Spinal Pain Experienced as a Visceral Referral
The lumbar spine is a frequent site of low back pain.

Anatomy of the lumbar spine
The lumbar spine is made up of 5 vertebrae, separated by intervertebral discs, and the sacrum, which consists of 5 bones fused together.

Each lumbar vertebra has a structure sharing common factors
- Body (bearing the weight)
- Pedicles
- Laminae
- Transverse processes
- Spinous process
- Superior and inferior articular processes

The pedicles and laminae form an arch, the neural arch, and the vertebral foramen. When all the vertebrae are aligned, this foramen forms the spinal canal and forms a body corridor for the spinal cord and the spinal nerves of the cauda equina.
**Intervertebral Disc**

It is said that the primary joint of the spine is the intervertebral disc.

The intervertebral disc consists of the annulus fibrosus, which consists of 10 - 20 concentric rings on the outside. These are known as lamellae and their fibres are arranged at right angles to each other. These are firmly attached to the adjacent vertebrae and have the function of holding the two vertebrae together.

The lamellae can suffer gap defects, not completely enveloping the nucleus pulposus and it might be that these defects contribute to painful annular tears and disc herniations.

The endplates of the discs are not as well attached as the annular fibres and some research has shown that even a mild trauma can cause its separation from the vertebra.

These endplates act mainly like a semipermeable membrane, allowing diffusion of nutrition and waste products. The nature of the endplate does not allow the movement of larger molecules, like the proteoglycans that maintain the nucleus pulposus. As the nucleus pulposus is avascular, it must get its nutrition via the blood supply through the segmental vertebral blood supply. Only the outer third of the annulus receives a blood supply and the endplate receives it from the vertebral body.
The centre of the disc is a jelly-like, almost liquid, substance called the nucleus pulposus, which acts to hold the two vertebrae apart and to permit movement between the two bones.

The discs of the lumbar spine are taller, as compared to the cervical and thoracic, and more vulnerable to injury. They contain 65% proteoglycans which have a great affinity for water (its water content can be about 80%).

Behind the disc are the zygapophyseal (facet) joints. The superior and inferior articular processes are bony projections that form these facets joints above and below. These are synovial joints, and the alignment of the facets defines what types of movement will be permitted at that level of the spine. In the lumbar spine, they are in the vertical plane on an antero-posterior alignment, allowing flexion/extension and sidebending, but very little rotation. The discs are cartilaginous and are mainly avascular and, like other cartilaginous tissues, are slow healing if damaged.

Zygapophyseal (Facet) Joints
In addition to this, the bones of the spine are shaped such that, where they normally aligned, there are holes for the mixed spinal nerves to emerge from the spinal canal – the intervertebral foraminae. This foramen enclosed the exiting nerve root and the delicate dorsal root ganglion (DRG).

The DRG contains all the sensory nerve cell bodies. Anatomical variations show that the DRG can be inside or outside the intervertebral foramen. Any restriction of the space of the intervertebral foramen (through ‘wear and tear’ changes or disc lesion: herniation of prolapse, can upset the course of these delicate nerves.

Figure 6 Lumbar intervertebral disc - longitudinal section

Figure 7 Diagram showing articular processes forming facet joints

Figure 8 Diagram demonstrating how the nerves emerge from the spinal canal
Nerves of the Lumbar Spine

The vertebral canal encloses the thecal sac (aka dura mater) which, in turn, houses the spinal cord and spinal nerves. It extends from the base of the brain stem (the foramen magnum) down to the level of L1. There are ‘expansions’ in the cervical and the lumbar regions, these reflecting the greater activity and demand through those regions

Within the spinal canal is found the dura mater, which is a waterproof sac enveloping the spinal cord and spinal nerves. The arachnoid mater is on the inside of the dura and held out against it by the presence of CSF in the subarachnoid space. The pia mater is on the outside of the spinal cord and follows it convolutions. Even though the dura is a robust structure enveloping all this, there are specializations of the pia mater that penetrate the dura appearing as 21 pairs of denticulate ligaments. These act to help anchor the spinal cord in the spinal canal.

