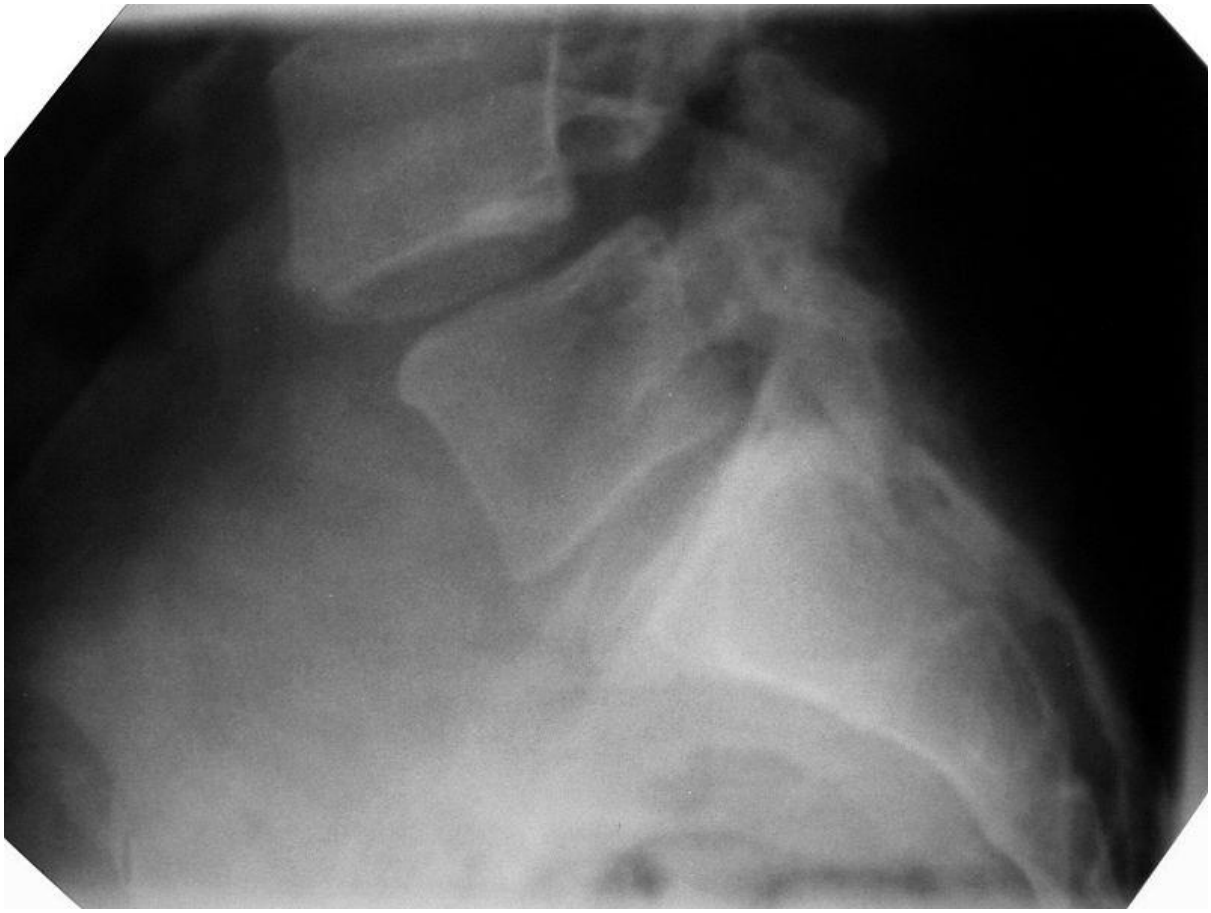
VERTEBRAL COLUMN DISORDERS



OCTOBER 28, 2020
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Vertebral Column

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Vertebral Column Disorders

Arnold-Chiari Malformation

What are Chiari malformations?

Arnold-Chiari (more commonly known today as Chiari malformations) are structural defects in the base of the skull and cerebellum, the part of the brain that controls balance. Normally the cerebellum and parts of the brain stem sit above an opening in the skull that allows the spinal cord to pass through it (called the foramen magnum). When part of the cerebellum extends below the foramen magnum and into the upper spinal canal, it is called a Chiari malformation (CM).

Chiari malformations may develop when part of the skull is smaller than normal or misshapen, which forces the cerebellum to be pushed down into the foramen magnum and spinal canal. This causes pressure on the cerebellum and brain stem that may affect functions controlled by these areas and block the flow of cerebrospinal fluid (CSF) - the clear liquid that surrounds and cushions the brain and spinal cord. The CSF also circulates nutrients and chemicals filtered from the blood and removes waste products from the brain.

What causes these malformations?

CM has several different causes. Most often it is caused by structural defects in the brain and spinal cord that occur during foetal development. This can be the result of genetic mutations or a maternal diet that lacked certain vitamins or nutrients. This is called primary or congenital Chiari malformation. It can also be caused later in life if spinal fluid is drained excessively from the lumbar or thoracic areas of the spine either due to traumatic injury, disease, or infection. This is called acquired or secondary Chiari malformation. Primary Chiari malformation is much more common than secondary Chiari malformation.

What are the symptoms of a Chiari malformation?

Headache is the hallmark sign of Chiari malformation, especially after sudden coughing, sneezing, or straining. Other symptoms may vary among individuals and may include:

- Neck pain
- Hearing or balance problems
- Muscle weakness or numbness
- Dizziness
- Difficulty swallowing or speaking
- Vomiting
- Ringing or buzzing in the ears (tinnitus)
- Curvature of the spine (scoliosis)
- Insomnia
- Depression
- Problems with hand coordination and fine motor skills

Some individuals with CM may not show any symptoms. Symptoms may change for some individuals, depending on the compression of the tissue and nerves and on the build-up of CSF pressure.

Infants with a Chiari malformation may have difficulty swallowing, irritability when being fed, excessive drooling, a weak cry, gagging or vomiting, arm weakness, a stiff neck, breathing problems, developmental delays, and an inability to gain weight.

How are CMs classified?

Chiari malformations are classified by the severity of the disorder and the parts of the brain that protrude into the spinal canal.

Chiari malformation Type I

Type 1 happens when the lower part of the cerebellum (called the cerebellar tonsils) extends into the foramen magnum. Normally, only the spinal cord passes through this opening. Type 1—which may not cause symptoms—is the most common form of CM. It is usually first noticed in adolescence or adulthood, often by accident during an examination for another condition. Adolescents and adults who have CM but no symptoms initially may develop signs of the disorder later in life.

Chiari malformation Type II

Individuals with Type II have symptoms that are generally more severe than in Type 1 and usually appear during childhood. This disorder can cause life-threatening complications during infancy or early childhood, and treating it requires surgery.

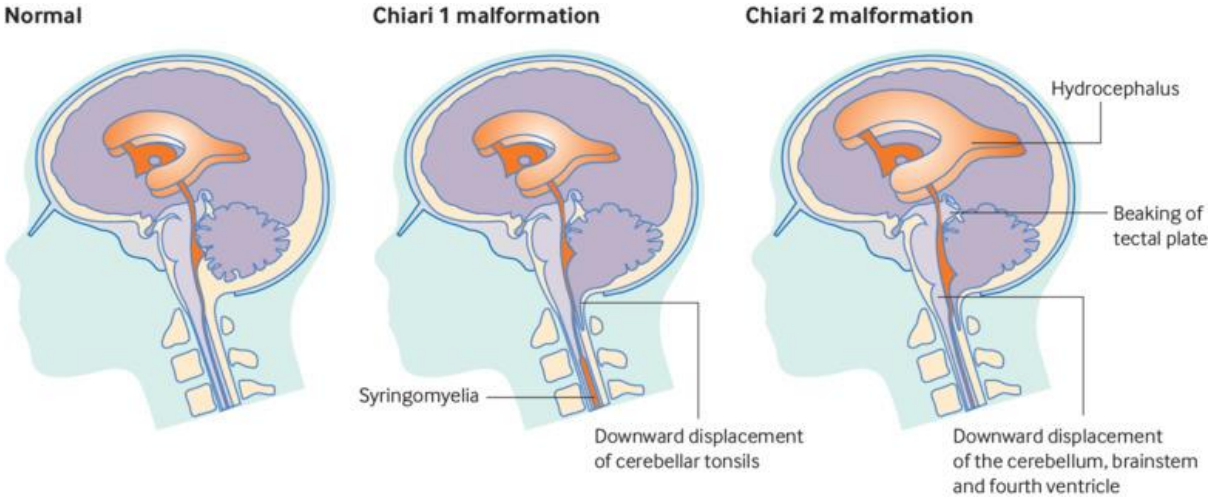


Figure 1 Chiari Malformations Type I and II

In Type II, also called classic CM, both the cerebellum and brain stem tissue protrude into the foramen magnum. Also, the nerve tissue that connects the two halves of the cerebellum may be missing or only partially formed. Type II is usually accompanied by a myelomeningocele—a form of spina bifida that occurs when the spinal canal and backbone do not close before birth. (Spina bifida is a disorder characterized by the incomplete development of the brain, spinal cord, and/or their protective covering.) A myelomeningocele usually results in partial or complete paralysis of the area below the spinal opening. The term Arnold-Chiari malformation (named after two pioneering researchers) is specific to Type II malformations.

Chiari malformation Type III

Type III is very rare and the most serious form of Chiari malformation. In Type III, some of the cerebellum and the brain stem stick out, or herniate, through an abnormal opening in the back of the skull. This can also include the membranes surrounding the brain or spinal cord.

The symptoms of Type III appear in infancy and can cause debilitating and life-threatening complications. Babies with Type III can have many of the same symptoms as those with

Type II but can also have additional severe neurological defects such as mental and physical delays, and seizures.

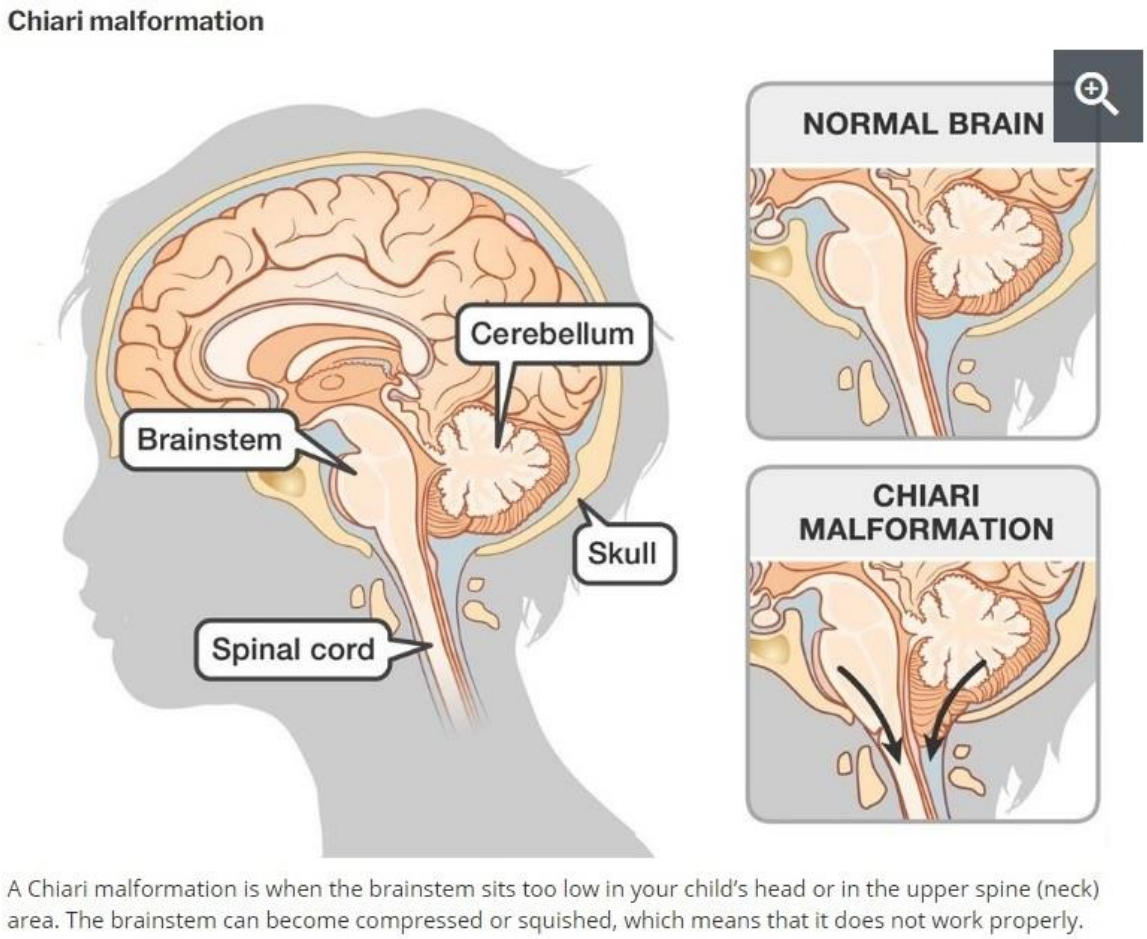


Figure 2 Arnold-Chiari Malformation

Chiari malformation Type IV

Type IV involves an incomplete or underdeveloped cerebellum (a condition known as cerebellar hypoplasia). In this rare form of CM, the cerebellum is located in its normal position but parts of it are missing, and portions of the skull and spinal cord may be visible.

What other conditions are associated with Chiari malformations?

Hydrocephalus is an excessive build-up of CSF in the brain. A CM can block the normal flow of this fluid and cause pressure within the head that can result in mental defects and/or an enlarged or misshapen skull. Severe hydrocephalus, if left untreated, can be fatal. The disorder can occur with any type of Chiari malformation, but is most associated with Type II.

Spina bifida is the incomplete closing of the backbone and membranes around the spinal cord. In babies with spina bifida, the bones around the spinal cord do not form properly, causing defects in the lower spine. While most children with this birth defect have such a mild form that they have no neurological problems, individuals with Type II Chiari malformation usually have myelomeningocele, and a baby's spinal cord remains open in one area of the back and lower spine. The membranes and spinal cord protrude through the opening in the spine, creating a sac on the baby's back. This can cause a number of neurological impairments such as muscle weakness, paralysis, and scoliosis.

Syringomyelia is a disorder in which a CSF-filled tubular cyst, or syrinx, forms within the spinal cord's central canal. The growing syrinx destroys the center of the spinal cord, resulting in pain, weakness, and stiffness in the back, shoulders, arms, or legs. Other symptoms may include a loss of the ability to feel extremes of hot or cold, especially in the hands. Some individuals also have severe arm and neck pain.

Tethered cord syndrome occurs when a child's spinal cord abnormally attaches to the tissues around the bottom of the spine. This means the spinal cord cannot move freely within the spinal canal. As a child grows, the disorder worsens, and can result in permanent damage to the nerves that control the muscles in the lower body and legs. Children who have a myelomeningocele have an increased risk of developing a tethered cord later in life.

Spinal curvature is common among individuals with syringomyelia or CM Type I. The spine either may bend to the left or right (scoliosis) or may bend forward (kyphosis).

How common are Chiari malformations?

In the past, it was estimated that the condition occurs in about one in every 1,000 births. However, the increased use of diagnostic imaging has shown that Chiari malformation may be much more common. Complicating this estimation is the fact that some children who are born with this condition may never develop symptoms or show symptoms only in adolescence or adulthood. Chiari malformations occur more often in women than in men and Type II malformations are more prevalent in certain groups, including people of Celtic descent.

How are Chiari malformations diagnosed?

Currently, no test is available to determine if a baby will be born with a Chiari malformation. Since Chiari malformations are associated with certain birth defects like spina bifida, children born with those defects are often tested for malformations. However, some malformations can be seen on ultrasound images before birth.

Many people with Chiari malformations have no symptoms and their malformations are discovered only during the course of diagnosis or treatment for another disorder. The doctor will perform a physical exam and check the person's memory, cognition, balance (functions controlled by the cerebellum), touch, reflexes, sensation, and motor skills (functions controlled by the spinal cord). The physician may also order one of the following diagnostic tests:

Magnetic resonance imaging (MRI) is the imaging procedure most often used to diagnose a Chiari malformation. It uses radio waves and a powerful magnetic field to painlessly produce either a detailed three-dimensional picture or a two-dimensional "slice" of body structures, including tissues, organs, bones, and nerves.

X-rays use electromagnetic energy to produce images of bones and certain tissues on film. An X-ray of the head and neck cannot reveal a CM but can identify bone abnormalities that are often associated with the disorder.

Computed tomography (CT) uses X-rays and a computer to produce two-dimensional pictures of bone and blood vessels. CT can identify hydrocephalus and bone abnormalities associated with Chiari malformation.

How are Chiari malformations treated?

Some CMs do not show symptoms and do not interfere with a person's activities of daily living. In these cases, doctors may only recommend regular monitoring with MRI. When individuals experience pain or headaches, doctors may prescribe medications to help ease symptoms.

Surgery

In many cases, surgery is the only treatment available to ease symptoms or halt the progression of damage to the central nervous system. Surgery can improve or stabilize symptoms in most individuals. More than one surgery may be needed to treat the condition.

The most common surgery to treat Chiari malformation is posterior fossa decompression. It creates more space for the cerebellum and relieves pressure on the spinal cord. The surgery involves making an incision at the back of the head and removing a small portion of the bone at the bottom of the skull (craniectomy). In some cases, the arched, bony roof of the spinal canal, called the lamina, may also be removed (spinal laminectomy). The surgery should help restore the normal flow of CSF, and in some cases, it may be enough to relieve symptoms.

Next, the surgeon may make an incision in the dura, the protective covering of the brain and spinal cord. Some surgeons perform a Doppler ultrasound test during surgery to determine if opening the dura is even necessary. If the brain and spinal cord area is still crowded, the surgeon may use a procedure called electrocautery to remove the cerebellar tonsils, allowing for more free space. These tonsils do not have a recognized function and can be removed without causing any known neurological problems.

The final step is to sew a dura patch to expand the space around the tonsils, like letting out the waistband on a pair of pants. This patch can be made of artificial material or tissue harvested from another part of an individual's body.

Infants and children with myelomeningocele may require surgery to reposition the spinal cord and close the opening in the back. Findings from the National Institutes of Health (NIH) show that this surgery is most effective when it is done prenatally (while the baby is still in the womb) instead of after birth. The prenatal surgery reduces the occurrence of hydrocephalus and restores the cerebellum and brain stem to a more normal alignment.

Hydrocephalus may be treated with a shunt (tube) system that drains excess fluid and relieves pressure inside the head. A sturdy tube, surgically inserted into the head, is connected to a flexible tube placed under the skin. These tubes drain the excess fluid into either the chest cavity or the abdomen so it can be absorbed by the body.

An alternative surgical treatment in some individuals with hydrocephalus is third ventriculostomy, a procedure that improves the flow of CSF out of the brain. A small hole is made at the bottom of the third ventricle (brain cavity) and the CSF is diverted there to relieve pressure. Similarly, in cases where surgery was not effective, doctors may open the spinal cord and insert a shunt to drain a syringomyelia or hydromyelia (increased fluid in the central canal of the spinal cord).

Deforming Diseases

Scheuermann's disease

Scheuermann's disease is a self-limiting skeletal disorder of childhood. It is also known as **Scheuermann's kyphosis,** as it results in kyphosis, **Calvé disease**, and **idiopathic juvenile kyphosis of the spine**. Scheuermann's disease describes a condition where the vertebrae grow unevenly with respect to the sagittal plane; that is, the anterior angle is often greater than the posterior. This uneven growth results in the signature "wedging" shape of the vertebrae, causing kyphosis.

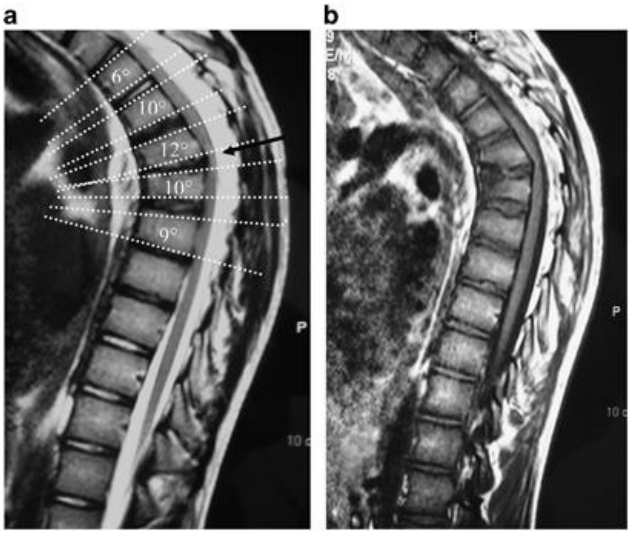


Figure 3 Scheuermann's Disease

It is found mostly in teenagers and presents a significantly worse deformity than postural kyphosis. Patients suffering with Scheuermann's kyphosis cannot consciously correct their posture. The apex of their curve, located in the thoracic vertebrae, is quite rigid. This rigidity is notorious for causing lower and mid-level back and neck pain, which can be severe and disabling. The sufferer may feel pain at the apex of the curve, which can be aggravated by physical activity and by long periods of standing or sitting. In addition to the pain associated with Scheuermann's disease, many sufferers of the disorder have loss of vertebral height, and depending on where the apex of the curve is, may have a visual 'hunchback' or 'roundback'. It has been reported that curves in the lower thoracic region cause more pain, whereas curves in the upper region present a more visual deformity.

The seventh and tenth thoracic vertebrae are most commonly affected. It causes backache and spinal curvature. In very serious cases it may result in internal problems and spinal cord damage, but these cases are extremely rare. The curvature of the back can put pressure on internal organs, wearing them out more quickly than the natural aging process; surgical procedures are almost always recommended in this case.

Along with this wedging of the vertebra there is also a change to the interface between the disc and the vertebra called endplate irregularities. Some of the disc then pushes into the vertebra and these are called **Schmorl's nodes** and are typically seen on an X-Ray.

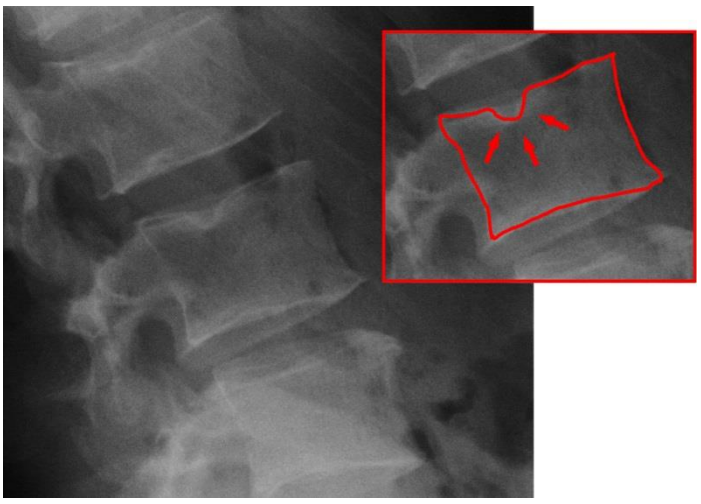


Figure 4 Schmorl's nodes

These Schmorl's nodes are present for life but are do not appear to cause any problems in the future. People may have an X-

Ray when they are older for an unrelated condition and find that they have Schmorl's nodes but have never experienced back pain.

Its causes are unknown, though it isn't unreasonable to see tension patterns within the chest pulling the spine into a kyphosis, this being particularly significant during the growth years. The tension pattern pulling the thoracic spine into a kyphosis will also contribute to the deformation of the thoracic vertebrae and their consequent wedge shape.

The main focus of any therapy is to help maintain mobility of the thoracic region.

Torticollis

Torticollis, also known as **wry neck** or **loxia**, is a dystonic condition defined by an abnormal, asymmetrical head or neck position, which may be due to a variety of causes. The term *torticollis* is derived from the Latin words *tortus* for twisted and *collum* for neck.

It can appear in various forms:

- Congenital
- Spasmodic
- Acquired

Figure 5 Torticollis

Congenital

The aetiology of congenital muscular torticollis is unclear. Birth trauma or intrauterine malposition is considered to be the cause of damage to the sternocleidomastoid muscle in the neck. This results in a shortening or excessive contraction of the sternocleidomastoid muscle, which curtails its range of motion in both rotation and lateral bending. The head typically is tilted in lateral bending toward the affected muscle and rotated toward the opposite side. It can be an expression of upset at the cranial base, not only at the occipitoatlantal joint, but tension patterns affecting the junction of the occiput and temporal bone, with the jugular foramen between the two (from whence emerges the ascending spinal accessory nerve) supplying sternocleidomastoid and trapezius.

The reported incidence of congenital torticollis is 0.3-2.0%. Sometimes a mass, such as a sternocleidomastoid tumour, is noted in the affected muscle at the age of two to four weeks. Gradually it disappears, usually by the age of eight months, but the muscle is left fibrotic.

Initially, the condition is treated with physical therapies, such as stretching to release tightness, strengthening exercises to improve muscular balance, and handling to stimulate symmetry. A TOT Collar is sometimes applied. About 5–10% of cases fail to respond to stretching and require surgical release of the muscle.

Spasmodic torticollis

This is a torticollis with recurrent, but transient contraction of the muscles of the neck and especially of the sternocleidomastoid. Synonyms are "intermittent torticollis", "cervical dystonia" or "idiopathic cervical dystonia", depending on cause.

Acquired torticollis

Non-congenital muscular torticollis may result from scarring or disease of cervical vertebrae, adenitis, tonsillitis, rheumatism, enlarged cervical glands, retropharyngeal abscess, or cerebellar tumours. It may be spasmodic (clonic) or permanent (tonic). The latter type may be due to Pott's disease (tuberculosis of the spine).

- A self-limiting spontaneously occurring form of torticollis with one or more painful neck muscles is by far the most common ('stiff neck') and will pass spontaneously in 1–4 weeks. Usually, the sternocleidomastoid muscle or the trapezius muscle is involved. Sometimes draughts, colds, or unusual postures are implicated; however, in many cases no clear cause is found. These episodes are commonly seen by physicians.
- Tumours of the skull base (posterior fossa tumours) can compress the nerve supply to the neck and cause torticollis, and these problems must be treated surgically.
- Infections in the posterior pharynx can irritate the nerves supplying the neck muscles and cause torticollis, and these infections may be treated with antibiotics if they are not too severe but could require surgical debridement in intractable cases.
- Ear infections and surgical removal of the adenoids can cause an entity known as Grisel's syndrome, a subluxation of the upper cervical joints, mostly the atlantoaxial joint, due to inflammatory laxity of the ligaments caused by an infection. This bridge must either be broken through manipulation of the neck, or, surgically resected.
- The use of certain drugs, such as antipsychotics, can cause torticollis.[9]
- Antiemetics - Neuroleptic Class - Phenothiazines
- There are many other rare causes of torticollis.

Spondylopathies

In medicine, **spondylopathies** is a general term for disorders of the vertebrae. The word derives from *spondylos* (vertebra) and *pathos* (suffering). When involving inflammation, it can be called **spondylitis**. In contrast, a **spondyloarthropathy** is a condition involving the vertebral joints, but many conditions involve both spondylopathy and spondyloarthropathy.

Conditions like this include:

- **Ankylosing Spondylitis**
- **Spondylosis**

Ankylosing Spondylitis

Ankylosing spondylitis (Greek *ankylos*, fused; *spondylos*, vertebra; *-itis*, inflammation), previously known as **Bechterew's disease** (or syndrome) and **Marie-Strümpell disease**, is a chronic inflammatory disease of the axial skeleton, with variable involvement of peripheral joints and non-articular structures.

It is a member of the group of the spondyloarthropathies, with a strong genetic predisposition. It mainly affects joints in the spine and the sacroiliac joint in the pelvis. In severe cases, it can eventually cause complete fusion and rigidity of the spine.

"Bamboo spine" develops when the outer fibres of the fibrous ring of the intervertebral disks ossify, which results in the formation of marginal syndesmophytes between adjoining vertebrae.

