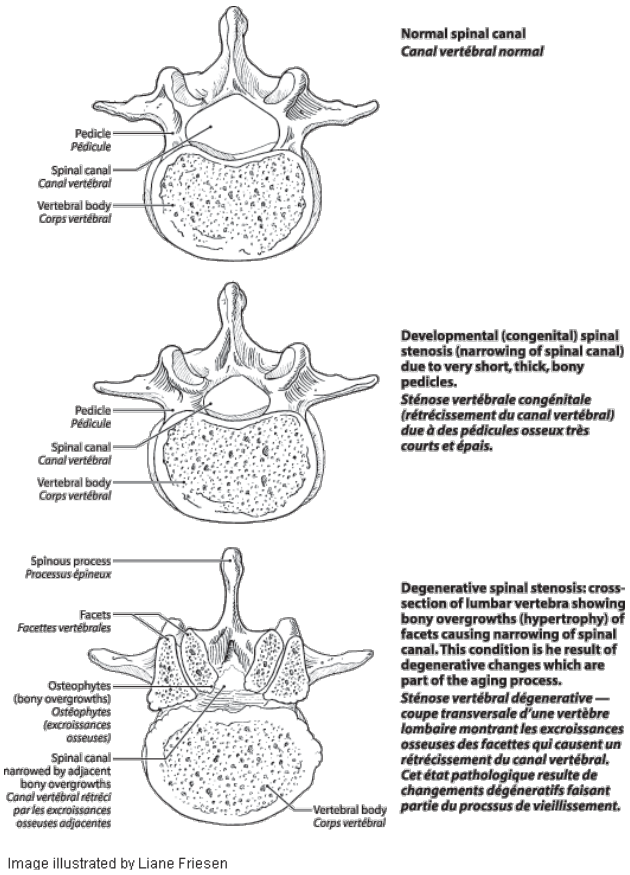


Figure 22a. Top image shows the normal spinal canal; middle image shows the developmental (congenital) spinal stenosis (narrowing of spinal canal) due to very short, thick, bony pedicles; bottom image shows degenerative spinal stenosis; cross-section of lumbar vertebra showing bony overgrowths (hypertrophy) of facets causing narrowing of spinal canal. This condition is the result of degenerative changes which are part of the aging process, the most common cause of spinal stenosis.

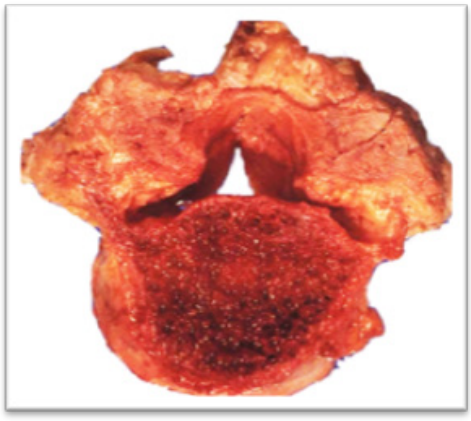


Figure 22b. Cross section of lumbar vertebra showing extreme narrowing of the spinal canal by encroachment by outcropping bone from the vertebral body anteriorly and the facet joints posteriorly. **Figure 22c.** CT showing severe triangular (i.e.trefoil shape) spinal canal of stenosis.

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. This claudication (crampy) type leg pain may be confused with vascular claudication; diagnosis is often difficult because vascular lesions are also common in this age group (see 4.2).

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 (see 9.2)).

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 (see 4.2.A) and neurogenic claudication secondary to spinal stenosis (see 4.2.B).

As previously stated degenerative changes in investigations, including spinal stenosis, disc herniation, and nerve root compression may be observed/reported in up to 60% of asymptomatic individuals 60 years, increasing to 80% at age 80; almost universal (see 3.0).

Correlation to clinical symptoms and signs by trained physician and health 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. The radiologic report should indicate if this patient has a congenital component to their spinal stenosis.

To reiterate again, since all the changes described in the spine, including aging changes in the intervertebral disc, facet joint arthritis, lumbar spondylolysis, spondylolisthesis and/or spina stenosis may be asymptomatic, only by correlating of these imaging findings with the clinical history, physical examination and laboratory findings is meaningful clinical diagnosis and treatment possible.

7.0 Other Back Pain Syndromes, Non-Motion Segment Related (Non-Discogenic)

7.1. Muscle (Soft Tissue Strain)

Soft tissue muscle sprain/strain is over diagnosed and does not necessarily relate to a disc or facet problem, that is, the discogenic pain we have been discussing. Pain following this type of injury is commonly felt directly in the back.

There may be several potential sources for this pain. A muscle strain may result in injury of the muscle - such as stretching and minor tears in the muscle fibres (Figure 23). 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.

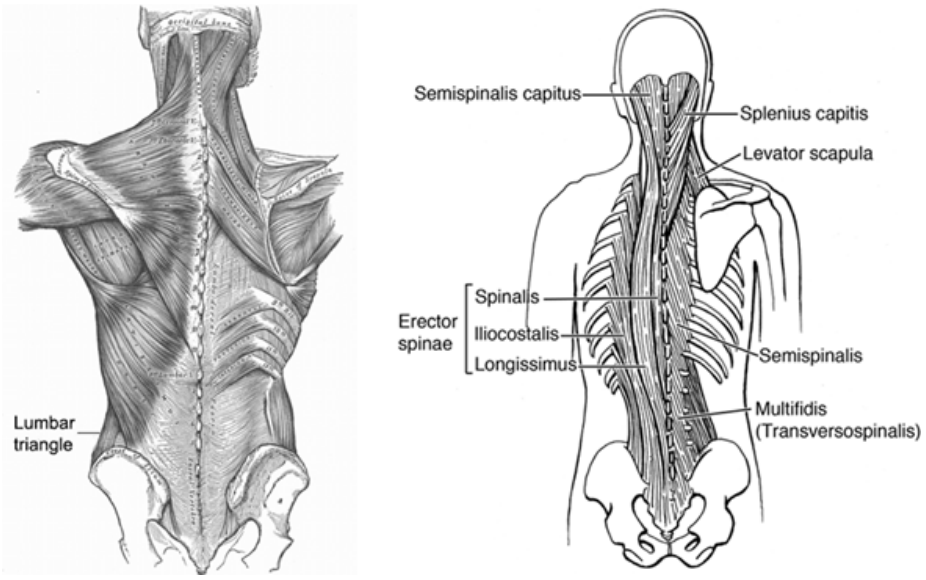


Figure 23. Diagram showing the muscles of the back, Superficial (left) and Deep (right) that may be involved in back pain caused by muscle strain

7.2. Vertebral Compression Fractures in Osteoporosis,

Vertebral compression fractures in the mid lumbar vertebra in patients with significant vertebral osteoporosis are common occurrences in our aging population, especially women.

They usually occur with minimal trauma or often spontaneously with no trauma.

Pain is often acute and localized to the back, and usually the pain settles down in a few days to 6 weeks with simple care and medication.

7.3. Ankylosing Spondylitis (Seronegative inflammatory arthropathies) (see MDP, Trauma and Inflammatory Arthritis, Dr. Dafna Gladman, 2008) [21]

This is an inflammatory arthritis that affects the spinal column, sacroiliac joints and sometimes the hips. It occurs almost exclusively in young males. It is one of a family of inflammatory arthritis called seronegative arthropathies, and includes, Reiter's disease, arthritis associated with inflammatory bowel disease (Crohn's) and psoriatic arthritis.

The cause of ankylosing spondylitis is unknown although it is associated with a white blood cell surface marker human leukocyte antigen B27 (i.e. HLA-B27).

The condition may result in fusion of the spinal column, sometimes in a flexed (i.e. kyphotic) position so that patients with the condition have trouble seeing where they are going (i.e. difficulty with horizontal gaze). It is characterized by intermittent flare-ups 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 24).

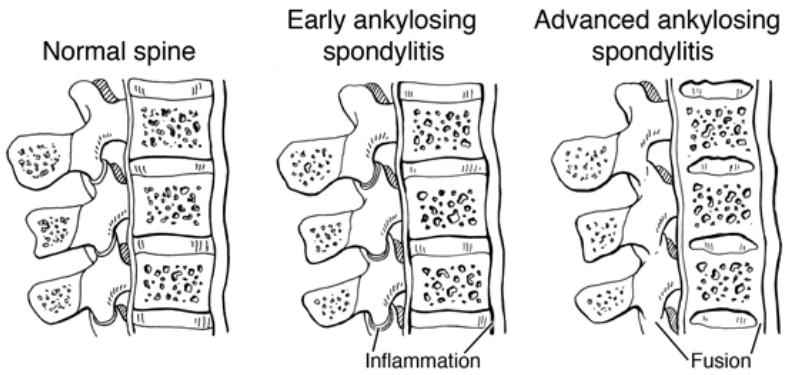


Figure 24. Ankylosing Spondylitis is an inflammatory arthritic condition that often affects the spinal column. Later stages of the condition result in auto-fusion of the spine (i.e. bamboo appearance of the spine on an x-ray) which limits spinal motion and may be associated with a spinal deformity (often kyphosis which is a deformity of the spine in the sagittal plane).

