

LOW BACK PAIN: CLINICAL EVIDENCE FOR “THE DURAL CONCEPT”

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Lumbogluteal pain is considered nowadays as having many different possible causes or even being multifactorial in origin. Literature shows that backache is a-specific in 85 % of the cases.^{24 80} Because each tissue in the back is innervated, in theory any of this can be responsible for pain. As a result, every tissue has been inculpated as a source of back pain: disc, zygapophyseal joints, ligaments, sacroiliac joints, muscles, nervous structures. In the last decade also the biopsychosocial aspect has been stressed.⁹⁰

We believe that this lack of consensus about a clear diagnosis for patients with low back pain has two reasons. First, there is no agreement about how to examine patients who suffer from the back.^{89 24 60} A solid history and a standardized clinical examination should lead to diagnosis or at least to a classification which allows precise judgement. Secondly, the technical investigations are not reliable enough to diagnose low back pain. Imaging studies commonly display pathology that is not responsible for the patient's symptoms.^{8 9 35 93} They rather serve to confirm a tentative diagnosis or to exclude serious pathology. Provocative discography, which has been put forward as the primary tool to determine the true pain generator in case of disc lesions⁷⁵, also has clear limitations. Although a good indicator for disc degeneration, discography has been criticised as being an accurate test for discogenic pain⁷³, mainly because pain reports are often influenced by the personality of the patient and chronicity or litigation claims.^{14 15 16 7} In individuals with disc degeneration and annular defects, discography may elicit pain with injection whether the patient is symptomatic with serious low back pain or not.¹⁶ False negative discograms have been reported when the protrusion or prolaps involves the annulus fibrosus rather than the nucleus.⁹⁹

Three premises

Cyriax²³ has put forward three premises:

1. Of all cases of low back pain, with or without radiation down the lower extremity, a majority is caused by the disc.
2. The disc displacement is not in itself a painful condition (“the intervertebral joint is insensitive to internal derangement”).
3. The pain is the result of an interaction between the disc and the dura mater (“the pain mechanism is dural”).

This “dural concept” is based, on the one hand, on the recognition of a clinical picture which is compatible with a disc (“It behaves like a disc”), and, on the other hand, on the analogy with the picture of a sciatica, which is caused by a disc. We do not discuss here the minority of patients with non-discal sciatica (e.g. in lateral recess stenosis) or with pseudoradicular pain (e.g. referred pain from another soft tissue lesion in the hip region).

The role of the intervertebral disc

The ‘disc theory’ is not generally accepted. Many authors, for example, stress the importance of the facet joints^{52 72}(manual therapy) or of the sacroiliac joints^{100 25 50 77} (osteopaths). Among the ‘disc-believers’ Cyriax and McKenzie are the main protagonists. They consider the disc as an important cause of back problems, but do not agree about the explanation of the sources of pain. Cyriax believes the pain is generated from the dura mater²², whereas McKenzie⁵⁷ accepts the theory of primary discogenic pain. Although nerve endings have

been described in the outermost lamellae of the normal human disc^{27 67}, and nerve ingrowth was observed in degenerated human discs^{28 29 36 17}, it is still not fully clear if the disc by itself can be responsible for back pain or leg pain. It suffices to look at the high rate of false positive results with imaging techniques as MRI and CT-scan^{8 9}, or discography⁹⁹. The internal disc disruption theory is also challenged by the natural evolution of disc disease which shows that, from the age of 60 –when the degeneration gets ‘at the top’ – the frequency of symptomatic periods drops rapidly⁴⁴, demonstrating that internal degeneration of the (innervated) disc cannot be the main reason for low back pain.

It behaves like a disc

Many patients with low back pain have a clinical picture that is compatible with a disc lesion. The problem is episodic in nature and the presentation may differ from one attack to another. The onset is known: symptoms mostly start as the result of a precipitating posture, movement or activity. The pain is usually exacerbated in sustained sitting or forward bending of the trunk, positions which are known to increase intradiscal pressure^{59 74}, relief is found in horizontal positions. There is a clear inherent dynamic which shows as the symptoms change (centralize, peripheralize) under the influence of movement or posture. The picture of an internal derangement in the intervertebral joint supervenes: the patient gets painful twinges on movement and the articular movements may become locked in some directions and not in others. Limitation can be minor or major dependent on the volume of the displaced fragment of disc.