The spinal cord proper ends at the level of L1. The remainder of the spinal canal is occupied by the spinal nerves, these continuing caudad to their level of exit from the spinal canal.

The dura ends at S2, with its other points of fixation at the foramen magnum and C2. The pia pierces the dura and continues to its final attachment at the coccyx. The cord proper ends at the conus medullaris at the level of L1. Below that the spinal nerves continue down to their level of exit as the cauda equina.

The nerves that leave and enter the lumbar spine control the trunk and lower extremities, as well as autonomic function. If injury occurs to any of these nerves there can be a consequent effect: pain, loss of motor (muscle) function, loss of sensory sensation, like light touch, pressure, temperature, vibration, and autonomic function (control of blood vessels size and sweating), or even cauda equina...
syndrome (loss of bladder and bowel control). Or even complex regional pain syndrome (CRPS), which is a chronic pain condition most often affecting one of the limbs (arms, legs, hands, or feet), usually after an injury or trauma to that limb. CRPS may cause the lower extremity to swell, be very hot, sensitive to touch and sweat.

**Spinal nerves**

The spinal nerves originate as a confluence of rootlets emerging from the spinal cord. These rootlets emerge from both the anterior and posterior aspects of the cord, the anterior roots are motor and the posterior are sensory. These anterior and posterior rootlets converge forming the anterior and posterior roots. The roots then form a mixed spinal nerve of both sensory and motor fibres.

*Figure 12 Diagram showing the formation of spinal nerves*

This mixed spinal nerve then emerges from the spinal canal, piercing the theca at about half a lumbar vertebra above that level.

*Figure 13 Diagram of top of cauda equina and descending spinal roots*

Below L1 there are four roots that are going to exit the thecal sac at each level and these are referred to as traversing nerve roots. This suggests that they cross the posterior of the next caudad disc en route to their level of exit, whilst still inside the thecal sac.

*Figure 14 Traversing roots of cauda equina*

The S1 root is an exception to this general format. It emerges from the thecal sac more cephalad than the other nerve roots, there is already in nerve root form at it crosses the posterior aspect of the L5 disc.

*Figure 15 S1 nerve roots as they cross L5 disc*

As the roots emerge from the spinal canal, the dura mater changes into the epineurium around the peripheral nerves. The mixed spinal nerve, on emergence, immediately divides into a ventral (anterior) ramus that supplies motor and
sensory function to the lower extremities, and dorsal (posterior) ramus that supplies the muscles and skin over the lower back and buttocks.

Nerves from the ventral rami form the lumbar and lumbosacral plexus form the femoral, obturator and sciatic nerves.

The ventral ramus also gives off a small branch that helps form the sinuvertebral nerve, which carries pain signals from the posterior aspect of the disc and gives off another branch, the grey ramus communicans, connecting to the sympathetic trunk of the autonomic nervous system.

The dorsal ramus also gives off the medial branch of the dorsal ramus giving sensory supply to the facet joints.

**Nerves supplying the disc**

Everyone who presents with back pain announces that they have a ‘disc out of place’. This is an over-generalization, but if a genuine disc pathology is present, what are the mechanics of low back pain?

A different nerve pathway innervates each region of the disc; a phenomenon that occurs in no other structure in the body.

- The posterior and posterolateral regions of the disc are innervated by the sinuvertebral nerve
- The lateral disc by the grey ramus communicans (a sympathetic nerve of the autonomic system)
- The anterior disc by the sympathetic branches of the sympathetic trunk or ganglion

![Figure 16 Diagram of the sinuvertebral nerve and its distribution](image)

The meningeal branches of the spinal nerves (also known as **recurrent meningeal nerves**, **sinuvertebral nerves**, or **recurrent nerves of Luschka**) are several small nerves that branch from the spinal nerve near the origin of the anterior and posterior rami, but before the rami communicantes branch. They then re-enter the intervertebral foramen, and innervate the facet joints, the *outer third* of the anulus fibrosus of the intervertebral disc, and the ligaments and
periosteum of the spinal canal, carrying pain sensation. The nucleus pulposus of the intervertebral disk has no pain innervation, it being both avascular and aneural.