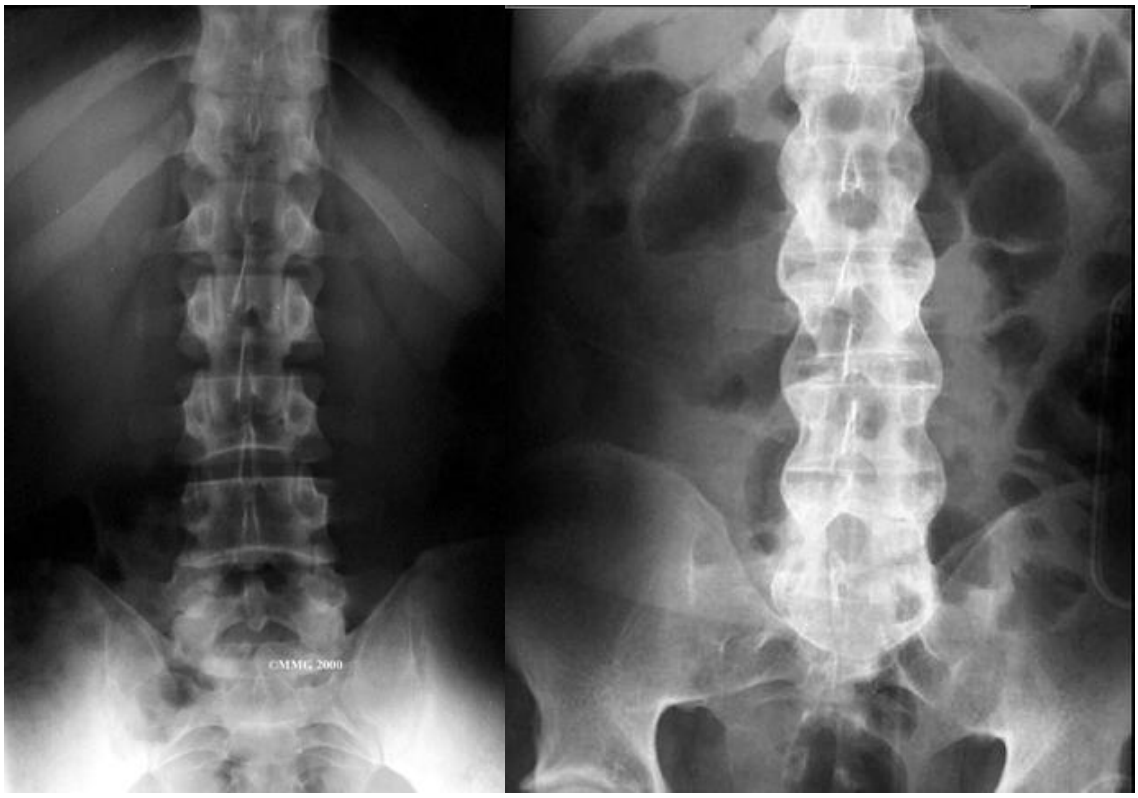


Figure 6 Ankylosing Spondylitis beside Normal Spine

The pictures above show a normal spine beside a spine with ankylosing spondylitis. Note:

- The absence of any outline of the sacroiliac joints
- The absence of any disc space, as outer fibres of the intervertebral disc has ossified, causing the characteristic ‘Bamboo spine’ effect, see here

Symptoms

- Symptoms appear gradually, most commonly between 15 and 45 years of age, more often in males
- Since the initial signs and symptoms are not specific for ankylosing spondylitis, there is a lag-time between onset of disease and diagnosis, which averages between 8.5 years and 11.4 years

- Sacroiliitis is usually one of its earliest manifestation.
- The initial symptom is usually a typical chronic dull pain, insidious in onset, felt deep in the lower lumbar or gluteal region, accompanied by low-back morning stiffness. It can occur in in the middle part of the spine or the entire spine, often with pain referred to one or the other buttock or the back of the thigh from the sacroiliac joint. This pain is often severe at rest but improves with physical activity. However, many experience inflammation and pain to varying degrees regardless of rest and movement.
- As the inflammation spreads up the spine, the costovertebral joints can also be involved, with pain on deep breathing and a reluctance to move into extension. This can result in a significant kyphosis before the spine fuses. The end result can be a straight lumbar spine and a kyphotic upper thoracic spine

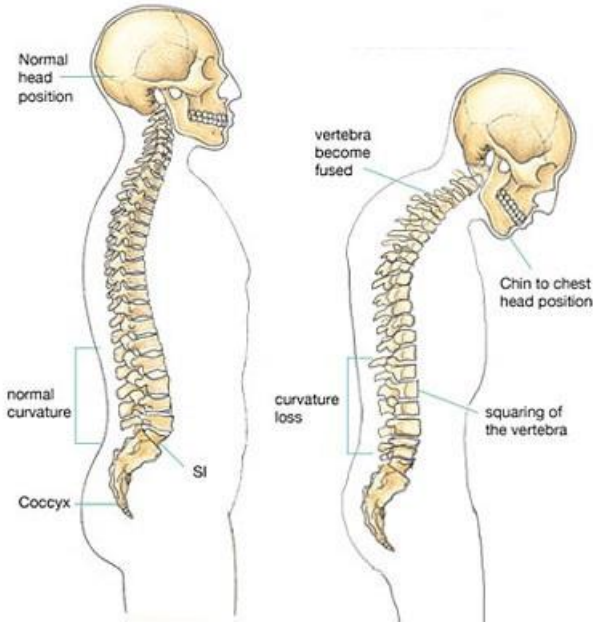


Figure 7 Ankylosing Spondylitis

- Loss of spinal mobility, with limitation of anterior flexion, lateral flexion, and extension of the lumbar spine, is seen with chest pain and generalized fatigue.
- The most serious complication of the spinal disease is spinal fracture, which can occur with even minor trauma to the rigid, osteoporotic spine.
- Arthritis in the hips and shoulders may occur.
- The most common extra-articular manifestation is acute anterior uveitis which can antedate the spondylitis. About 40 percent of AS patients experience inflammation in the anterior chamber of the eye (uveitis), causing redness, eye pain, floaters and photophobia. Visual acuity is usually maintained, and the fundus is normal. This is thought to be due to the association that both AS and uveitis have with the inheritance of the HLA-B27antigen.
- Pulmonary involvement is characterized by slowly progressive apical lung fibrosis. There is limitation of chest expansion. Recurrent chest infection is the most common cause of death.
- Aortitis, aortic valve insufficiency or cardiac conduction disturbances.
- Prostatitis occurs with increased frequency in men.

- Mortality attributable is largely the result of spinal trauma, aortic insufficiency, respiratory failure, amyloid nephropathy, or complications of therapy such as upper gastrointestinal haemorrhage.
- When the condition presents before the age of 18, it is relatively likely to cause pain and swelling of large limb joints, particularly the knee. In prepubescent cases, pain and swelling may also manifest in the ankles and feet, where calcaneal spurs may also develop.

It is a systemic rheumatic disease, meaning it affects the entire body. Approximately 90% of AS patients express the HLA-B27 genotype, meaning there is a strong genetic association. 1-2% of individuals with the HLA-B27 genotype contract the disease. Tumour necrosis factor-alpha (TNF α) and IL-1 are also implicated in ankylosing spondylitis (both are cytokines of the acute inflammatory reaction).

As there is no cure, orthodox treatment usually focusses on pain reduction medication and physiotherapy.

Scoliosis

Curvatures of the Spine

In the normal adult there are four curvatures in the vertebral column in the anteroposterior (A/P) plane. These serve to align the head with a vertical line through the pelvis.

- A curvature concave anterior is a kyphosis
- A curvature concave posterior is a lordosis

The function of these curvatures is help create and maintain a physiologic efficient posture. It might also be seen that this forms the axis of rotation for the vertebral column.

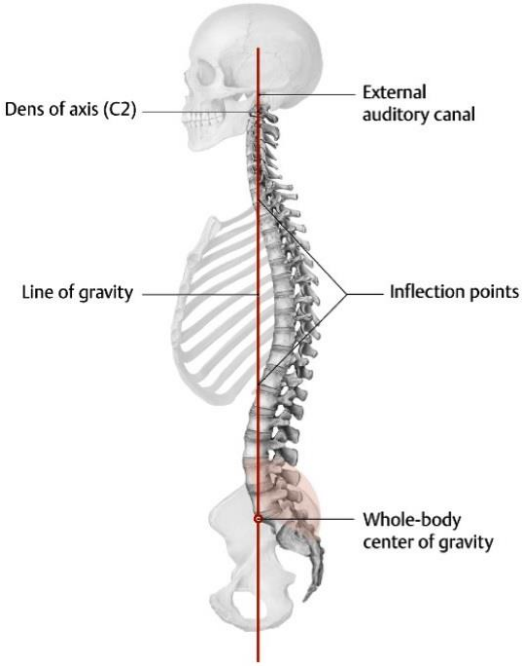


Figure 8 Spinal centre of gravity

In an ideal anatomically efficient posture, the centre of gravity falls through certain points:

- Through the occipital base
- Through the dens
- Through the anterior edge of the body of C7
- Through the body of L1
- Through the anterior edge of S1 (sacral promontory)
- Through the hip
- Through the knee
- Through the ankle

This format of spine is only the 'finished item', as it were. It wasn't born like this, though, the curves grew and developed.

In foetal life the whole of the spine was in a kyphosis. Then we learned to hold our head upright and the first secondary curvature developed; this is a reverse curvature, a lordosis - here in the cervical spine. Then we learned to walk on our hind limbs and our second secondary curvature, the lumbar lordosis, developed. Hence the normal kyphosis remains in the thoracic and sacral spines.

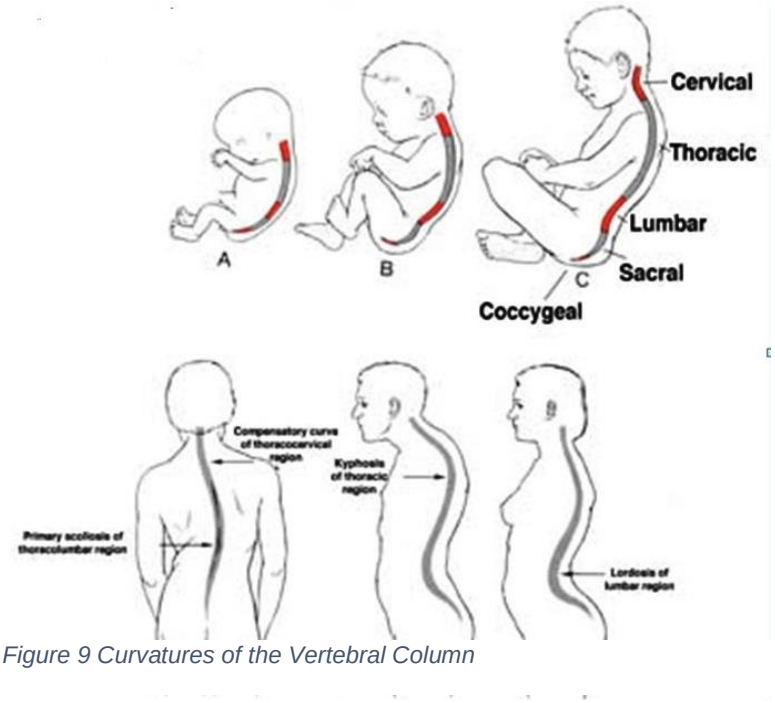


Figure 9 Curvatures of the Vertebral Column

Deviations away from this physiologically efficient posture are usually defined in the adjective:

- **Kyphotic** - an exaggerated curvature concave anteriorly
    - If this is of significance it can create a hunchback deformity
- **Lordotic** - An exaggerated curvature concave posteriorly
    - If this is of significance it can manifest as a swayback deformity

Exaggerated kyphosis or lordosis can occur under some normal conditions (e.g. increased lumbar lordosis in pregnancy).

Scoliosis

Any curvature of the vertebral column laterally away from the midline can occur normally or pathologically and is known as a **scoliosis**. A scoliosis can be both functional and organic.

Functional scoliosis

A functional scoliosis is a normal aberration of the curvature of the spine away from the line of centre of gravity. If you were walking along a bank, or even just stood on one foot, the centre of gravity would shift. The head would want to stay over the pelvis, so a curvature would appear in the spine to achieve this. If there is side bending in the normal spine, there will be contralateral rotation (to the opposite side). However, all the curvatures will normalise when both feet are returned to a level surface.

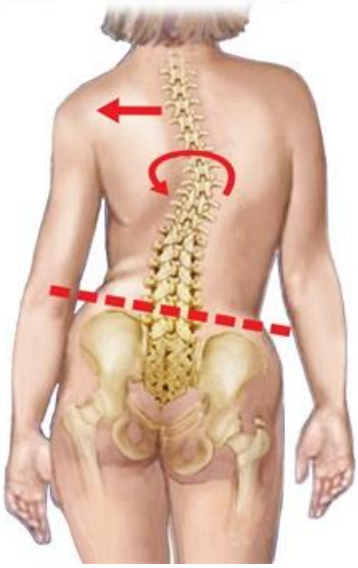


Figure 10 Functional scoliosis

Organic Scoliosis

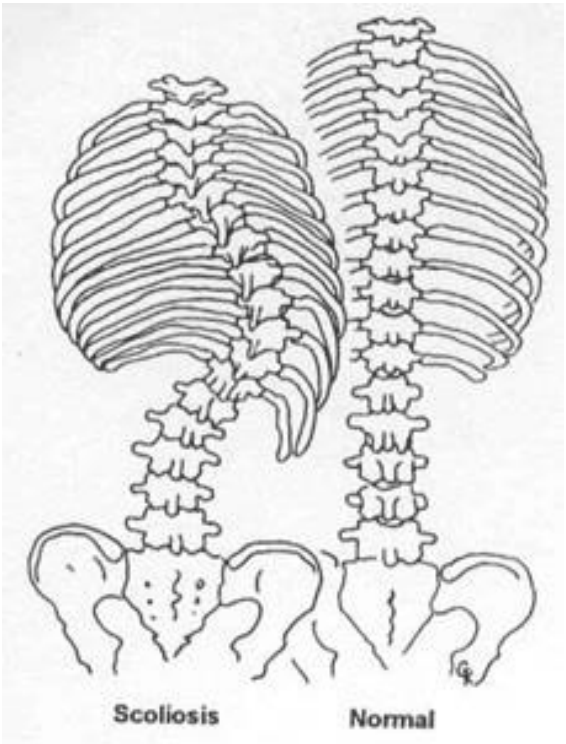


Figure 11 Organic Scoliosis

An organic scoliosis is a curvature that develops during the growth process. Theories on causative factors vary, but it can be seen as persistent tension patterns within the fascia along, and even within, the axis of the spine. Such tension patterns will define how the bones (vertebrae, ribs and pelvis) grow.

Note the curvature of the spine and ribs. This can cause compression of internal visceral organs with their consequent dysfunction.

Rotation of the spine, particularly in the thoracic region, can have 'knock on' effects in attachments and adnexa in that region. With reference to the ribs the tension patterns that define how the spine develop and grow also effect the growth and development of the attached ribs.

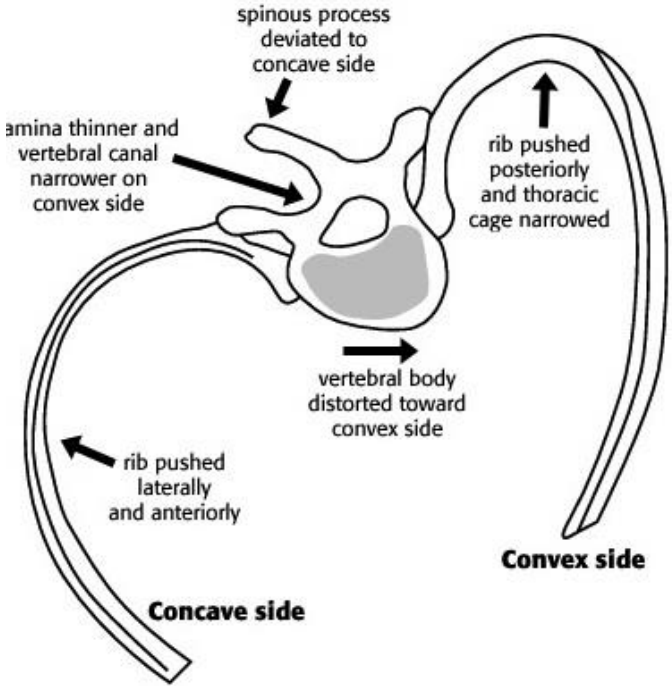


Figure 12 Scoliosis and ribs

Fig 12 here shows a level of the thoracic spine with its associated ribs.

Note:

- The rotation of the vertebral body.
- The difference in the angles of the ribs. Here the rib on the right of the diagram has a more acute angle compared with the rib on the left.
- Note that the spinous process is also being pulled over to the concave side, which also supports the concept of tension patterns of **pulling**.
- The bony configuration can affect the structure and function of the organs and viscera adjacent to the ribs
- Any organic scoliosis defines how each level of the spine grows and develops. Hence the facet joints grow and develop in response to this persistent, on-going, tension pattern and the facets do not have a normal configuration and alignment. Every vertebra will have an aberrant movement compared to normal

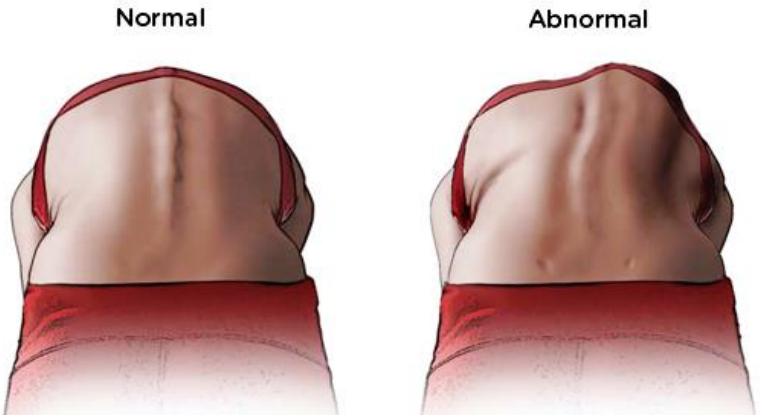


Figure 13 Scoliotic spine and ribs

Spondylosis

Spondylosis (usually called ‘wear and tear’ of the disc, or disc degeneration, is an old term (no longer used) referring to degenerative osteoarthritis of the joints between the centre of the spinal vertebrae and/or neural. If this condition occurs in the zygapophyseal joints, it can be considered facet syndrome, or spondylarthritis. If severe, it may cause pressure on nerve roots with subsequent sensory and/or motor disturbances, such as pain, paraesthesia, or muscle weakness in the limbs.

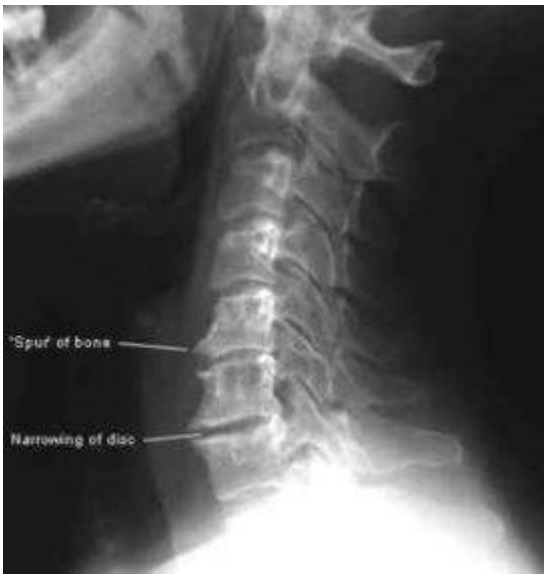


Figure 14 Disc degeneration in the cervical spine

It is seen as a degenerative condition, that the discs have a structure that keeps the vertebral bodies apart, but with time there is loss of the disc space. It is seen as wear and tear, or degenerative, as it usually occurs later in life. It usually manifests as chronic, sometimes disabling, low back or neck pain (depending upon the area affected). Pain such as this is usually treated with pain medication, but if persists further examination is called upon in the form of X-ray or MRI (see right). The x-ray usually reveal narrowing off the disc space and the presence of osteophytes (outgrowths of bone around the periphery of the vertebral body).

When the space between two adjacent vertebrae narrows, compression of a nerve root emerging from the spinal cord may result in radiculopathy (sensory and motor disturbances, such as severe pain in the neck, shoulder, arm, back, and/or leg, accompanied by muscle weakness). Less commonly, direct pressure on the spinal cord (typically in the cervical spine) may result in myelopathy, characterized by global weakness, gait dysfunction, loss of balance, and loss of bowel and/or bladder control. The patient may experience a phenomenon of shocks (paraesthesia) in hands and legs because of nerve compression and lack of blood flow. If vertebrae of the neck are involved, it is labelled cervical spondylosis. Lower back spondylosis is labelled lumbar spondylosis.

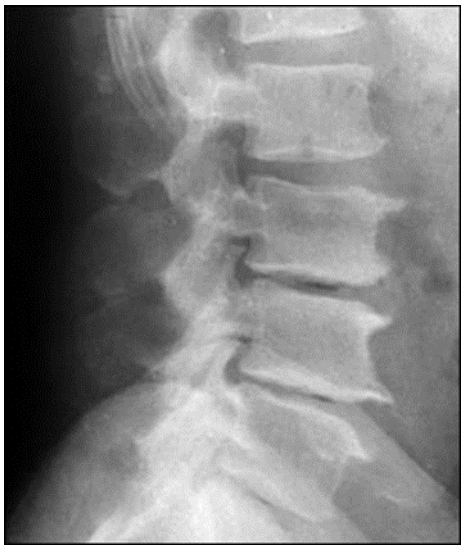


Figure 15 Lumbar spondylosis

Causes of Spondylosis

It is seen as a degenerative condition, but it is not really known what causes it.

Normally there is compression of the disc with weight bearing and this can lead to some loss of disc space over the course of a day. This compression can be relieved by lying down (in bed at night). Hence, we are probably taller in the morning than we are at night.

It might be good to remind ourselves of the oblique and deepest muscles of the spine here. One way of looking at a possible cause of such 'wear and tear' is to see a chronic tension in these groups of muscles (and more particularly **the fascia** around these muscles). It can result in a chronic compression along the axis of the spine with a consequent compression of the intervertebral discs. Such a tension will not be relieved by rest or lying down. It manifests as chronic pain that may, or may not be relieved by pain-killers. The X-rays taken demonstrate bony changes and the characteristic osteophyte formation. Osteophytes are bony outgrowths that form at the periphery of the vertebral bodies and can be seen in the pictures above. They are diagnostic of the condition, but to see these suggests that such a condition has been present for 5-10 years.

Another way of seeing it is again via fascial tension patterns. Here though they can express themselves over regions of the spine allowing one or two levels to move. This creates what could be seen as a 'hinge point', with the adjacent bones touching off each other more readily. Such 'touching' will again create bony changes with osteophyte formation.

Spondylolisthesis

Spondylolisthesis is defined as forward translation of a vertebral body with respect to the vertebra below. The term is derived from the Greek roots *spondylos,* meaning spine, and *listhesis,* meaning to slide down a slippery path.

Spondylolisthesis can occur at any level of the spinal column, although it is most common in the lower lumbar spine. Most cases are thought to result from minor overuse trauma, particularly repetitive hyperextension of the lumbar spine. Spondylolysis, a break in the vertebra typically in the region of the pars interarticularis, may or may not be associated with a spondylolisthesis. If the pars defect is bilateral, it may allow slippage of the vertebra, typically L5 on S1, resulting in spondylolisthesis.

Both spondylolysis and spondylolisthesis are often asymptomatic, and the degree of spondylolisthesis does not necessarily correlate with the incidence or severity of symptoms, even when a patient is experiencing back pain. However, these 2 entities have been reported to be the most common underlying causes of persistent low back pain among children and adolescents, despite the fact that most cases are asymptomatic.

Spondylolisthesis can be classified into the following 6 distinct categories.

- Type I
  - Congenital (dysplastic)
  - Caused by agenesis of the superior articular facet.
- Type II
  - Isthmic (spondylolytic)
  - Caused by pars interarticularis defects.
- Type III
  - Degenerative
  - Secondary to articular degeneration
- Type IV
  - Traumatic
  - Caused by fracture or dislocation of the lumbar spine, not involving the pars.
- Type V
  - Pathologic
  - Caused by malignancy, infection, or other types of abnormal bone.

- Type VI
    - Postsurgical (iatrogenic)

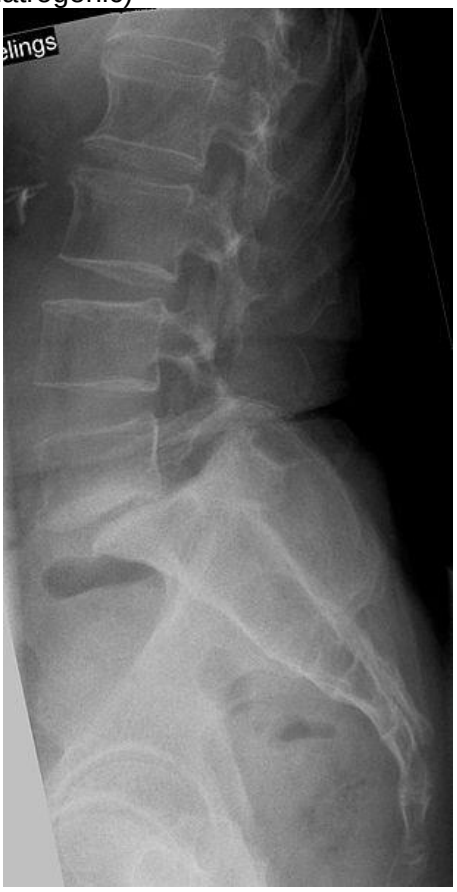


Figure 16 Spondylolisthesis at lumbosacral junction

Epidemiology

Frequency

United States

The prevalence rate of isthmic spondylolisthesis is approximately 5% at age 5-7 years, with an increase to 6-7% by age 18 years. This condition is twice as common in males as in females, and the prevalence is lower in blacks (2.8%, black men; 1.1%, black women) than in whites (6.4%, white men; 2.3%, white women). Despite the higher prevalence in males, progression, although still rare, has been reported to be more common in females. Additional risk factors include having a first-degree relative with a slip, occult spina bifida at S1, and the presence of scoliosis.

Functional Anatomy

Mechanical stresses play an important role in this process. Erect posture produces a constant downward and forward thrust on the lumbar vertebrae. Stresses on the pars interarticularis are accentuated during repetitive hyperextension, which results in increased contact of the caudal edge of the L4 inferior articular facet with the L5 pars interarticularis. This collective trauma may eventually result in a stress fracture of the pars interarticularis.

Spondylolisthesis may occur when bilateral pars defects are present, which allows forward slippage of the vertebra (typically L5 on S1). Spondylolisthesis has never been reported in quadrupeds or people who are chronically bedridden.

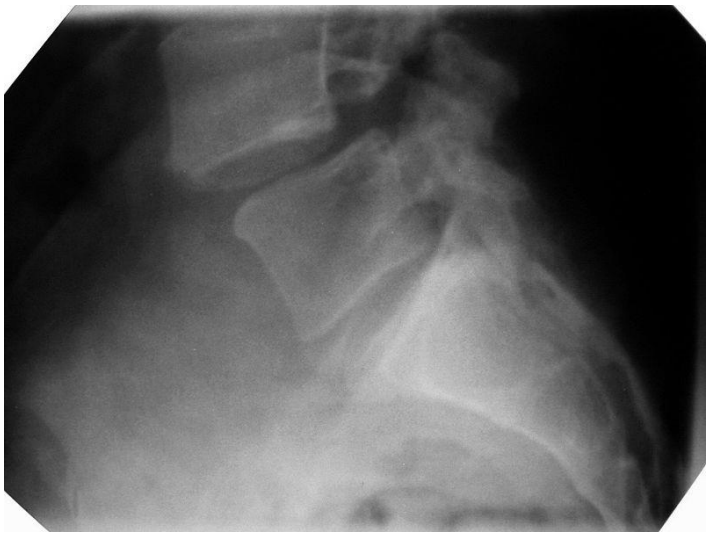


Figure 17 Spondylolisthesis at L4/5

Sport-Specific Biomechanics

Sports that involve repetitive hyperextension and axial loading of the lumbar spine may result in repetitive microtrauma to the pars interarticularis, resulting in spondylolysis and sometimes spondylolisthesis. Examples of such activities include gymnastics, football (lineman), wrestling, weight lifting (particularly standing overhead presses), rowing, pole vaulting, diving, hurdling, swimming (especially the butterfly stroke), baseball (especially pitching), tennis (especially serving), sailing (particularly the hiking manoeuvre), and volleyball. Gymnastics and football are generally considered the highest risk sports

History

Typical findings when obtaining the history from a patient with spondylolisthesis may include the following:

- The patient is usually asymptomatic.
- The onset usually occurs during the growth spurt in late childhood and early adolescence, probably due to increased participation in strenuous sports during this period.
- Spondylolisthesis is an unlikely cause of back pain in adults (especially after age 40 y) with no history of symptoms before age 30 years; usually, another cause is identified (e.g., disc, strain).
- Low back pain is the usual symptom reported, and it is often exacerbated by motion, particularly lumbar extension and twisting. Radiation of pain into the buttocks is not uncommon. The patient may report relief of pain with extended periods of rest.
- Rarely, associated leg pain is present in the L5 or S1 distribution as a result of nerve root compression.
- Symptoms are often more severe during the advanced months of pregnancy

Physical Examination

Findings noted during the physical examination may include the following:

- With high-grade slips, a palpable step-off may be felt over the spinous process at the level above the slipped vertebra because the posterior arch of the forward translated vertebra remains in place.
- Tenderness to deep palpation of the spinous process above the slip (typically L4) may be present. This palpation occasionally causes radicular pain.
- A positive one-leg hyperextension test (stork test) suggests a diagnosis of spondylolysis, but it is a nonspecific test with low sensitivity and low specificity.
- Hamstring tightness that is associated with all grades of symptomatic spondylolisthesis (see Grading) occurs at a rate of 80%. It commonly results in an abnormal gait, typically waddlelike, due to the inability of the patient to flex the hip with the knees extended.
- Paraspinal muscle spasm and tenderness are usually present.
- In advanced cases, a relatively short torso with a low rib cage, high iliac crests, and heart-shaped buttocks are noted.
- Limited forward flexion of the trunk is common with reduced straight-leg raising, which may cause pain but rarely any signs of nerve root tension.
- Postural deformity and a transverse abdominal crease are seen as a result of the pelvis being thrust forward.
- A thorough neurologic evaluation should be performed, including sensation in the sacral region to check for cauda equina compression.
- Weakness in the tibialis anterior muscle (L4 nerve root) is common.

Lumbosacral Spondylolisthesis Differential Diagnoses

Diagnostic considerations include:

- Discogenic
- Infectious (discitis, osteomyelitis)
- Mechanical low back pain (acute or chronic musculotendinous or ligamentous injuries, overgrowth syndrome, postural deformities)
- Neoplastic (osteoid osteoma, aneurysmal bone cyst, chondroblastoma)
- Spondylolysis / spondylolisthesis (acute [rare] vs chronic)
- Vertebral growth plate injuries (growth plate fractures, Scheuermann’s)

Differential Diagnoses

- Degenerative Lumbar Disc Disease in the Mature Athlete
- Lumbar Disk Problems in the Athlete
- Lumbosacral Disc Injuries
- Lumbosacral Discogenic Pain Syndrome
- Lumbosacral Facet Syndrome
- Lumbosacral Radiculopathy
- Lumbosacral Spine Acute Bony Injuries
- Lumbosacral Spine Sprain/Strain Injuries
- Lumbosacral Spondylolysis
- Myofascial Pain in Athletes
- Pars Interarticularis Injury
- Sacroiliac Joint Injury

Radiography

- Standing lateral radiographs are the preferred method of evaluating slippage of the vertebrae in persons with spondylolisthesis, and they are an excellent means of monitoring for progression of the condition.
- The standing lateral view is best because the translation occurs in the sagittal plane and is often accentuated during standing (due to the oblique orientation of the lower lumbosacral intervertebral disc spaces).
- Standing flexion/extension films should be obtained to assess the degree of instability of the involved vertebrae. These radiographs are also useful in detecting an occult spondylolisthesis.
- The anteroposterior view offers limited information in mild cases of spondylolisthesis; however, in cases of severe slips, this view may reveal the so-called reverse Napoleon hat sign because the L5 vertebra is viewed end-on through the sacrum, giving rise to the appearance of an upside-down Napoleon hat.
- Oblique films are best for evaluating the integrity of the pars interarticularis. A defect is seen as a collar on the neck of the Scotty dog.