These individuals 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 (including advanced CT/MRI imaging) to rule out an occult fracture.

7.4. Scoliosis

This is sideways curvature of the spine (Figure 25). It can be congenital, secondary to paralysis (such as poliomyelitis or other neuromuscular diseases) 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.

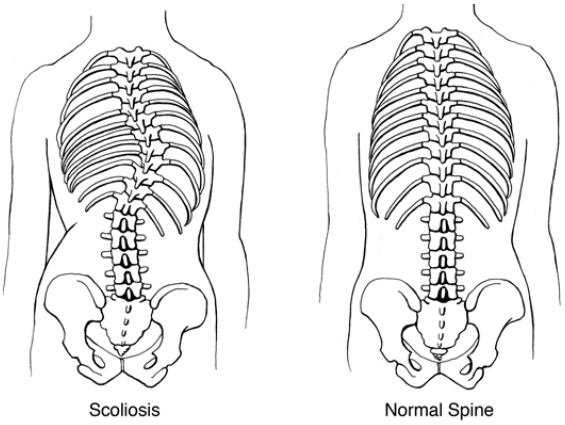


Figure 25. This figure illustrates the appearance of scoliosis which is a coronal plane imbalance of the spine.

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.

Patients with severe acute discogenic back pain may show rotation of their spine from muscle spasm; this not a structural scoliosis and disappears when the pain and muscle spasm are relieved.

7.5. 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.

7.6. Tumors and infections

Vertebral infections, including tuberculosis or tumours, both primary and metastatic; intervertebral disc infection (discitis) all cause back pain. The pain may be severe and unremitting, present at rest and at night and they are readily diagnosed by appropriate investigation such as lab studies and especially advanced imaging, CT, MRI.

7.7. Complex Regional Pain Syndrome

Complex Regional Pain Syndrome, now replaces several common causes of extremity pain as noted (see MDP, Complex Regional Pain Syndrome, Dr Anthony Weinberg, 2010)[22]

Complex regional pain syndrome (CRPS) CRPS bears several synonyms: causalgia; shoulder– hand syndrome; reflex sympathetic dystrophy syndrome (RSDS); Sudeck's atrophy, transient osteoporosis, and acute bone atrophy.

The terminology was changed because the pathophysiology of CRPS is not known with certainty. It was decided that a descriptive term such as CRPS was preferable to the term 'reflex sympathetic dystrophy,' which carries with it the outdated assumption that the sympathetic nervous system is important in its causation.

The consensus definition of CRPS is as follows: "CRPS describes an array of painful conditions that are characterized by a continuing (spontaneous and/or evoked) regional pain that is seemingly disproportionate in time or degree to the usual course of any known trauma or other lesion. The pain is regional (not in a specific nerve territory or dermatome) and usually has a distal predominance of abnormal sensory, motor, sudomotor, vasomotor, and/or trophic findings. The syndrome shows variable progression over time."[23]

It is defined a chronic pain syndrome with two forms.

CRPS 1 currently replaces the term 'reflex sympathetic dystrophy syndrome' and corresponds to patients with CRPS without evidence of peripheral nerve injury and represents approximately 90 percent of clinical presentations.

It is a chronic nerve disorder that occurs most often in the arms or legs after a minor or major injury. CRPS 1 is associated with severe pain; changes in the nails, bone, and skin; and an increased sensitivity to touch in the affected limb (see MDP,CRPS, Weinberg[22], UpToDate 1, 2[24])

CRPS 2 replaces the term causalgia, and refers to cases in which an identifiable peripheral nerve injury is present.[24]

7.8. Somatic Disorders Syndrome (Non-Organic Factors)

According to the American Psychiatric Association, "Somatic Symptom Disorder involves a person having a significant focus on physical symptoms, such as pain, weakness or shortness of breath that results in major distress and/or problems functioning. The individual has excessive thoughts, feelings and behaviors relating to the physical symptoms. The physical symptoms may or may not be associated with a diagnosed medical condition, but the person is experiencing symptoms and believes they are sick (that is, not faking the illness). A person is not diagnosed with somatic symptom disorder solely because a medical cause can't be identified for a

physical symptom. The emphasis is on the extent to which the thoughts, feelings and behaviors related to the illness are excessive or out of proportion."

Careful clinical evaluation is essential, including psychological consultation and testing when indicated. Also, when indicated, a psychiatric consultation is important.

Nonorganic findings are physical findings that do not have a direct anatomical cause and are distinct from physical findings of organic pathology. They were identified in 1980 by Waddell and colleagues. Although these findings were initially described in patients with low back pain, they may be adapted to patients with neck pain—such as those injured in a car crash. According to Waddell, nonorganic findings "provide a simple and rapid screen to help identify the few patients who require more detailed evaluation." The observation of nonorganic signs is one aspect of the physical examination and is independent of the anatomic and physiological components (the Waddell signs are reviewed in 9.2 D).

Other non-organic pain syndromes include:

- Malingering, Malingering may be described as pretending to have or exaggerating an illness in order to gain a benefit. The feigned illness may be mental or physical.
- Munchhausen Disorder now called Factitious Disorder

Also considered in this non-organic pain section is Munchhausen Syndrome or Factitious Disorder characterized by intentional falsification of physical and/or mental signs and symptoms in oneself or in another individual for no obvious external gain or reward. These individuals frequently visit different hospital emergency departments; recent electronic records across Ontario makes tracking them much easier than in the past (see DSM-5, 5th edition).[26]

8.0 Conditions That Physicians May Erroneously Consider to Be the Cause of Pain

8.1 Sacrolisation of the Fifth Lumbar Vertebra

In this congenital condition, the lowest (5th) 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 5th lumbar vertebra articulates with the ala of the sacrum (pelvis) by means of a false joint (pseudarthrosis) but this does not typically cause pain. In some instances, the transverse process of the 5th lumbar vertebrae is fused to the sacrum (Figure 26).

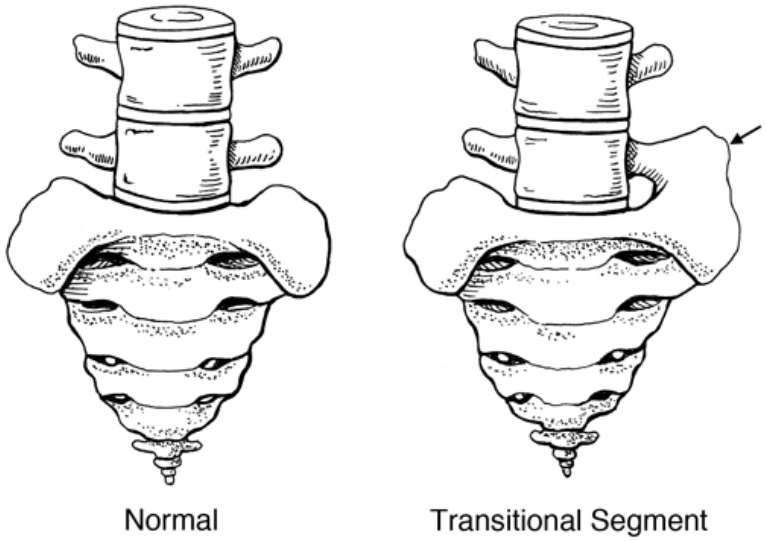


Figure 26. This demonstrates normal lumbosacral anatomy on the left image. Anatomical variations (which may or may not be associated with back or sacroiliac joint related symptoms) such as a transition segment is depicted on the right image – on one side (see black arrow), the transverse process appears fused to the sacrum

8.2. Lumbarization of the First Sacral Segment

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 27).