The true pain sensors of the disc (the free nerve endings of the nociceptors) are shown as yellow dots, and have a greater concentration in the posterior and posterolateral aspects of the annulus of the disc, as compared to the lateral and anterior regions. This suggests that the posterior and posterolateral regions of the disc are more sensitive to pain and possibly to the development of chronic back pain.

Annular tears and other types of disc lesion are found most frequently on the posterior and posterolateral regions of the disc, it would seem appropriate to focus on the sinuvertebral nerve, the primary nerve that carries pain sensation away from this area.

![Figure 17 Diagram showing the sinuvertebral nerve and its bilateral distribution](image)

The sinuvertebral nerve has a bilateral origin and looks like a wishbone, for it arises from both the ventral ramus (a somatic nerve) and the grey ramus communicans of the sympathetic ganglion (an autonomic nerve). This nerve not only innervates the posterior outer third of the disc at the same level, but also the same areas of the disc of the level above, e.g. the L4 sinuvertebral nerve innervates the posterior annulus of both L3 and L4. Other opinions claim that one nerve can innervate three levels. This suggests that there can be ‘confusion’ in the subjective experience of a painful disc, as L3 and L4 are both wired together (so if a person points at the pain in their back, the lesion may not be at that level).

Hence, if a disc lesion is present, the sensation can enter the cord at one or more levels (myelomeres). From here the nerves decussates and thence travels up to the thalamus and on to the sensory cortex.

Despite extensive research, the precise pathway of the pain signals from the sinuvertebral nerves is uncertain. (This is frustrating to the orthodox medical world and its endeavours in injectable pain reduction). Logic would have us believe that the pain signals would take the shortest pathway to the dorsal root ganglion (DRG), but apparently, it takes a more convoluted route.
Using L4 as an example:

- The sinuvertebral nerve goes to the grey ramus communicans (GRC)
- The GRC to the sympathetic ganglion of L4
- The sympathetic ganglion of L4 up to the sympathetic ganglion of L1 or L2
- Across the white ramus communicans (WRC) (which are only present at L1 and L2)
- The WRC to the same level ventral root of the spinal nerve of L1 or L2 to the DRG of L1 or L2
- The DRG of L1 or L2 to the spinal cord at the L1 or L2 myelomere
- The spinal cord to the sensory cortex.

This hypothesis seems to be confirmed by Nakamura (1996) with L4 and L5 discogenic low back pain. 33 patients with diagnosed L4 and L5 annular tears or herniation (all confirmed on MRI) were treated with selective nerve root block, using lidocaine injected at the L2 intervertebral foramen (i.e. close to the DRG). The theory behind this was that blocking the L2 DRG would block pain sensation from L4 and L5 disc lesions; the patient would experience less pain and an increase in function. This was shown to be true, as in 15 minutes of the selective nerve root block, all the patients had experienced a reduction in subjective low back pain. They also had an increased ability to remain flexed at the waist in the standing position (this test is called the static flexion test and is difficult to perform in patients with disc lesions. 79% of this group experienced a complete, or near complete, reduction of low back pain.

The authors suggested that discogenic low-back pain is transmitted non-segmentally by visceral sympathetic afferents mainly through the L2 spinal nerve root, and that this may be perceived as referred pain in the L2 dermatome (Fig. 18). Discogenic low-back pain may be a type of visceral pain. Injection of the L2 spinal nerve root is a possible diagnostic tool and could be used for the conservative treatment of discogenic low-back pain. As such pain is being transmitted via visceral afferents, it can be interpreted (felt) as a type of visceral pain. Visceral pain, such as tummy ache, can be quite vague and tends to move around a bit in its location. Does this sounds familiar?