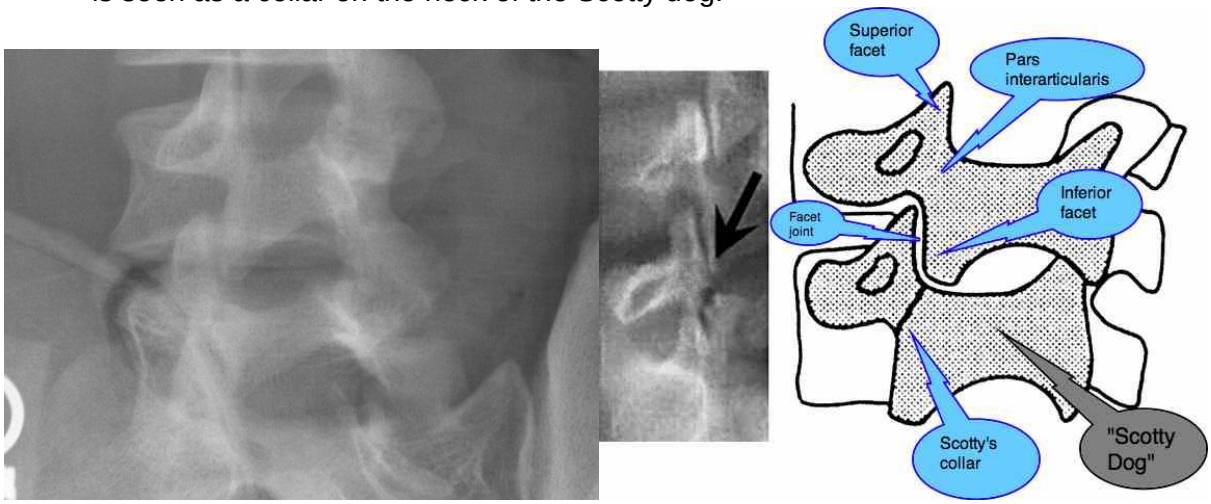


Figure 18 Spondylolysis showing 'scotty dog'

Grading spondylolisthesis

Meyerding technique: This involves dividing the superior aspect of the vertebra below the slip into 4 equal divisions, as is observed on a lateral radiograph. Assess where the posterior arch of the slipped vertebral body lies with respect to these 4 quadrants.

- Grade 1: Less than 25% slippage
- Grade 2: Between 25% and 50% slippage
- Grade 3: Between 50% and 75% slippage
- Grade 4: Between 75% and 100% slippage
- Grade 5: Greater than 100% slippage (also called spondyloptosis)

Lumbosacral Spondylolisthesis Treatment & Management

Acute Phase Physical Therapy

As a general rule, physical therapy should not be started until after an adequate rest period and once pain with daily activities has subsided.

The goals of physical therapy are to decrease extension stresses of the lumbar spine and to strengthen elements that promote an antilordotic posture. This consists of exercises to

strengthen the abdominal muscles (e.g., William flexion-type exercises) and flexibility programs to stretch the spinal extensor muscles, hamstrings, and lumbodorsal fascia.

Bracing with a thoracolumbosacral orthosis (e.g., Boston antilordotic brace) may offer relief for those who do not respond to activity restrictions or whose daily activities are producing symptoms. This type of bracing is usually effective in most patients with less than 50% slippage. The brace is generally worn for 3-6 months and may be worn during activity.

If the slippage is less than 50% but the patient is symptomatic, then non-operative therapy (e.g., stretching and strengthening exercises, antilordotic brace and activity modification) is instituted. If pain continues to persist, then a spinal fusion is recommended.

Occupational Therapy

Avoidance of heavy-duty labour or activities with repetitive lumbar extension is necessary to allow healing to occur. An occupational therapist can be very beneficial for those individuals who need instructions and compensatory strategies for activities of daily living.

Recreational Therapy

Restriction from sports and other activities that require repetitive hyperextension may be sufficient treatment in young athletes. Patients with grade 2 slippage are generally instructed to avoid hyperextension loading of the spine after symptoms resolve with conservative treatment.

Medical Issues/Complications

Younger patients require more careful observation, even if the initial symptoms resolve, because of their greater risk for progression. In an asymptomatic child with slippage up to 25% (grade 1), initially observe with radiographs every 4-6 months if younger than age 10 years, semi-annually until age 15 years, then annually until the end of growth. No limitation of activities is required, but the patient is advised to avoid occupations that entail heavy labour. If the slippage is 26-50% (grade 2) and the patient is asymptomatic, then the treatment is the same as for the grade 1 slippage but with a warning against participation in contact sports or sports requiring lumbar hyperextension (e.g., football, gymnastics). In general, the results of conservative management are good in most athletes with Grade I or II slips.

Complications include slip progression, loss of motion segments, neurologic deficit (e.g., cauda equina syndrome, radiculopathy [greatest risk with >50% slippage]), and residual deformity (following fusion of a high-grade spondylolisthesis).

Surgical Intervention

Surgery is indicated for skeletally immature patients with greater than 30-50% slippage (with or without symptoms) because they are at greater risk for progression, in the event of progressive neurologic deficit, or in those with pain persisting for more than 6-12 months that has not been relieved with rest and immobilization with any degree of slip. Spondylolysis or low-grade spondylolisthesis may be managed non-operatively.

Options for operative management include direct repair of the spondylolytic defect, fusion in situ, reduction and fusion, and vertebrectomy. Ideally, repair of a pars defect is for young patients with spondylolysis but no spondylolisthesis. Best results are observed in those with a lytic defect between L1 and L4. L5 defects yield less predictable results. Disc degeneration as seen on MRI is a relative contraindication. Slippage of greater than 2 mm decreases the likelihood of successful repair.

Fusion in situ at the involved level is the criterion standard of surgical treatment for most patients in whom conservative management fails. Fusion in situ is recommended for patients with persistent, symptomatic, low-grade spondylolisthesis and for patients who are not candidates for repair of the pars defect. The desire to participate in a contact sport should not be the sole indication for a fusion.

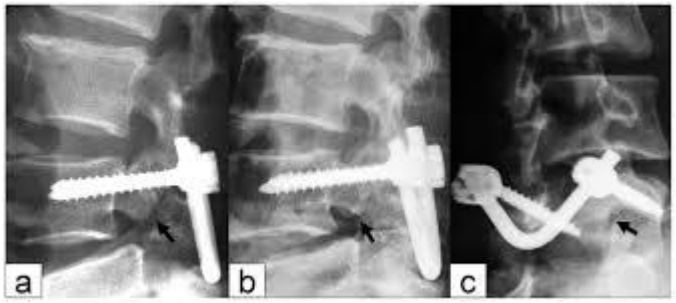


Figure 19 Spondylolysis fixation

Decompression and fusion are typically performed in cases of dural sac compression with the presence of bowel or bladder dysfunction or significant motor deficits. Decompression is never performed without concomitant fusion. Pedicle screw fixation enables rapid mobilization and early ambulation after decompression and fusion. Fixation may be beneficial in repairing pseudoarthrosis and, in the face of laminectomy, in preventing further slippage while awaiting fusion.

Spondylolisthesis reduction is performed either through closed or open procedures. Reduction serves to correct lumbosacral kyphosis and to diminish sagittal translation observed in high-grade slips. Vertebrectomy may be used to treat spondyloptosis (grade 5 spondylolisthesis), as an alternative procedure to reduction or fusion in situ. The postoperative rate of permanent neurologic deficits is high (25-30%), although many are pre-existent. This does not appear to be balanced by improved results; fusion in situ has achieved similar clinical outcomes with a lower complication rate.

Spondylolysis

Spondylolysis is a defect of a vertebra. More specifically it is defined as a defect in the pars interarticularis of the vertebral arch. It can progress until one or more vertebrae slip out of place which is then called **spondylolisthesis** (see above).

The great majority of cases occur in the lowest of the lumbar vertebrae (L5), but spondylolysis may also occur in the other lumbar vertebrae, as well as in the thoracic vertebrae.

Spondylolysis occurs in three to six percent of the population

Spondylolysis pain can lead to reduced mobility and inactivity. Inactivity can result in weight gain, loss of bone density, and loss of muscle strength and flexibility of other areas of the body.

It is diagnosed via oblique X-ray (Scotty dog), or CT scan, showing a flaw or damage of the pars interarticularis.

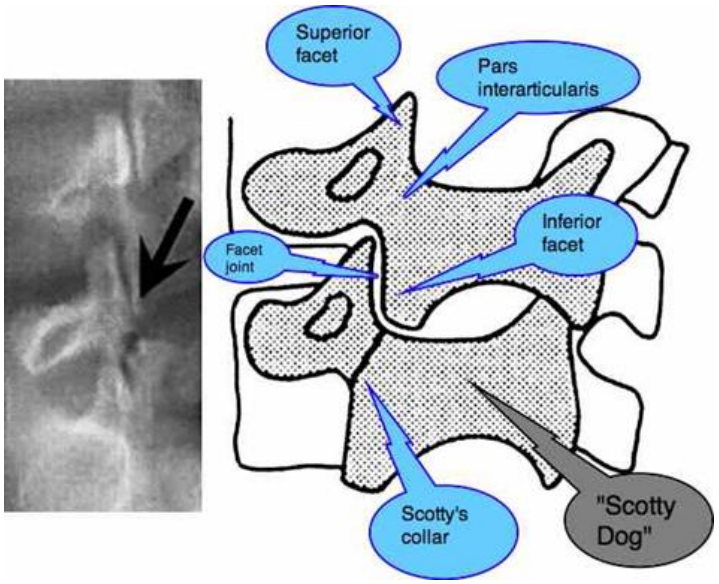


Figure 20 Spondylolysis on oblique X-Ray

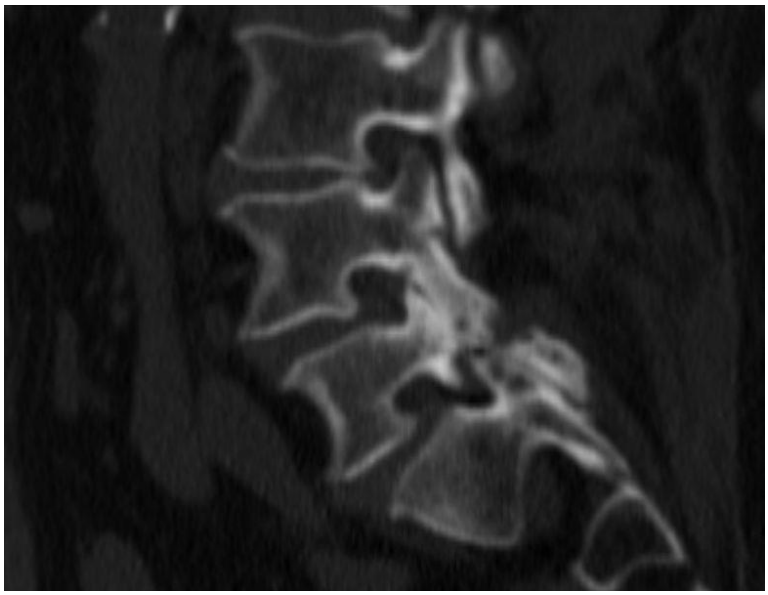


Figure 21 Spondylolysis on CT scan

Spinal stenosis

Spinal stenosis is an abnormal narrowing (stenosis) of the spinal canal that may occur in any of the regions of the spine. This narrowing causes a restriction to the spinal canal, resulting in a neurological deficit.

Symptoms include:

- Pain
- Numbness
- Paraesthesia
- Loss of motor control

The location of the stenosis determines which area of the body is affected. With spinal stenosis, the spinal canal is narrowed at the vertebral foramen, which is a foramen between the vertebrae where the spinal cord (in the cervical or thoracic spine) or nerve roots (in the lumbar spine) pass through. There are several types of spinal stenosis, with lumbar stenosis and cervical stenosis being the most frequent. While lumbar spinal stenosis is more common, cervical spinal stenosis is more dangerous because it involves compression of the spinal cord whereas the lumbar spinal stenosis involves compression of the cauda equina.

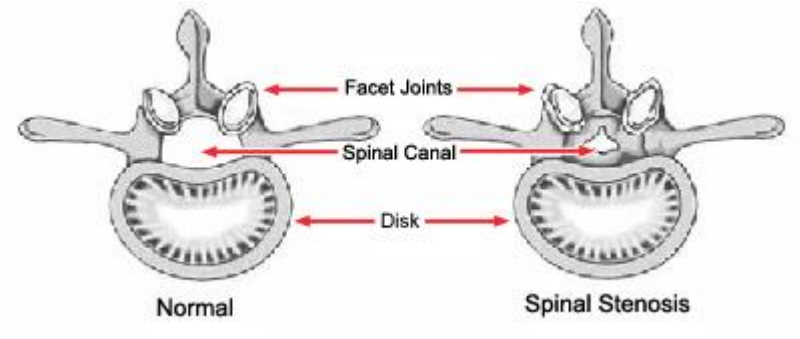


Figure 22 Spinal Stenosis

Types

The most common forms are cervical spinal stenosis, at the level of the neck, and lumbar spinal stenosis, at the level of the lower back. Thoracic spinal stenosis, at the level of the mid-back, is much less common

In lumbar stenosis, the spinal nerve roots in the lower back are compressed which can lead to symptoms of sciatica (tingling, weakness, or numbness that radiates from the low back and into the buttocks and legs).

Cervical spinal stenosis can be far more dangerous by compressing the spinal cord. Cervical canal stenosis may lead to serious symptoms such as major body weakness and paralysis. Such severe spinal stenosis symptoms are virtually absent in lumbar stenosis, however, as the spinal cord terminates at the top end of the adult lumbar spine, with only nerve roots (cauda equina) continuing further down. Cervical spinal stenosis is a condition involving narrowing of the spinal canal at the level of the neck. It is frequently due to chronic degeneration but may also be congenital or traumatic. Treatment frequently is surgical

What Causes Spinal Stenosis?

There are many potential causes for spinal stenosis, including:

- Aging: With age, the body's ligaments (tough connective tissues between the bones in the spine) can thicken. Spurs (small growths) may develop on the bones and into the spinal canal. The cushioning disks between the vertebrae may begin to deteriorate. The facet joints (flat surfaces on each vertebra that form the spinal column) also may begin to break down. All of these factors can cause the spaces in the spine to narrow.
- Arthritis: Two forms of arthritis that may affect the spine are osteoarthritis and rheumatoid arthritis.
- Heredity: If the spinal canal is too small at birth, symptoms of spinal stenosis may show up in a relatively young person. Structural deformities of the involved vertebrae can cause narrowing of the spinal canal.
- Instability of the spine, or spondylolisthesis: When one vertebra slips forward on another, which can narrow the spinal canal.
- Tumours of the spine: Abnormal growths of soft tissue may affect the spinal canal directly by causing inflammation or by growth of tissue into the canal. Tissue growth may lead to bone resorption (bone loss due to over-activity of certain bone cells) or displacement of bone and the eventual collapse of the supporting framework of the spinal column.
- Trauma: Accidents and injuries may either dislocate the spine and the spinal canal or cause burst fractures that produce fragments of bone that penetrate the canal.

When Should Surgery Be Considered and What Is Involved?

In many cases, the conditions causing spinal stenosis cannot be permanently altered by nonsurgical treatment, even though these measures may relieve pain for a period of time. To determine how much nonsurgical treatment will help, a doctor may recommend such treatment first. However, surgery might be considered immediately if a patient has numbness or weakness that interferes with walking, impaired bowel or bladder function, or other neurological involvement. The effectiveness of nonsurgical treatments, the extent of the patient's pain, and the patient's preferences may all factor into whether or not to have surgery.

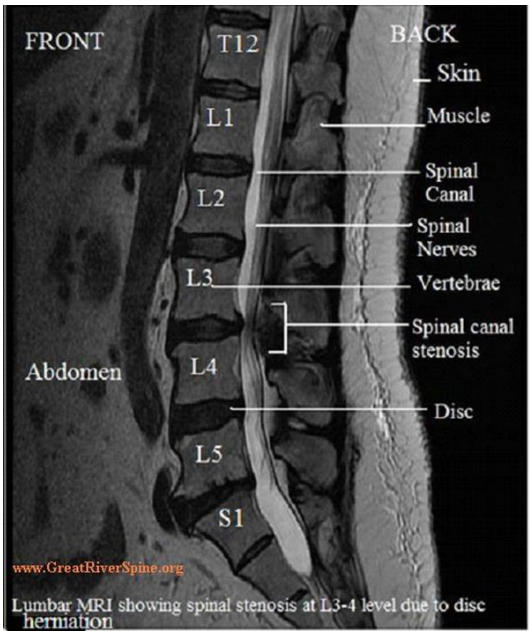


Figure 23 MRI scan of spinal stenosis at L3/4

The purpose of surgery is to relieve pressure on the spinal cord or nerves and restore and maintain alignment and strength of the spine. This can be done by removing, trimming, or adjusting diseased parts that are causing the pressure or loss of alignment. The most common surgery is called decompressive laminectomy: removal of the lamina (roof) of one or more vertebrae to create more space for the nerves. A surgeon may perform a laminectomy with or without fusing vertebrae or removing part of a disk. Various devices may be used to enhance fusion and strengthen unstable segments of the spine following decompression surgery.

Patients with spinal stenosis caused by spinal trauma or achondroplasia may need surgery at a young age. When surgery is required in patients with achondroplasia, laminectomy (removal of the roof) without fusion is usually sufficient.

Facet syndrome

Facet syndrome is a syndrome in which the zygapophyseal joints cause back pain. In this respect it may also be known as **spondylarthosis**; a condition affecting the facet joints of the spine.

55% of facet syndrome cases occur in cervical vertebrae, and 31% in lumbar. Facet syndrome can progress to spinal osteoarthritis, which is known as spondylosis. Pathology of the C1-C2 (atlantoaxial) joint, the most mobile of all vertebral segments, accounts for 4% of all spondylosis.

It can be generally regarded as a progression of ‘wear and tear’ of the spine; narrowing of the disc space. If the weight bearing part of the vertebral column, the disc, is narrowed, then the facet joints may begin to bear weight. The problem is that the facet joints are not designed to bear weight and they protest.

It might be said that inflamed facets can cause a powerful muscle spasm. On the other hand, any muscle tightness can cause compression along the axis of the spine, resulting in facet syndrome.

Intervertebral Disc Herniation

Spinal disc herniation (commonly called a *slipped disc,* though this term is not medically accurate as the spinal discs are firmly attached between the vertebrae and cannot "slip") is a medical condition affecting the spine in which a tear in the outer, fibrous ring (*annulus fibrosus*) of an intervertebral disc allows the soft, central portion (*nucleus pulposus*) to bulge out beyond the damaged outer rings.

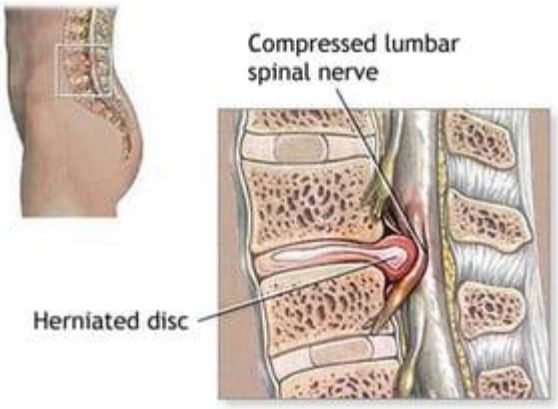


Figure 24 Diagram of herniated intervertebral disc

Disc herniation is usually due to age related degeneration of the annulus fibrosus, although not always so and trauma, lifting injuries, or straining have been implicated. Here, though, it is important to say that we don't really know as there is not camera during the damage process; we only take photographs 'afterwards' to try to explain the pain. It is a working, structural, model that justifies our treatment protocol (structural repair).

Tears are almost always posterolateral in nature owing to the presence of the posterior longitudinal ligament in the spinal canal. This tear in the disc ring may result in the release of inflammatory chemical mediators, which may directly cause severe pain, even in the absence of nerve root compression.

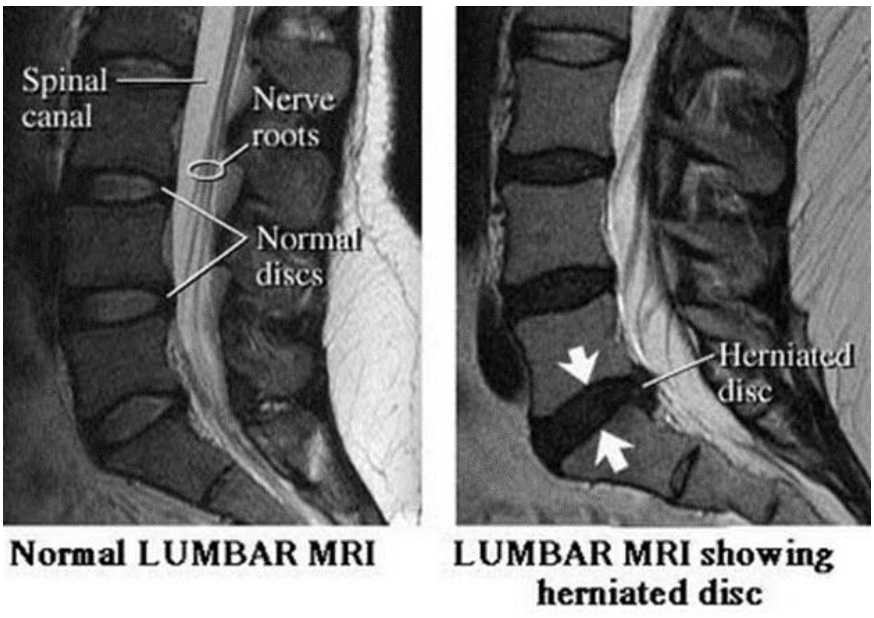


Figure 25 Prolapsed intervertebral disc

Disc herniations are normally a further development of a previously existing disc "protrusion", a condition in which the outermost layers of the anulus fibrosus are still intact but can bulge when the disc is under pressure. In contrast to a herniation, none of the nucleus pulposus escapes beyond the outer layers. Most minor herniations heal within several weeks. Anti-inflammatory treatments for pain associated with disc herniation, protrusion, bulge, or disc tear are generally effective.

Severe herniations may not heal of their own accord and may require surgical intervention.

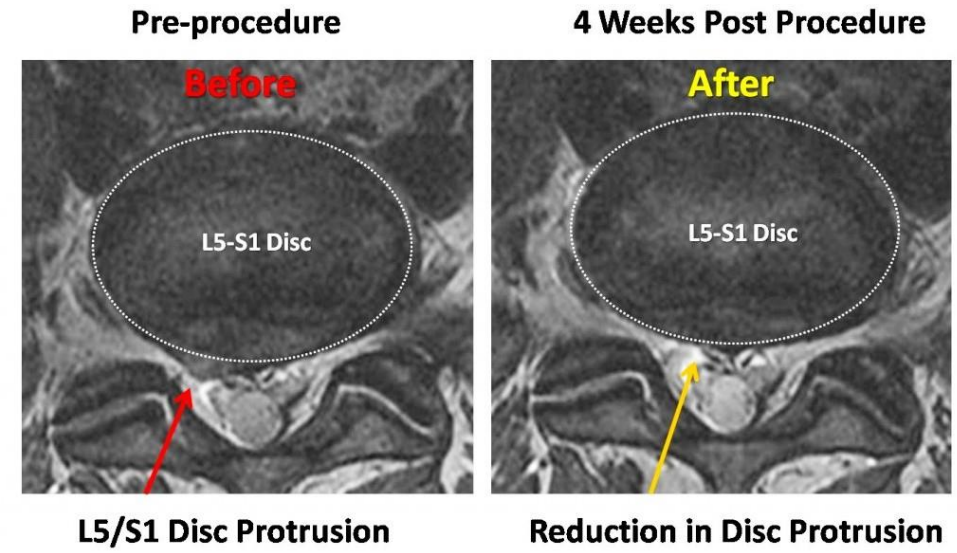


Figure 26 Disc prolapse; pre and post-operation

Osteitis Condensans Ilii

Osteitis condensans ilii is a rare, benign, cause of low back pain, which is self-limiting and has an incidence rate of 0.9%-2.5%, mostly among women in the prepartum and postpartum period. Although osteitis condensans ilii is still an orthopaedic mystery, mechanical stress across the joint is a significant triggering factor according to the prevailing theories.

The traditional location of involvement is around the ileum and can be misinterpreted as sacroiliac joint involvement. Osteitis condensans ilii is one of the benign aetiologies of chronic axial low back pain. Most of the time, osteitis condensans ilii is an incidental finding on plain X-ray with ileal sclerosis. Even though osteitis condensans ilii is predominantly found in women of childbearing age in the prepartum or postpartum period, it can also present in nulliparous women and men. It is commonly misdiagnosed as sacroiliitis, with the aetiology being around the Ileum.

In general, osteitis condensans ilii is asymptomatic, but in a few patients, it manifests with low back pain at a young age which mimics axial spondyloarthropathy. Aetiologies involving sacroiliac joint needs to be considered in differential diagnosis.

Initially, osteitis condensans ilii was considered as one variety of ankylosing spondylitis, though human leukocyte antigen (HLA) B27 is predominantly negative among the patients and, with exceptional cases, is generally not associated with elevated inflammatory markers and hence not classified as inflammatory arthritis.

The diagnosis of osteitis condensans ilii is commonly made on radiological findings which should be differentiated from spondyloarthropathies, inflammatory arthritis, and malignancy. A typical radiological finding to distinguish osteitis condensans ilii from other sacroiliac abnormalities is the triangular shape of sclerosis at the iliac border with preserved joint space. The hallmark of osteitis condensans ilii is sclerosis of the articular portion of the iliac bone.

The figure shows an anterior-posterior view showing significant sclerosis at the iliac border of the sacroiliac joints.

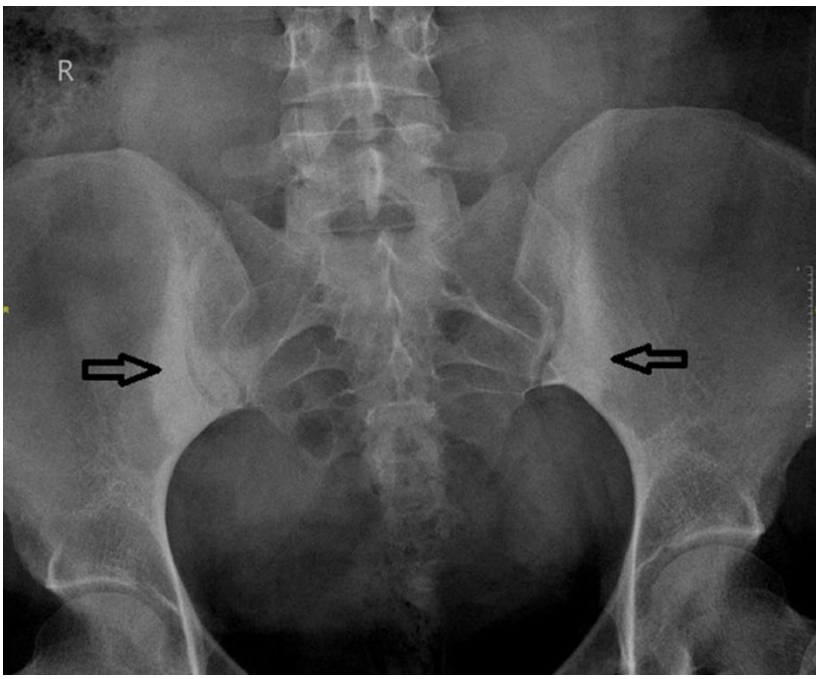


Figure 27 X-ray through the sacroiliac joints showing osteitis condensans ilii

The increased vascularity in the ileum leads to remodelling of bone causing sclerosis. Histopathology of the sclerosed bone showed increased lamellar bone in biopsies of the affected region.

Still, the pathophysiology of osteitis condensans ilii is not clearly understood, but the increased mechanical stress on ileum is considered one of the causative factors of osteitis condensans ilii in pregnant women.

In general, osteitis condensans ilii is asymptomatic, but in a few patients, it manifests with low back pain at a young age which mimics axial spondyloarthropathy.

The pain of osteitis condensans ilii might radiate to the bilateral gluteal area and posterior aspect of the thighs along with positive SIJ tenderness as confirmed by a positive Faber's test.

Management involves physical therapy and the use of non-steroidal anti-inflammatory drugs and muscle relaxants as needed. Though osteitis condensans ilii symptoms are self-limiting and even radiological findings can disappear with time, it is essential to diagnose osteitis condensans ilii as refractory cases can cause a varying degree of disabilities and might require surgical intervention.

Lumbosacral transitional vertebrae

The design of the spine is complex enough already, with the majority of the body’s weight being transferred through the L5/S1 junction. Normally there are 5 vertebrae in both the lumbar and sacral regions of the spine, with the bones of S1-5 fused together. To make this more awkward, congenital variation can occur at the L5/S1 level.