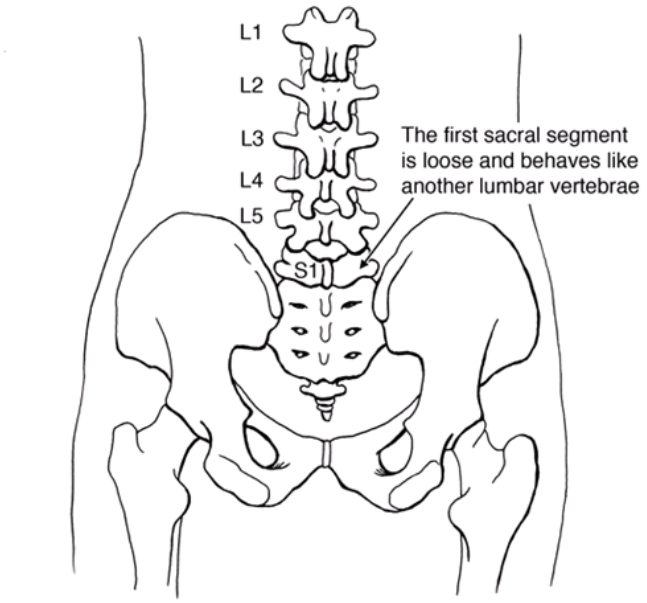


Figure 27. This diagram illustrates where the first sacral segment (S1) behaves like a lumbar vertebra

8.3. 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 misinterpreted as compression fractures. Clinical correlation is required. An assessment of global sagittal spine alignment may be helpful to determine if there are biomechanical risk factors that may be associated with symptoms.

8.4. Schmorl's Nodules

These are indentations of the nucleus pulposus into the body of the vertebra above. They are seen commonly and are not a source of pain.

8.5. 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.

9.0 Assessment and Diagnosis of Low Back Pain

As in all individual medical assessment and diagnosis, the cause of low back pain is determined by taking a detailed history, where possible; performing a careful physical examination and correlating the clinical findings to the investigation, such as laboratory tests and imaging studies as indicated.

Where available, radiologic imaging may support a clinical diagnosis, but without clinical correlation, imaging studies alone do not explain the cause of the pain and therefore will not help in the management of the patient.

9.1. History

Taking an accurate history, which includes getting to know the patient, is the most important assessment tool; the history will lead the examiner to the relevance of the findings, especially the imaging findings in the patient's symptoms of Low Back Pain.

A review of the file will help focus on the important aspects of assessment and diagnosis.

The history of an injury, the location and onset of back-related symptoms following injury as well as the pattern of pain is important.[26]

As academic surgeons teaching medical students, residents and orthopaedic/ neurosurgical fellows, we have emphasized repeatedly the dictum: “listen to the patient.”

By listening, the observer will learn the following important details.

a) Know the patient, past and present. A careful assessment of the patient includes the demographics, age, gender, education, occupation, employment history and any other important details. Also important are marital status, family relationships, and living quarters.

   Also, any past medical health concerns, surgery, and specifically any previous back injuries, any third party claims, including work related WSIB.

   Also, specifically, any psychologic disorders either expressed by the patient or documented.

   The present state of general health (co-morbidities), BMI, medications, (opiate addiction), activity levels, including work, sports, exercise.

b) The symptoms (clinical complaints). In this situation, the complaint is low back pain. Therefore the following are important:

   - Did the pain begin suddenly, spontaneously or with injury?
   - Did the pain begin at work?
   - Location of pain, back dominant or leg dominant
   - If there is both back and leg pain, which started first and the temporal course?
   - Quality of pain, severe, moderate, mild; is it achy or sharp; is it caused by movement or present at rest; is it present mainly at night;
   - Is the pain relieved by rest, by drugs, anti-inflammatories or are opiates required.
   - If leg pain, is it generalized to the anterior or posterior aspect of the leg, does it go beyond the knee? Does it follow a nerve pathway such as into the big toe, or the heel beginning at the buttock or sacroiliac joint?
   - Is the pain associated with neurologic symptoms such as numbness or loss of motor power? Is there loss of bowel or bladder function?

Consider the following not infrequent scenarios outlined by the patient.

> “I’ve never had any back or leg pain, I was lifting a large rock and twisted to move it onto a wheelbarrow. I got a sudden pain in my back, and then noticed the pain going down my right leg. It was in my buttock, back of knee and then into my big toe.

In the E.R, I noted my big toe and top of foot becoming numb and I could not pull my ankle up to a right angle (impending foot drop)."

That history is compatible with an L5 neuropathy, and can be corroborated by the physical examination and CT/MRI.

Very different than a history of chronic pain, non-anatomic radiation patterns, and many other health and social related factors such as high BMI, diabetes, family, psychological and more.

c) The degree of disability. In other words, "are you coping with what's going on?" Is the pain affecting your ability to work, to cope with activates of everyday living or sporting activities. How is this pain affecting your life?

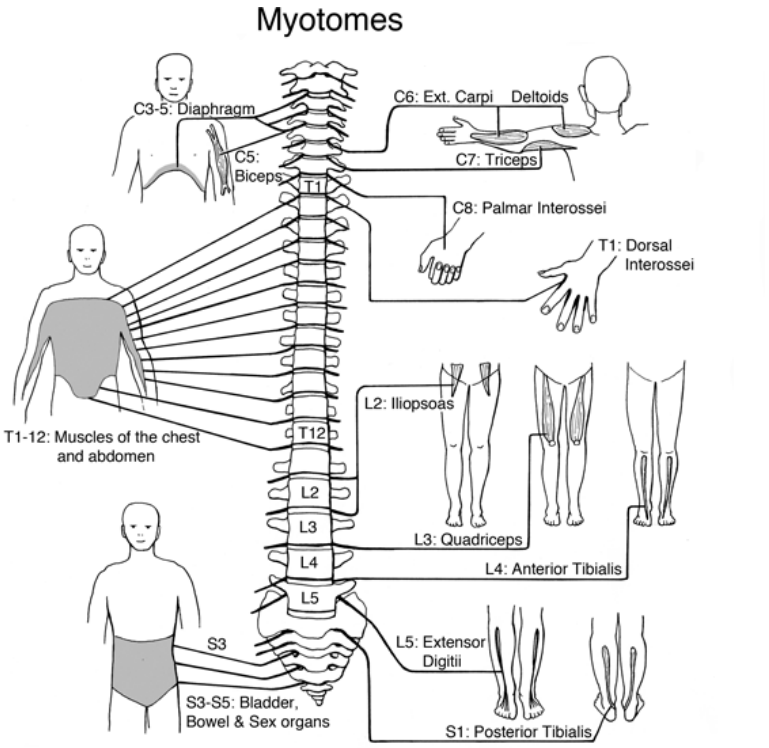


Figure 28. Diagram showing the myotomes (muscle groups and the supplying nerve roots)

9.2. Physical Examination

A. Look

Physical examination focuses on consistent reproducible findings. Observation of the patient, inspection of the area of injury, and specific examination tests. Following an injury, there may also be visible signs of acute trauma (bruising in the back region, abnormal swelling, gaps or steps in the back).

B. Move

The examiner will record the range of movement, (ROM), including flexion, extension and lateral (side to side motion); also, which movements exacerbate or relieve the pain. This raw recording of the range as a percentage of normal or as degrees of motion cannot be taken at face value, as there are many nuances noted by an experienced examiner.

A simple test and recording for accuracy of ROM would be to perform the range with the patient standing, then repeat asking the patient to sit or lie on the stretcher. In the standing position, patients may guard and move barely 10-20 degrees, often with grimacing and expressions of pain whereas in the sitting position, raising both legs may be possible to normal, above 100 degrees with no complaints; the implications' are obvious.

C. Touch

The examiners test and recording of 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; again, there are nuances that may be recorded by the expert examiner.

For example, discrepancies in tenderness when using the examining finger and a tuning fork to test tenderness may be striking, "the rule of inverse intensity."

Most discogenic back patients have little or no tenderness.

If the examiner touches an individual's back just with a finger, just a little touch, and they fly off the table, this would be considered clearly discordant to what would be anticipated. But people who have true involvement in their spine, unless it's a spinal infection or acute trauma, will have very little tenderness.

If the examiner applies a tuning fork and asks, "can you feel the vibration," and the answer was "No, I don't feel the vibration". And the examiner was pushing so hard on the tuning fork that the tuning fork would almost come out through the abdomen; that's what is meant by about inverse intensity (See 9.3.3.Waddell's non-organic signs).

D. Special tests

1. Neurologic Examination: Neurological changes including objective evidence of motor weakness anatomically linked to specific nerve roots (i.e. myotomes, Figure 28); reflex changes, which are linked to nerve roots and numbness in a nerve root distribution (i.e. dermatomes, Figure 29) is more concerning if objectively reproducible (see 4.2, and 5.1,C,b).

   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').

