REVIEW ARTICLE

Physiopathology of Lumbar Spine Degeneration and Pain

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Low back pain is a very frequent condition in our country and has great social and occupational repercussions. With advancing age, a degenerative cascade occurs in the lumbar spine, which starts at the intervertebral disc and subsequently involves the facet joints and other vertebral elements. This spinal degeneration is part of the normal ageing process, although it can sometimes cause pain and/or neurological alterations.

To understand the physiopathology of lumbar pain, it is necessary to know that the innervation of the lumbar spine occurs mainly through three nerve rami: dorsal ramus of the lumbar spine nerves (or «dorsal rami»), Luschka's sinuvertebral nerve and the ventral rami of the sympathetic chain. There are two types of pain pattern in the lumbar spine: irradiated pain and referred pain.

The degeneration cascade is divided up into three stages. The first is that of dysfunction, whereby the *annulus fibrosus* is fissured and can no longer contain the nucleus pulposus. This leads, first to the so-called *disc disruption syndrome* and, if the nucleus exceeds the contour of the *annulus*, to *disc herniations*. The second stage, also called the Kirkaldy-Willis stage, is that of instability. At this stage, the mobility of the mobile segment increases pathologically. We describe the radiological signs associated to this instability. The third phase is that of stabilization and is characterized by stenosis, which may or may not be associated to instability.

Key words: *low back pain, lumbar discopathy, lumbar spine degeneration.*

Fisiopatología de la degeneración y del dolor de la columna lumbar

La lumbalgia es una patología muy frecuente en nuestro medio, con una enorme repercusión sanitaria y sociolaboral. Con la edad, se inicia una cascada degenerativa en la columna lumbar, que comienza en el disco intervertebral, continuando por las facetas articulares y demás elementos vertebrales. Esta degeneración del raquis forma parte del envejecimiento normal del individuo, aunque en ocasiones puede causar dolor y/o alteraciones neurológicas.

Para comprender la fisiopatología del dolor lumbar, será preciso conocer que la inervación de la columna lumbar se hace fundamentalmente por tres ramos nerviosos: ramo dorsal de los nervios espinales lumbares (o rami dorsal), nervio sinuvertebral de Luschka y ramos ventrales de la cadena simpática. Existen dos tipos de patrones de dolor en la columna lumbar: el dolor irradiado y el dolor referido.

La cascada de la degeneración consta en tres estadios. El primer estadio sería la disfunción. El anillo fibroso se fisura y pierde la capacidad de contener al núcleo pulposo. Esto ocasiona primero el síndrome de disrupción discal y, si el núcleo supera el contorno del *annulus*, las hernias discales. El segundo estadio de *Kirkaldy-Willis* es el de inestabilidad. En este estadio la movilidad en el segmento móvil aumenta de forma patológica. En esta revisión describiremos los signos radiológicos asociados a esta inestabilidad. La tercera fase es la de estabilización, caracterizada por la estenosis, asociada o no a inestabilidad.

Palabras clave: *lumbalgia, discopatía lumbar, degeneración columna lumbar.*

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Low back pain is a frequent pathology that bears enormous socioeconomic consequences. Two thirds of the adult population suffer from pain in the lower back at some point in their lifetime[1]. In Spain, the pathology affects four and a half million people, with a prevalence of almost 15 percent in the population of over 20 years of age[2].

PHYSIOPATHOLOGY OF DISC DEGENERATION

Disc degeneration conditioning factors

There are many factors that lead to the degeneration of the intervertebral disc; they can be grouped into the following categories: age, genetic factors and environmental factors.

Age

The degenerative phenomena of the spine are part of the normal ageing process. Degeneration begins in the second decade of a man's life and in the third decade in the case of women. At the age of forty, 80% of the discs in men and 65& in women are degenerated[3].

Genetic factors

These factors have been proven in studies on twins[4], as well as in studies of the relatives of patients treated for lumbar disc herniation.

Environmental factors

The percentages for disc degeneration observed through magnetic resonance imaging (MRi) are notably higher in smokers than in non-smokers. Tobacco reduces the vascular supply of the disc through the endplates, causing hypoxia and degeneration, as well as a reduction in the production of type II collagen in the nucleus[5]. Other conditioning factors are heavy work, the practice of certain sports, etc.