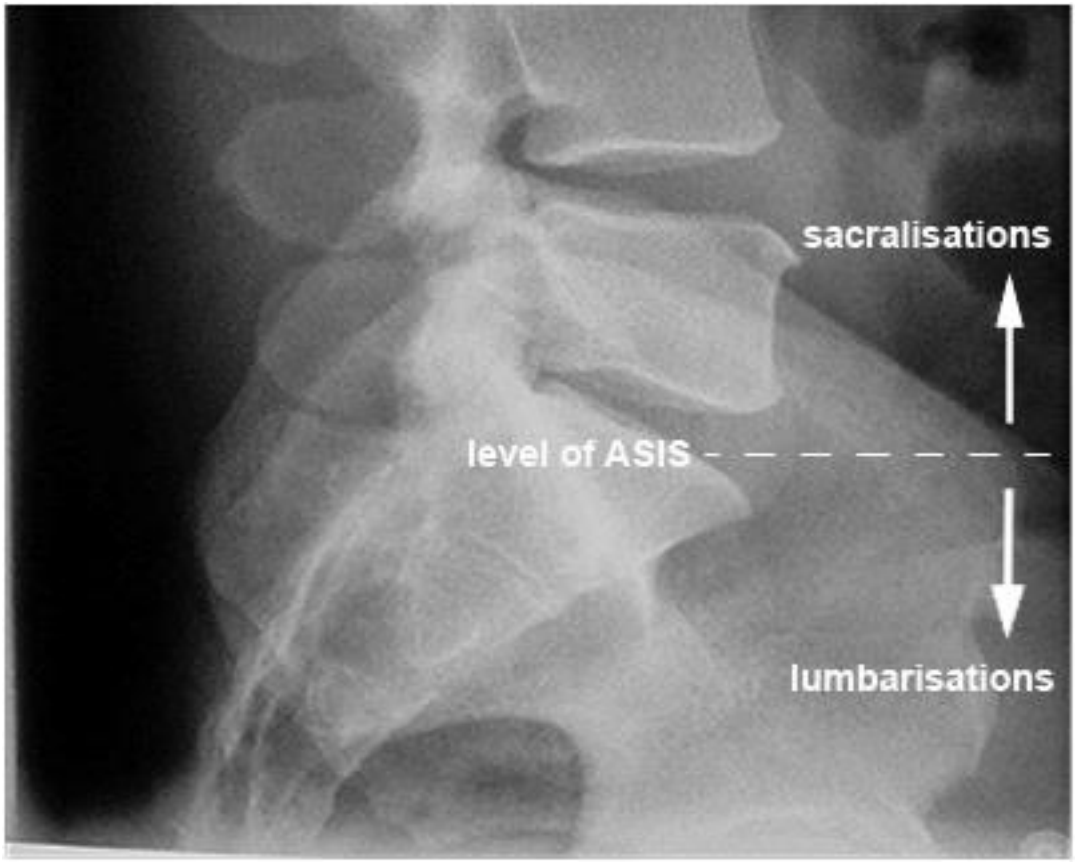


Figure 28 X-ray of lumbosacral region

The picture above demonstrates the principles of **lumbarisation** and **sacralisation.**

With all this, though, there are two main possibilities for a lumbosacral transitional vertebra:

- **Lumbarisation of S1**
- **Sacralisation of L5**

These sound like deliberately confusing terms, but remember that the number of bones in each section of the spine remain the same, any changes in this number causes changes that can occur in the function of the region:

- In a lumbarised S1, the S1 vertebra functionally becomes part of the lumbar spine
    - Effectively creating a longer lumbar spine
- In a sacralised L5, the L5 vertebra functionally becomes part of the sacrum
    - Effectively creating a shorter lumbar spine

The **Castellvi classification** is used for lumbosacral transitional vertebra

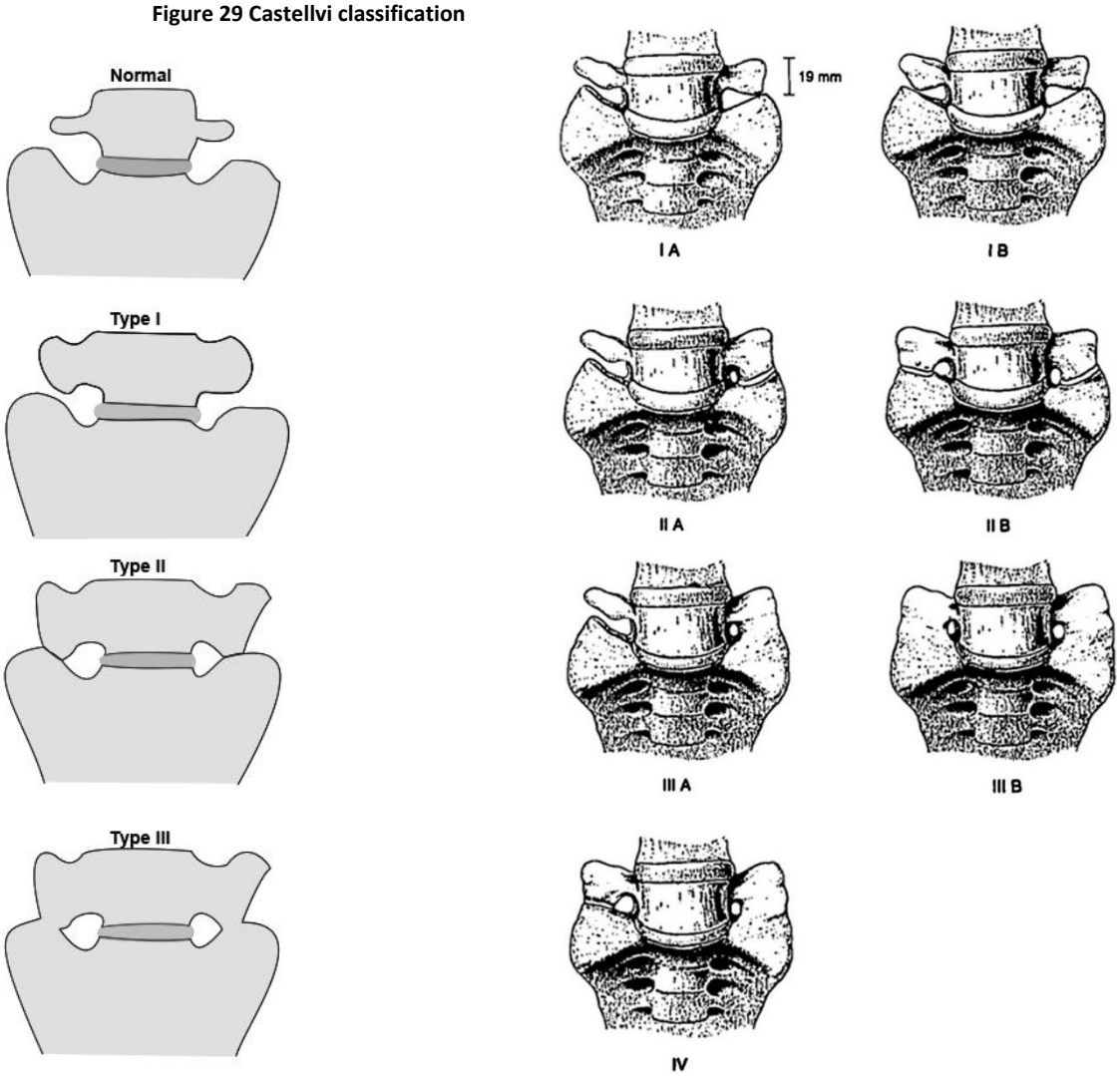
- **Type I -** enlarged and dysplastic transverse (at least 19 mm)
    - **Ia** - unilateral
    - **Ib** - bilateral

- **Type II -** pseudoarticulation of the transverse process and sacrum with incomplete lumbarisation/sacralisation; enlargement of the transverse process with pseudoarthrosis
    - **IIa** - unilateral
    - **IIb** – bilateral

- **Type III -** transverse process fuses with sacrum and there is complete lumbarisation or sacralisation, enlarged transverse process with complete fusion
    - **IIIa** - unilateral
    - **IIIb** - bilateral

- **Type IV -** type IIa on one side and type III on contralateral side

Figure 29 Castellvi classification



Lumbarisation of S1

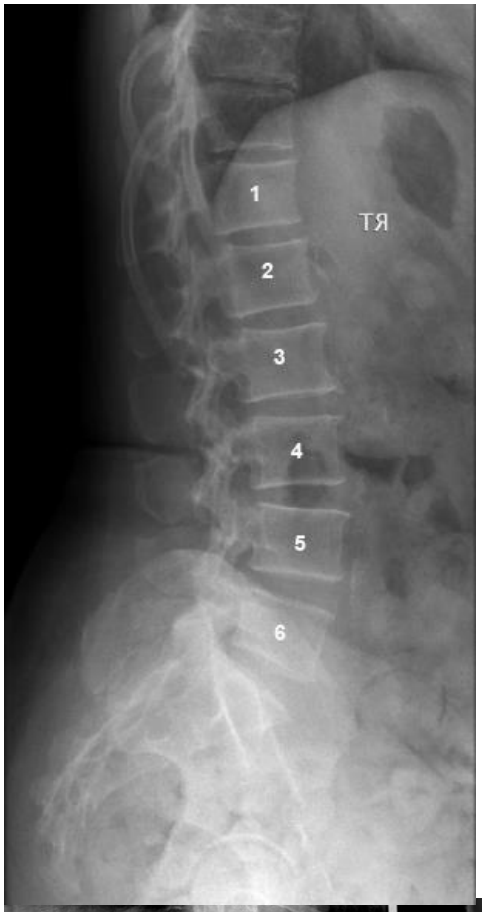


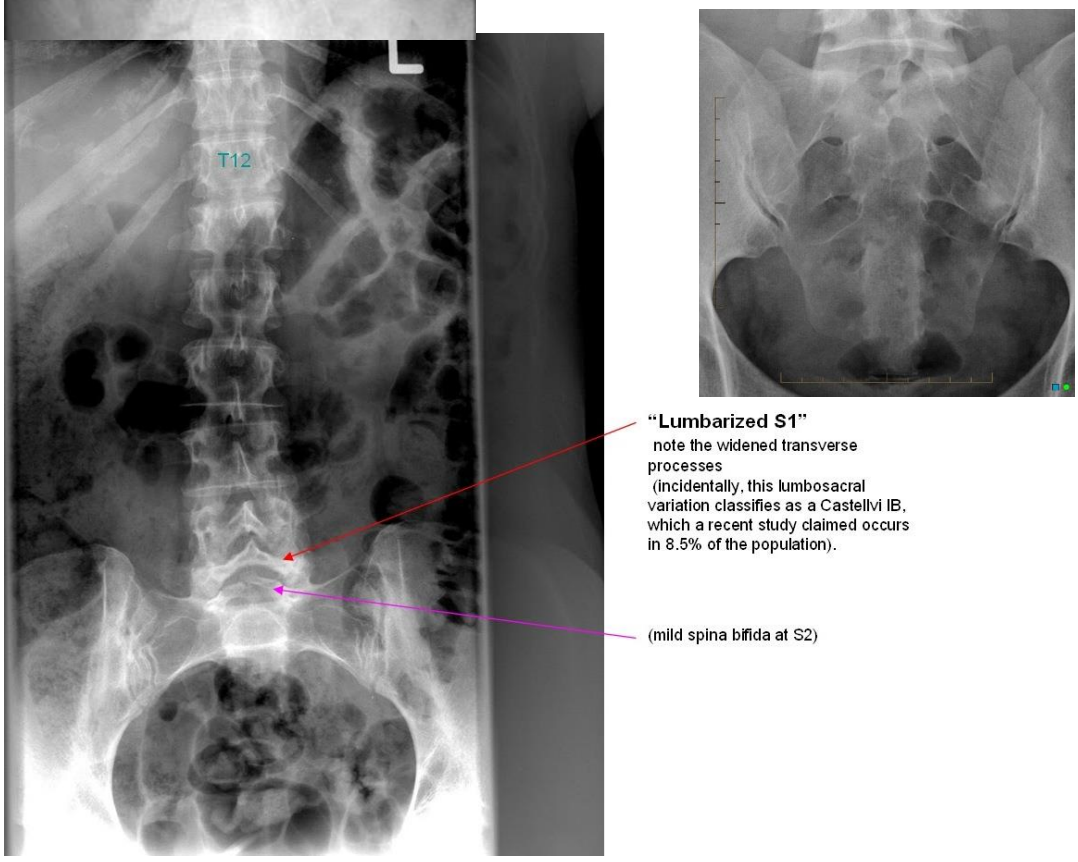
Figure 30 Lumbarised S1 - lateral view

This congenital situation can cause confusion, even to the experienced eye. This is one of those confusing transitional vertebra cases that defies a clear categorisation (left).

There are 6 vertebra that demonstrate lumbar characteristics. The verterbra above the vertrebra annotated with "1" has associated ribs. Are these the 12th ribs or are these the 11th ribs with congenital absence of the 12th ribs?

Seeing this picture, and recalling that the sacroiliac joints (see inset) extend between S1-3, questions arise about whether this condition can be blamed on low back pain when it occurs. This X-ray clearly shows that the S/I joints are of reasonably normal configuration, but the bony apparatus does not extend to include S1. It is sometimes referred to as ‘L6’, but this is not strictly true as there is no L6 nerve root.

Figure 31 Sacralised L5 - A/P view



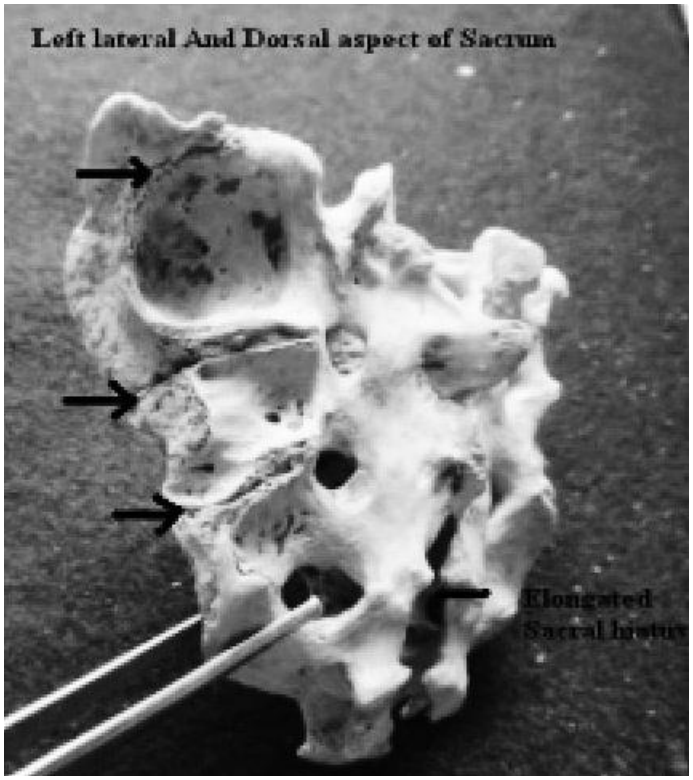


Figure 32 Sacrum showing vestigial joint spaces

The association of lumbosacral transitional vertebra and low back pain is known as **Bertolotti's syndrome**. This syndrome was characterized by the presence of a variation of the fifth lumbar vertebra having a large transverse process, either articulated or fused with sacral base or iliac crest and producing a chronic, persistent low back pain due to arthritic changes occurring at the site of pseudoarthrosis.

It may be a sweeping statement, but it might be said that such a condition only becomes a problem if the person has overt symptoms. Lumbarisation of S1 can potentially lead to instability, especially if associated with a pars interarticularis defect, leading to a spondylolisthesis, as seen here.

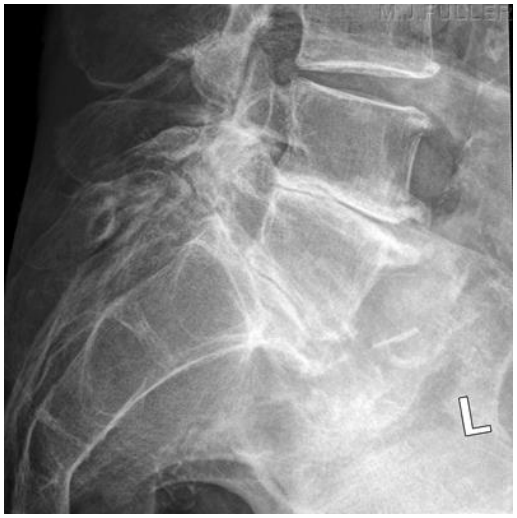


Figure 33 Spondylolisthesis with lumbarisation of S1

Sacralisation of L5

Sacralisation of L5 is the other variant of lumbosacral transitional vertebrae**.**

Lumbosacral region is one such region there are so many puzzling stresses and strains, renders the question more than ordinary interest. Occult sacralisation has been described by O'Connell 1951, where the sacrum is high in the pelvis and the spinous process of the last lumbar vertebra may be with or just below the iliac crests.
The lumbar spine shows five vertebrae, but the sacrum may be composed of six vertebral segments. As these are not cases of frank sacralisation of fifth lumbar vertebra, the term "Occult Sacralisation" has been used to describe the abnormality.

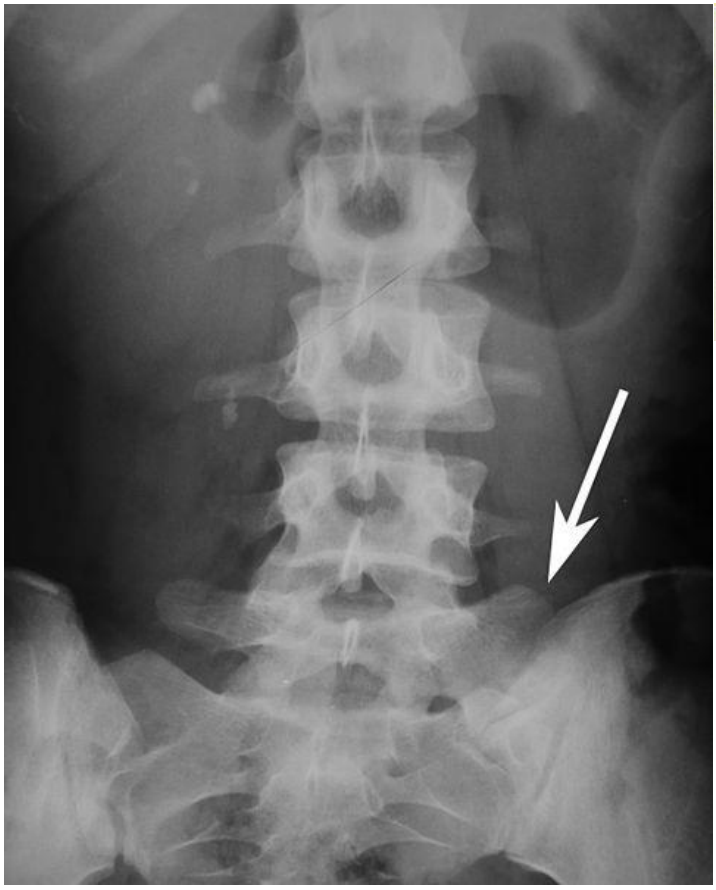


Figure 34 Sacralisation of L5

The upper picture demonstrates a type iv, according to Castellvi, whereas on the left is a type iiA.

Both will significantly reduce any motion between the two bones. With the two examples, the types iiA may permit some movement on the left (the right demonstrating at least a pseudoarthrosis on the right, but the type iv, as it is fused bilaterally, will allow no movement at all.

This type of condition reduces the number of bones in the lumbar spine, possibly reducing the overall movement, but also possibly putting a greater demand upon the lumbar structures and joints that are remaining.

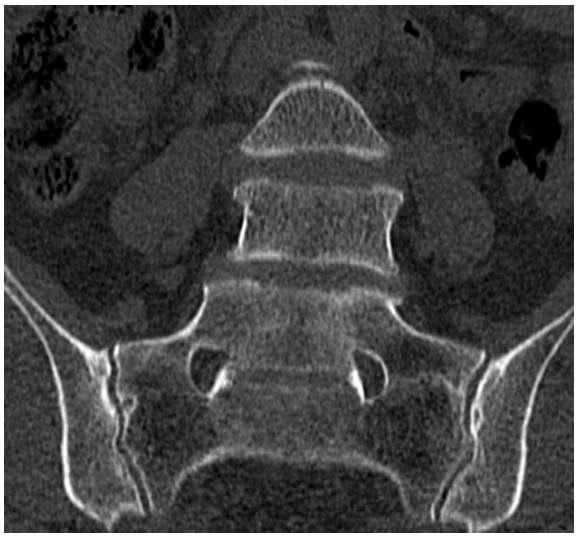


Figure 35 Sacralisation of L5 on CT scan

Disc bulge / herniation occurs nearly nine times more commonly at the interspace immediately above the lumbar transitional vertebra than at any other level.

Lumbosacral transitional vertebra increases the risk of early degeneration in the upper disc. The presence of a transitional vertebra should be noticed when morphologic methods are used in research on lumbosacral spine.

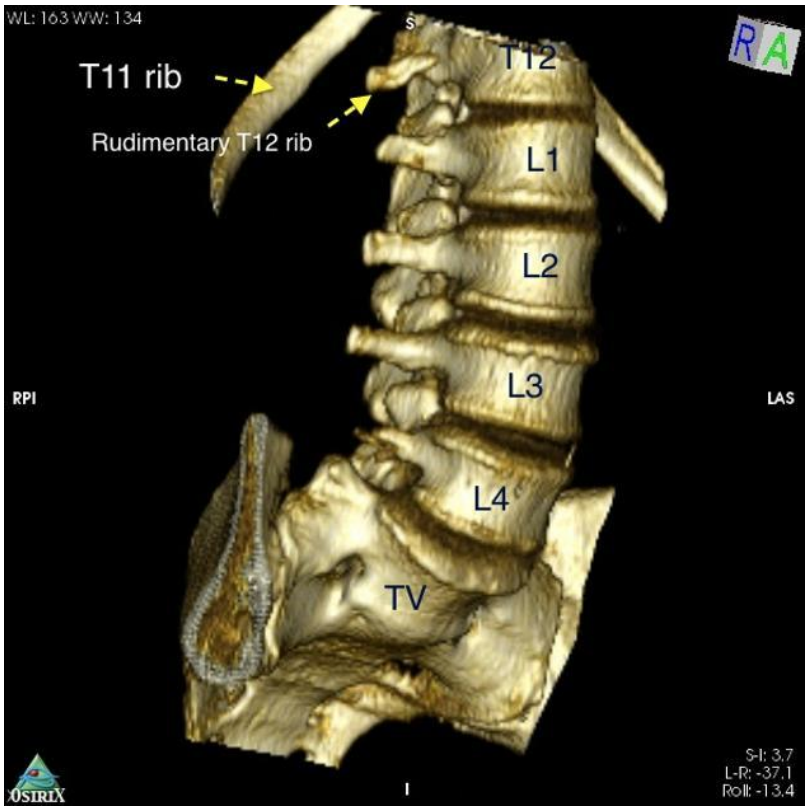


Figure 36 3D virtual reality scan of sacralised L5

This CT virtual reality scan shows four rib-free lumbar type vertebrae and one transitional vertebra characterised to assimilation of L5 to the sacrum.

There are also rudimentary T12 ribs

Whiplash and Cervical Spine Injuries

The majority of road traffic accidents precipitate a reactive spasm and chronic painful tension in the cervical spine and shoulders. This possibly associated with a mild, non-inflammatory, sprain of the associated ligaments. The bulk of these can be treated with soft tissue and myofascial techniques. There will always be the incident of severe injury.

This review is based on a presentation given by Adam Flanders and adapted for *the Radiology Assistant* by Robin Smithuis.

- Approximately 3 % of patients who present to the emergency department as the result of a motor vehicle accident or fall have a major injury to the cervical spine. 10-20% patients with head injury also have a cervical spine injury.
- Up to 17% of patients have a missed or delayed diagnosis of cervical spine injury, with a risk of permanent neurologic deficit after missed injury of 29%.
- Most cervical spine fractures occur predominantly at two levels.
- One third of injuries occur at the level of C2, and one half of injuries occur at the level of C6 or C7.

In this overview we will discuss the most common cervical spine injuries.

Flexion injuries

The most common fracture mechanism in cervical injuries is hyperflexion.

- **Anterior subluxation** occurs when rupture of the posterior ligamentous complex (PCL). Since the anterior and middle columns remain intact, this fracture is stable.

- **Simple wedge fracture** is the result of a pure flexion injury. The PCL remain intact. Anterior wedging of 3mm or more suggests fracture. Increased concavity along with increased density due to bony impaction. Usually involves the upper endplate.
- **Unstable wedge fracture** is an unstable flexion injury due to damage to both the anterior column (anterior wedge fracture) as the posterior column (interspinous ligament).
- **Unilateral interfacet dislocation** is due to both flexion and rotation.
- **Bilateral interfacet dislocation** is the result of extreme flection. BID is unstable and is associated with a high incidence of cord damage.
- **Flexion teardrop fracture** is the result of extreme flection with axial loading. It is unstable and is associated with a high incidence of cord damage.
- **Anterior atlantoaxial dislocation**

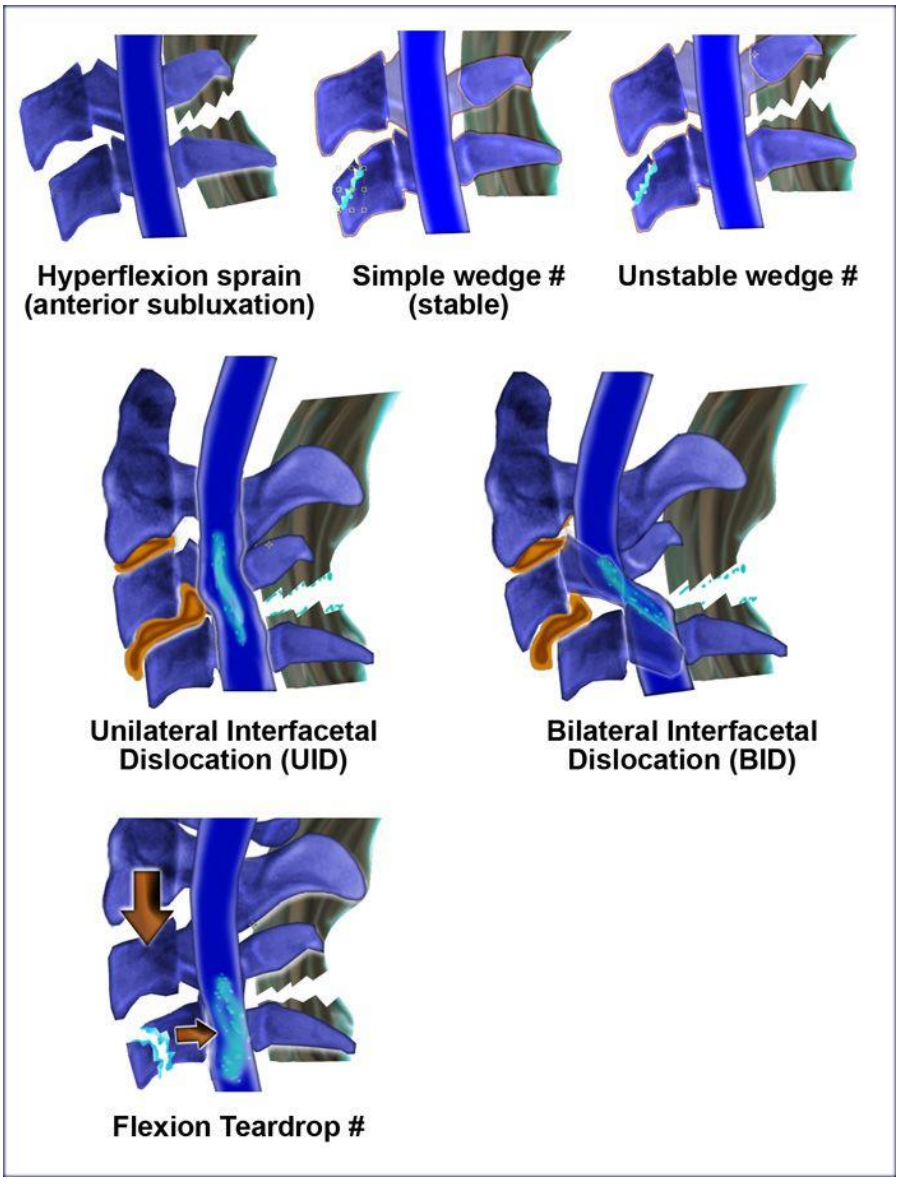


Figure 37 Cervical flexion injuries

Extension injuries

- **Hangman's fracture**
  - Traumatic spondylolisthesis and fracture of C2
- **Extension teardrop fracture**
  - Avulsion fracture via ALL on anterior body of C3
- **Hyperextension in pre-existing spondylosis**
  - 'Open mouth fracture'
  - Disruption of anterior ligaments and intervertebral disc (here) at C3/4

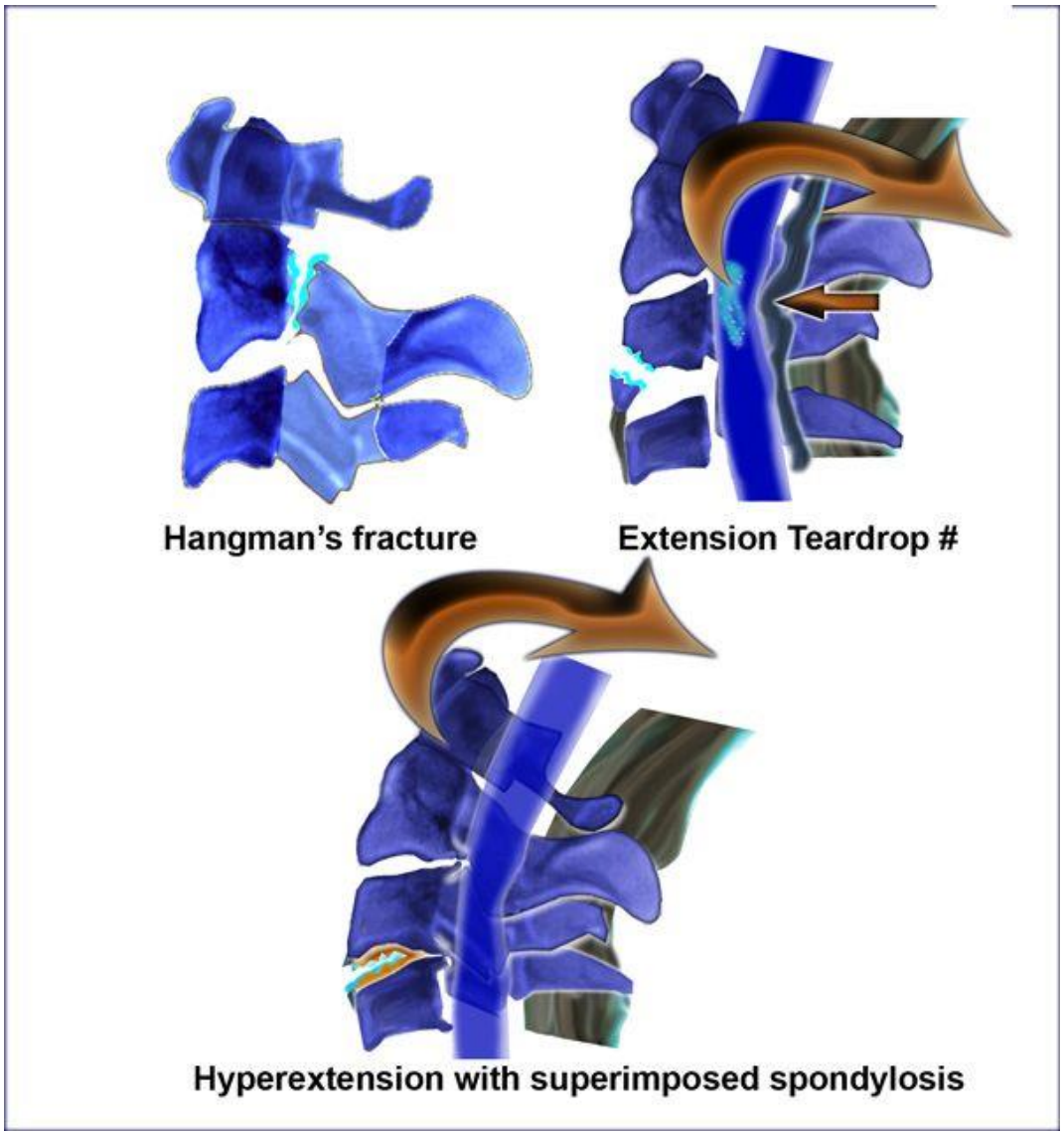


Figure 38 Cervical extension injuries

Flexion tear drop fracture

This fracture is the result of a combination of *flexion* and *compression*, which is usually the result of a motor vehicle accident.

The teardrop fragment comes from the anteroinferior aspect of the vertebral body. The larger posterior part of the vertebral body is displaced backward into the spinal canal.

On X-rays the facet joints and interspinous distances are usually widened and the disk space may be narrowed. 70% of patients have neurologic deficit.