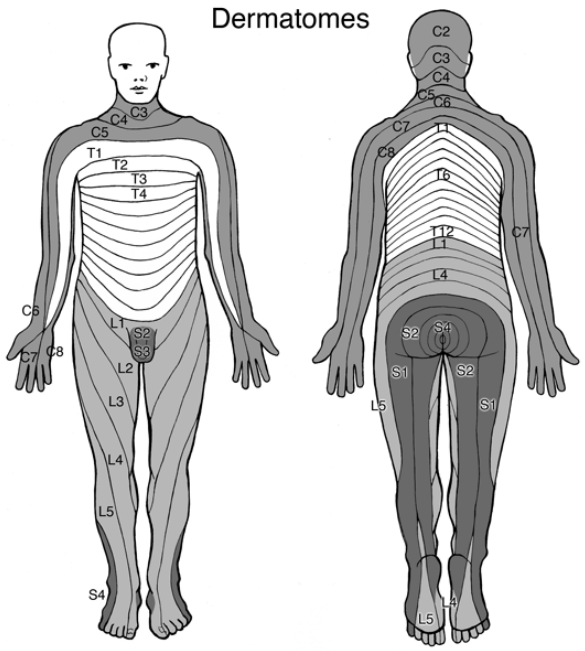


Figure 29. Diagram showing the dermatomes (regions of the skin and the respective nerve roots)

2. Nerve root irritative tests (see 5.1 C.a). There are several nerve root irritative tests which require special consideration in performing testing and its related interpretation.

   The straight leg raise test (and its variants including the Lasegue test, cross-over sign, well-leg raise) are aimed to detect nerve root irritation of lumbosacral nerve roots innervating into the sciatic nerve. These tests are often not performed correctly, and it is not uncommon for aggravation of back pain to be incorrectly mis-interpreted as a positive test. A true positive test is when the test (when performed correctly) reproduces the radiating radicular leg pain / sciatica that a patient may be experiencing. This suggests potential nerve root irritation most commonly associated with the L4/5-L5/S1 lumbosacral motion segments.

   Mid-lumbar nerve root irritation (e.g. L2/3, L3/4 levels) cause referred leg pain that is often more anterior in the leg (proximal thigh, anteromedial shin). The nerve irritative test that is provocative in these circumstances is the femoral nerve stretch test (performed with the patient lateral or prone) with passive extension of the hip and knee reproducing the radiating referred anterior thigh / anteromedial shin pain.

   Tests such as the straight leg raise, Lasegue, can be performed supine or sitting. Significant discrepancy between testing supine versus sitting can be a warning sign cautioning the conclusion of a true positive sign of nerve root irritation. The Lasegue straight leg raising technique is so important; it should be done in the

sitting position as well as supine, and therefore when you're seeing it on a report it may or may not be a true bill. For example, if done with the patient lying on a stretcher and you raise the leg to 20 degrees and the patient pushes down, tells you to stop and vocalizes pain and then you sit the patient up by the side of the stretcher and you can get the leg up beyond 90-100 degrees with no pain, and no pushing down, that obvious discrepancy is important and negates any sign of nerve root irritation in the sciatic nerve. Unfortunately, most reports do not record this detail and therefore are of little help in diagnosis.

3. Waddell's five non-organic signs in low back pain,
    i. Tenderness tests: superficial and diffuse tenderness and/or nonanatomic tenderness

    ii. Simulation tests: these are based on movements which produce pain, without actually causing that movement, such as axial loading and pain on simulated rotation

    iii. Distraction tests: positive tests are rechecked when the patient's attention is distracted, such as a straight leg raise test

    iv. Regional disturbances: regional weakness or sensory changes which deviate from accepted neuroanatomy such as "my entire arm is numb, my entire leg from the knee down. That doesn't follow an anatomic pattern." There are definite anatomic patterns to all the nerve roots, (see 4.2.A) and overreaction and bizarre behavior are not anatomical by definition.

    v. Overreaction: subjective signs regarding the patient's demeanor and reaction to testing

Any individual sign marks its category as positive. When three or more categories were positive, the finding was considered clinically significant.[27] However, assessing the patient on the basis of overreaction has raised concerns regarding observer bias and idiosyncrasies related to the patient's culture. Consequently, a practitioner may assess the patient on the remaining four categories, with two or more positive categories being considered clinically significant.[28]

One or two Waddell's signs may be found even when there is not a strong non-organic component to pain. Three or more are positively correlated with high scores for depression, hysteria and hypochondriasis on the Minnesota Multiphasic Personality Inventory.[29]

9.3. Investigation

A. Laboratory tests

Laboratory tests should be recorded on file. Tests that may have been performed if clinically indicated include a complete Blood Count (CBC) including routine hemoglobin to rule out anemias, a blood smear, WBC for inflammation and neoplasms, ESR and CRP for inflammation including infection.

Blood chemistry including A1C for diabetes, electrolytes, calcium, phosphorus, PSA (prostate cancer), serum protein electrophoresis (myeloma). Also routine urinalysis.

In older patients with LBP and red flag symptoms, these tests may serve as red flags to rule out more sinister causes of low back pain.

B. Imaging studies.

Although not recommended unless red flag symptoms are present or they are required for particular evidence-based therapeutic intervention, imaging tests include: routine Lumbar Spine x-ray includes a standard anteroposterior and lateral view (Figure 29a) as well as a pelvic AP view. If instability is suspected, lateral view in flexion and extension are indicated.

Practice guidelines in Canada and other developed countries often say these tests are not necessary because they say “the tests don’t mean anything given the incidence of almost universal degenerative change by age 80.”

As described by Choosing Wisely Canada:

> Don’t routinely image patients with low back pain regardless of the duration of symptoms unless: (a) there are clinical reasons to suspect serious underlying pathology (i.e., red flags), or (b) imaging is necessary for the planning and/or execution of a particular evidenced-based therapeutic intervention on a specific spinal condition.

But most patients over age 60 are very concerned they have a serious cause of their pain; they come to be reassured and if so, may return to work and other activities. As back specialists, one feels the relief in those patients after they are told after negative routine tests that showed no major sinister cause of their pain.

CT scans are superb for outlining bony pathology. Modern scanners have improved resolution and in some patients can outline soft tissue pathology like a disc herniation well.

MR is the best imaging for looking at the neural elements. (Figure 30) The use of MRI with Gadolinium IV contrast is typically reserved for situations where patients have had prior surgery (i.e. differentiating scar tissue from a recurrent disc herniation) or in suspected cases of neural tumours).

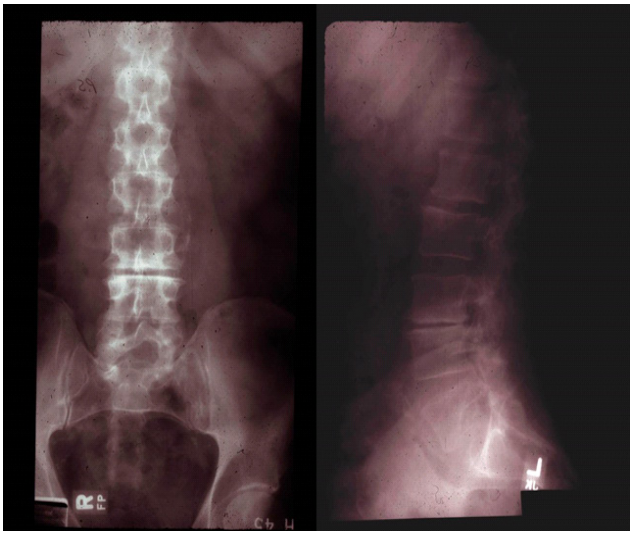
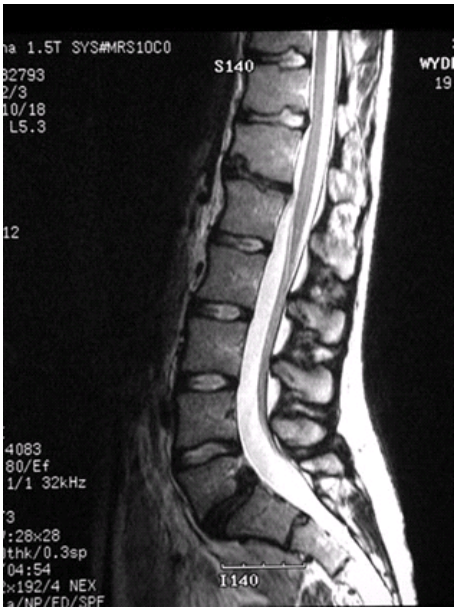


Figure 30a. An example of the AP (frontal) and lateral (side view) plain lumbosacral spine X-ray in a patient. The bone is seen clearly as is the narrowed degenerative L3-4 intervertebral disc level. The neural elements cannot be seen.