Insofar as lumbar spine degeneration is a habitual ageing process, it is the appearance of certain symptoms—mainly pain—that indicates that the so far innocuous physiological process has become pathological. Most publications explain that the earliest degenerative changes occur in the intervertebral disc and subsequently affect the facet joints[6].

Intervertebral disc composition

Discs function as stabilizing and shock-absorbing intermediaries between two vertebrae. Hence, they must possess certain mechanical properties that derive from their composition. A disc is formed by three parts: (i) the *nucleus pulposus*, or central portion. The *nucleus pulposus* is a gelatinous highly hydrated matrix formed by proteoglycans, collagen and a low number of cells. Proteoglycans are extremely hydrophilic and they regulate the water content in the nucleus. The degree of hydration will have a direct effect on the resistance to compression of the nucleus. Proteoglycans also regulate the passage of solutes through the extracellular matrix. Having a negative charge, they facilitate the passage of small molecules (mainly glucose) and of positively charged ions (sodium and calcium). Collagen provides the nucleus with a framework in which both proteoglycans and cells can settle. It also plays a major role in the transmission of pressure within the nucleus. Collagen type II predominates in the nucleus (80%), the remaining part being formed by collagen types VI, IX and XI. The scant number of cells has a fundamental role in the maintenance of the extracellular matrix, which provides the *nucleus pulposus* with its mechanical properties.

The *annulus fibrosus is the external part of the disc. It is formed by several concentric fibrous layers that surround the nucleus pulposus; these layers are called «lamellas». Each lamella is positioned perpendicularly to the following one. The main element in their composition is collagen. Type I collagen predominates in this area of the disc (80%), accounting for 70% of the dry weight of the fibrous ring (it constitutes only 20% of the nucleus pulposus). The high density of the collagen together with its spatial arrangement lends the fibrous ring its high tensile strength. It can be seen that the proportions of collagen types I and II vary inversely between the innermost area of the disc (80% type II in the nucleus pulposus) and the external area (80% type I in the outermost areas of the fibrous ring)*[7].

The third element of the disc is the *hyaline cartilage of the intervertebral plate*. This cartilage is formed by chondrocytes and an extracellular matrix (collagen, proteoglycans, and water). It plays a major role in the transportation of solutes from the vertebra (which is a vascularized element) to the disc (an avascular element). The main mechanism of transportation is diffusion. Because of this, the alteration produced by the degenerative phenomena in this structure will play a fundamental role in the degeneration of the disc.

Intervertebral disc nutrition

The intervertebral disc is the largest avascular structure in the human body[8]. Within the intervertebral disc, there is low oxygen tension that gives rise to a cellular metabolism based on anaerobic glycolysis and hence a low local pH due to the high lactate production. It is fundamental that there should be a balance between pH, oxygen tension and glucose concentration. This last factor is the most decisive one in regard to cellularity[9].

In the vertebral body there are capillaries that penetrate the subchondral space of the endplates. Nutrients are delivered to the disc by diffusion from the capillaries and through the cartilage. Only the most peripheral zones of the disc (outer zone of the annulus) are nourished by direct blood supply.

Intervertebral disc degeneration

Biochemical disc changes

During the process of degeneration, discs undergo a reduction in height. This phenomenon causes a loss of vol-

ume, principally due to a decrease in the water content in the extracellular matrix. This event is a consequence of the decline of the synthetic process and of an increase in protein degradation in the extracellular matrix.

One of the first changes is the decline in the synthesis of proteoglycans and, as a result of this, the loss of the capacity to retain water. This change is observed mostly in the *nucleus pulposus*. Together with degeneration there is also an alteration in the production of collagen and an increase in the production of abnormal collagen[10].

It is at present believed that the process of disc degeneration originates in the endplate. In fact, the degeneration of the proteoglycans of the facet joint cartilage precedes that of the disc proteoglycans[11]. Another mechanism that is involved in the reduction of cellularity is apoptosis or programmed cellular death.

Collagen is the most abundant protein in the disc matrix. A local, abnormally intense activity of collagen-degrading enzymes, the metalloproteinases, has been observed. This activity is fundamental in the process of disc degeneration[12]. The development of this type of protease in the disc has been recently connected with tears and common disruptions in degenerate discs. This finding will open up a new path for therapeutic investigation in disc degeneration directed particularly towards the search for metalloproteinase-inhibiting substances[13].