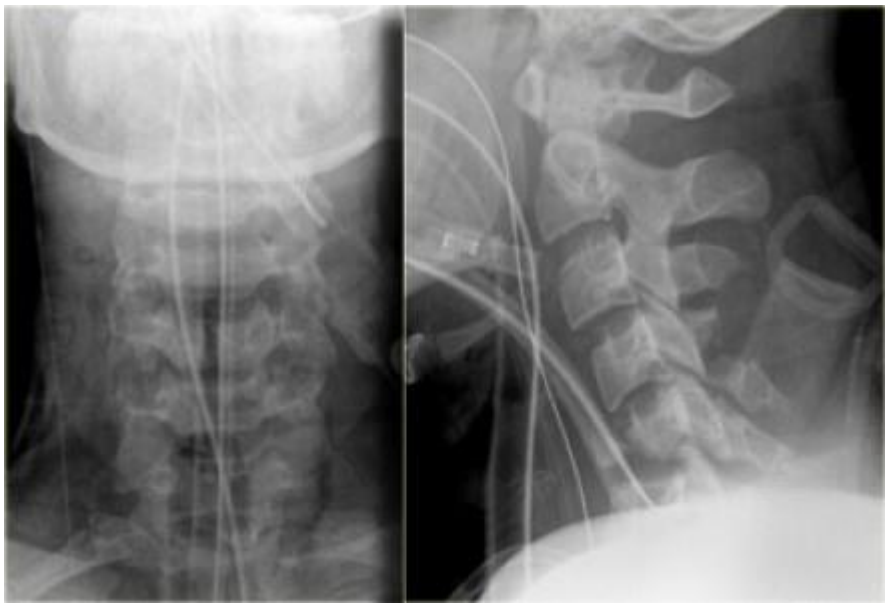
It is an unstable fracture associated with complete disruption of ligaments and anterior cord syndrome.

Figure 39 Cervical flexion tear drop fracture

Below are mages of a 21-year-old male who sustained a diving injury, striking his head in a swimming pool.
He had immediate onset of upper and lower extremity weakness.

The findings are:

- Fracture of the body of C5 with a small fragment anteriorly
- Fracture of the spinous processes of C4
- Acute angulation at the level of C5C6 with displacement of C5 in posterior direction



Some would just call this a severe hyperflexion injury, but this entity is better known as a 'flexion tear drop' fracture.

Figure 40 X-ray tear drop fracture

Additional findings on the CT-images:

- Abnormal positioning of some of the facet joints due to distraction but no dislocation
- Additional fracture of the body of C4 (blue arrow)
- The vertical orientation of the fractures of the bodies of C4 and C5 indicate that there was severe axial loading (red arrow)
- In fact these vertebral bodies kind of 'exploded' with propulsion of a bone fragment anteriorly (teardrop) and the larger part posteriorly against the spinal cord.

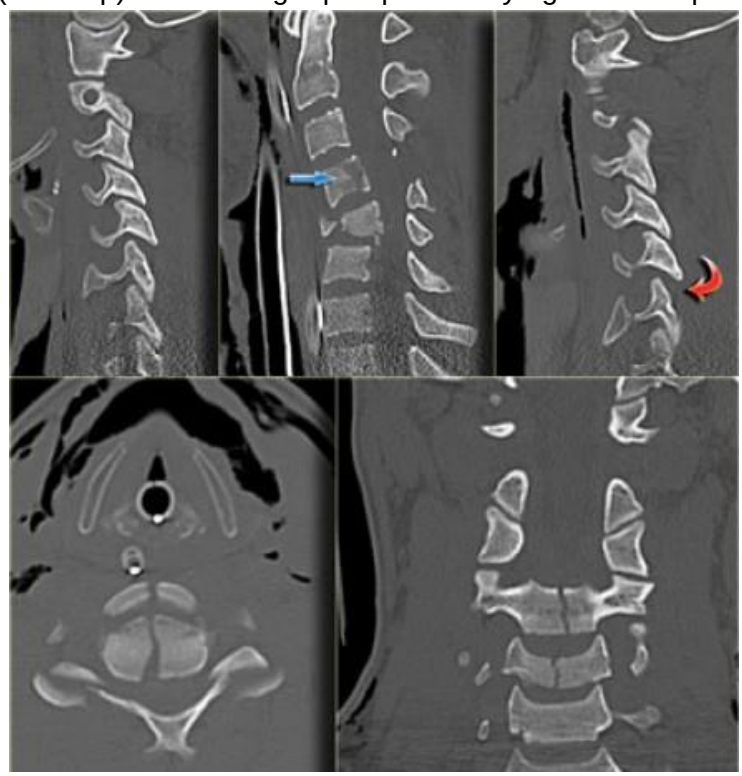


Figure 41 CT scan of tear drop fracture

The MRI findings are:

- Soft tissue injuries anteriorly and posteriorly with flavum and interspinous ligament rupture and CSF leakage.
- Haemorrhagic spinal cord injury!

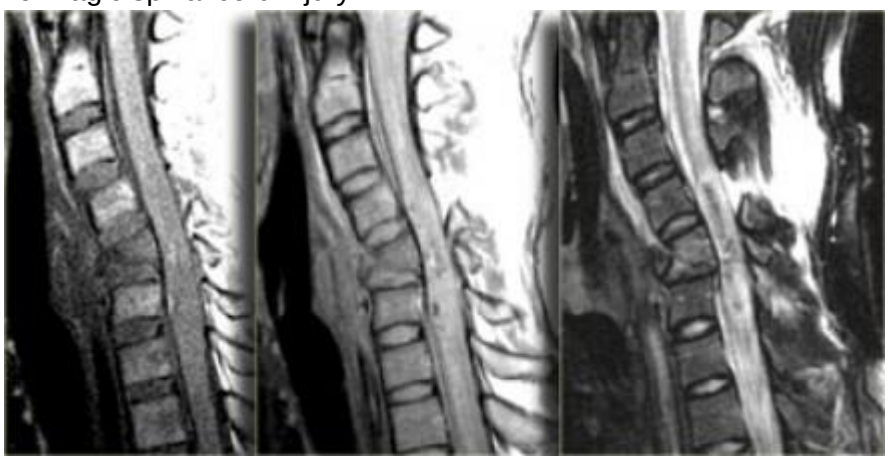


Figure 42 MRI scan of tear drop fracture

Spinal Fractures

Compression Fractures of the Spine

The vertebral bodies and processes consist of compact bones surrounding trabecular bone. In cases of osteoporosis and/or trauma, fractures can occur. The vertebral bodies in the lower thoracic and upper lumbar spine have mainly parallel superior and inferior epiphyseal plates

The type of fracture in the spine that is typically caused by osteoporosis is generally referred to as a **compression fracture**.

- A compression fracture is usually defined as a vertebral bone in the spine that has decreased at least 15 to 20% in height due to fracture.
- These compression fractures can occur in vertebrae anywhere in the spine, but they tend to occur most commonly in the upper back (thoracic spine), particularly in the lower vertebrae of that section of the spine (e.g. T10, T11, T12).
- They rarely occur above the T7 level of the spine, occurring in the upper lumbar segments as well, such as L1.

Classification of Fractures

Classifying the various types of spinal fractures can be confusing because there are several classification methods. Here the focus will be on the most basic and the most generally accepted classifications for spinal fractures.

Denis Classification: The Three-column Concept

The three-column spine was first introduced by Dr. Francis Denis in his aptly named paper, "The Three Column Spine and its Significance in the Classification of Acute Thoracolumbar Spinal Injuries". This paper, published in 1983, proposed a new biomechanical model for spinal stability that challenged Dr. Frank Holdsworth's two column model from the 1960s.

Denis' three column model proposes that the thoracolumbar spine can be divided into three columns. The first column includes the anterior longitudinal ligament (ALL) up to the first half of the bony vertebral body. The second column includes the second half (ie: more posterior half) of the vertebral body, up to, and including the posterior longitudinal ligament (PLL). The third column includes the pedicles, spinal cord/thecal sac, lamina, transverse processes, facet joints, spinous process, and the posterior ligaments (i.e. supraspinous, interspinous, and ligamentum flavum).

The purpose of Denis' model was to delineate which injuries to the thoracolumbar spine were considered "unstable". This delineation was important because it determined which patient's required operative treatment of their spine.

Simply stated, an unstable spine was present if two or more of the columns were involved in the injury. However, it is important to note that every rule can be broken, so not all injuries to the thoracolumbar spine follow this rule. Part of the "art" of practicing spine surgery is

determining which two-column injuries can be left alone and managed non-operatively, and which truly require operative intervention.

Based on Denis' model he classified specific types of injuries. Injuries to the anterior column only were called "compression fractures". Damage to the anterior and middle columns were known as "burst fractures". Injury to the middle and posterior column were known as "flexion-distraction" injuries, or more colloquially as "seat-belt type" injuries. Finally, damage to all three columns were classified as "fracture-dislocation" injuries.

Classification of Thoracolumbar Injuries Using the Three Column Model		
Columns	**Name**	**Stability**
Anterior	Compression fracture	Stable
Anterior and Middle	Burst fracture	Unstable
Middle and Posterior	Flexion-distraction injury (aka: seat-belt type)	Unstable
Anterior, middle, and posterior	Fracture-dislocation	Unstable

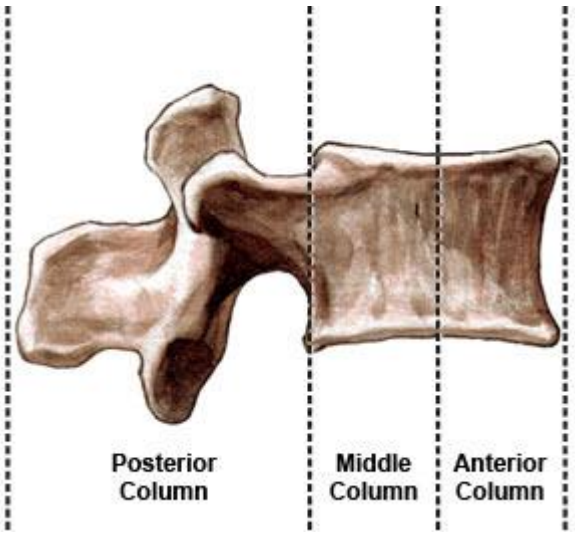


Figure 43 Denis' three column concept

This system divides the spine into three columns (when viewing the spine from the side):

- **Anterior Column:** This is the front part of the vertebra—the part that faces in towards your body. The anterior column is the front half of the vertebral body and intervertebral disc. There's a ligament on the front of the spine called the anterior longitudinal ligament; that's part of the anterior column as well.

- **Middle Column:** This is the key part of spinal stability. It's the back half of the vertebral body and intervertebral disc. There's a ligament on the back of the vertebral body called the posterior longitudinal ligament; that's part of the middle column as well. If there's a fracture in the middle column in addition to the anterior or posterior column, you're much more likely to have nerve damage and spinal instability. As long as the middle column stays intact, you have a better chance of having a stable fracture.
- **Posterior Column:** All the parts of the vertebra that are on the back side make up the posterior column. This includes the pedicles, lamina, facet joints, and spinous process (that's the bony part you can feel when you run your finger up your spine).

The three-column concept makes it easier to visualize spinal fractures. It also makes it easier to understand why some fractures are more stable than others. For example, if you have a fracture just in the anterior column, the spine should be able to still carry your weight well enough.

However, if you fracture the anterior *and* middle column, it's more likely that your spine will be unstable. Since the middle column connects the anterior and posterior columns, if that part is fractured, it will be harder for the spine to function well.

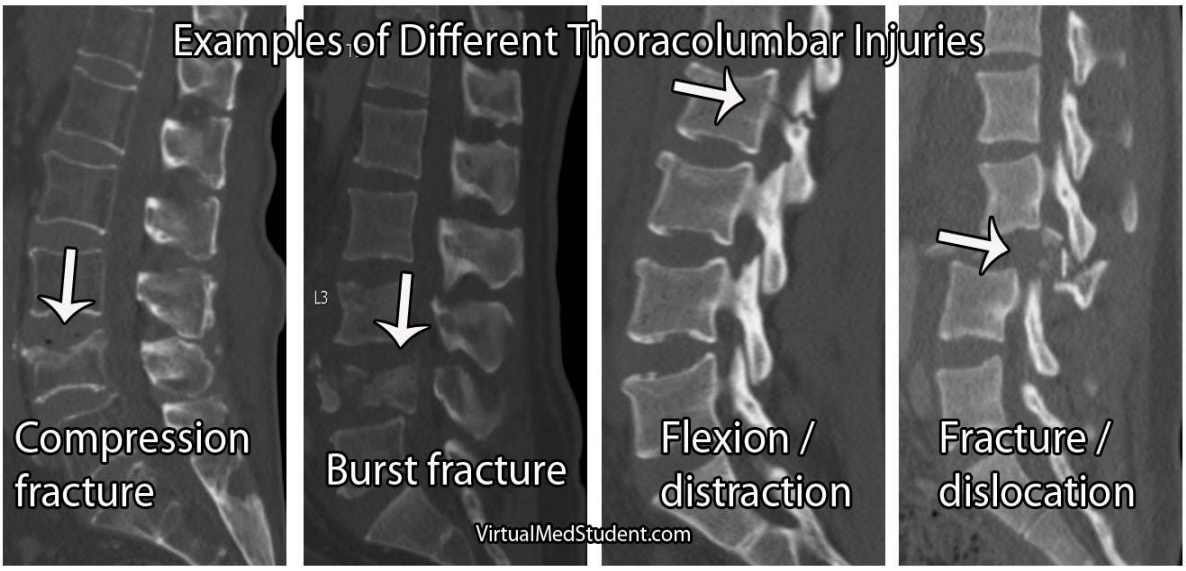


Figure 44 Examples of different types of fracture

In very simple terms, unstable fractures require definitive treatment. Treatment for unstable spine injuries is usually operative, although rigid immobilization with bracing is sometimes used in certain circumstances.

The treatment of each fracture type is beyond the scope of this article (and discussed in more detail elsewhere on this site), but suffice it to stay that for the most part (and again, every rule was made to be broken) two or three column injures = unstable = surgery.

Major and Minor Fractures

This is the simplest way to talk about fractures: major or minor.

A minor fracture means that a part of the posterior (back side) elements of the vertebra has broken—the parts that aren't as vital to spinal stability. The posterior elements include the spinous process and the facet joints (also called the articular processes). If you fracture this part of the vertebrae, it's usually not too serious.

A major fracture means that part of the vertebral body, the pedicles, or the lamina has fractured. Fracturing the vertebral body is considered major because it helps carry so much weight and distribute the force of your movements. If it's broken, you can have serious problems with the vertebrae lining up correctly. Fracturing the pedicles or lamina is dangerous because of the increased possibility of nerve damage. Additionally, the pedicles and lamina provide a lot of necessary support to keep your spine stable. If they fracture, your spine may be unstable.

Stable and Unstable Fractures
Stable and unstable is another basic classification for spinal fractures.

Stable fractures don't cause spinal deformity or neurologic (nerve) problems. With a stable fracture, the spine can still carry and distribute your weight pretty well (not as well as if there weren't a fracture, but it's still able to function with a stable fracture).

Unstable fractures make it difficult for the spine to carry and distribute weight. Unstable fractures have a chance of progressing and causing further damage. They may also cause spinal deformity.

Specific Fracture Types
Beyond whether a fracture is stable or unstable, major or minor, you should understand the different ways a vertebra can break. As your doctor describes your fracture, you may hear terms such as:

- **Compression Fracture:** This type of fracture is very common in patients with osteoporosis, or patients whose bones have been weakened by other diseases (such as bone cancer). The vertebra can absorb so much pressure; if there's a sudden force of a lot of pressure, the bone may not be able to handle the stress. The vertebra can fracture then.
    - A wedge fracture is a subtype of compression fracture. With a wedge fracture, part of the vertebra—usually the anterior (front) part—collapses under pressure and becomes wedge shaped.
- **Burst Fracture:** Burst fractures are caused by severe trauma (e.g., car accident). They happen when the vertebra is essentially crushed by extreme forces. Unlike compression fractures, it's not just one part of the vertebra that's fractured. In a burst fracture, the vertebra is fractured in multiple places. Because the vertebra is crushed completely, bony fragments can spread out and cause spinal cord injury. Burst fractures are more severe than compression fractures.
- **Flexion-distraction Fractures:** If you're in a car accident where your body is pushed forward, you may get a flexion-distraction fracture. Your spine is made to flex forward, but if there's a sudden forward movement that places incredible stress on

the spine, it may break a vertebra or vertebrae. Thinking of the three-column concept, a flexion-distraction fracture usually has fractures in the posterior and middle column.

- **Fracture-dislocation:** If you have any of the above fractures *and* the vertebra(e) moves significantly (dislocation), you have a fracture-dislocation. Usually, these fractures involve all three columns from the three-column concept, and they make your spine very unstable.

The term "wedge fracture" is used because the fracture usually occurs in the front of the vertebra, collapsing the bone in the front of the spine and leaving the back of the same bone unchanged. This process results in a wedge-shaped vertebra. A wedge compression fracture is generally a mechanically stable fracture pattern.

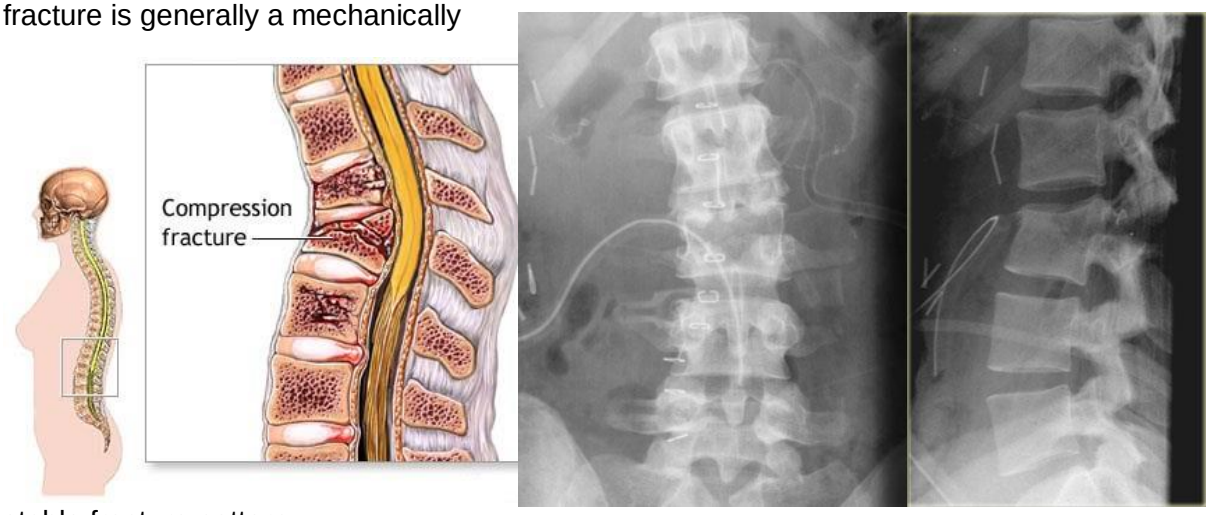


Figure 45 Wedge fracture of L1

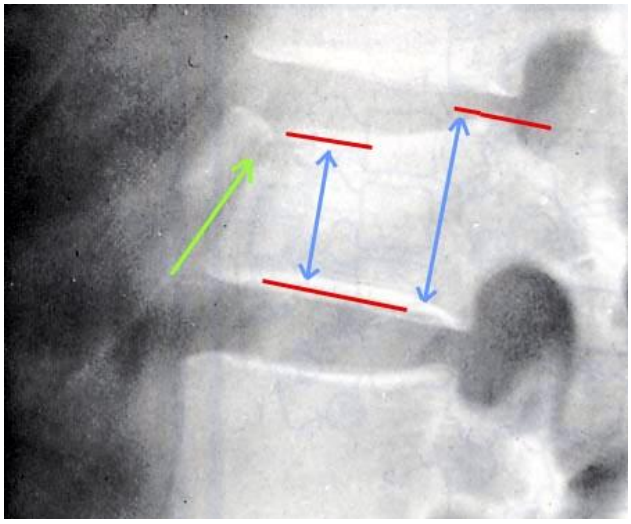


Figure 46 Wedge fracture parameters

A wedge fracture occurs when the anterior of the vertebral body is crushed in relation to the posterior, as seen here. It may result in a flexion deformity of the spine.

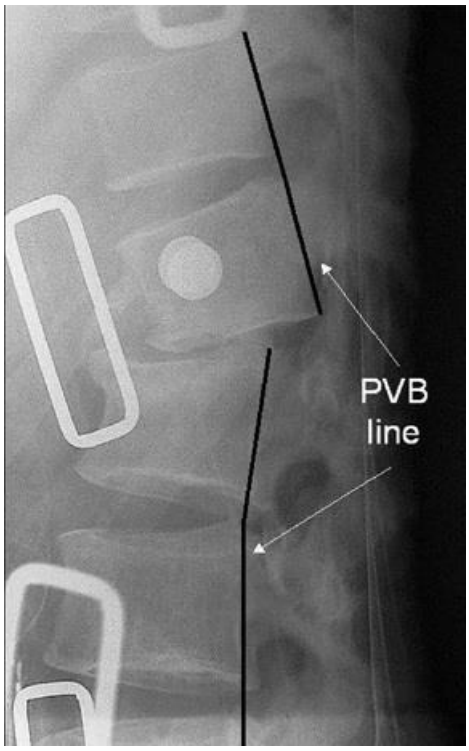


Figure 47 Wedge fracture parameter lines

If a wedge fracture causes a flexion deformity, it can be assessed by drawing lines along the posterior vertebral bodies (PVB). A line drawn along these should be continuous. A break suggests a translational deformity, as seen here in this lateral lumbar X-ray.

While wedge fractures are the most common type of compression fracture, there are other types as well, such as:

- **Crush fracture** - If the entire bone breaks, rather than just the front of the vertebra, it may be called a crush fracture.

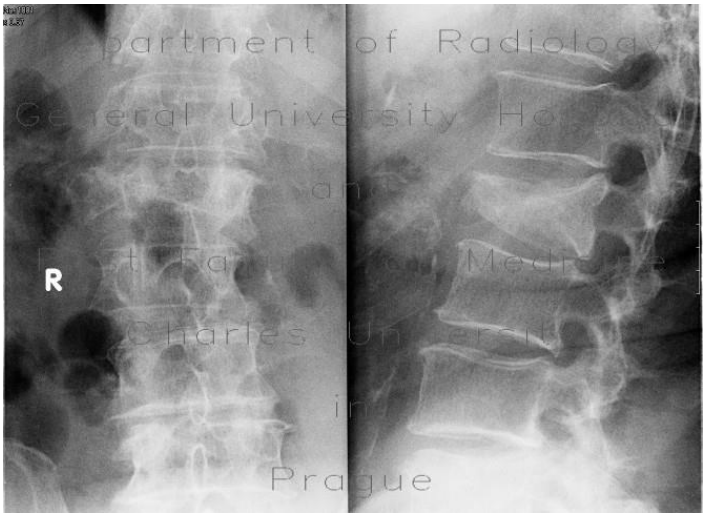


Figure 48 Crush fracture

Depending upon the severity, this may result in a reduction in the length of the spine.

- **Burst fracture** - This type of fracture involves some loss of the height in both the front and back walls of the vertebral body (rather than just the front of the vertebra). It may even cause detached fragments, as seen here in the anterior part of the vertebral body. Making this distinction is important because burst fractures can be unstable and result in progressive deformity or neurologic compromise.

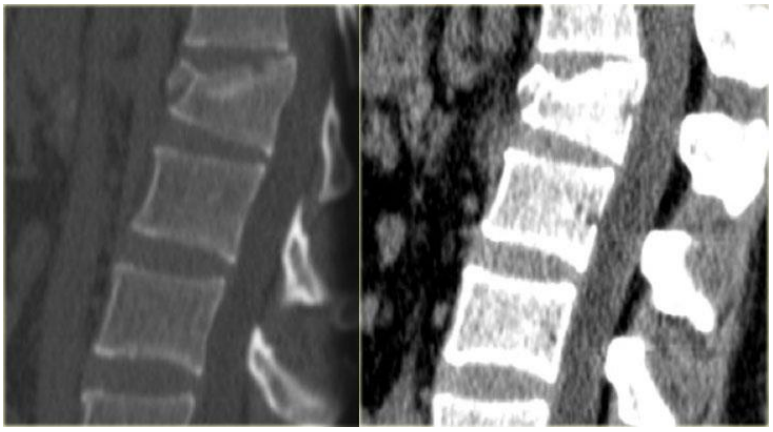


Figure 49 Burst Fracture

As these X-rays demonstrate, they are easily seen on lateral X-ray. They can also be seen on A/P X-ray as well

This X-ray is marked with arrows showing the positions of the pedicles in the lumbar spine. The crush fracture has occurred at the level of L3, with the pedicles being pushed outwards, bilaterally. This arrangement fails to follow the normal arrangement of the pedicles gradually widening from top to bottom.

Compression Fracture Symptoms

Vertebral fractures are usually followed by acute back pain, and may lead to chronic pain, deformity (thoracic kyphosis, commonly referred to as a dowager's hump), loss of height, crowding of internal organs, and loss of muscle and aerobic conditioning due to lack of activity and exercise. A combination of the above problems from vertebral fractures can also lead to changes in the individual's self-image, which in turn can adversely affect self-esteem and ability to carry on the activities of daily living.

Because the majority of damage is limited to the front of the vertebral column, the fracture is usually stable and rarely associated with any nerve or spinal cord damage.

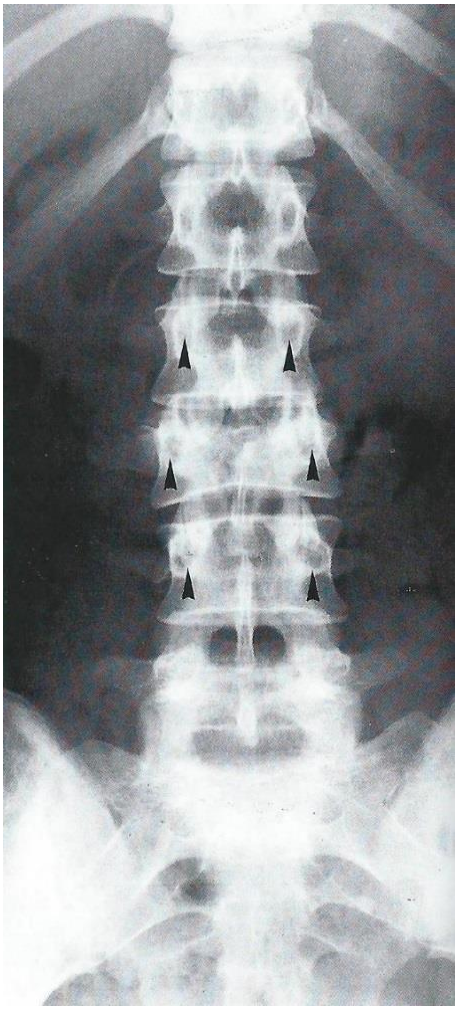


Figure 50 Crush fracture L3

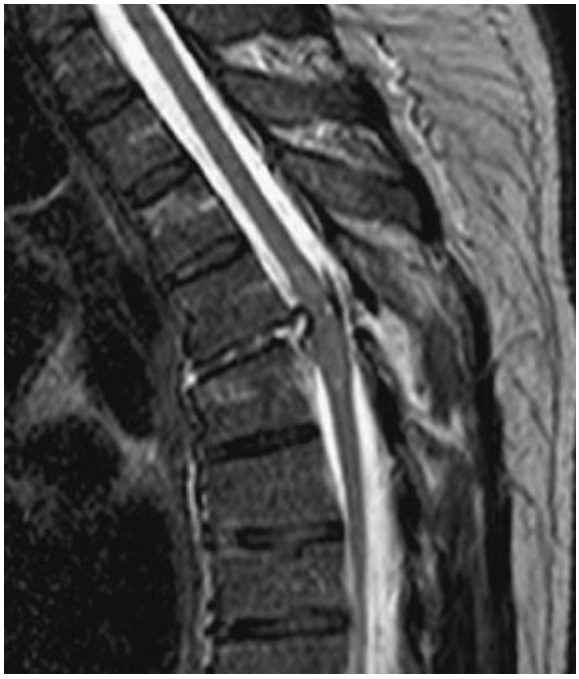


Figure 51 Crush fracture with spinal cord damage

This MRI scan shows a crush fracture. It also demonstrates a fusiform swelling of the spinal cord at that level, suggesting substantial swelling there. It is here that the MRI scan is very useful, as X-ray or CT examination would not show this.

Fracture-Dislocations

Fracture-dislocations of the thoracolumbar spine are highly unstable injuries. Although they may display various combinations of damage to both the anterior and posterior elements, the unique injury feature is translational deformity, which can occur in the sagittal and/or coronal planes.

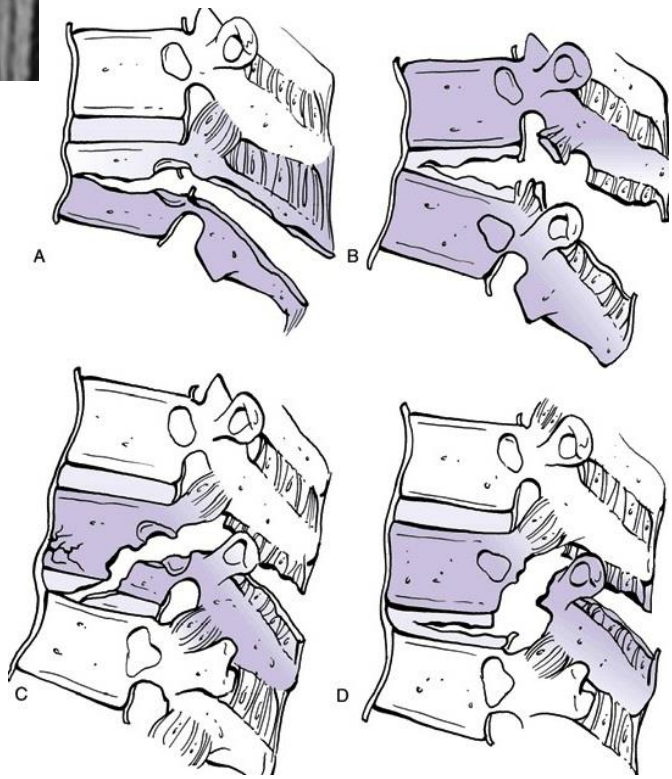


Figure 52 Flexion traction injury

Type A and B occur at one level.
Type C and D occur at two levels.