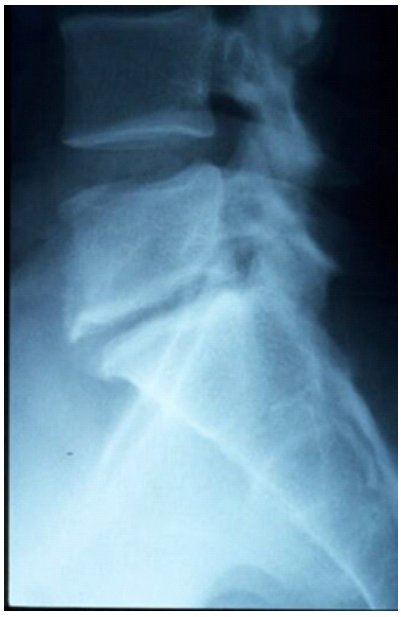


Figure 30b. In another patient, the neural elements (cauda equina) are clearly seen on this MRI image (T2 weighted) with no compression, as well the degenerative L5-S1 intervertebral disc is better outlined on MRI than the plain X-ray. **Figure 30c.** The corresponding lateral lumbosacral view of this patient showing loss in L5-S1 disc height with osteophytes.

Nuclear bone scans can be helpful to screen the appendicular and axial skeleton to localize tumour, infection or other pathologies.

SPECT (Single Photon Emission CT) and PET (Positron Emission Tomography) scanning are nuclear medicine imaging techniques which provide metabolic and functional information unlike CT and MRI. They have been combined with CT and MRI to provide detailed anatomical and metabolic information.

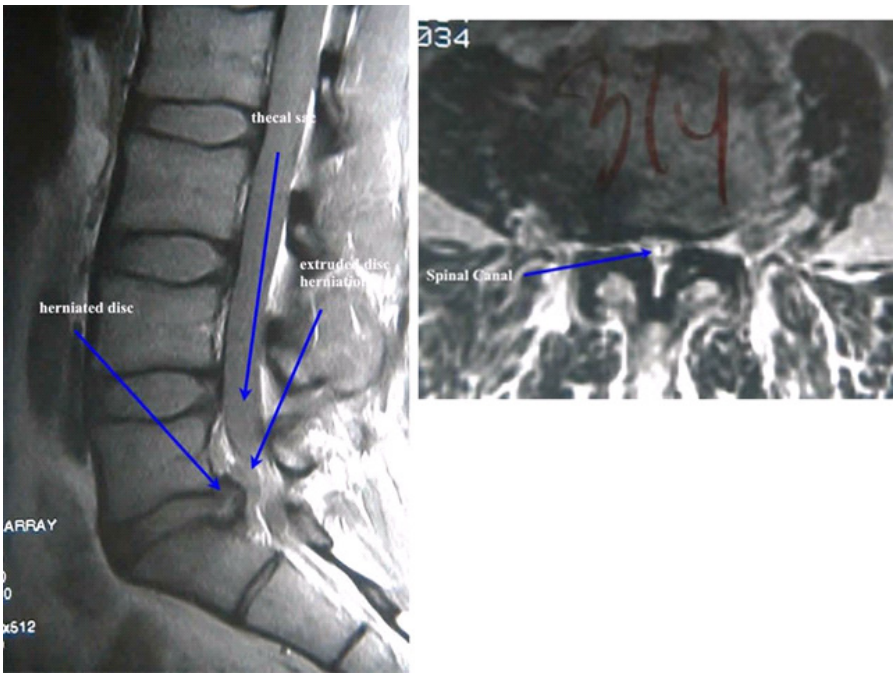


Figure 31. MRI images (T1 weighted) with abnormal findings: Left, sagittal view showing a disc herniation at L5-S1 with compression of the nerve roots. Right, T2 weighted Axial MRI view in another patient showing severe central and lateral recess spinal stenosis at the level L3-4.

C. Nerve conduction studies, EMG

If physical examination findings are not conclusive, or if reported symptoms do not match available radiologic imaging (Figures 20 and 21), additional investigations may be necessary. For example, a neurologist or physiatrist may also perform Electromyography (EMG) and Nerve Conduction Studies (NCS) that may complement history, physical examination, and imaging results.

10.0. Treatment of Low Back Pain

10.1. Non-Operative:

Treatment, to begin with, in an acute syndrome is nonoperative, whatever works – tincture of time and educational support, temporary modification of activity but definitely avoid complete bed rest. If severe leg pain, encourage the patient to find a body/leg position that may ease symptoms, in the later phase education,

active exercise complementing judicious but not isolated use of passive physical modalities. Evidence-based studies, especially by our Swedish colleagues, have indicated that it may not be as much as what therapy is received, the outcomes and natural history is that most patients improve over time and often within 4-6 weeks (see 3.0, 5.0).

10.2. Operative

Operative treatment may be proposed with a failure of non-operative care to relieve the symptoms, especially the pain and is rarely indicated. Careful selection is important.

If the patient feels they are no longer coping, and their expectations are realistic, then surgery is indicated if the clinical picture matches the investigation. Indications are typically recalcitrant referred radicular leg pain and/or progressive neurologic deficit, with correlative physical and imaging findings consistent with nerve root compression.

A. Leg Pain Dominant.

Urgent surgery may be indicated for severe nerve root compression causing severe painful motor loss (e.g. L5, Foot drop) or cauda equina compression causing bowel and bladder dysfunction, with acute cauda equina syndrome also presenting with severe bilateral leg sciatica and progressive lower extremity neurologic deficit.

Nerve compression and/or claudication syndromes including herniated discs, root stenosis, spinal stenosis, are all possible operative indications.

Surgery for nerve compression syndrome involves decompression of the neural elements involved, be it a nerve root, or nerve roots within the cauda equina.

Disc herniation decompression involves removal of a small portion of bone and ligamentum flavum (laminotomy, hemi-laminotomy) to expose the nerve roots and removal of the disc fragment compressing the nerve root. It's now done minimally invasive, and depending on the structural changes can be with a short one-inch incision under microscopic visualization. (see Figure16).

For spinal stenosis, at one level a similar procedure may be done, usually with more bone removed (laminectomy, laminoplasty, laminotomy). Many are now done as outpatients, with a 90% good outcome for relief of acute leg pain.

For multi-level stenosis, the decompression requires more levels and the results less promising.

If decompression extends laterally to removal of facet joints, instability may result often requiring an added spinal fusion.

B. Back Pain Dominant

For back dominant pain, careful selection is indicated, and results are good if done for the correct indications. For chronic low back pain with an unclear cause, the results are generally not predictably good, surgery should be avoided. Indications include these instability syndromes.

Spondylolisthesis:

- In young patients with isthmic types and active slip not coping with the pain (low back and/or radicular leg). Local spinal fusion is indicated (L5-S1); there is a trend to direct repair of the isthmic defect (often with graft and instrumentation to encourage defect healing) if there is an isolated spondylolysis without spondylolisthesis and a morphologically healthy intervertebral disc on MRI imaging.
- In older patients with degenerative spondylolisthesis usually at L4-5, with definite instability on dynamic flexion/extension films, decompression followed by local fusion is indicated following a trial of conservative treatment. Also, in older patients where decompression has caused instability (e.g. violation of the pars Interarticularis, cumulative removal of a facet joint at a particular level), spine fusion is indicated where necessary.

C. Spine Fusion.

Spine fusion, especially at one level may be done without internal devices, but more often, implants are used.

Also now available are several types of allograft as well as the time honoured autograft bone. Types of artificial collagen-hydroxyapatite, and other calcium sulphates/phosphates are in constant development and testing. These synthetics are typically osteoconductive and are often used to supplement autograft bone as a bone graft extender. Demineralized bone matrices (DBMs) incorporate processed allogeneic bone and are primarily osteoconductive and weakly osteoinductive. Bone morphogenic proteins (BMPs) are growth factors that are highly osteoinductive and in recalcitrant cases (recurrent non-unions, revision procedures) can function as a bone graft substitute.

Several types of pedicle screw fixation or more commonly screw-rod combinations are available. Also in common use are intervertebral metal (titanium, trabecular metal) or medical grade polymer (Polyetheretherketone, PEEK cages), which can be filled with bone graft material. This cage, which could be filled with bone graft material, can be inserted from the front (anterior) or from the side (oblique/lateral), or commonly from the back (posterolateral or transforaminal) (Figure 32).