Structural disc changes

When it is undergoing the process of degeneration the *nucleus pulposus* acquires a fibrous texture and a darker pigmentation. The boundary between the nucleus and the fibrous ring becomes fuzzy and there begins a delamination in the outer zones of the *annulus fibrosus*. These delaminations of the outer layers of the *annulus fibrosus* might constitute a precursory stage in the formation of concentric fissures[14]. Under microscopic observation, various alterations have been found: a reduction in the caliber of the collagen fibers, an increase in the production of type II collagen, a decrease in the production of type I collagen[15], a decrease in the amount of collagen in the nucleus, as well as apoptosis phenomena in the local chondrocytes, with pigmented cell inclusion bodies. The first change observed in the endplate is its separation from the adjacent subchondral bone. After adulthood, the cartilage of the growth plate undergoes a process of extensive mineralization, occasionally being substituted by bone[16].

A healthy disc is avascular; however, blood vessels may also be found in discs with severe degeneration[17]. These capillaries are thought to penetrate into the disc through fissures in the endplate. Angiogenic factors, inflammatory cells (macrophages) and proteases have also been found.

STAGES IN DISC DEGENERATION

According to Kirkaldy-Willis[18], disc degeneration can be divided into three stages:

Dysfunction

This is the first phase. It occurs between ages 20 and 45. The disc begins to lose its capacity to bear axial weight and its height diminishes. This event is related to the decrease in the water content of the *nucleus pulposus*, which is in turn due to the reduction in the number of proteoglycans. The degeneration of the disc will be followed by the degeneration of the facet joints. In this phase signs of synovitis in the joints can already be observed.

Instability

This is the second phase. Normally, it affects people of between 45 and 60-70 years of age. When a person is standing, a disc bears 80% of the axial weight and the facet joints bear the remaining 20%. The loss of height of the disc during degeneration gives rise to the redistribution of weight and, in some cases, 70% of the axial weight is passed onto the facet joints[19]. This produces a vertical subluxation of the facet joints and instability. The overloading of the facet joints is inversely proportional to disc height. Thus, as the disc collapses, arthritic phenomena appear in the facet joints, together with a loss of tension and the thickening of the ligamentum flavum and the posterior longitudinal ligement. All these elements contribute to the formation of soft stenosis. The loss of sagittal stability also determines stenosis, which is dynamic at first, and increases both with extension and on standing.

Stabilization

This is the third phase. It appears in people over 60 years of age. Due to the overloading of the facet joints, bone spurs, or osteophytes, are formed. These increase the contact area and thus stabilize the area. Bone spurs are localized in the endplates and in the facet joints, and they contribute to the stenosis of the spinal canal. This is a rigid stenosis, which will be added to the soft stenosis caused by the ballooning of the disc together with the thickening and the creasing of the ligamentum flavum. Structural stenosis will be followed by a stenosis caused by static instability, which may occur either with degenerative listhesis, in which the posterior facet joints fail symmetrically or asymmetrically in the form of rotational dislocations.

Several papers have shown significant variations—dependent on the position of the spine—regarding the dimensions of the central medullary canal and the foramen. It has been proven that transition from flexion to extension reduces the central sagittal diameter and the area of the

medullary canal. There occur similar variations in the volume of the dural sac, which increases significantly during flexion[20].The area, height and volume of the foramen increase during flexion and decrease during extension[21].The thickness of the ligamentum flavum increases during extension and decreases during flexion[22]. Disc protrusion behaves in the same way. All this explains the improvement of clinical symptoms in cases of stenosis in which the lumbar spine is placed in kyphotic angulation.

INNERVATION OF THE LUMBAR SPINE. SOURCES OF PAIN

The innervation of the lumbar spine branches out into three rami[23] (Fig. 1).

Dorsal branch of the spinal lumbar nerves or dorsal rami

This branch develops from the thick anterior branch at the exit of the foramen. It runs dorsally, between the transverse processes, innervating the most posterior structures: facet joints, ligamentum flavum, supra and interspinous ligaments, spinal muscles and adjacent dorsal skin.