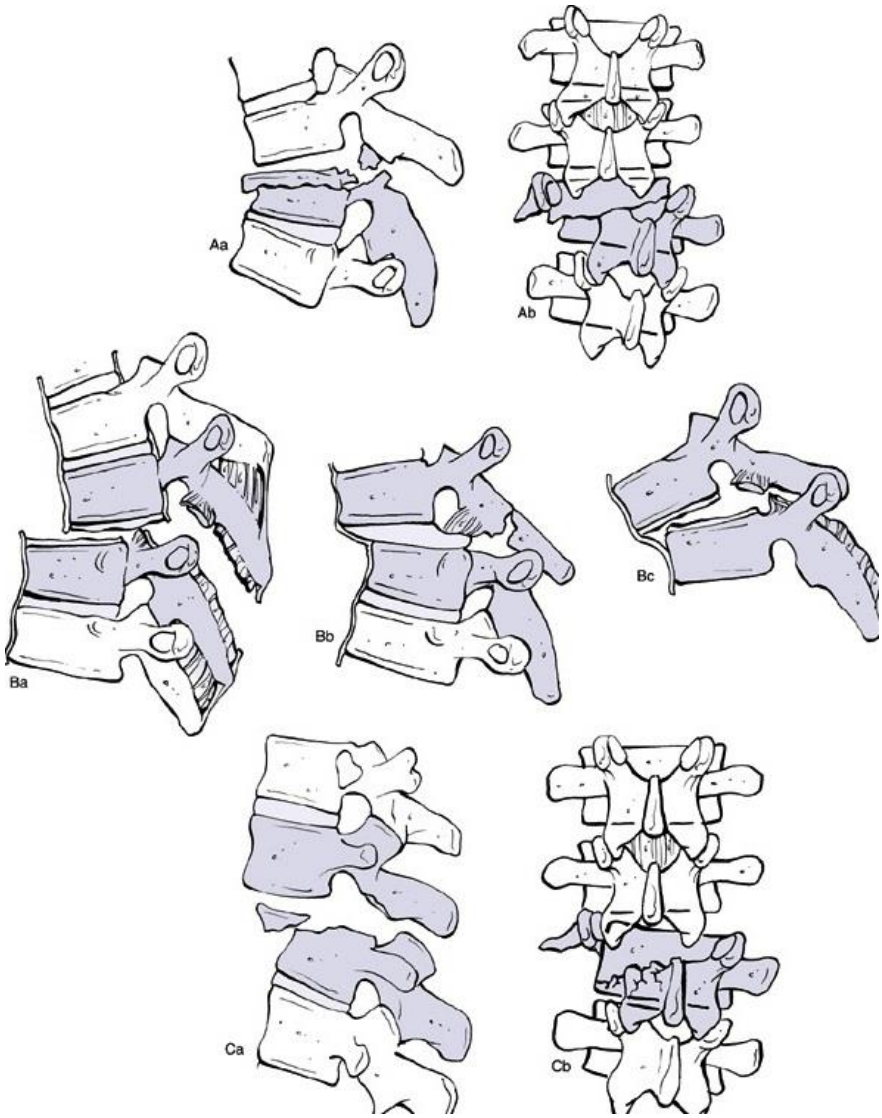


Figure 53 Traction dislocation

Type A are bony one-level injuries.

Type B are one-level ligamentous injuries.

Type C injuries are two-level injuries that occur through bone and/or ligament.

Any and all of these are likely to cause spinal cord damage.

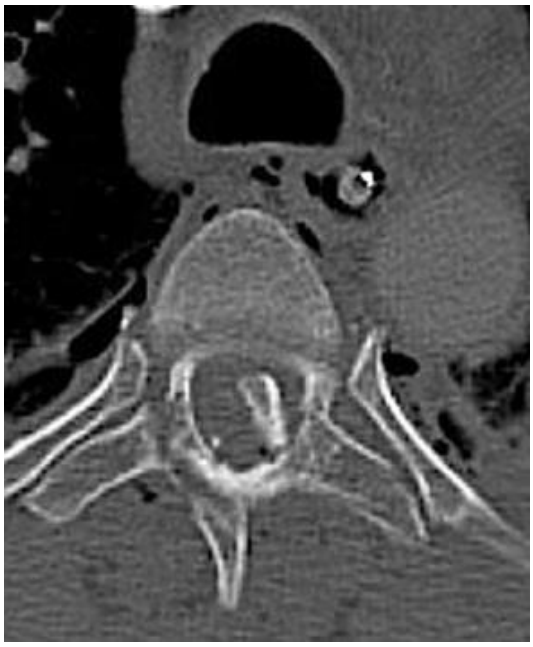


Figure 54 Fragment of bone in spinal canal

Canal compromise rarely occurs from displaced fractures of the posterior elements. In the axial CT scan here, a large piece of fractured lamina intruded on the spinal canal of this patient who had sustained a high-energy injury to the thoracic spine.

Treatment

A careful review of the data suggests that the nonsurgical management of stable thoracolumbar burst fractures (defined as fractures that have no PLC injury without neurologic deficit) can provide good results. Although many investigators have suggested the use of various x-ray measurement criteria for non-operative treatment such as less than 25 degrees to 30 degrees of kyphosis, less than 50% height loss, the absence of interspinous process widening, and less than 50% canal compromise, we believe that MRI evidence of discontinuity or continuity of the posterior ligamentous complex (PLC) is very important.

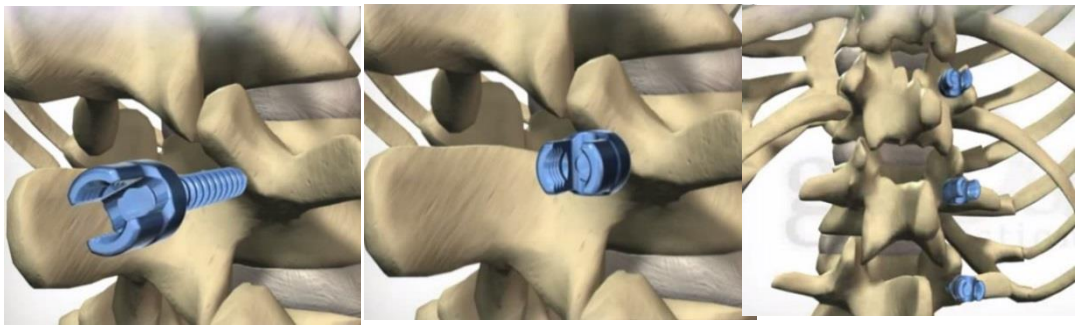
Provided the patient is neurologically intact and the PLC is intact, greater amounts of canal compromise or height loss can be accepted. Doctors prefer to keep the patient flat and on log-roll precautions until a thoracolumbar sacral orthosis (TLSO) [brace] is in place. The patient then undergoes a trial of standing x-rays before ambulation is permitted. X-ray and clinical follow-up examinations are scheduled at 2 weeks, 1 month, 2 months, and 3 months. At the 3-month follow-up, x-rays are made out of the brace to ensure stable alignment.

Surgical Treatment

Anterior and posterior approaches have been advocated for decompression and stabilization; both having advantages and disadvantages. Although there is uncertainty regarding the optimal surgical treatment of thoracolumbar burst fractures, so fixation is favoured in cases of neurological compression of the spinal cord and increasing kyphotic deformity. In addition, fractures in multiple injured patients or those in whom bracing cannot be effectively employed, due to other injuries or body habitus, may benefit from internal stabilization.

Posterior Surgery

Advocates of posterior surgery cite various advantages compared to anterior surgery for thoracolumbar burst fractures. First, it avoids the morbidity of anterior exposure in patients who potentially have concomitant pulmonary or abdominal injuries and shorter operative times and decreased blood loss, and functional outcomes are similar to those following anterior surgery.



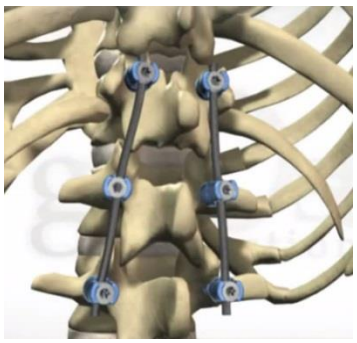


Figure 55 posterior spinal fixation

Here, special screws are inserted through the axis of the pedicles, into the bodies of the vertebrae, at and either side of the damaged vertebra. Then rods are attached and secured, fixing the vertebrae together. The role of posterior surgery for burst fractures is primarily for realignment and stabilization.

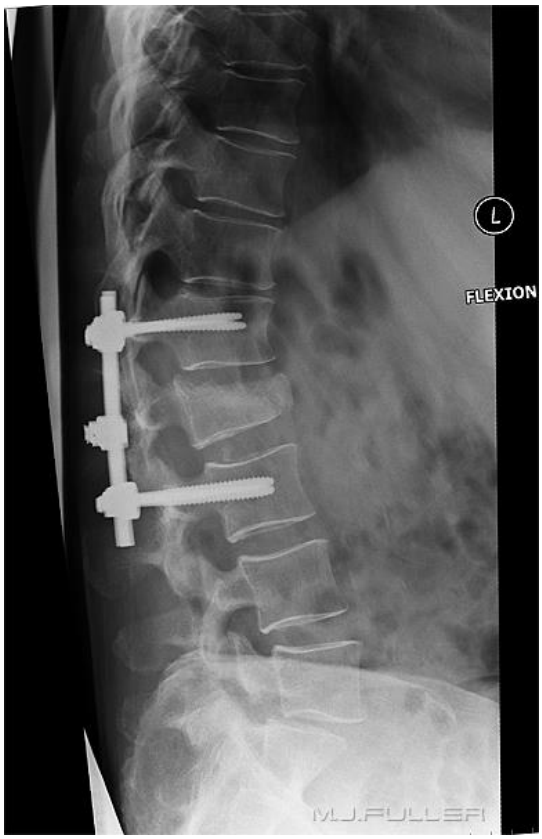


Figure 56 posterior spinal fixation X-ray

The X-ray here demonstrates such posterior fixation of the spine. Posterior fixation alone cannot reconstitute anterior column support, however, and is therefore somewhat weaker in compression than anterior fixation. This has led to a higher incidence of progressive kyphosis and instrumentation failure when treating highly comminuted fractures.

Anterior Surgery

Anterior surgery for thoracolumbar burst fractures is primarily indicated for decompression of the neural elements.

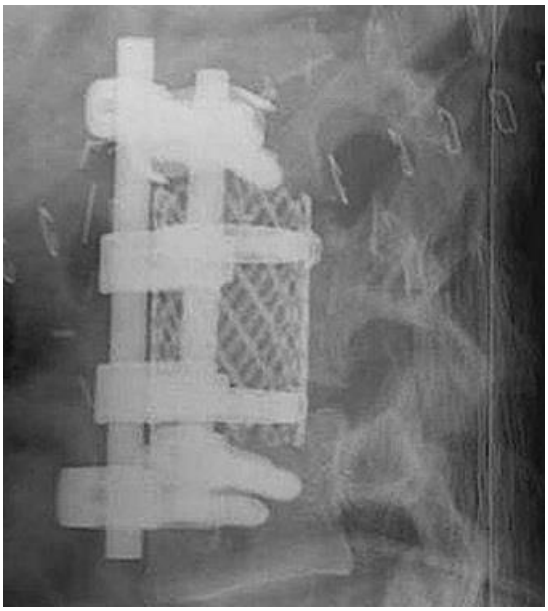
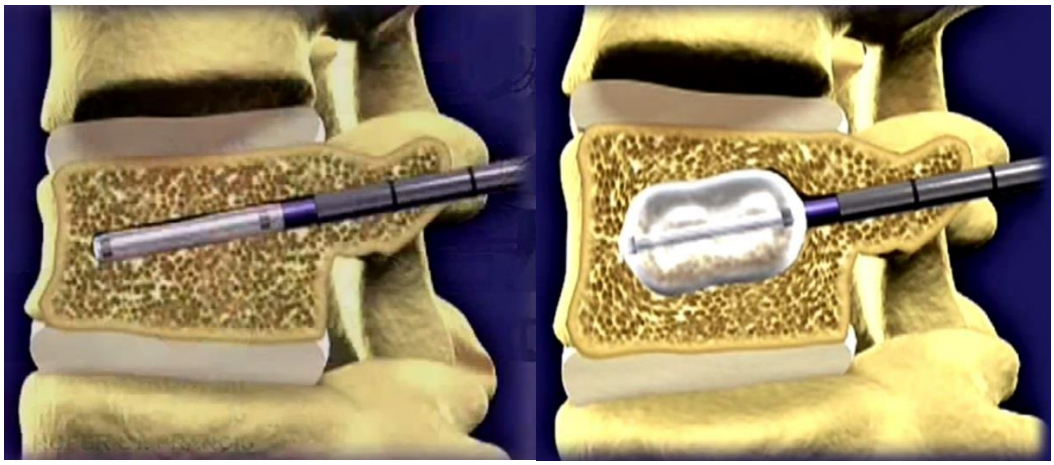


Figure 57 Anterior spinal fixation with Kaneda device

Note that a titanium mesh cage (filled with salvaged fractured bone and supplemented by allograft) was used to reconstruct the anterior column. If an anterior corporectomy (removal of vertebral body) is undertaken to decompress the spine, it must be understood that the spine is always destabilized. To remedy this situation the vertebral body defect must be replaced with a supportive strut that will also result in bony fusion. A structural, bony, autograft can be harvested from rib, fibula, or the iliac crest and inserted.

Another method of repairing crush fractures of the spine, if the alignment or length is compromised, or if there is neurological deficit. There is a technique for injection of bone cement into the body of the vertebra itself. The technique is called kyphoplasty.



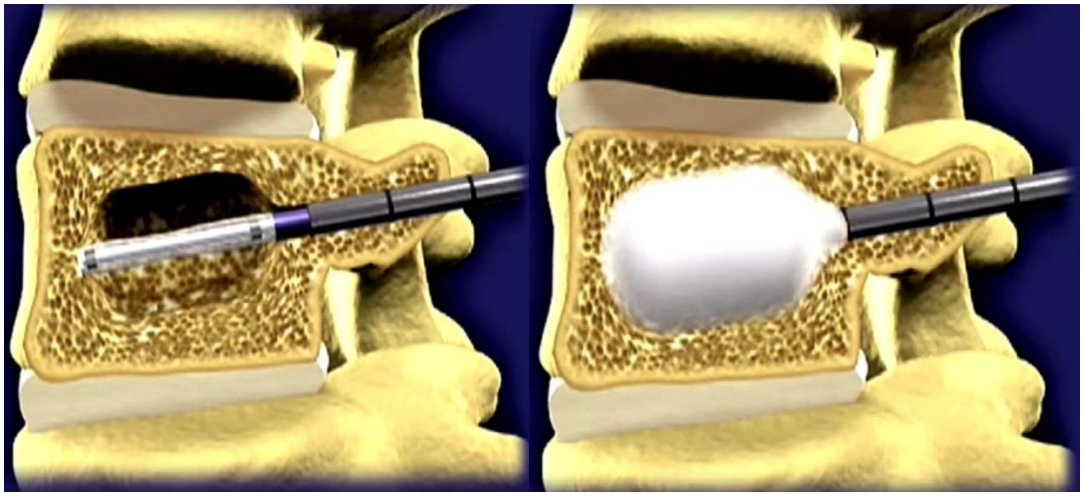


Figure 58 Kyphoplasty repair of crush fracture

The series of diagrams demonstrate that a hole is drilled into the vertebra, through the pedicle (the same route as the screw insertion of spinal fixation). Then a balloon is inserted and inflated (similar to angioplasty with the insertion of a stent), then the bone cement is injected. This technique helps maintain the structure and width of the vertebral body and help minimise any kyphosis resultant of the crush fracture.

Axial compression injuries

- **Jefferson fracture** is a burst fracture of the ring of C1 with lateral displacement of both articular masses.
- **Burst fracture at lower cervical level.**

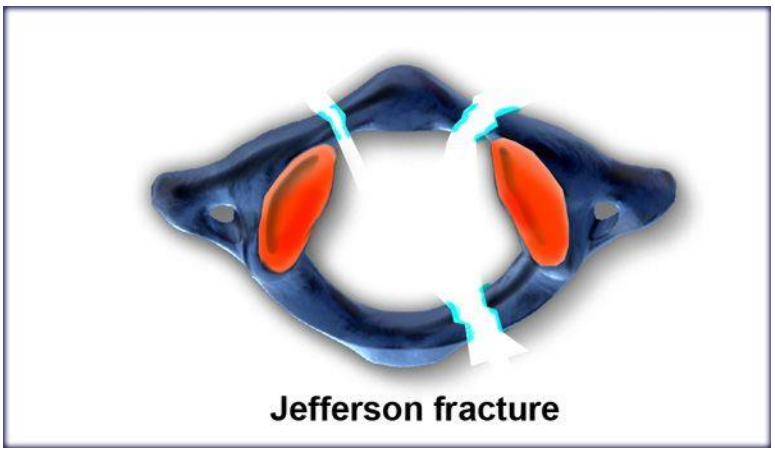


Figure 59 Jefferson fracture of C1

Stability

With bony damage and/or disruption of ligaments, stability at the level of injury may be compromised, possibly with consequent spinal cord injury

Unstable fractures:

- **Flexion**
  - Bilateral interfacetal dislocation
  - Flexion teardrop fracture
  - Wedge fracture with posterior ligamentous rupture
- **Extension**
  - Odontoid fracture type II
  - Hangman's fracture
  - Extension teardrop fracture
- **Vertical compression**
  - Burst fracture, e.g., Jefferson fracture.

Fracture of Odontoid Process (Dens)
Aetiology

The Odontoid Process, or Dens, is a short peg of bone projecting up from the anterior aspect of the Atlas, C1. It supposedly occupies the space where the body of C1 ‘should’ be.

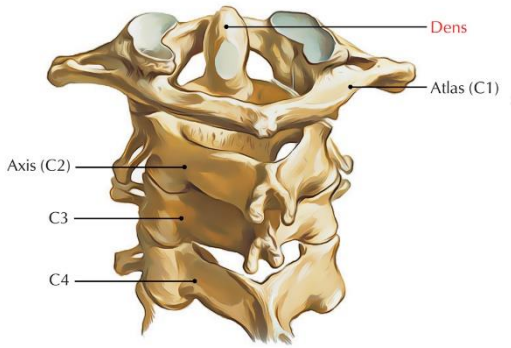
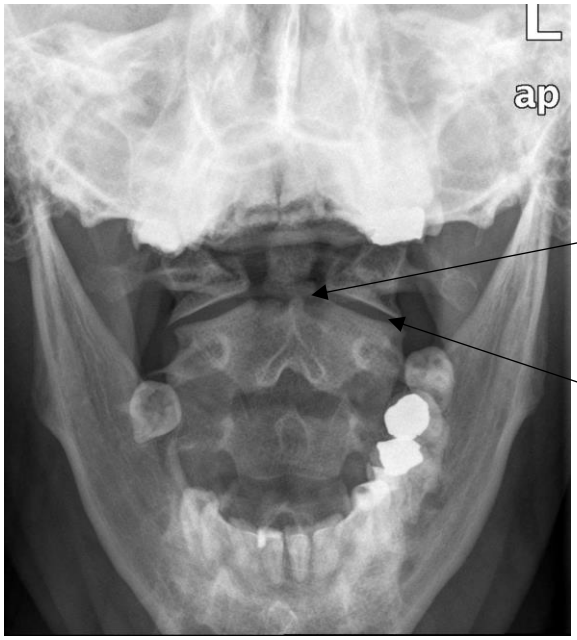


Figure 60 Odontoid process (dens)

Odontoid fractures occur as a result of trauma to the cervical spine. In younger patients, they are typically the result of high-energy trauma, which occurs as a result of a motor vehicle or diving accidents. In the elderly population, the trauma can occur after lower energy impacts such as falls from a standing position. The most common mechanism of injury is a hyperextension of the cervical spine, pushing the head and C1 vertebrae backward. If the energy mechanism and resulting force are high enough (or the patient's bone density is compromised secondary to osteopenia/osteoporosis), the odontoid will fracture with varying displacement and degrees of comminution.

Odontoid fractures account for 10% to 15% of all cervical spine fractures. Of the types of odontoid fractures, type II is the most common and accounts for over 50% of all odontoid fractures. Type III (see later) odontoid fractures make up most of the remaining percentage of odontoid fractures. Type I odontoid fractures are rare.



Fracture seen at base of odontoid process.

Joint between C1 and C2

Figure 61 Classic X-ray of Odontoid process fracture, taken through open mouth.

The odontoid fracture can also occur with hyperflexion of the cervical spine. The transverse ligament runs dorsal to (behind) the odontoid process and attaches to the lateral mass of C1 on either side. If the cervical spine is excessively flexed, then the transverse ligament can transmit the excessive anterior forces to the odontoid process and cause an odontoid fracture.

Pathophysiology

Type I Odontoid Fracture

A type I odontoid fracture occurs when the rostral tip of the odontoid process is avulsed (broken or torn off). This injury commonly occurs due to pulling forces from the apical ligament attachment to the odontoid process. The apical ligament attaches the tip of the odontoid process to the foramen magnum (skull base).

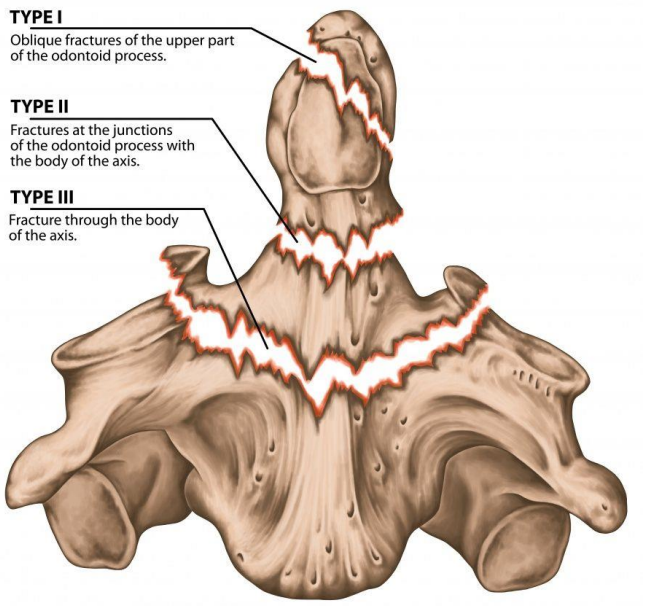


Figure 62 Types of odontoid fracture

Type II Odontoid Fracture

A type II odontoid fracture is a fracture through the base of the odontoid process. This injury occurs most typically when there is an excessive extension of the cervical spine, and the anterior arch of C1 pushes dorsally (backward) with sufficient force on the odontoid process (dens) to fracture the odontoid process at its base. Type II odontoid fractures can also occur

with hyperflexion of the neck and the transverse ligament, pushing the odontoid process forward to the point of fracture.

Type III Odontoid Fracture

A type III odontoid fracture is a fracture through the body of the C2 vertebrae and may involve a variable portion of the C1 and C2 facets. Type III odontoid fractures occur secondary to hyperextension or hyperflexion of the cervical spine in a similar manner to type II odontoid fractures. The difference is where the fracture line occurs.

History and Physical

Younger patients with an odontoid fracture typically have identifiable recent trauma (motor vehicle accident, sports-related impact, diving accident, fall from a height or downstairs). Older patients tend to have less resilient bones and can sustain an odontoid fracture after minor trauma, including fall from ground level or running into a door or cabinet. However, older individuals can also sustain an odontoid fracture from recent injuries similar to those of younger people.

On physical exam, patients may note cervical neck pain, which is worse with motion. The person may even may want to hold onto, and support, their head with their hands. They can also have dysphagia due to a retropharyngeal hematoma or associated parapharyngeal swelling. Less commonly, the patient may have myelopathic spinal cord injuries such as paraesthesias in the arms and/or legs, weakness of the arms and/or legs, or other neurologic dysfunctions. There are fewer spinal cord injuries in odontoid fractures due to the relatively large cross-sectional diameter of the spinal canal at the level of the odontoid process compared to the diameter of the spinal cord.

Evaluation

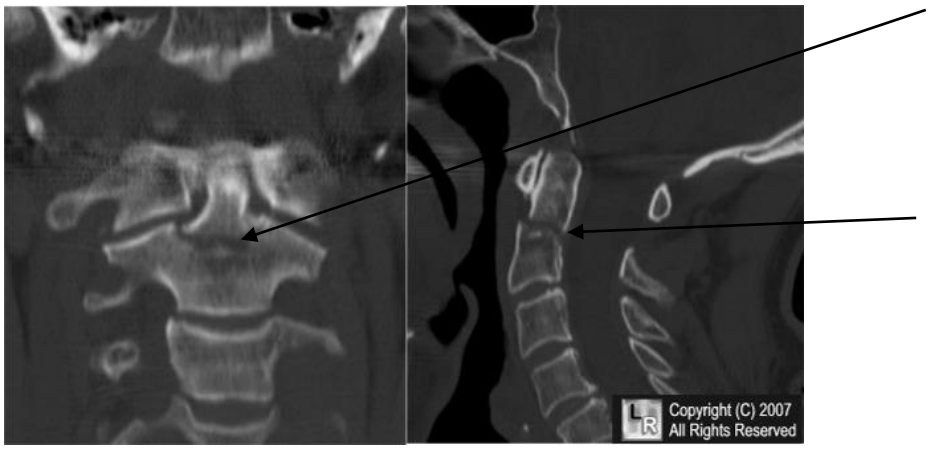
In hospitals and countries without readily available advanced imaging capabilities, radiographs are critical to evaluate and assist in ruling out potential odontoid fractures. Recommended views include:

- AP C-spine
- Lateral C-spine
- Open-mouth odontoid view

Although radiographs yield lower sensitivity and specificity rates when compared to computed tomogram (CT) scans, experienced clinicians and practitioners can still appreciate suspected injury without CT utilization. In addition, in the setting of suspected occipitocervical instability (useful in type I odontoid fractures or the setting of os odontoideum), flexion-extension radiographs should be obtained.

Advanced Imaging modalities

The imaging modality of choice is a CT of the cervical spine.



The CT provides the best resolution of the bony elements allowing for identification and characterization of an odontoid fracture. If there is neurologic injury (paraesthesia, weakness), then magnetic resonance imaging (MRI) without contrast of the cervical spine should be obtained to assess the cervical cord for injuries.

Some surgeons also order a CT angiogram of the cervical spine if posterior instrumentation is planned. The CT angiogram allows for better identification of the course of the vertebral arteries for surgical planning of posterior instrumentation.

Treatment / Management

The treatment of an odontoid fracture depends on the type of fracture and age of the patient.

Most consider a type I odontoid fracture a stable fracture and treat for six to 12 weeks in a rigid cervical orthosis (hard cervical collar). Some have suggested that rarely a type I odontoid fracture may be unstable secondary to more extensive and unrecognized ligamentous injury, and flexion/extension x-rays should be obtained at the time of removal of the cervical collar after six to 12 weeks to ensure cervical stability.

Type II Odontoid Fracture

Type II odontoid fractures are inherently unstable and have a lower union rate than type III odontoid fractures due to the lower surface area of fractured bone in type II versus type III odontoid fractures. The configuration of type II odontoid fracture and age of patient also play important roles in treatment decisions. The current treatment options for a type II odontoid fracture include rigid cervical orthosis, halo vest immobilization, odontoid screw, transoral odontoidectomy, and posterior instrumentation.

Rigid Cervical Orthosis

A type II odontoid fracture is inherently unstable, and a rigid cervical orthosis is not the ideal treatment for such an injury. In the elderly population, many are not surgical

candidates (due to comorbidities or poor bone quality), and the elderly typically poorly tolerate a halo vest immobilization. In such situations, a practitioner may attempt a rigid cervical orthosis, although union rates are low. Some authors have argued that a fibrous union will form with the use of a rigid cervical orthosis with time, and this may provide sufficient stability in the elderly population while avoiding the morbidity of surgery or halo vest immobilization. Note that many elderly patients also poorly tolerate rigid cervical orthosis because of pressure ulcers from the cervical collar and difficulties in eating. In some situations, the patient and/or family may elect to forego all treatment while understanding the unstable nature of the spine and risk of progressive deformity and/or cervical cord injury.

Halo Vest Immobilization

If a patient is relatively young and healthy, and there is low risk for non-union, then halo vest immobilization may be the best treatment for a type II odontoid fracture. Risk factors for non-union include a fractured space greater than a few millimetres between the odontoid process and vertebral body, poor alignment of the odontoid process with respect to the vertebral body, and poor bone quality and/or health status of the patient. Elderly patients poorly tolerate halo vest immobilization and have increased the risk of death with halo-vest immobilization. Younger patients typically spend six to 12 weeks in halo vest immobilization with frequent x-rays to check alignment and healing.

Odontoid Screw

An anterior odontoid osteosynthesis (odontoid screw) is a screw placed from the inferior anterior aspect of the C2 vertebral body, in a superior trajectory, and capturing the odontoid process and affixing it in place to allow bony fusion to occur.

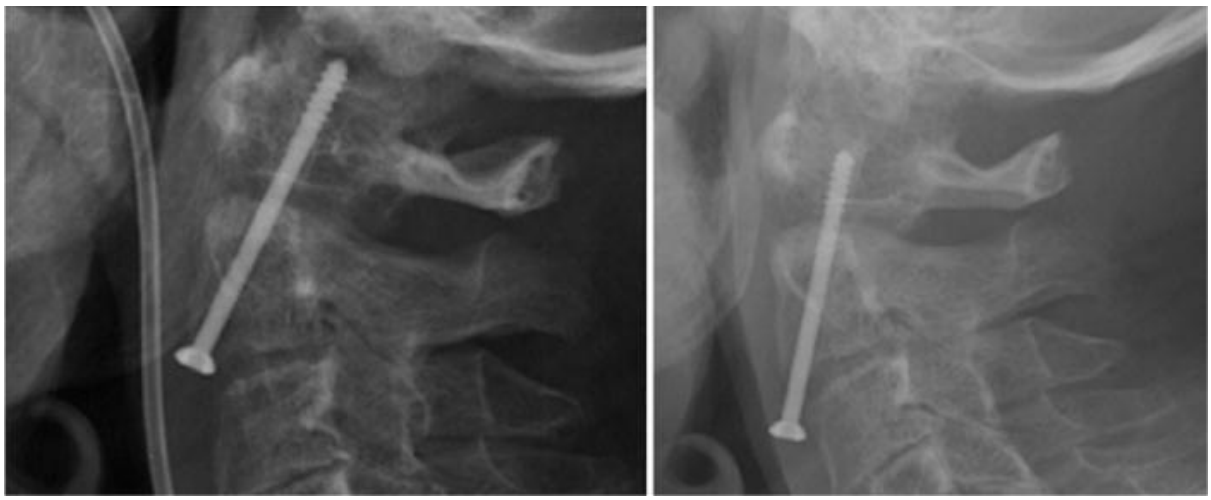


Figure 63 Odonoid screw

The odontoid screw has an advantage of relative preservation of motion of the upper cervical spine while treating a type II odontoid fracture. A surgeon can only place the odontoid screw if there are acceptable alignment and minimal displacement of the odontoid process, the fracture line is oblique or perpendicular to the screw trajectory, the injury is relatively recent, and the patient has acceptable body habitus to place

the odontoid screw. Due to the relatively vertical orientation of an odontoid screw, a person with a short neck or large chest or sternum may not allow the surgeon an adequate trajectory for placement of the odontoid screw. Odontoid screws have a lower union rate and a higher failure rate than posterior instrumentation.