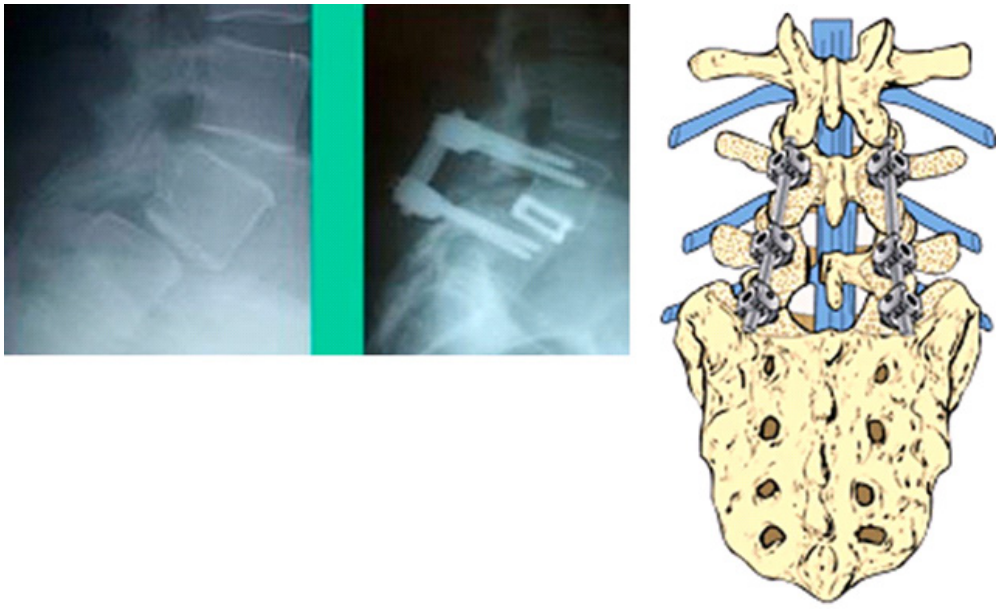


Figure 32. Unstable L5-S1 motion segment after spinal fusion using pedicle screws as well as a cage. The drawing on the right shows the position and direction of pedicle screws, in this drawing from L4-S1.

D. Artificial Intervertebral Disc

The modern era artificial disc now has been around for 15 to 20 years, and is used extensively in Asia, especially in the cervical spine (Figure 33). It has been used sparingly in Canada which may be due, in part, to costs of the implant. Studies have shown their use where indicated did not cause more complications than standard techniques of fusion, but nor did most studies show significant advantage. The jury is still out, but their use is increasing.

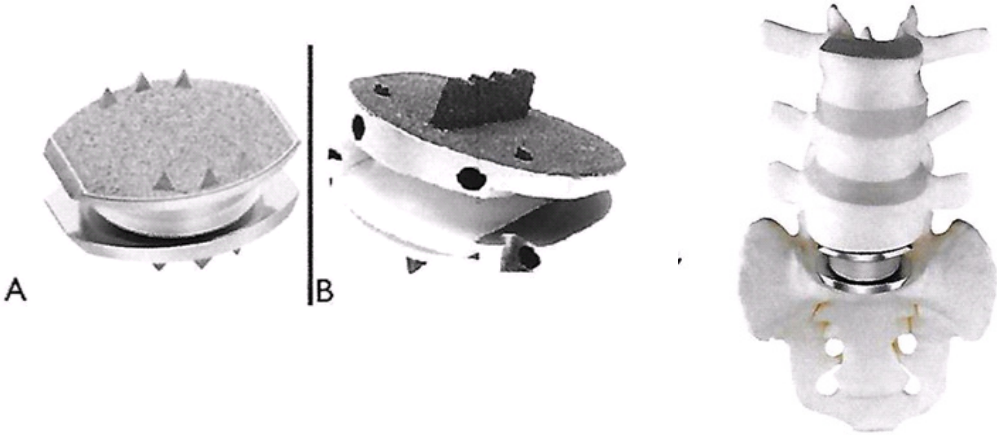


Figure 33. Artificial disc as inserted at L5-S1.

E. Failed Back Syndrome

This is a challenging condition to treat and is a catch-term syndrome for when treatment, including surgery, has not been successful. There are patient, perioperative, and structural factors that contribute to this syndrome.

Patient factors include psychological, social issues, and opioid addiction.

Surgical factors include consideration of poor patient selection, revision surgery, and other iatrogenic issues (eg. inadequate surgical decompression, instability due to excessive decompression, wrong-level surgery, failure to consider or restore sagittal plane alignment (ie. flat back syndrome)).

With multiple decompressive back surgeries, the nerve roots and cauda equina can also become covered in an inflammatory membrane, called arachnoiditis (Figure 33). This may result in intractable causalgic nerve pain and be very difficult to manage. Epidural fibrosis is also not uncommonly reported on MRI scans performed after surgery. Exuberant scar/fibrosis has also been suggested potentially to contribute towards residual neuropathic pain that may still persist.

Occasionally, symptoms can relate to sagittal plane imbalance that may be pre-existing, or further exacerbated following treatment. With aging, there is typically a loss in physiologic sagittal plane balance and flat-back syndrome (back pain, adaptive hip and knee flexion/contracture in attempts to maintain horizontal gaze and increased difficulty with ambulatory function) can result. The importance of sagittal plane balance and spinopelvic anatomy to physical functioning of the spine is increasingly being recognized and considered in related spinal treatments.

Longer term effects of spinal fusion include adjacent segment degeneration which may also cause recurrent low back and radicular leg symptoms

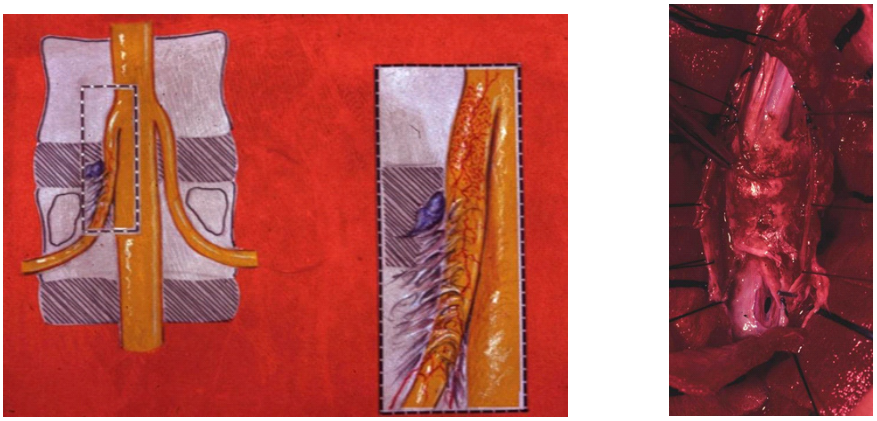


Figure 34. Arachnoiditis after multiple back surgeries (left). In this case, the appearance of the cauda equina at surgery. Matched that of the drawing, severe matting of the nerves from a thick fibrotic membrane (right). This patient had suffered significant burning type nerve pain in her legs.

11.0. Summary Statement.

Optimal treatment depends on getting to know the patient, individualizing the treatment, understanding the background, the natural history, and biomechanics of the spine. The treating physicians, surgeons and all health care workers must have a clear vision for the goals of the treatment, both short and long-term. It remains paramount to always involve the patient in the final decision and especially to learn if the expectations of the person meet the reality of the treatment.[30]

As always in individual patient care, do the benefits of treatment outweigh the risks?

12.0 Questions and Answers

1. What is a Spinal Motion Segment?

See 2.1. Motion Segment Anatomy

Understanding mechanical back pain requires an understanding of the complex anatomy of the spine, especially that of the Spinal Motion Segment (also known as the Functional Spinal Unit, FSU) (Figure 1).

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.

See Figure 2. Spinal Motion Segment, showing the intervertebral disc with the nucleus pulposis and annulus fibrosis and the ligaments, i) from above, ii) from the side, lateral, iii) from the front, anterior, also with cut out to show the posterior structures surrounding the cauda equina (neural).

See Figure 3. Spinal Motion Segment, showing the intervertebral disc with the nucleus pulposis and annulus fibrosis and the ligaments, i) from above, ii) from the side, lateral, iii) from the front, anterior with the neural elements, caudal equina and exiting nerve roots

2. How common are these age related changes in the population; prevalence?

See 1.2. Clinical Epidemiology with respect to Low Back pain and 3.1. Prevalence

In summary of 1.2, as noted above, these aging changes are almost universal occurring in 50% of people at age 50 and 80% at age 80. They are most often seen in the lumbar spine at the L4-5 and L5-S1 motion segments, and in the cervical spine at C5-6 and C6-7.

3. What happens to the Spinal Motion Segment with Aging; Age related or degenerative changes

See 3.2. summary statement.

Aging Changes in the motion segment: the anatomical changes alter the normal biological and biomechanical environment of the spinal motion segment. These aging (degenerative) changes are an incremental process which occurs gradually over time (see 2.1, 3.1). Degenerative change apparent on imaging shortly after the accident, was not the result of the accident.