Luschka's synovertebral nerve or meningeal nerve

This nerve is the result of the junction of a small nerve of the anterior branch with another nerve from the sympathetic system. It penetrates retrogressively through the joint cavity in the spinal canal. It innervates the dura mater, the posterior common vertebral ligament and the posterior area of the disc.

Ventral branches of the sympathetic system

They innervate the anterior common vertebral ligament and the anterior and lateral zone of the disc.

PAIN PATTERNS IN LUMBAR SPINE

Various structures may be sources of pain in the lumbar spine. The vertebral foramen and the facet joints have often been found to be involved in the production of low back pain.

Vertebral foramen

The nervous root and the dorsal spinal ganglion are situated at the level of the foramen. Several studies have shown the sensibility to mechanical compression of the spinal ganglion[24]. Certain neuropeptides have been found to be involved in the production of pain, chiefly substance P and the neurological growth factor (NGF) [25], both of which perform as nociceptive mediators.

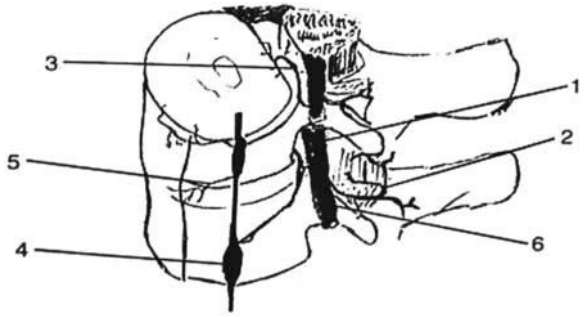

Figure 1. *Innervation of lumbar spine. 1: spinal nerve; 2: dorsal branch of the spinal nerve; 3: Luschka's synovertebral nerve; 4: sympathetic trunk; 5: ventral branches of the sympathetic trunk; and 6: ventral branch of the spinal nerve.*

The intervertebral disc is an avascular structure. In degenerated discs, however, there is an anomalous proliferation of vessels and intradiscal nerve terminations. In these terminations, both substance P and the genetically calcitonin-related peptide (GCRP) were found[26]. It is theorized that these substances perform a local role as pain mediators.

Facet joints

Facet joints may also be a source of pain. They are innervated by the fine dorsal branch of the spinal lumbar nerves. The pain may have a mechanical or an inflammatory origin. As was discussed above, as the disc loses height, the axial weight on the facet joints may increase from 20% to 70%. This mechanical overload causes different changes in the joints. The following are the changes that bring about pain: hyperpressure of the subchondral bone, trabecular microfractures, capsular distension or impingement of the synovial villi. Proinflammatory substances (substance P, etc.) are also released here. The distribution of pain in the spinal column takes place according to one of the following typical patterns:

1. Irradiated pain. The compression of the anterior spinal branch will produce irradiated pain. This is true or sciatic radiculalgia. It might cause a motor or a sensitive deficit and abolition of the osteo-tendinous reflex (OTR). Lasegue's sign is usually positive. This test is positive if there is radicular or sciatic pain when elevating the leg between 35 and 70°, with the knee fully extended[27]. The test is not positive if it only produces low back pain, since the pain should reach the whole dermatome that was affected in the corresponding lower limb.

2. Referred pain. The irritation of the synovertebral nerve of Luschka is caused by the ballooning of the outermost region of the annulus fibrosus, and also by irritation on the adjacent dura mater. The irritation of the dorsal rami occurs at the level of the facet joints. This causes referred

pain, which is also known by other names. This pain does not reach the end of the dermatome and its location varies depending on the level involved: L4-L5 (greater trochanter), L5-S1 (groin). This distribution is misleading in the sense that it can make the problem be mistaken for a hip problem or for trochanteritis. The nociceptive information of these branches is confused at central nervous level with that of the thick anterior spinal branch. This causes a kind of pain, called «pseudoradiculalgia» that is similar to radicular pain. In this condition, there is no sensitive, motor or reflex deficit and Lasegue's sign is negative.

Table 1. Syndromic groups, correlated with studies described by Kirkaldy-Willis

1. Stage I.	Dysfunction, discopathies and tears caused by overloading
	Crock's internal disruption syndrome
	Lumbar disc herniation
	Facet joint syndrome
2. Stage II.	Instability, dynamic instabilities
3. Stage III.	Stabilization, stenosis
	With static instability
	Degenerative listhesis
	Rotational subluxation
	Degenerative scoliosis
	Without static instability
	Lumbar canal stenosis

SYNDROMIC GROUPS OF THE DEGENERATE LUMBAR SPINE

They are correlative with the three stages described by Kirkaldy-Willis[18] (Table 1).