Sciatica: a discoradicular interaction

Since the famous article by Mixter and Barr in 1934⁵⁸, actualized by numerous later publications, sciatica has widely been accepted as being the result of a discoradicular interaction. A posterolateral displacement of disc tissue impinges and/or irritates the dural sleeve of the nerve root⁸². This leads to segmental pain which is projected in a dermatomal area^{42 62}. Most commonly the L4 or L5 disc interacts with the L4, L5 or S1, S2 nerve root⁴². Root pain as the result of an L3 prolaps is much less common. Increase of the compression may also affect the nerve root parenchyma leading to sensory, motor and reflex disturbances.

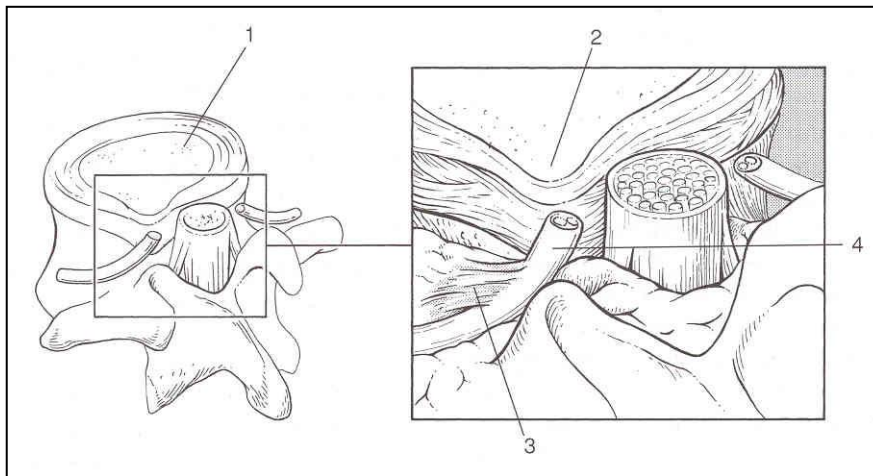
The clinical picture of a discoradicular sciatica is well known^{2 21}.

In the *history* the patient mentions previous episodes of acute lumbago or backache^{88 60}. The pain often started in the centre of the back after which there was a tendency to unilateralize, but not necessarily at the same side. The pain was clearly related to movements or activity. During the present attack, which also began in the back, the pain peripheralized completely, went into the buttock region and further into the leg. By the time the pain reached the leg, the backache had disappeared or at least diminished substantially – shifting pain. The actual leg pain is quite severe, burning and confined to one aspect of the lower extremity (lateral – L4 or L5, or posterior – S1 or S2) and may spread as far as the foot. Coughing may hurt the leg^{42 40}, as do some movements, i.e. flexion. Once the pain is experienced distally it is very often accompanied by sensory disturbances in the same area (paraesthesia, hypoaesthesia) and in a number of patients neurological deficit supervenes.

On *functional examination* an antalgic posture may be found, usually away from the painful side^{81 54}. Movements influence the symptoms in an asymmetrical way and especially the nerve tension tests increase the symptoms: neck flexion may hurt the leg and so does SLR which is usually painful and limited, unilaterally and sometimes bilaterally^{43 42}. Manual neurological screening may reveal sensory and motor deficit and impaired reflexes⁸⁵.

Because a duality is found in the symptoms and signs, interpretation leads inevitably to the conclusion that a discoradicular interaction is responsible. This duality shows at the level of the symptoms (history) and the signs (examination). The articular symptoms and signs build a picture which ‘behaves like a disc’: lumbar movements, i.e. flexion, give rise to painful twingic pain down the leg. The severe pain may force the patient in an antalgic

position and the movements are painful and/or limited in an asymmetrical way. The radicular symptoms (pain and paraesthesia) and signs (pain on neck flexion and SLR, and possibly deficit) have a segmental distribution and thus indicate fairly accurately which nerve root is involved^{70 42}.