Understanding the anatomy of the normal motion segment is the key to understanding these aging changes and are seen in Figures 1-5 and Figures 7-8.

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 in mid to advanced stages on plain x- rays. Resultant discovertebral changes include the development of vertebral endplate osteophytes. Additionally degenerative facet osteoarthritis occurs with loss of joint space, osteophytes, and thickening of facet capsule as well as ligamentum flavum tissues. Vertebral endplate subchondral sclerosis also commonly occurs (and is visualized on MRI scans as Modic endplate changes).

For full response to question 3, see 3.2. Aging Changes in the Motion Segment:

See Figure 7. Anatomy of a normal Spinal Motion Segment, two vertebra, posterior arch and facet joints, intervertebral disc, neural elements.

See Figure 8. Anatomy of the normal Intervertebral disc showing complex anatomy of the annulus fibrosis (a, below) and the properties of the normal nucleus pulposis (b, above) Proteoglycans are mucopolysaccharides (MPS) bound to protein are commonly found in connective tissues and abundant in the nucleus pulposus (NP) of the intervertebral disc (IVD). Proteoglycans are comprised of glycosaminoglycans (GAGs) which have strong water (H2O) binding abilities. Negatively charged chrondroitin sulphate and keratin sulphate GAG complexes attached to core proteins exert osmotic swelling pressure to draw water to support the hydrostatic properties of the IVD under mechanical loading.

See Figure 9. Aging changes in the nucleus pulposis, including loss of water content (wrinkled skin analogy) and change in chemical content (see Figure 8) as well as degenerative changes in the annulus fibrosis, causes loss in intervertebral disc height and facet arthrosis (osteoarthritis) which further leads to narrowing of the intervertebral foramen (i.e. foraminal stenosis) which may impinge upon the existing nerve root at that motion segment.

See Figure 10. Biomechanical and Biological abnormal motion segment in close proximity to the exiting nerve root in the intervertebral foramen is the result of these aging changes.

4. What causes the pain syndromes arising from the lower back motion segment? (Discogenic, Mechanical, low back pain.)

See 5.0, Pain Syndromes Arising from the Lower Back Motion Segment.

Low back pain may arise without injury, or with relatively minor trauma, such as bending over and tying one’s shoe laces. People may present to a health professional with acute low back pain which we can arbitrarily define as pain arising acutely and lasting no more than 8-12 weeks before resolution; or more chronic pain which may be recurrent or chronic unremitting pain.

This pain is often referred to in medical notes as mechanical back pain, meaning pain with movement of the spine. It is a grab bag term, and has no meaning with respect to causation. Pain with movement may occur with discogenic pain, but also muscular strain and other causes.

Acute back pain arising from a lumbar motion segment (discogenic) may arise spontaneously without trauma or may arise following injury. The resultant pain may be back pain only, back pain associated with referred pain into the proximal lower extremities, or neurogenic (radicular) lower extremity pain.

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

See also 5.1., Acute Low Back Pain; Spontaneous Onset; the Role of Inflammation, Enthesopathy.

Spontaneous onset pain without injury is common throughout the musculoskeletal (MSK) system. This occurs where tendons, ligaments or fascia attach to bone in the region of a joint and they are collectively called enthesopathies or tendon into bone syndromes. Pathological inflammation (enthesitis) as in conditions such as ankylosing spondylitis, psoriatic arthritis, and rheumatoid arthritis can also occur with the umbrella term of enthesopathy.

Common examples of enthesopathy include shoulder pain (rotator cuff,) elbow pain (tennis elbow), hip pain (trochanteric tendinitis), heel pain (Achilles tendonitis), foot pain (plantar fasciitis) and in all other anatomical areas where tendon, ligament or fascia attach to bone (e.g. wrist, hand, etc.).

This is also true in acute low back pain; most patients who seek help from health care providers for low back pain have no history of injury. As in most enthesopathies, the pain arises in regions of aging or degenerative changes, or as we have described previously, in a biological and biomechanical abnormal motion segment. (see Figures 10,11)

See also 5.2. Acute Low back pain; associated with a trauma event, as discussed in Questions 5, 6 below.

5. Where in a Spinal Motion Segment affected by age related degenerative changes does the pain arise? The pain receptors are located in the musculoskeletal system and/or the neurological system.

See 4.0 Where Does Low Back Pain Come From?

Given the almost universal nature of these aging changes in people with and without back pain, where does the pain come from?

The origin of pain requires a knowledge of the location of pain receptors.

In the lower back or lumbar area, these receptors may be found in the musculoskeletal system and the neurological system.

See Figure 11. Discogenic pain arising in a Spinal motion segment may arise in the pain receptors located in the musculoskeletal system or from the neurological system.

Pain receptors in the musculoskeletal system are found in the posterior longitudinal ligament and in the outer fibres of the annulus fibrosis, in the neurologic system, they are related to the nerve root in or beyond the intervertebral canal or to the cauda equina in the spinal canal. The pain may be triggered by trauma, inflammation (e.g. infection, chemical, diabetes).

6. Can injury or vocation influence the aging process; causation vs aggravation?

See 5.2. A. Can Injury or Vocation Influence the Aging Process; Causation vs Aggravation (also see 1.2 C)

i) Causation:

Given that degenerative change is so pervasive in our society, it is unlikely that one's occupation causes early disc degeneration. There are few credible scientific papers supporting that theory (see Andersson, Epidemiology, chapter 1.2, D).

These aging (degenerative) changes are an incremental structural process which occurs gradually over time and may or may not be symptomatic. Degenerative change apparent on imaging studies such as X-Ray, CT or MRI shortly after the accident, was not the result of the accident.

ii) Aggravation:

Age related degenerative change results in a biomechanical and biological abnormal motion segment in close proximity to the exiting nerve root in the intervertebral foramen.

Although the individuals with these changes do not necessarily have any symptoms, they are at risk of becoming symptomatic often with no injury. On occasion, individuals with pre-existing degenerative changes may be vulnerable to symptoms arising from work injury – for example, the development of lumbar radiculopathy following injury in a situation where there was significant pre-existing degenerative

changes that had already resulted in nerve root compression. This would be considered an aggravation with symptoms arising from a pre-existing condition with structural changes that were previously asymptomatic or minimally symptomatic. Such symptoms (eg. lumbar radiculopathy in the example provided) would typically arise within a short period of time (ie. within days up to ~1-2 weeks). Such injury may arise by repetitive bending and twisting movements, usually in the flexed position and/or associated with heavy lifting.

Some occupations, such as construction work and other manual employment, e.g. garbage collectors, warehouse workers, nurses, Personal Support Workers (PSWs) which involve repetitive bending, heavy lifting and twisting, may put the workers at risk as they age, thereby acting as aggravating factors for this pre-existing condition. Sitting for long periods may also contribute to symptoms of low back pain.[19]

See Figure 10. Biomechanical and Biological abnormal motion segment in close proximity to the exiting nerve root in the intervertebral foramen is the result of these aging changes.

7. How may injury aggravate or accelerate the onset of low back pain in people with age related or degenerative changes in the Spinal Motion Segment?

In pain arising from an injury to an abnormal motion segment, either work related or not, the forces are usually low energy, even relatively minor, and usually involve flexion and rotation (twist). Common scenarios include lifting and rotating, often with the legs extended. Even the motion of bending over to tie one's shoe laces may have the same effect.

(For Biomechanics. Type of Forces associated with injury to the lumbar spine; low energy, high energy. See also 5.2B.)

Since all epidemiologic studies have shown the prevalence of the abnormal degenerative disc at 50% of the population at age 50, many of these injuries occur in the 40-60 age groups, and are not gender specific.

In non-work related instances, the injuries are often sports related, such as hitting a golf ball or related to activities of daily living around the house involving lifting and rotation.

Work related injuries are also usually minor, as compared to high energy motor vehicle crashes or falls from heights, and often involve lifting and rotation.

Common examples of such injuries include the following:

- Construction worker lifting a heavy board from the ground in the flexed position and twisting to install it.

- Landscape worker planting a tree; pain occurs as the worker lifts the root ball, twists to put the ball into the prepared hole in the ground.
- Nurse or Personal Support Worker (PSW), lifting a heavy patient from bed while in a flexed position and twisting.
- Bending and pulling to put on a work boot.

Since the motion segment is biologically and biomechanically abnormal in this situation, minor trauma usually involving flexion and rotation with or without a lift, may further injure the disc and/or spinal soft tissues and cause pain (see 3.2, and Figures 10, 11b).