Discopathies and tears caused by overloading

This stage correlates with disc dysfunction, without there yet being any signs of instability. There are different modes of presentation, which may occur in isolation or simultaneously: Crock's internal disruption syndrome, lumbar disc herniation, and facet joint syndrome.

Crock's internal disruption syndrome[28]

There is a rupture in the internal structure of the disc that occurs together with discogenic pain, and without compression of the nervous root. The degenerated *nucleus pulposus* produces inflammatory mediators that reach the nervous terminations in the periphery of the *annulus fibrosus* and irritate them.

The clinical situation is not specific. Patients present with pseudoradicular pain: negative Lasegue, normal OTR and irradiation extending, maximally, to the knee.

Most authors agree that discography is the fundamental diagnostic test. The contrast injection in the involved disc should cause a type of pain that is similar to the one suffered habitually by the patient. As regards the morphology of the disc, it should have a disruption extending up to the outer area of the *annulus fibrosus*. In fact, and with the aim of obtaining greater precision, the diagnosis should include a painless discography of the healthy disc as well. This test has several drawbacks: false positives, discitis (in a recent review of 12,700 discographies carried out on over 4,800 patients the percentage for discitis was found to be 0.25[29]). Moreover, a positive preoperative discography does not ensure better surgical results[30].

These results have led to the use of MRi. However, it has been found that his method, when carried out in isolation, cannot diagnose the internal disruption syndrome: there are data showing asymptomatic patients with signal changes at T2 weighted MRi. In one of these studies, Boden[31] found that, out of a group of individuals under 60 years of age, 20% had discopathies and, in the case of those of over 60 years of age, 36% had discopathies and 21% also showed signs of having a narrow lumbar canal. With the aim of finding a solution to this problem, some studies have emphasized the significance of the relationship between lumbar pain, disc signal change in MRi and positive discography[32].

Disc herniation

We consider disc herniation to be the migration of the *nucleus pulposus* outside of its normal location. This egression may be towards the periphery, through a torn *annulus fibrosus*, or cranio-caudal (Schmorl's herniations). The levels most frequently involved are: L4-L5 and L5-S1.

The terminology for the different kinds of herniation and disc lesions is the following[33]:

1. Disc tears. This is a localized disruption (radial, concentric or horizontal) of the *annulus fibrosus*. There is no migration of disc material outside the limits of the intervertebral disc.

2. Disc herniation. In disc herniation there is migration of disc material outside of the normal confines of the disc. The morphology of disc herniation varies (Fig. 2): a) a *bulging* disc refers to a symmetrical and vast enlargement of the disc (over 50% of the disc perimeter) that is not considered pathological; b) *disc protrusions* are common in asymptomatic individuals and involve displacement of the disc outside of its normal boundaries, the implantation base having the greatest diameter (sessile aspect); c) *disc extrusions* are displacements of the disc outside its normal boundaries in which the implantation base does not have the largest diameter (pedicled aspect) and which are usually found in symptomatic individuals; and d) *disc sequestration* is a severe kind of herniation found in symptomatic individuals in which the expelled material loses contact with the disc and remains free, and in which spontaneous resorptions

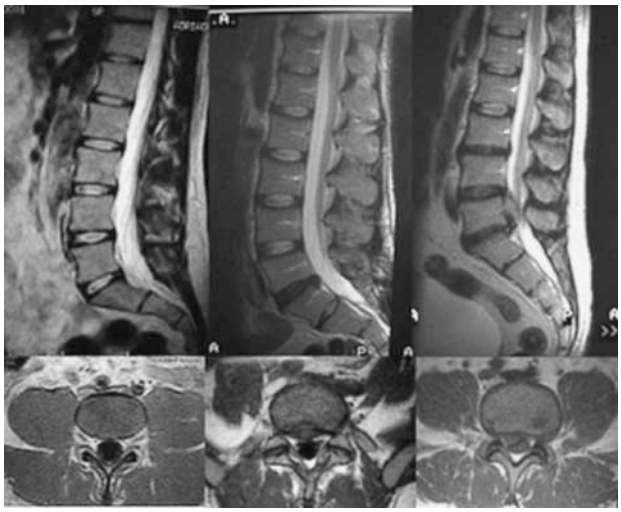

Figure 2. *Degrees of discopathy. T2-weighted magnetic resonance imaging with sagittal and coronary planes in three patients. Left-hand column: normal discs. Central column: bulging (symmetrical enlargement, over 50% of the disc, not deemed pathological). Right-hand column: true disc protrusion with compression of the dural sac.*