Acute lumbago: a discodural interaction

What strikes is the similarity of the clinical picture. The only difference between the sciatica and the acute lumbago is the difference in localization of the pain: the sciatica-patient has mainly pain down the leg, the lumbago-patient has severe pain in the back and buttock region. The similar clinical picture forces us to believe that we are faced to the same pathology, i.e. an internal derangement in the intervertebral joint, caused by a displacement of discal tissue. In the same way as in sciatica the disc interacts with an irritated nerve root sleeve, it would seem logical to state that in lumbago the interaction occurs between the disc and the dura mater and its surrounding structures.

Acute lumbago also 'behaves like a disc'. The *history* also discloses an episodic condition. Lumbago-patients get an attack from time to time and in between the attacks they are often completely painfree. They also mention a shifting of pain: from the centre to one side or from one side to the other, not necessarily during the same attack. Pain is exacerbated by coughing and sneezing. The *functional examination* shows a deviated patient^{84 69} with gross, but asymmetrical, limitation of movements. SLR is often bilaterally limited. All these tests cause severe pain in the lower back with possible radiation into buttock and hip.

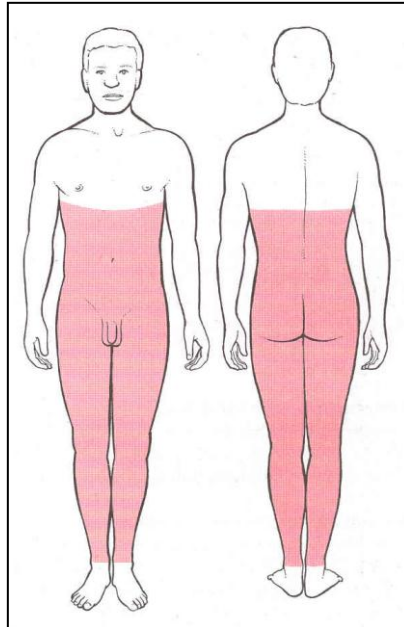
Interpretation of the clinical picture in acute lumbago

The different elements in the clinical picture of an acute lumbago can be interpreted logically and this leads to the following conclusions.

Multisegmental pain

Contrary to sciatica where the pain is confined to a dermatomal area ('segmental' pain), acute lumbago gives rise to vague widespread pain, i.e. the lower back, buttock, groin, lower abdomen and upper thigh. This multisegmental pain reference is the result of involvement of the dura mater which is multisegmentally innervated by a dense network of branches from sinuvertebral nerves from different levels^{31 32}. Immunohistochemical studies of the dura mater have illustrated a significant number of nociceptive fibers^{37 98 1}, and clinical experiments have demonstrated that the ventral dura is sensitive both to mechanical and chemical stimulation^{45 26 95 79}. Numerous studies point to the particularly noxious effect on the dura by inflammatory material produced by injured discs^{18 33 64 65 48 56}. Recent studies also report a sympathetic innervation of the dura^{97 41 78}. Extensive ligamentous connections exist between the ventral dura and the posterior longitudinal ligament (PLL)^{5 94 68}. These ligaments may play a role in dissipation of forces⁸³, and may also be responsible for pain if

movement causes them to pull on the inflamed PLL or dura^{5 68 45}. In conclusion it could be said that “the mechanism of discodural interactions is a conflict between an inert an mostly painless structure (nucleus and inner part of the annulus) and a pain-sensitive duroligamentous complex (outermost rim of annulus, PLL, dura mater and dural ligaments), all innervated by the sinuvertebral nerves”⁶⁶.



Shifting pain

The unilateral displacement of pain whereby the pain disappears at its original site to appear at another site instead is defined as “shifting”. And “shifting pain” would be synonymous to “shifting lesion”. This clinical situation is comparable to the shifting of symptoms when a renal calculus shifts from the kidney into the ureter: the pain moves as the lesion moves. In order to be able to shift there has to be a cavity. And because in lumbago the pain shifts from the centre to one side, it has to be a central cavity. The only central cavity is the spinal canal and the only structure capable to shift in this area is the disc.

Relation to activity, movement and posture

The condition is activity-related and can therefore be classified under the ‘mechanical backache’. Worst are those movements – bending, lifting, sitting – that induce raising of the intradiscal pressure and kyphosis which pushes the disc in posterior direction where it threatens the sensitive structures in the spinal canal, i.e. the dural sac containing the cauda equina.