The injury to the preexisting degenerate intervertebral disc may be a further tear to that already present in the annulus fibrosis. It may cause the nucleus pulposis to herniate through the annulus fibrosis occasionally leading to nerve root irritation and/or compression.

This injury is often called by the public a “slipped disc”, more formally, a “herniated intervertebral disc” (see 5.3).

This pain is often referred to in medical notes as mechanical back pain, meaning pain with movement of the spine. It is a grab bag term, and has no meaning with respect to causation. Pain with movement may occur with discogenic pain, but also muscular strain and other causes.

8. Timing of symptoms following injury; can there be a delay in the onset of symptoms after back injury? If so to what extent?

See 5.2.D.Timing of the symptoms.

Most patients with an injury to the disc complex will have immediate pain at the time. The pain may be minor, often shrugged off, but may often become worse, especially in the first 24-48 hours after injury.

If the pain is noted at a later time, for example 2-4 weeks later, only a careful history of all the facts, especially documentation, will determine any relationship of pain to alleged injury. Also important would be a review of a detailed physical examination and the results of any investigations that may have been done and recorded. The pain may be low back dominant if there is no concomitant nerve root irritation or it may be associated with referred radicular leg (neurogenic) pain in some cases where there is nerve root compression by discal changes. The neurogenic leg pain component can follow the onset of low back pain from injury, and if related to injury the neuropathic component would typically arise within 2-4 weeks from injury if not already present from the immediate time of injury. Most patients’ symptoms with a disc injury following trauma will resolve over a period of several weeks and

by three months at least 90 percent of patients will be significantly better (see 1.2, Epidemiology).

9. What is the effect of previous back surgery on the Motion Segment and low back pain?

Previous back surgery may have a considerable effect on further low back pain.

The effect may be in the nervous system and/or the adjacent motion segments following spine fusion over time.

If the original surgery is performed for a herniated disc causing nerve root compression, the early results are excellent relieving the leg pain (sciatica) in more than 90% of cases. This is also true in nerve decompression for spondylolisthesis at one level. In a small number of patients, usually less than 5% with nerve decompression surgery, early postop pain may result from damage to the nerve tissue, inflammation and arachnoiditis which may become chronic.

In more extensive nerve or cauda equina decompression at several levels, this syndrome may occur more frequently, beginning shortly after surgery or over time.

Patients may 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.

If the original surgery is 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, ASD). ASD over time also typically narrows the spinal canal/foramens at the adjacent level so apart from chronic low back pain, patients may develop recurrent or new neuropathic leg symptoms including claudication or sciatica which may lead to future surgery.

See Figure 32. Spine Fusion and Figure 33. Artificial Intervertebral Disc. See also 10.2, Failed Back Syndrome.

This is a challenging condition to treat and is a catch-term syndrome for when treatment has not been successful. With multiple back surgeries, the nerve roots and cauda equina may be covered in an inflammatory membrane, called arachnoiditis. (Figure 33). This may result in intractable causalgic nerve pain and be very difficult to manage. Epidural fibrosis is also not uncommonly reported on MRI scans performed after surgery. Exuberant scar/fibrosis has also been suggested potentially to contribute towards residual neuropathic pain that may still persist.

Occasionally, symptoms can relate to sagittal plane imbalance that may be pre-existing, or further exacerbated following treatment. With aging, there is typically a loss in physiologic sagittal plane balance and flat-back syndrome (back pain, adaptive hip and knee flexion/contracture in attempts to maintain horizontal gaze and increased difficulty with ambulatory function) can result. The importance of sagittal plane balance and spinopelvic anatomy to physical functioning of the spine is increasingly being recognized and considered in related spinal treatments.

See Figure 34. Failed back syndrome, Arachnoiditis

10. What is the relationship of low back pain and/or neurological symptoms with the following structural spinal syndromes?

Spondylolisthesis, see 6.3., Spinal Stenosis, see 6.4. and Scoliosis, see 7.4.

11. 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. Differentiating low back pain radiating to the buttocks arising from the lumbar spine as versus other areas such as the sacro-iliac joints can be challenging.

See also 4.2. Clinical symptoms. Neurogenic pain

Clinical symptoms. Neurogenic pain

Lumbar disc herniation may become symptomatic involving the neural elements (neurogenic, or neuropathic pain); the result may be nerve root irritation and/or nerve root compression (Figure 16).

Nerve root irritation if often seen in the acute phases of a lumbar disc herniation causing nerve root compression. Nerve root compression simply refers to the structural discal-vertebral changes that cause physical compression of the nerve(s). The presence of and extent of nerve root irritative symptoms or related physical signs depending on the acuity or chronicity of symptoms and also the degree of structural nerve compression.

a) **Nerve root irritation:** is characterized by referred pain along the distribution of the affected nerve, often associated with nerve root irritative physical signs (see straight leg raise, SLR below). Pain distribution along affected nerves include:

- (4th nerve root, femoral nerve), the anterior thigh/shin,
- L4-5 the lateral calf to the big toe (5th nerve root, Sciatic nerve),
- L5-S1, (S1 nerve root, Sciatic nerve branch), the posterolateral calf to the heel and 5th toe.

These 3 nerve roots L4, L5, and S1 account for most of the cases of low back symptoms associated with nerve involvement as these are the most common vertebral levels for aging degenerative lumbar spine changes (L5-50%, S1-45%, L4-5%).

For the upper lumbar nerve roots, extending the hip with the knee flexed may cause severe anterior thigh pain (i.e. positive Femoral nerve stretch test); whereas patients with sciatic nerve pain have restriction of straight leg raising (SLR) because of pain – symptomatic leg elevation in either the supine or sitting position is limited and reproduces the individuals reported radiating neurogenic leg pain.

Many do not progress beyond the irritation phase, but ultimately neurological abnormalities may occur.

The nerve syndromes may develop slowly, or very rapidly, within a few hours.

b) **Nerve root compression**: is characterized by sensory, motor, and reflex changes; these changes are present on a careful neurological examination, and are diagnostic when the clinical findings match the imaging studies (CT, MRI).(Figure 16)

- L4; a history of anterior thigh pain and/or numbness that may radiate into the anterior shin below the knee, and the neurological examination showing anterior thigh/shin numbness, a weak quadriceps muscle (knee extension), and an absent or diminished knee reflex.
- L5, a history of posterior thigh and lateral calf pain that may radiate into the dorsal foot and big toe and/or numbness, and the neurological examination showing numbness on the dorsum of the foot to the big toe, weakness in the toe

extensors to a complete inability to raise (i.e. dorsiflex) the foot at the ankle (eg. a drop foot), no reflex change.

- S1, a history of posterior thigh pain, lateral/posterior calf pain to the heel and 5th toe/sole of foot area and/or numbness, and the neurological examination showing numbness along the lateral calf and heel to the little toe/sole of foot, weakness in the calf muscles (inability to raise one's body weight standing on the tip toes), and a diminished or absent ankle reflex.

Often, the pain may lessen or even disappear without surgical removal of the disc herniation; the neurological abnormalities may improve, or not, usually the absent reflexes do not return.

Continuing radicular pain with nerve deficit and a large compressive disc herniation on MRI matching the clinical finding is the usual indication for surgical disc removal. This surgery is now done with minimally invasive techniques as an outpatient procedure with excellent results for pain relief and improvement of the neurological picture (see 9.2).

Saddle anal numbness (i.e. skin area overlying the saddle seating region around the anus) and/or loss of bowel and bladder function, the so-called cauda equina syndrome requires urgent clinical and imaging assessment and possibly emergent spinal decompression.

12. How is back injury diagnosed? In clinical medicine, diagnosis follows the time honoured tradition of:

- History, listening to the patient
- Physical Examination, done carefully by an experienced physician or surgeon
- Investigation, to corroborate the clinical findings including laboratory tests, imaging, including plain radiographs, CT, MRI, ultrasound and more,
- Review of past health care records including other special tests such as nerve conduction studies/EMG.

This is all reviewed in detail in 9.0, Assessment and Diagnosis of Low back Pain.

As in all individual medical assessment and diagnosis, the cause of low back pain is determined by taking a detailed history, where possible; performing a careful physical examination and correlating the clinical findings to the investigation, such as laboratory tests and imaging studies as indicated.

Where available, radiologic imaging may support a clinical diagnosis, but without clinical correlation, imaging studies alone do not explain the cause of the pain and therefore will not help in the management of the patient.

13.0 Selected Glossary of Terms

(Fardon, DF et al., Spine, Vol. 26(5), pp E93-113, 2001; Fardon, DF et al., Spine J, 14(11), pp 2525-45, 2014)

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.

anulus, 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 intravertebral 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 spondyloarthropathies.

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.

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