are frequent. In our environment, and due to the fact that it is considered a serious condition, the term «disc herniation» is used only in the case of extrusions and sequestrations.

In the MRi axial planes we can observe the localization of disc herniation: central, foraminal and extraforaminal. Apart from providing information about the disc morphology, MRi also informs about changes in the signal of the disc. With degeneration, discs dehydrate. The loss of water makes the disc signal in T2-weighted sequences diminish and the disc is obscured. Authors such as Lam[34] and Milette[35] have noted that, in the case of individuals with low back pain, these signal changes are almost as meaningful as a pathological discogram.

Endplates also undergo changes caused by the process of degeneration. Modic[36] classified them into three types. Type I (hyposignal in the T1-weighted image and hypersignal in the T2-weighted image) is also known as «edema-like endplate signal change» and is characterized by severe disc degeneration. Type II (hypersignal in the T1-weighted image and iso/hyposignal in the T2-weighted image) is also known as «fat-like endplate signal change». The latter is more frequent and stable in time and has been found to be associated with other degenerative changes already visible on x-rays. Lastly, type III (hyposignal in T1 and T2-weighted images) is notably less frequent than the first two and associated with bone sclerosis-type changes in x-rays. Type I is more often associated with low back pain and type II is most frequently found in asymptomatic patients.

Facet joint syndrome

Facet joints obtain rich innervation from the dorsal branch of the lumbar spine nerves, each facet thus being innervated from two different levels of the spine. The synovial membrane contains abundant nervous terminations that supply the facet joint with nociception and mechanoreception.

Isolated prevalence of facet pain is low—between 7 and 15%. Lumbar facet pain is localized in the low lumbar area, in the gluteus area and in the upper-posterior thigh. It is a referred pain or pseudoradiculalgia with characteristics similar to those of the internal disruption syndrome pain. The pain worsens during extension of the spine and during lateralization towards the affected side.

Saline injections have been used as a challenge test. Corticoids, especially methylprednisolone, have been used in the treatment of the facet joint syndrome. The results obtained with this technique, as far as improvement of pain is concerned, have been poor. Denervation through radiofrequency has also been used in patients with facet joint syndrome and it has been partially successful[37]. It was observed that the patients that respond best to this treatment are the same ones that improve with the corticoid injection[38]. After this process, there is frequently re-innervation of the facet joint one year later approximately. These clinical results can be used to strengthen rehabilitation treatments.

Dynamic instabilities

Instability was described by Knutsson in 1944. Later work by Morgan attributed 25% of the cases of low back pain to instability phenomena. Works by Kirkaldy-Willis and Farfan, which considered instability as the second stage in disc degeneration, contributed to the final consolidation of this concept.

Junghanns mobile segment comprises two adjacent vertebrae and the soft tissue between them. It is the smallest functional unit in the spine that maintains all its biomechanical properties. The global mobility of the spine is the sum of the movements of all its mobile segments. The disc and facet joints are its most important elements. Instability is generated within this mobile segment. As the disc degenerates various events take place:

1. Shear stress on the disc and the facet joints increases. The orientation of the facet joint is important: The more sagittal it is, the greater the likelihood of listhesis in the segment[39].

2. There is abnormally high traction on the edges of the disc ring, due to the ligaments that are attached to it, giving rise to deformities that Macnab has termed traction entophytes.