Such pain can cause guarding of the muscles in those areas of referral, causing a persistent tension pattern therein, even establishing reflex patterns to effectively maintain these patterns. Persistent tension patterns can become chronic and predispose to degenerative changes in the areas affected: low back, hips etc. (The next question, from this, is what is causing the pressure in the disc and how can it be reduced?)

How do we know this for certain?

So, how do we know that pain signals are not transmitted via the simple route described: same level disc, sinuvertebral nerve, same level ventral ramus, same level DRG?
There have been some disc surgeries performed under local anaesthetic (i.e. the patient was awake). One subject was experiencing sciatica along with low back pain. During the discectomy procedure, it was demonstrated that by anaesthetizing the inflamed nerve root at the same level of the disc herniation (i.e. the one causing the sciatica), the patient felt relief of the radiating leg pain, but no relief of the low back pain. They concluded from this that the low back pain signals were not travelling through the same level nerve root that was anaesthetized; if were, the low back pain would also have been relieved.

There is evidence from modern techniques performed: transforaminal epidural steroid injections (TFESIs) and selective nerve root blocks (SNRBs), that anaesthetizing the spinal nerve (adjacent/distal to the DRG) have very little effect on a patient’s nerve root pain and these procedures are for radicular pain (sciatica, lower extremity pain) only.

Nakamura et al has suggested these results are from the convergence projection theory of Ruch (1955). This is because visceral and somatic afferent fibres have synapses to common posterior-horn cells (Cervero and Tattersall 1985; Ammons 1987; Cervero 1987; Yokota et al 1988). Pain caused by disease of abdominal organs may be perceived as if it comes from somatic tissues. One example is that pain during delivery is referred to the T10 to L1 dermatomes through ‘sympathetic’ hypogastric nerve fibres (Bonica 1984). Colicky low-back pain due to ureteric calculus is relieved by a thoracolumbar sympathetic block (MacLean, Carroll and Graves 1949). If discogenic pain is transmitted by sympathetic afferent fibres through the sinuvertebral nerves, this explains why some patients with lumbar radiculopathy have only leg pain, and also why low-back pain can be produced by stimulation of an intervertebral disc even when the corresponding nerve root is blocked (Wiberg 1949).

Low-back pain is usually exacerbated in sustained sitting or forward bending of the trunk, both of which are known to increase intradiscal pressure (Nachemson 1976). Such pain is often relieved by lying on the side and gentle movement of the trunk; joint pain is usually made worse by such movement. Visceral pain can be induced by maintaining the intraluminal pressure of a hollow visceral organ above a certain pressure (Lipkin and Sleisenger 1957; Ness and Gebhart 1990); the latency from the onset of such a phasic stimulus to perception of pain by the patient is directly related to the intensity of the distending stimulus (Lipkin and Sleisenger 1957). It is assumed that the ‘flexion test’ which can be used increased the intradiscal pressure and distends the outer layer of the annulus fibrosus. The reduction of this pain after infiltration implies that the low back pain which we elicited originates mainly from the intervertebral discs.

The Basivertebral Nerve – a Cause of Vertebrogenic Back Pain?
In the last decade, or so, a new potential diagnosis has been suggested for chronic and intractable low back pain – vertebrogenic pain syndrome (VPS).

VPS can occur when the vertebral endplates of the disc, along with the adjacent subchondral bone of the vertebra. The layer of bone here is very thin and can move with pressure. This can cause them to become irritated and inflamed and result in chronic pain.

The science behind this is, as yet, uncertain. Two papers (2, 3) have been published, demonstrating that the basivertebral nerve, of
the vertebral body, supply branches that innervate the subchondral bone of the vertebral endplates with a pain carrying fibre.

It is also known from the work of Bogduk (4, 5) that the basivertebral nerves leave the basivertebral foramen and turn into divisions of the sinuvertebral nerves. From here it sends the pain signals through the grey ramus communicans to the sympathetic trunk.

References

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