Chronology

The pain starts in the centre, then spreads widely in different dermatomes, either bilaterally or unilaterally. There is a tendency to unilateralization and peripheralization. Further displacement in posterolateral direction would menace the nerve root and set up a series of symptoms, again in chronological order: pain – paraesthesia – deficit (see further).

The duality in the symptoms and signs of an acute lumbago

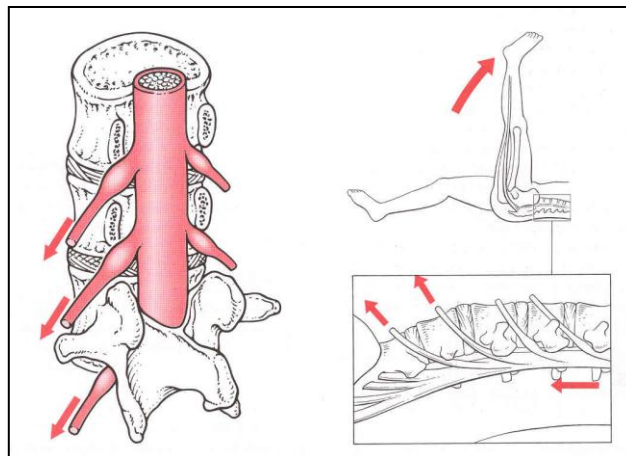
The dual symptoms and signs (radicular and articular), which were present in sciatica, are also recognizable in acute lumbago (dural and articular).

Dural symptoms and signs

Interaction with the sensitive dura mater leads to the ‘dural symptoms’: *pain* with a multisegmental reference. The *tender spots* on palpation that are found in the painful area are considered referred phenomena from the dura. A reason for this hypothesis lies in the

fact that these 'trigger points' also tend to centralize as the symptoms abate. Pain on coughing and sneezing is the result of an abrupt increase of compression: the high intra-abdominal pressure leads to sudden increase of the intravenous pressure, i.e. in the dural venous plexus.

The 'dural signs' develop as the result of interaction with the mobility of the dura mater. This mobility becomes impaired when a space-occupying lesion in the spinal canal compresses the dura, in this case the disc. Pain in the back is provoked on *neck flexion*, on *SLR* – which can also become limited – and possibly on the *N. femoralis stretch*. The vertebral canal lengthens considerably during flexion⁴⁷. The dura mater, a structure situated in the vertebral canal but anchored at the top and at the bottom will consequently move in the spinal canal. During flexion the stretched dura shifts towards a position of less tension and is pulled against the anterior wall of the canal^{101 71}. Anatomical studies demonstrated that flexing the neck moves neural tissue in the lumbar spine^{46 10 11 83}. In fact, neck flexion has been accepted for decades to be positive in meningeal irritation (Kernig's sign)^{3 86 63}. Straight leg raising drags the nerve roots L4 to S2 downwards and forwards³⁰. Since the nerve root is connected through its dural investment with the distal part of the dura, the latter will also be involved in the downwards movement. Therefore, straight leg raising drags on the dura mater and pulls it caudally, laterally, and forwards^{38 53}.



Articular symptoms and signs

The following symptoms can be considered articular: back pain on *movement*, painful *twinges* and momentary *giving way*, f. ex. during sneezing.

Articular signs are the *deviation*, which is fairly common, and the *asymmetrical pattern of limitation* of movement.

When an acute lumbago turns into sciatica

After several attacks of acute lumbago, whereby the central pain most often becomes unilateral, a next attack may lead to further peripheralization which causes sciatica. In that case we can observe a shift from a discodural interaction into a discoradicular interaction. This change can be seen at the level of all the symptoms and signs.

Pain

The 'multisegmental' character of the pain (bilateral or unilateral, and felt in the back, buttock, groin and abdomen) becomes 'segmental'. The backache disappears or becomes very secondary and is replaced by pain from the buttock into the leg, confined to one dermatome in case of a monoradicular sciatica, or to two dermatomes in case of a biradicular sciatica.

Coughing

Coughing and sneezing originally caused severe pain in the back. If they hurt in a sciatica the pain will mostly be felt in the buttock and/or leg.