3. Disc degeneration phenomena (progressive dehydration caused by loss of proteoglycans, etc.) bring about a loss of disc height. We can measure the instability of a particular mobile segment by means of functional lateral x-rays of the lumbar spine, during flexion and extension. The shifting of vertebrae and angulation between adjacent vertebrae in the

Table 2. Limit values for angulation and shift according to different authors

Author	Shift (mm)	Angulation (degrees)
Hayes et al L1-5	2-3	7-13
White et al L1-5	3	13
Kanayama et al L1-5	4	10
American Medical Association	4	11

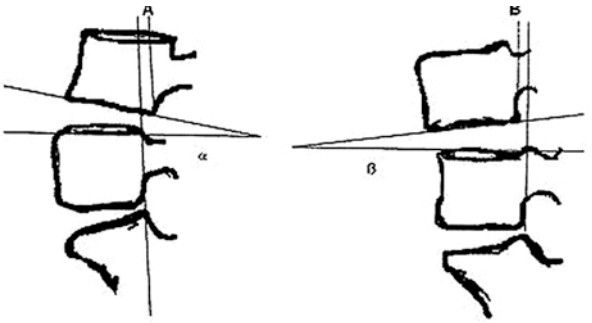

Figure 3. *The lumbar segments with a shift exceeding 4mm (distance A-B > 4 mm) and/or an oscilation of over 11 degrees (·-, > 11°) are rated as unstable.*

segment will be measured when they change their position. Table 2 shows the limit values for angulation and shifting accepted by various authors. We rate as unstable lumbar segments with shifts that exceed 4mm and/or an oscilation of over 11 degrees (Fig. 3).

Stenosis

This correlates with the third stage of stabilization described by Kirkaldy-Willis. The structuring of degenerative lumbar phenomena occurs with ageing (especially after the age of 55-60).

Stenosis with static instability

Stenoses are classified according to whether they entail static instability or not (Table 1). The Frymoyer and Selby[40] classification of degenerative instabilities encompasses all the phenomena of static instability. They are classified into primary and secondary. Secondary static instabilities occur after surgery (laminectomies, discectomies and failed fusions) or after percutaneous procedures (chemonucleolysis). The primary static instabilities are:

1. Type I. Rotational axial instability. It generates a rotational subluxation that can be observed in anteroposterior x-rays showing malalignment of the spinous processes and of various pairs of vertebral bodies.

2. Type II. Shift instability or degenerative spondylolisthesis. (Fig. 4). The following can be seen in lateral x-rays:

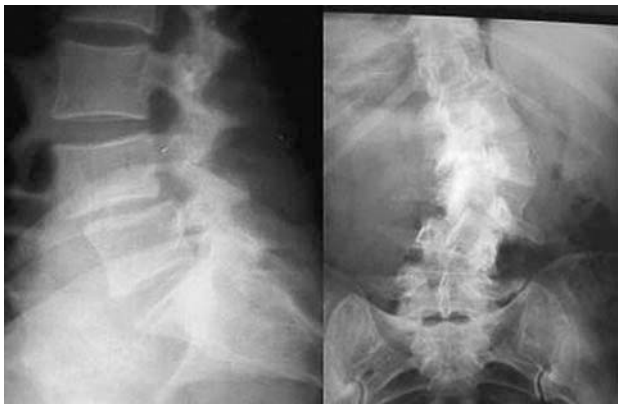

Figure 4. *Shift instability. Left-hand image: L4-L5. degenerative listhesis. Right hand image: degenerative scoliosis with rotational dislocation.*

traction osteophytes, a narrowing of the intervertebral space and displacement of one vertebra over the underlying one. The segment that is most frequently affected is L4-L5 and the condition rarely progresses from a degree I/II according to Meyerding[41]. This type of instability is more common in women, especially after age 60.

3. Type III. Retrolisthesic instability. It is frequently found in L5-S1 and is more common in men with a history of low back pain.

4. Type IV. Degenerative scoliosis (fig. 4). These scolioses develop *de novo* in individuals over 20-25 years of age. The global prevalence of this condition varies depending on the age range: 2% before 45 years of age and 15% after 60 years of age[42]. They are often associated radiologically with a loss of lumbar lordosis and with rotational dislocations (mainly in the middle lumbar zones L2-L3 and L3-L4). Recently, various radiographic signs in cases of degenerative scoliosis have been significantly related to a greater incidence of pain[43]: maximum degree of rotational dislocation, obliquity to the horizontal of endplates L3 and L4, trunk imbalance at T1-S1, etc.

In this classification we have omitted a fifth type, internal disc disruption, whose association with instability is at present under discussion.