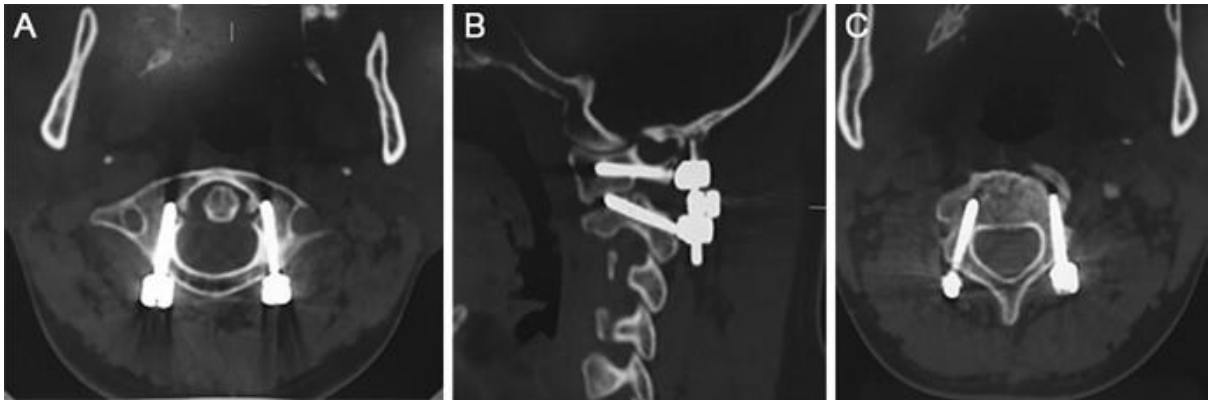


Figure 64 Posterior fixation of odontoid fracture

Transoral Odontoidectomy

In some situations, the odontoid process (dens) may be severely posteriorly displaced and compressing the spinal cord causing neurologic deficits. It is difficult and dangerous to reduce the odontoid process in a closed manner, so surgical removal of the odontoid process is required to relieve the compression of the spinal cord. This relief is commonly achieved through a transoral odontoidectomy, as the odontoid process commonly is located posterior to the oropharynx. If the odontoid process is removed, the cervical spine remains unstable, and the patient requires instrumented fusion, commonly from a posterior or combined anterior-posterior approach.

Posterior Instrumentation

If the patient has certain risk factors for non-union, then posterior instrumentation may provide the best treatment option for a type II odontoid fracture. The risk factors include:

- More than a few millimetres gap between the odontoid process and vertebral body
- Poor odontoid process alignment
- Poor bone quality, older fractures
- Older patients
- Failure of other treatment modalities
- Smoking

Posterior instrumented fusion techniques vary widely and include fusion limited to C1 and C2 as well as more extensive fusions. Fusion of only C1 and C2 will lead to approximately 50% reduction of the lateral rotation of the cervical spine, as it is at C1/2 where most of cervical rotation occurs. Advantages of posterior fusion include a higher rate of union and stabilization than other treatment options, less risk of swallowing or vocal cord issues than an anterior approach, and avoidance of rigid

cervical orthosis or halo immobilization. The major disadvantages of posterior fusion include loss of cervical spine motion and risk of damage to the vertebral arteries, exiting nerve roots or spinal cord.

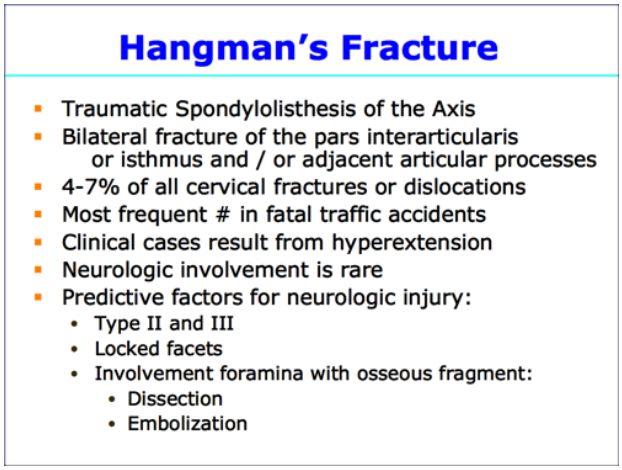
Just for completeness, here is a differential diagnosis list:

- Anterior cervical wedge fracture
- Atlanto-occipital dissociation
- Cervical burst fracture
- Cervical facet dislocation
- Cervical spinous process fracture
- Extension cervical teardrop fracture
- Flexion cervical teardrop fracture
- Hangman's fracture
- Isolated transverse process fractures
- Jefferson fracture

Hangman’s fracture

The Hangman’s fracture is the most common cervical spine fracture. Classically it is an extension-fracture as the hangman puts the knot under the chin to produce maximal extension-force. That is why the hangman’s fracture is discussed in hyperextension injuries. In some situations however it can also be the result of extreme flexion.

The hangman's fracture is common in diving accidents. Although considered an unstable fracture, it seldom is associated with spinal injury, since the anteroposterior diameter of the spinal canal is greatest at this level, and the fractured pedicles allow decompression. When associated with unilateral or bilateral facet dislocation at the level of C2, this type of hangman's fracture is unstable and has a high rate of neurologic complications.



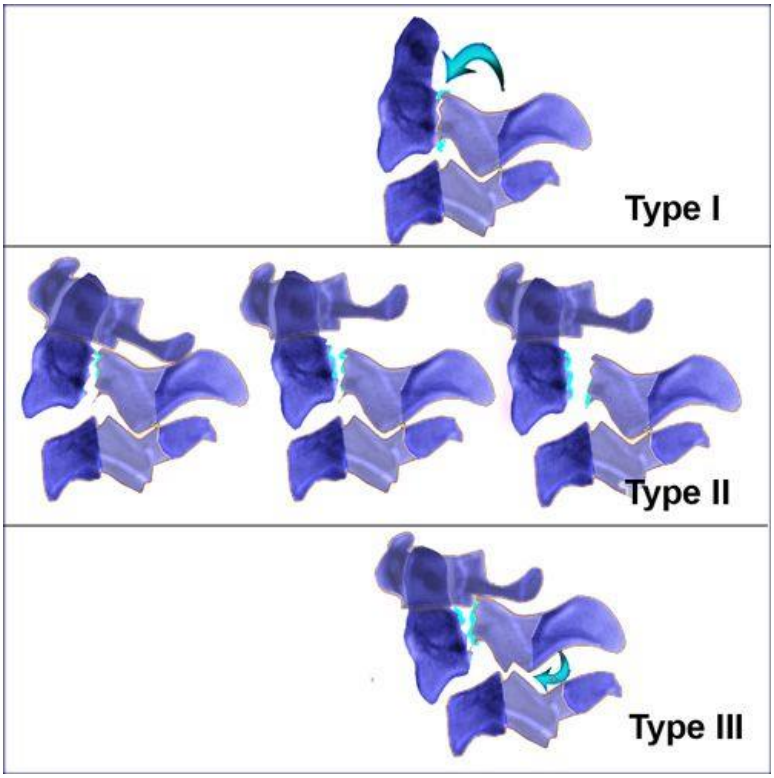


Figure 65 Hangman's fractures

Classification of Hangman's fractures

- **Type I** (65%)
  - hair-line fracture
  - C2-3 disc normal
- **Type II** (28%)
  - displaced C2
  - disrupted C2-3 disc
  - ligamentous rupture with instability
  - C3 anterosuperior compression fracture
- **Type III** (7%)
  - displaced C2
  - C2-3 Bilateral interfacet dislocation
  - Severe instability

Below are images of a restrained passenger in a vehicle going about 55 miles per hour. She ran into a tree at about 9 p.m. the previous night with questionable loss of consciousness. She had cervical tenderness to palpation but was alert and had no neurologic abnormalities on examination.

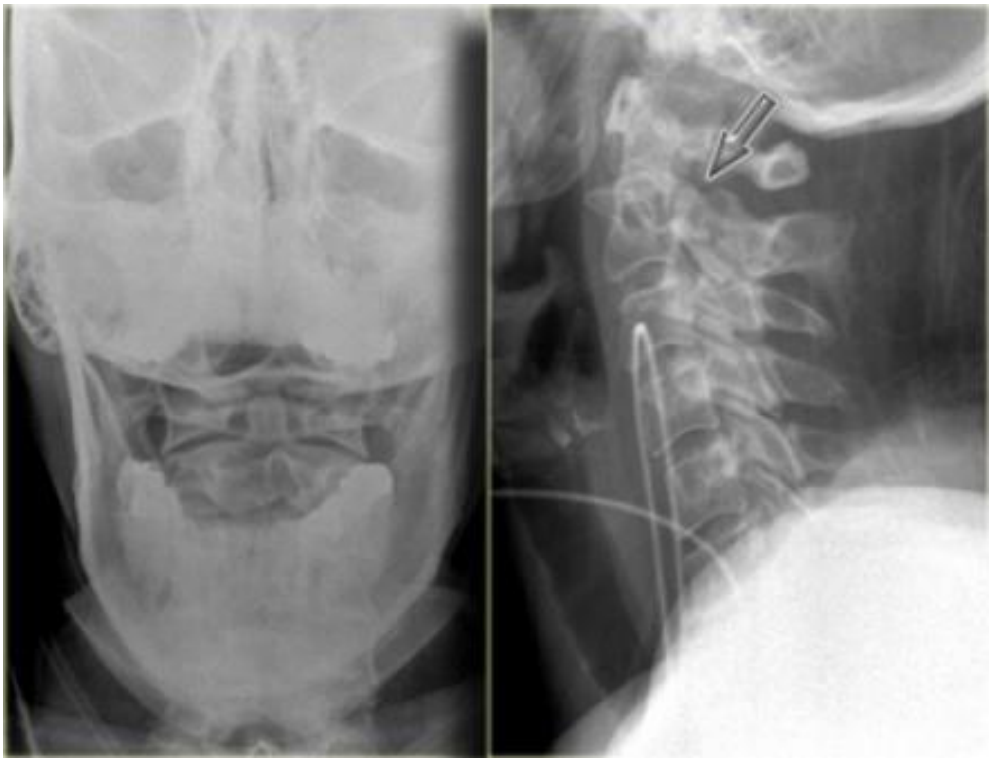


Figure 66 Hangman's fracture following road accident

The findings are:

- Subtle lucent line at the back of the corpus of C2 as seen on the lateral view (arrow).
- Subtle discontinuity of the arch of C2

The CT-images confirm the fracture-lines of the hangman's fracture. They run through the pars interarticularis resulting in a traumatic spondylolysis. In this case there was no neurologic deficit, because the spinal canal is widened at the level of the fracture.

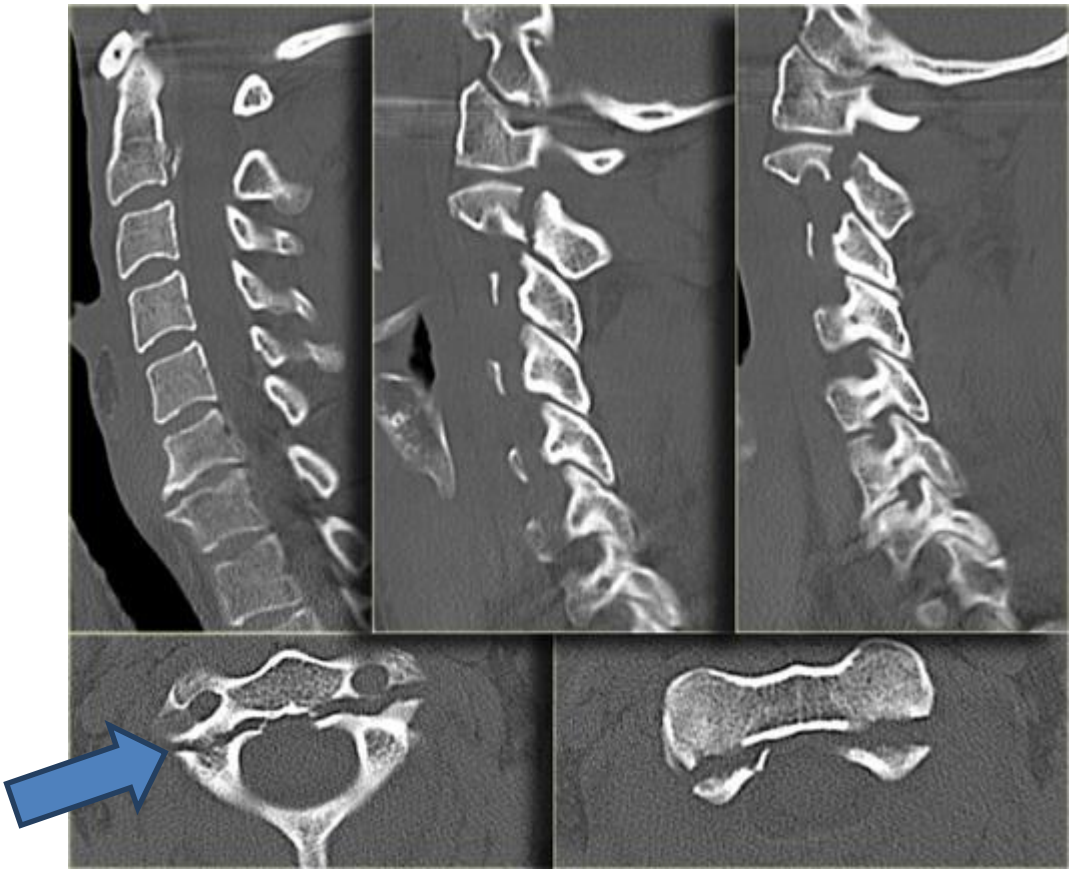


Figure 67 CT scan of hangman's fracture

Figure 68 Hyperextension showing open book lesion

The images below are of a 90-year-old male who tripped and fell on his back and the back of his head.
He had immediate quadriparesis after the event with no loss of consciousness

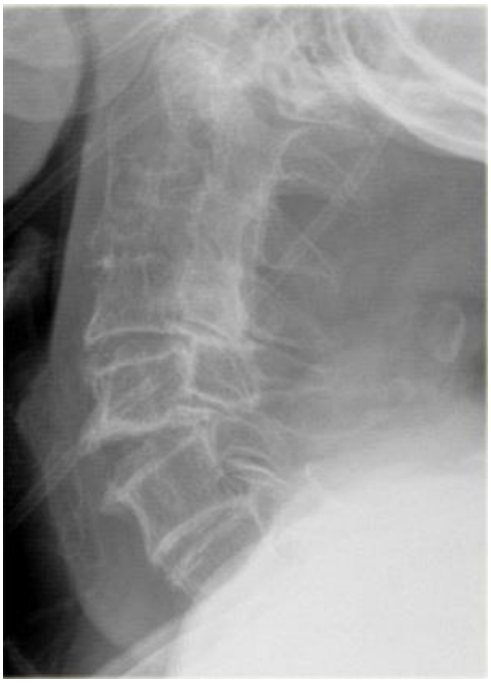
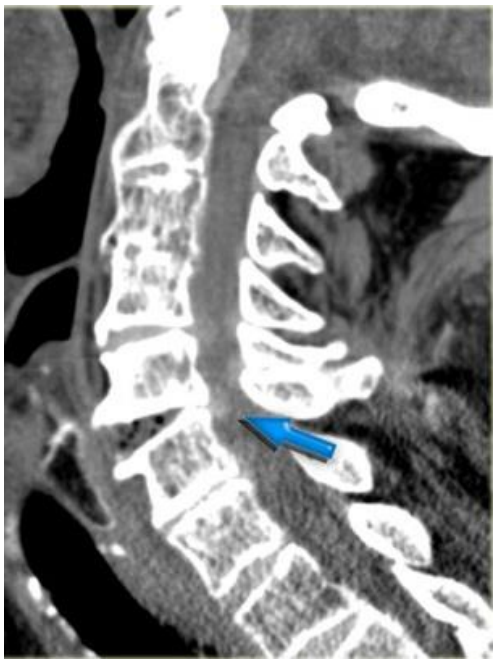


Figure 69 Cervical open book lesion

The (above) X-ray findings are:

- Widening of the disc space C5/6 in the front and narrowing in the back.
- This is also called 'an open book'.
- It tells us that there was a hyperextension injury



The CT scan (left) shows a hyperextension injury. The small black dots in the disc space are the result of a vacuum phenomenon. The negative pressure resulted in a vacuum phenomenon in the injured disc space.

There is also some hyperdensity at the back of C5/6, which could be a herniated disc or just pre-existing disc degeneration.

In such a patient with spondylosis which has led to narrowing of the canal, a low velocity injury can lead to spinal cord injury.

Figure 70 Cervical open book CT scan

The MRI, below, shows a subtle increase in signal intensity of the spinal cord. Most of the time these patients get a central cord injury. There is only injury to the central part of the cord and these patients have disproportioned weakness of their arms and normal strength in their legs. These injuries can be devastating, although it is uncommon that they are haemorrhagic.

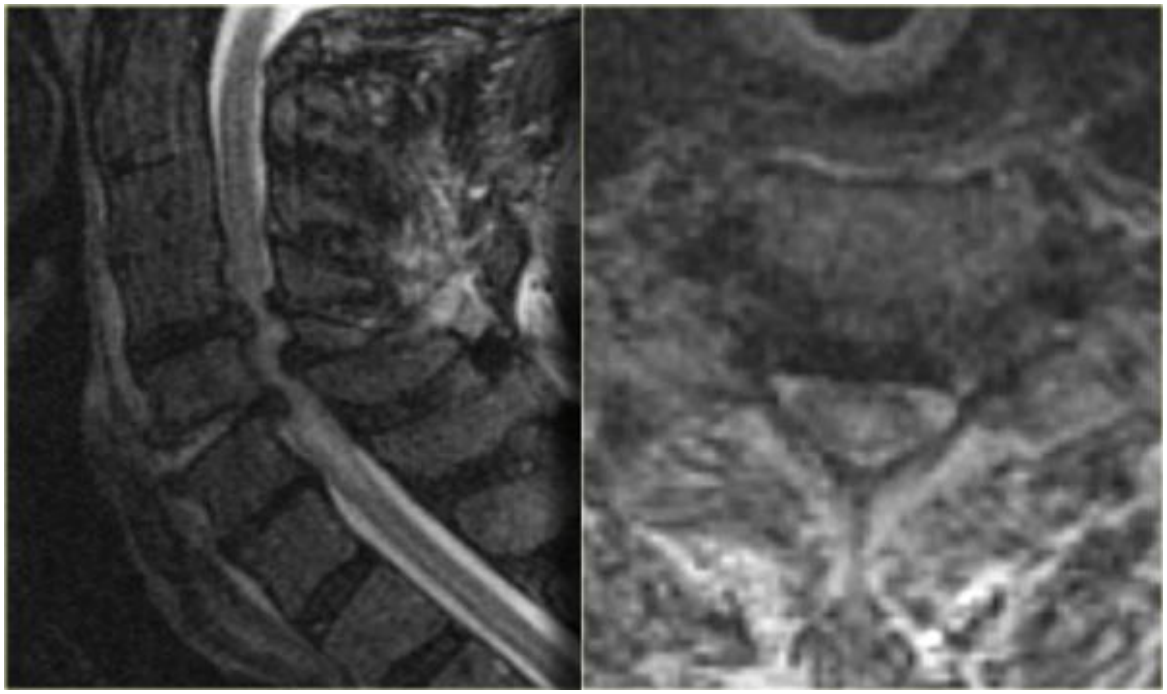


Figure 71 Cervical open book MRI

Extension teardrop fracture

As with flexion teardrop fracture, extension teardrop fracture also manifests with a displaced anteroinferior bony fragment. This fracture occurs when the anterior longitudinal ligament by creating an avulsion fracture by pulling a bony fragment away from the inferior aspect of the vertebra because of the sudden hyperextension. The fragment is a true avulsion, in contrast to the flexion teardrop fracture in which the fragment is produced by compression.

This type of fracture is commonly seen in diving accidents and tends to occur at lower cervical levels. It also may be associated with the central cord syndrome due to buckling of the ligamenta flava into spinal canal during the hyperextension phase of injury. This injury is stable in flexion but highly unstable in extension.

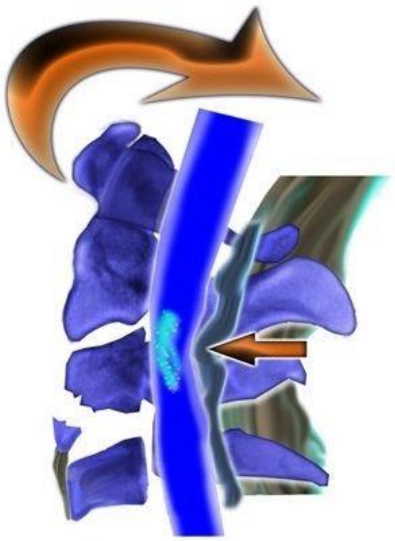


Figure 72 Extension teardrop fracture

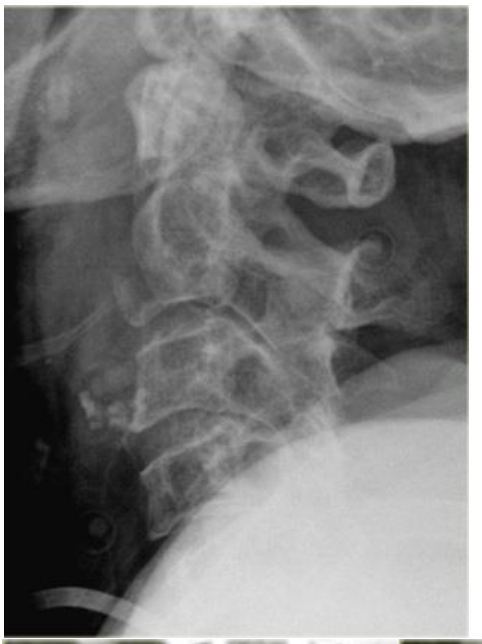


Figure 73 Extension teardrop fracture X-ray

On the left images of a 70-year-old female who fell down ten steps striking her head resulting in a subgaleal hematoma with possible loss of consciousness. There was no neurologic deficit. Notice the anteroinferior bony fragment of C2.

The MRI scan, below, also confirms that this is not a flexion injury, since the soft tissue injury is located anteriorly.

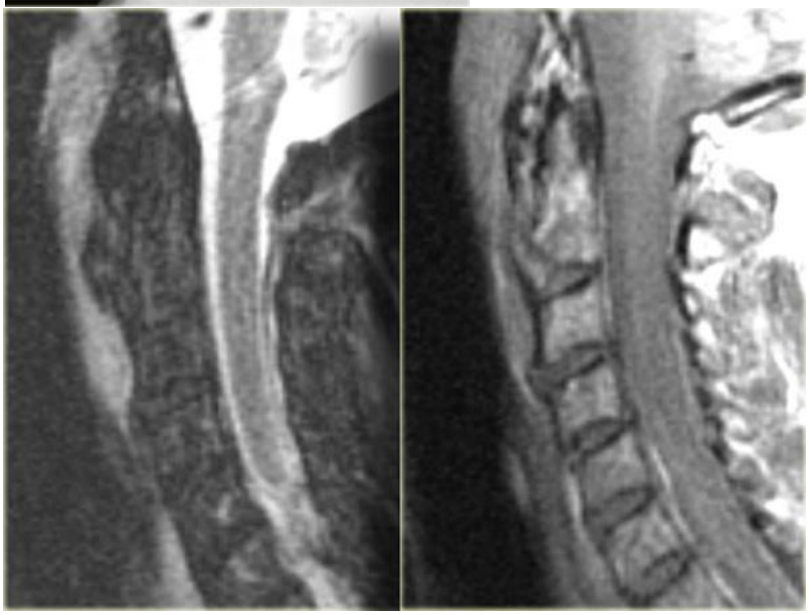


Figure 74 Extension teardrop MRI

Acute Cervical Injuries

Harris and Mirvis classification

Hyperflexion

- Hyperflexion sprain (anterior subluxation)
- Bilateral interfacetal dislocation (BID)
- Simple wedge (compression) fracture
- Clay Shoveler's (coal shoveler's) fracture
- Flexion teardrop fracture
- Unilateral interfacetal dislocation (UID, with rotation)

Hyperextension

- Hyperextension dislocation
- Avulsion fracture of the anterior arch of the atlas
- Fracture of the posterior arch of atlas
- Extension teardrop fracture
- Laminar fracture
- Hangman's fracture
- Hyperextension fracture-dislocation
- Pillar fracture (with rotation)
- Pedicolaminar fracture-separation (with rotation)

Vertical compression

- Jefferson burst fracture, C1
- Burst fracture, lower cervical spine

Lateral flexion

- Unilateral occipital condylar fracture
- Unilateral fracture, lateral mass, C1
- Uncinate process fracture
- Transverse process fracture

Others

- Occipitoatlantal dissociation
- Odontoid fractures
- Torticollis
- Atlantoaxial rotary dissociation

Figure 75 Acute cervical spine injury resume

Spinal cord injuries

There are two types of injury to the spinal cord:

- **Non-haemorrhagic** with only high signal on MRI due to oedema.
- **Haemorrhagic** with areas of low signal intensity within the area of oedema.

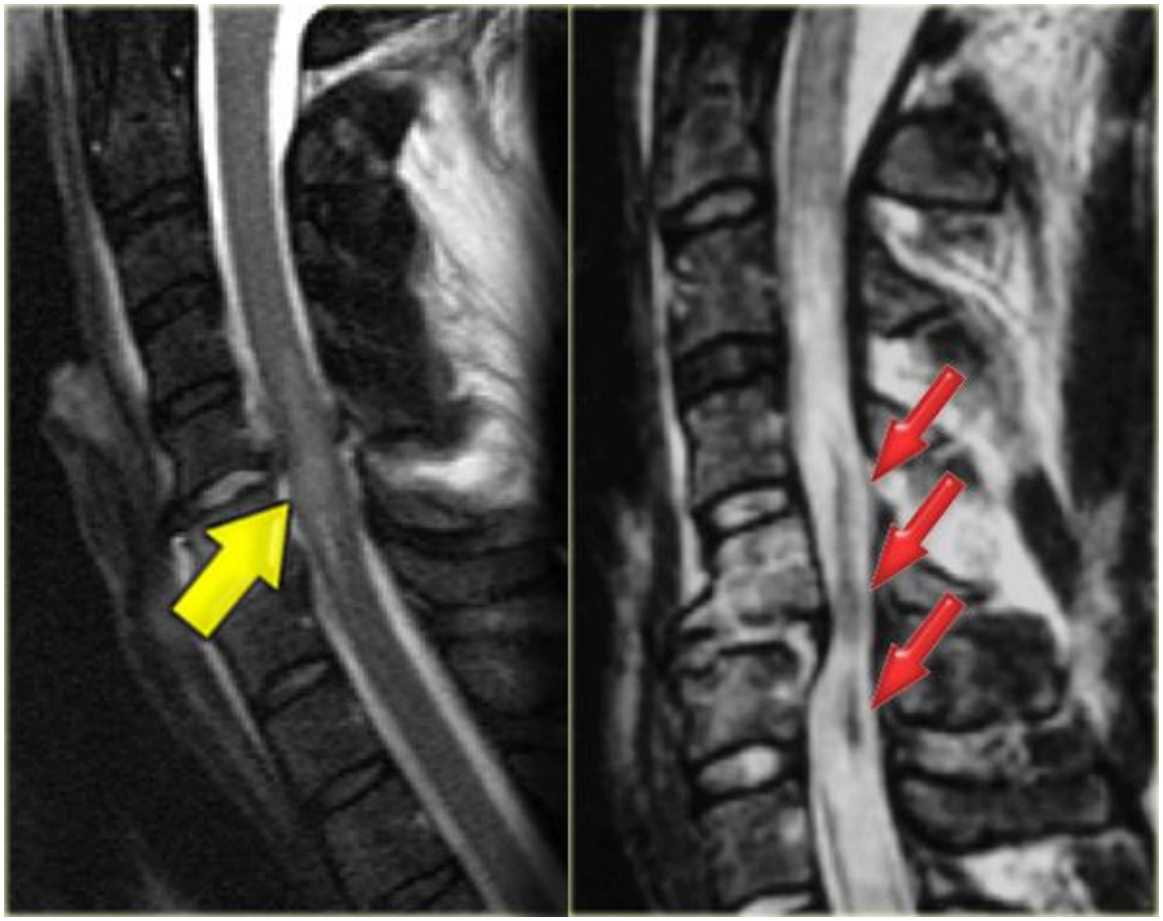
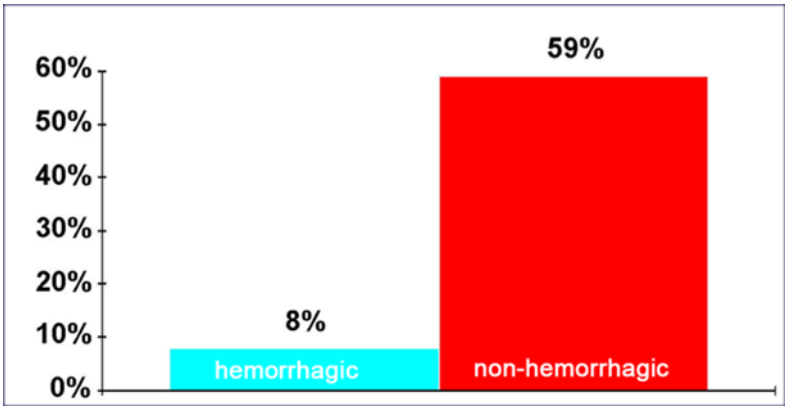


Figure 76 Cervical spinal cord injuries

Here, there is a strong correlation between the duration of the spinal cord oedema and the clinical outcome. The most important factor however is whether there is haemorrhage, since haemorrhagic spinal cord injury has an extremely poor outcome.
The chart below is showing the motor recovery rate for patients with oedema alone (in blue) versus oedema plus cord haemorrhage (in red). The motor recovery rate is for the legs only.

Figure 77 Incidence of haemorrhagic spinal cord injury



Hyperflexion Sprain

Hyperflexion sprain injuries are injuries to the soft tissues of the spine without fracture. X-rays of this can only suspect damage when there is angulation or translation, but MRI scans will demonstrate subtle injuries to the soft tissues.

On the left images of a patient who has been in a car accident and complained of neck pain. The x-rays were normal and there were no neurological symptoms.