Tension tests

Neck flexion and SLR, instead of hurting the back, will now affect the lower extremity. SLR – which mainly tests the nerve roots L4 to S2 – may also become limited. The N. femoralis stretch is painful in the anterior thigh in case of involvement of the L3 nerve root. The dural signs have turned into root signs.

Radicular symptoms and signs

The posterocentral disc prolaps turns into a posterolateral one, and this will affect the nerve root.

When the dural sleeve becomes compressed and/or irritated, apart from the segmental pain – which is the result of the segmental innervation by its own sinuvertebral nerve, the mobility can become impaired which results in limitation and pain on the nerve tension tests (see above).

Once the nerve root parenchyma becomes affected sensory and/or motor symptoms and signs may develop. They are all segmental: paraesthesia, numbness, sensory deficit, reflex disturbance and muscle weakness.

For example: the L5 disc affecting the S1 nerve root. The pain, paraesthesia and numbness are felt in the S1 dermatome: the calf, the outer side of the foot, as far as the 4th and 5th toe. The weak S1-muscles are: peroneal and calf muscles. The Achilles reflex is sluggish.

Diminution of the articular symptoms and signs

The disc tissue, which originally strongly disturbed the articular movements, shifts laterally and leaves the intervertebral joint. As a result the articular involvement becomes less important. This shows in the clinical picture. The deviation tends to diminish or becomes purely antalgic instead of merely mechanical. It increases as the nerve root becomes stretched, i.e. during flexion.

Less movements become positive. Often flexion is the most painful and limited movement, which we no longer consider an articular sign, but a root sign⁷⁶. The other movements (extension, side flexion) which were very limited in acute lumbago, in sciatica often become full range.

‘Comprehensible’ clinical situations

The acceptance of this ‘dural concept’ allows us to better understand some other clinical situations.

Asymptomatic disc bulging

Many imaging studies have stressed the frequent presence of disc displacements in an asymptomatic population^{8 9 35}. The internal derangement in itself does not seem to be painful. It is only when the disc enters in conflict with the sensitive tissue (either dura mater, or nerve root) that a symptomatic situation develops.

Painful arc during articular movements

During the functional examination of patients with low back pain a painful arc in one or more movements is a common finding. This situation of pain occurring half way a movement and disappearing again when the movement is continued, can only be explained by a temporary pinching. The dura mater enters in contact with a minor bulging disc and slips over the bulge whereafter it disengages again.

Root atrophy in severe sciatica

In cases of severe sciatica a sudden aggravation may occur. The patient experiences some hours or days of excruciating pain, whereafter all symptoms suddenly cease. The pain disappears and the SLR becomes full and painless. The nerve root has become ischaemic and the conduction stops: a gross palsy appears. The patient has become symptomatically better, but anatomically worse. There is a major protrusion, but no pain anymore.

Spontaneous recovery of acute lumbago and sciatica

Acute lumbago recovers spontaneously in one week to ten days¹⁹. The discodural interaction ceases as the disc tissue recedes under the influence of centripetal forces, partly originating in the tensed posterior longitudinal ligament.

The spontaneous cure of discoradicular interactions occurs as the result of three possible mechanisms: reduction, bony erosion or, most commonly, disc resorption^{34 87 49 12 61}. There is a cure of the pain, but also of the sensory and motor disturbances^{4 91}.

Diminution of backache as age advances

Disc lesions occur mainly in the age group between 30 and 60 years, after which this pathology tends to diminish or even disappear. This has to do with the normal ageing evolution of the spine. During the period of instability of the functional segment, also the disc becomes unstable. When, later in life, the spine stabilizes again – as the result of fibrosis of capsule and ligaments and the building of osteophytes – the chance of getting disc displacements becomes smaller.

Response to diagnostic epidural injection

A weak local anaesthetic, f.ex. procaine 0,5 %, is capable of causing a surface anaesthesia of the sensitive tissue, without interfering with the free nerve endings in the other structures in the anterior spinal canal – posterior longitudinal ligament, facet joint capsule – or with the sinuvertebral nerve. Disappearance of the pain inculcates the dura mater or the dural sleeve of the nerve root. Positive results with therapeutic epidural injections of local anaesthetics are reported for acute low back pain^{51 96 92 39 6}, and injections of a local anaesthetic in combination with hydrocortisone for sciatica^{13 55}.