Stenosis without static instability

Lumbar canal stenosis

It consists in the pathological narrowing of the spinal canal, of the lateral recess and/or the foramen, and in most cases it is of degenerative origin. The most widely accepted etiological classification is Arnoldi's[44] (Table 3).

Topographically, we can divide the lumbar canal into three areas: the central canal, the lateral recess and the foramen (Fig. 5).

At the level of the central canal, the normal value of the spinal canal at mid-sagittal level normally exceeds 15mm. When it is between 10 and 12mm, there is relative

Table 3. Arnoldi's classification of lumbar canal stenosis[44]

1. Congenital/ with ageing
Idiopathic
Achondroplasic/hypochondroplasic
2. Acquired
Degenerative
Congenital-degenerative combined
Spondylolythic/spondylolisthesic
Post-traumatic
Iatrogenic, postlaminectomy, post-lumbar fusion, post-chemonucleolysis
3. Metabolic
Paget's disease
Fluorosis

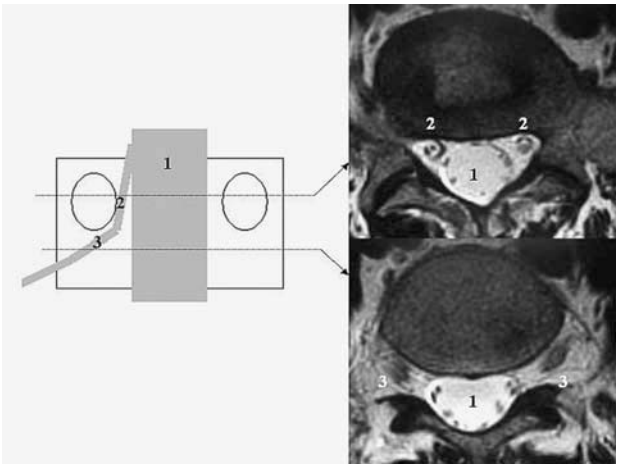

Figure 5. *Lumbar canal zones. 1: central canal with dural sac; 2: lateral recess; 3: foramen.*

stenosis, and when it is inferior to 10mm, there is absolute stenosis[45].

The lateral recess is the middle zone of the vertebral pedicle that is in close contact with the vertebral root emerging from the dural sac. The high lumbar spinal roots are parallel to the pedicle and at an almost perpendicular angle (L1- L2 at 80°) whereas the low roots are suprapedicular and oblique (L5-S1 at 45°). It is because of this that there exists a true lateral recess only at the levels of L4, L5 and S1, and only to a lesser degree at L3. Failure to decompress the root in the lateral recess has been mentioned as one of the principal etiologies of the *Failed Back Surgery Syndrome. Thus, the partial facetectomy of the articular process has been widely recommended to decompress the root at the level of the lateral recess.*

The foramen is the exit zone of the spinal roots of the vertebral bony structure. Its dimensions vary depending on the posture of the spine (Fig. 6). Disc height loss contributes to foraminal stenosis in several ways. Hasegawa considers the following situations to be critically important: a foramen

Figure 6. *The size, height and diameter of the foramen increase with flexion (right-hand image) and decrease with extension (left-hand image).*

height of under 15mm and a posterior disc height of under 4mm; the overgrowth of the boundary structures of the foramen (disc, ligamentum flavum, osteophytes, etc.) and the progressive subluxation of the facet joint above a given vertebra, which could contribute to the stenosing of the foramen.

Canal stenosis manifests clinically with pain, neurogenic claudication and/or radicular compression symptoms. Neurogenic claudication should be distinguished from vascular claudication, taking into account that it improves by positioning the lumbar spine in kyphotic angulation. This claudication occurs by stenosis of the central lumbar canal. The radicular symptoms appear by compression of the root at the level of the lateral recess or of the foramen.

CONCLUSION

In the first place, it must be stated that a thorough knowledge of the physiopathology of degeneration and of lumbar spine pain helps to distinguish between the physiological and the pathological aspects of this condition; and in the second place, it must be said that the understanding of the physiopathology of degeneration is useful for establishing the etiopathogeny of pain in each patient, thus allowing us to place them within one of the syndromic groups of degenerative lumbar spine. This will guide our decisions and help us offer the patient the most adequate treatment.

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Conflict of interests

The authors have declared to have no conflict of interests.