The hypothesis of the dural concept

Sciatica is generally accepted as being caused by an interaction between the disc and the nerve root, partly as a problem of compression and partly as a chemical process whereby certain products released from the damaged and degenerating disc cause irritation of the sensitive tissue.

There are strong clinical arguments to accept that acute lumbago, just like sciatica, is also the result of a conflict between two partners, on the one hand, the same disc, and, on the other hand, the sensitive tissue – dura mater.

There should be no problem then also to accept that common low back pain can have the same pathogenesis, provided the same duality in symptoms and signs is found (articular and dural).

If the end stage of a disc displacement (sciatica) is accepted as a discoradicular interaction, what arguments could be used not to accept the preliminary stages of acute lumbago or backache as a discodural interaction, when exactly the same clinical picture is found and a proper explanation for the difference in pain localization?

These ideas have forced Cyriax to put forward his “dural concept” in The Lancet in 1945²⁰.

Clinical examination: how to detect a discodural or discoradicular interaction?

The recognition of the above mentioned clinical picture is, of course, only possible if the clinical examination is constructed in such a way that it allows the recognition of the dural, articular and radicular symptoms and signs.

In orthopaedic medicine the clinical examination contains a detailed history-taking – looking for the localization and the behaviour of the symptoms – and a functional examination – based on the principles of applied anatomy, as well as selective tension of the inert and contractile tissues respectively. The clinical reasoning is based on pattern thinking and on the recognition of clinical pictures.

This approach is perfectly well possible in the peripheral joints, but encounters some difficulties in the spine where all structures overlap and cannot be tested selectively. That is a reason why history-taking becomes the main part of the examination procedure. In the spine it is fair to start from a premise and we therefore take the disc, because in the majority of cases this will be correct. Following a perfect logical approach we then look for arguments in favour of this premise: dural, articular, radicular, medullar symptoms and signs, as well as for arguments in favour of alternative causes of back and leg pain.

When sufficient correct arguments are found, it can safely be concluded that indeed a discodural or discoradicular interaction is present.

The diagnosis of a disc should be entirely based on clinical grounds. Technical investigations are not of very much help in these cases.

Absence of a clinical picture of discodural or discoradicular interaction

In those cases where no or insufficient arguments are found to diagnose the disc as a cause of the symptoms, we have to look for other possible origins of pain. In case a disc pattern is found, but no dural or radicular symptoms or signs, it could very well be that the pain is indeed primary discogenic. In the absence of arguments in favour of the disc, the examiner has to look for other pathologies, for which again there should be arguments. Facet joint lesions, sacroiliac lesions, ligamentous or muscular conditions, or any other possible organic or non-organic pathology should be recognized by their own specific clinical picture, which will be different from the discal one. As Cyriax said: "All discs are alike and all other conditions are different."

Differential diagnosis

In the differential diagnosis of low back pain the visceral and psychogenic problems should be ruled out. In the orthopaedic group a distinction should be made between spinal and non-spinal conditions. Amongst the pathologies affecting the spine the activity-related or mechanical ones form the most important group. There are a great number of non-activity-related conditions, but altogether they affect a smaller number of patients²⁴. Further differentiation is possible between the ligamentous concept (postural syndromes and dysfunction syndromes, f.ex. facet joint lesions, sacroiliac lesions, ligamentous lesions), the stenotic concept (canal stenosis and lateral recess stenosis) and the dural concept. The latter includes the discodural interactions and the discoradicular interactions, either primary or secondary.

Therapeutic consequences

Classification of an important number of patients under the dural concept must have therapeutic consequences. The interaction between two partners can be stopped by focusing the therapy on one of the partners.

When there is a chance to influence the disc displacement, this can be done by a mechanical approach, such as mobilization, manipulation or exercise therapy, including prevention.

For those cases where the disc cannot be treated, therapy is directed to the sensitive tissue by means of local epidural anaesthesia or infiltration of steroid around the nerve root.

Surgery is the last resort and the process of spontaneous recovery should not be forgotten as a possible form of passive treatment.

Conclusion

A systematized clinical evaluation of patients with low back pain may permit the recognition of clinical patterns or pictures that point towards the disc and/or the dura mater, independent of what may be seen during imaging procedures. The clinical approach prevails.

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