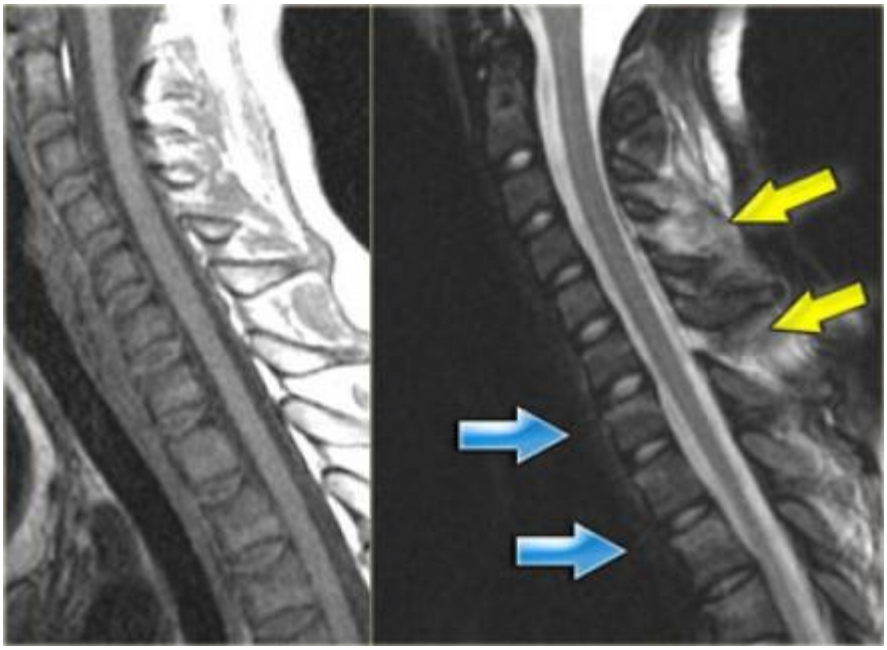


Figure 78 Cervical hyperflexion MRI

The findings are:

- Oedema in the posterior soft tissues indicating a hyperflexion injury.
- Oedema in the vertebrae of the lower C-spine and upper T-spine indicating bone bruise as a result of axial loading.

In this patient we can conclude that there was mild hyperflexion strain and we do not know if a special treatment is required, since these were isolated MR-findings without evidence of fracture or abnormal positioning. However, there is controversy regarding the meaning of soft tissue abnormalities detected *only* on MRI.

- Signal changes do not necessarily equate with structural failure.
- These findings still require better validation.
- In trauma centres up to 25% of all patients with neck injury have signal abnormalities on MRI and the significance is indeterminate.

Below are the images of a 44-year-old female, who sustained a fall on the ice. She subsequently had a second fall the following morning, where after she had complete loss of motor and sensation. On physical examination there was lower extremity paraparesis with some upper extremity weakness on the right. Central cord injury was proposed initially. The radiographs were normal.

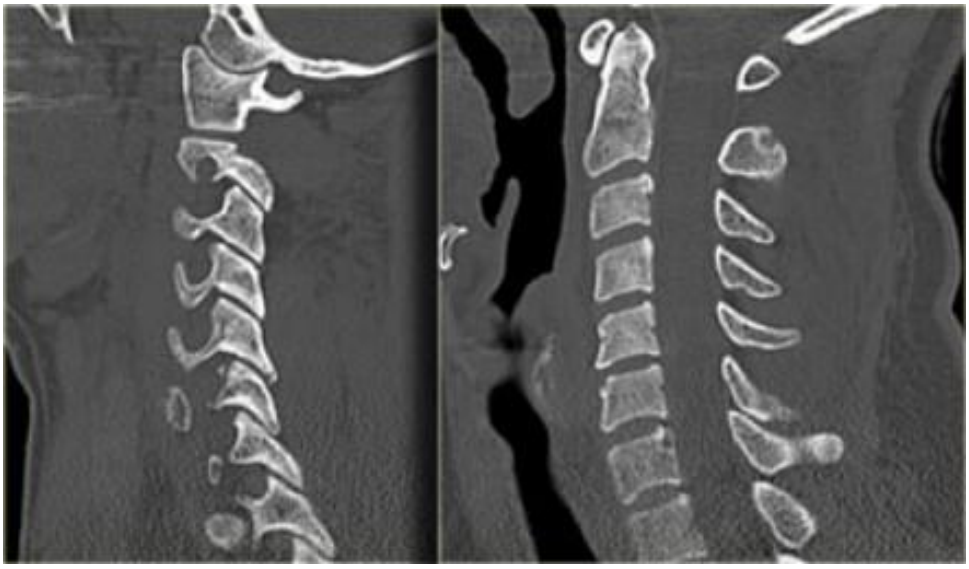


Figure 79 Cervical hyperflexion injury in 44-year-old

The findings are:

- Small bone fragments coming off the superior and inferior facets.
- Widened interspinous space at C5-6.
- Soft tissue swelling at this level posteriorly.
- Subtle narrowing of the disc space at the C5-6-level.

These CT-findings are very subtle and do not seem to match the neurological problem. In such a case MRI is the next step.

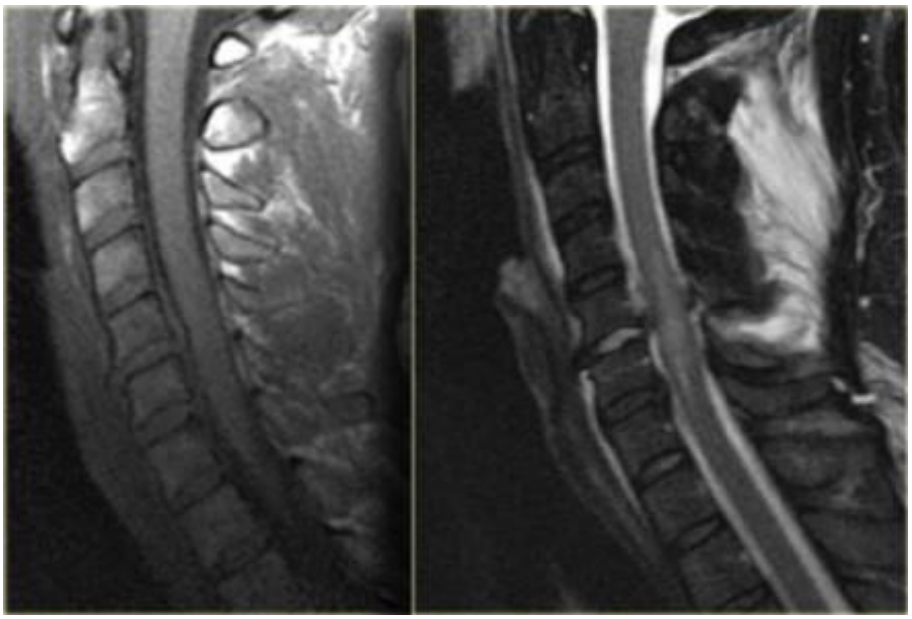


Figure 80 Cervical MRI scan

The MRI -findings are:

- Severe soft tissue injury of the posterior paraspinal structures, especially at the C5-6 level, where the interspinous ligament and the ligamentum flavum is ruptured.
- Disruption of the C5-6 disc with migration behind C5.
- Large amount of spinal cord oedema.

This type of cervical injury can also compromise the vertebral arteries and their blood supply for the brain stem and arterial circle of Willis. It is here that cervical manipulation is contra-indicated.

Unilateral interfacet dislocation

Unilateral interfacet dislocation is due to a hyperflexion injury with rotation. The superior facet on one side slides over the inferior facet and becomes locked. This results in an anterior subluxation of the upper vertebral body of about 25% of the A/P diameter of the body. Simple unilateral facet dislocation is a stable injury.
30% of patients have an associated neurologic defect. MRI plays an important role in the diagnosis in order to see if there is disc extrusion leading to cord compression.

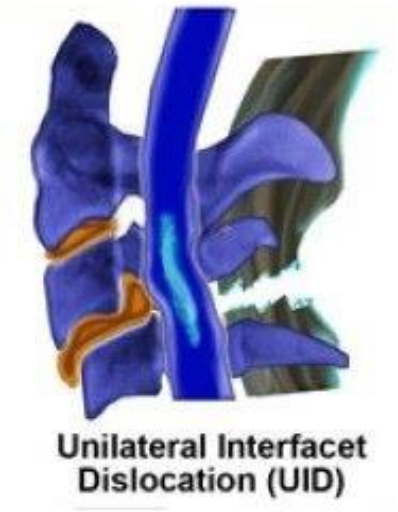


Figure 81 Cervical unilateral dislocation

The next example is a 20-year-old male who had a rollover motor vehicle accident.

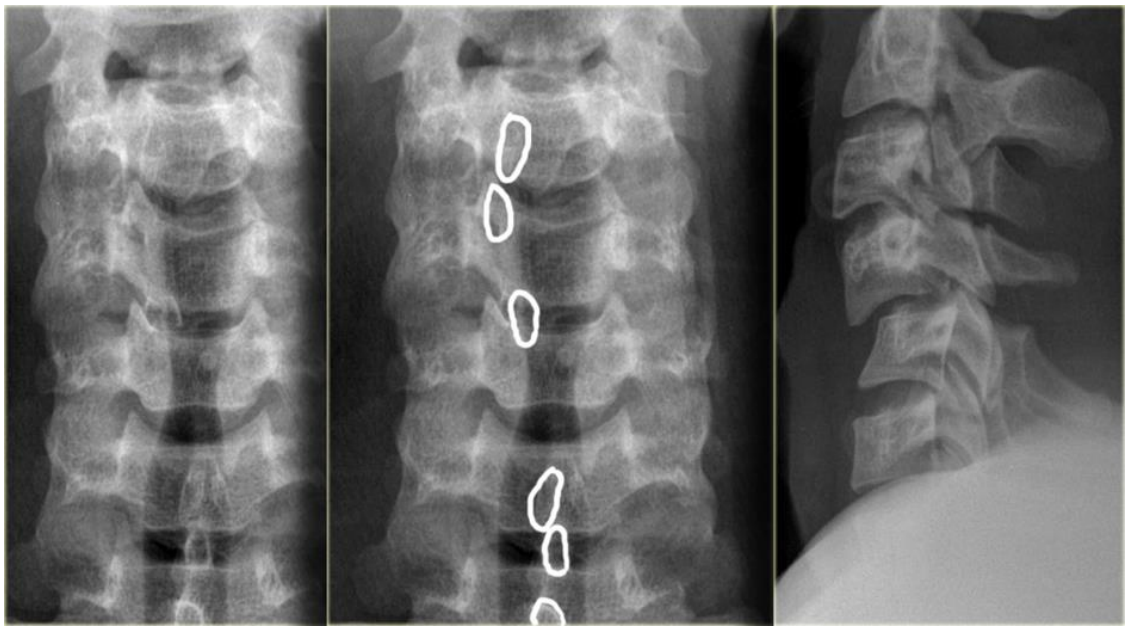


Figure 82 X-ray of 20-year-old - roll over injury

The radiographic findings are:

- Hyperflexion at the level of C4/5 with widening of the interspinous space.
- Subluxation at the level of C4/5 with about 25% translation (i.e., anteroposition of 25% of the AP diameter of the vertebral body)
- Malalignment of the spinous processes as seen on the A/P-view, which can only be produced by a rotatory injury. The involved spinous process points to the involved side
- Due to the rotation the spinous processes of C4 and C5 seem shorter on the lateral view.

The CT confirms the unilateral dislocation. The contralateral facet joint is only distracted.

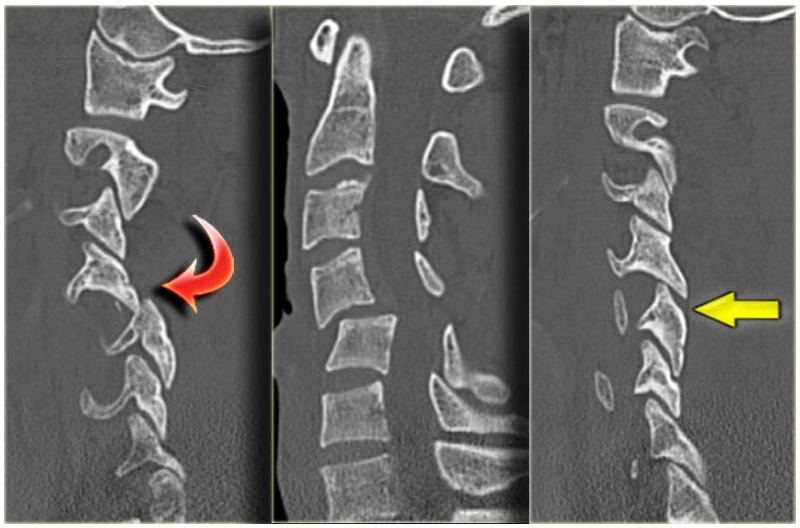


Figure 83 CT scan of unilateral facet dislocation

The MRI-findings are:

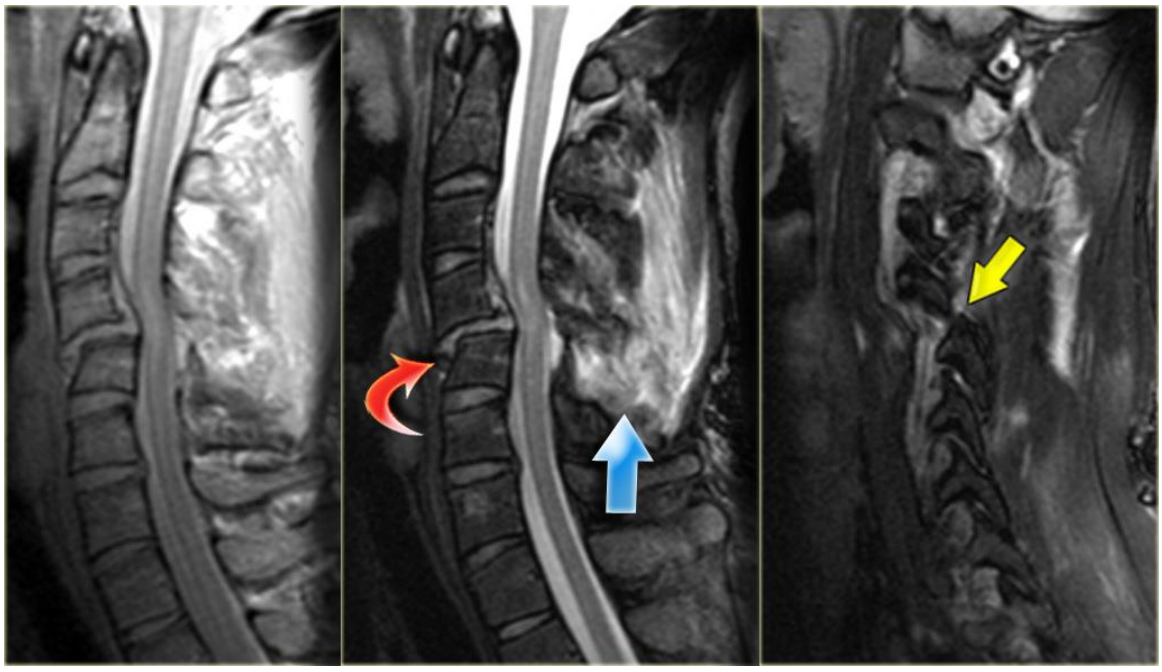


Figure 84 MRI of unilateral interfacet dislocation

- Spinal cord lesion, which can be described as contusion, oedema or non-haemorrhagic spinal cord injury.
- Dislocation of C4/5 facet (yellow arrow)
- Rupture of the spinous ligaments (blue arrow)
- Rupture of the ligamentum flavum.
- Rupture of the disc with migration of disc material on the posterior side of C4 and even on the anterior side of C5 (red arrow)
  The disc space is always disrupted in this kind of injury due to the extreme rotation.

Bilateral Interfacet Dislocation

Bilateral interfacet dislocation (BID) is the result of extreme hyperflexion. There is anterior dislocation of the articular masses with disruption of the posterior ligament complex, posterior longitudinal ligament, the disc and usually also the anterior longitudinal ligament.
When the dislocation is complete, the dislocated vertebra is anteriorly displaced one-half of the AP diameter of the vertebral body.
Because of its extensive soft tissue damage and dislocated facet joints, BID is unstable and is associated with a high incidence of cord damage.

Figure 85 Bilateral interfacet dislocation

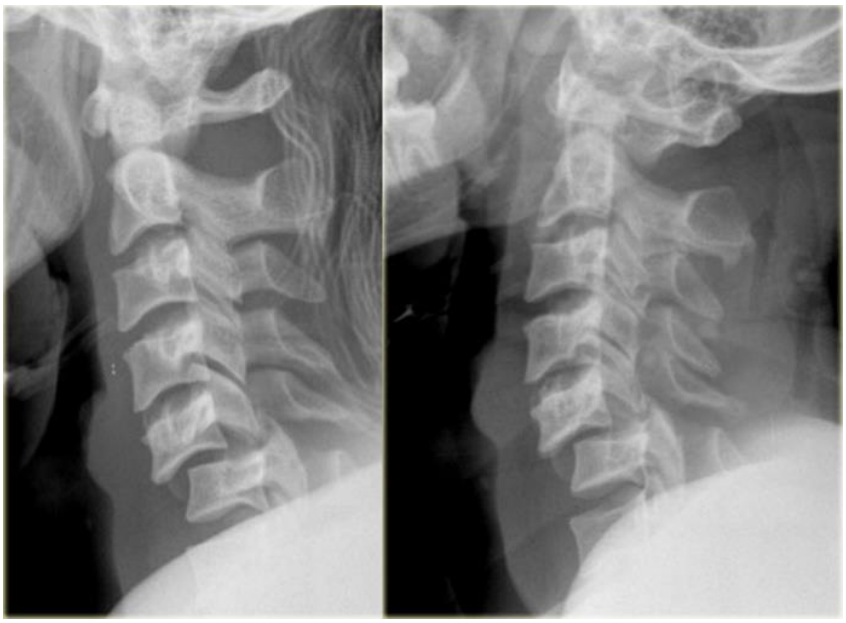


Figure 86 X-ray bilateral interfacet dislocation C6/7

The findings are:

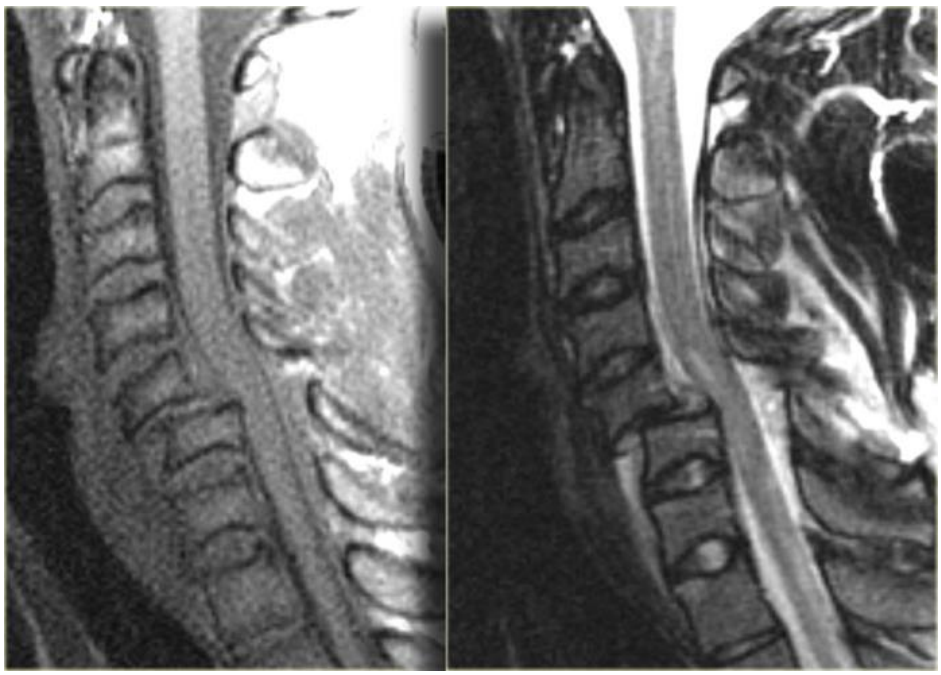
- Bilateral interfacet dislocation.
- 50% anteroposition C5/6 as a result of the dislocation.

    - In unilateral dislocation the anteroposition is usually only 25%.
- Widened space between spinous processes C5 and C6 due to ligament rupture.
- Ruptured disc space.

The MRI findings for the same patient are:

- Soft tissue swelling anteriorly.
- Disruption of the disc.
- Non-haemorrhagic cord injury.

Figure 87 Cervical bilateral dislocation MRI



Correction of this was performed via a traction technique In order to regain normal alignment. Progressive weights are used to lengthen the spine until reduction is achieved. In this case, with 60 pounds the facets start to move, but it finally takes about 110 pounds before the neck is reduced.
Because someone is holding on to the neck while more weight is added, an actual 'clunk' can be felt in the neck indicating that reduction is achieved.

Bilateral interfacetal dislocation

Below are images of a 15-year-old, who was injured during wrestling.

- There is 50% anteroposition of C3 on C4 as a result of bilateral interfacetal dislocation.
- There is complete disruption of the posterior complex.
- This boy had severe neurologic deficit.

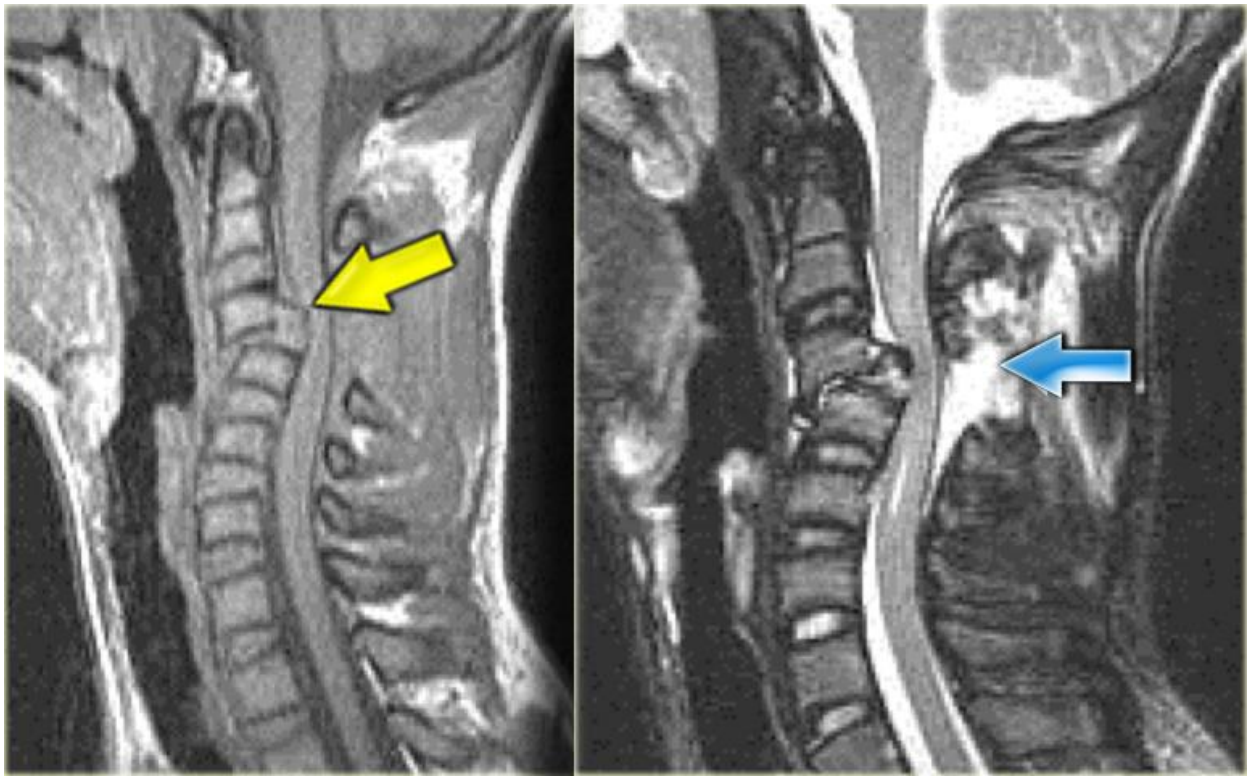


Figure 88 Cervical bilateral dislocation of 15-year-old

Spinal cord syndromes:

1. **Central cord syndrome**
    - Most common incomplete cord syndrome.
    - Frequently found in elderly with underlying spondylosis or younger people with severe extension injury (figure).
    - Upper extremity deficit is greater than lower extremity deficit, because the lower extremity corticospinal tracts are located lateral in the cord.
2. **Anterior cord syndrome**
    - Seen in flexion injuries e.g. burst fracture, flexion tear drop fracture and herniated disk.
    - Presents with immediate paralysis, because the corticospinal tracts are located in the anterior aspect of the spinal cord.
3. **Brown-Sequard syndrome**
    - Ipsilateral motor weakness and contralateral sensory deficit due to hemisection of the spinal cord.
    - Brown-Sequard syndrome may result from rotational injury such as fracture-dislocation or from penetrating trauma such as stab wound.
4. **Posterior cord syndrome**
    - Uncommon syndrome due to extension injury.
    - Loss of positioning sense due to disruption of dorsal columns.
    - Good prognosis.

5. **Complete spinal cord injury**
    - Total absence of sensation and motor function caudal to the level of injury.

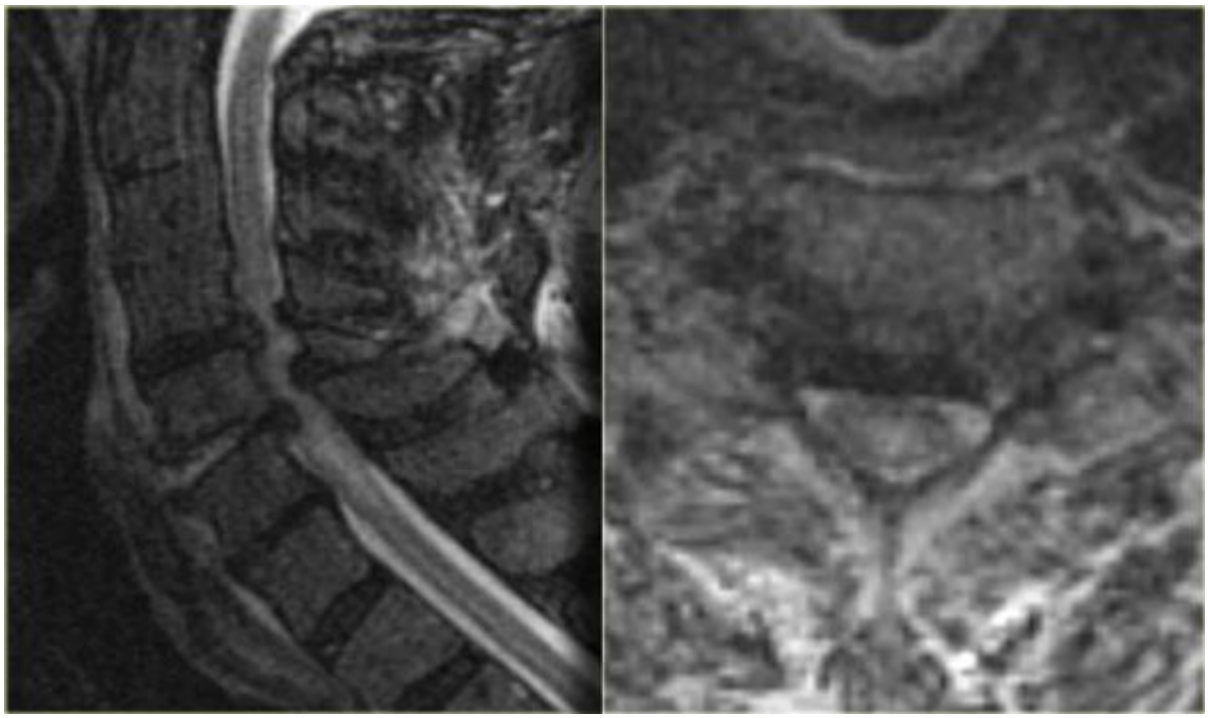


Figure 89 Cervical hyperextension injury with spinal stenosis

Cauda Equina Syndrome and Conus Medullaris Syndrome

Low back pain is quite common. It affects millions of people. In most cases, you do not need surgery for low back pain. But in rare cases, severe back pain can be a sign of **cauda equina syndrome** (CES), a condition that usually requires urgent surgical treatment. People with cauda equina syndrome often are admitted to a hospital as a medical emergency.

Cauda equina syndrome is a **rare disorder** that usually is a surgical emergency. In patients with cauda equina syndrome, something compresses on the spinal nerve roots. You may need fast treatment to prevent lasting damage leading to incontinence and possibly permanent paralysis of the legs.

In understanding the difference between pathological basis of any disease involving the **conus medullaris** and the cauda equina, keep in mind the level of this structure, it being at the level of L1 and constitutes part of the spinal cord (the distal part of the cord) and is in proximity to the nerve roots. Thus, injuries to this area often yield a combination of upper motor neuron (UMN) and lower motor neuron (LMN) symptoms and signs in the dermatomes and myotomes of the affected segments.

On the other hand, a **cauda equina lesion** is an LMN lesion because the nerve roots are part of the peripheral nervous system. So, as disc lesions usually occur low in the spine, cauda equina syndrome is commoner that conus medullaris syndrome.

Causes of Cauda Equina Syndrome

CES occurs more often in adults than in children. But it can occur in children who have a spinal birth defect or have had a spinal injury.

These are the most common causes of cauda equina syndrome:

- A severe ruptured disk in the lumbar area (the most common cause)
- Narrowing of the spinal canal (stenosis)
- A spinal lesion or malignant tumour
- A spinal infection, inflammation, haemorrhage, or fracture
- A complication from a severe lumbar spine injury such as a car crash, fall, gunshot, or stabbing
- A birth defect such as an abnormal connection between blood vessels (arteriovenous malformation)
- Pre-existing Spondylolisthesis

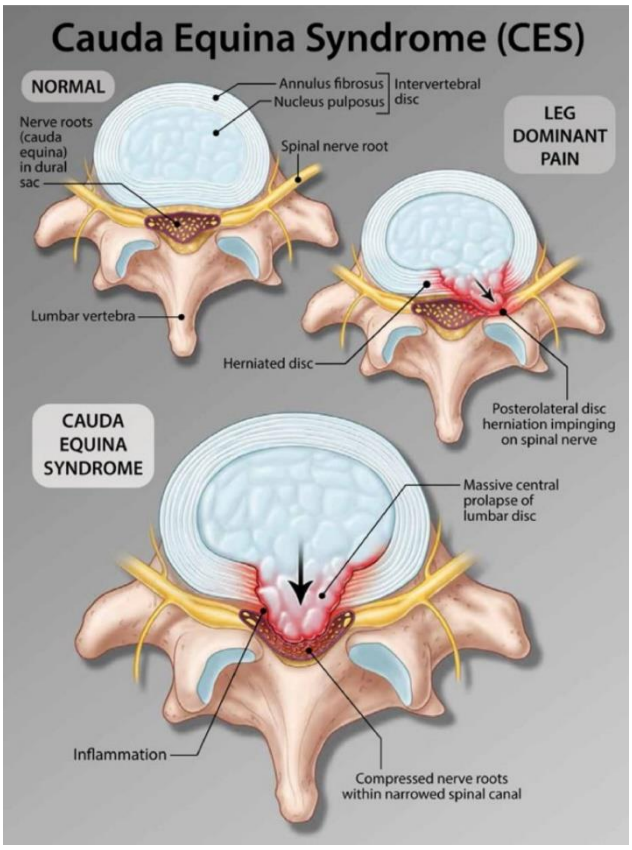


Figure 90 Diagram showing disc lesions of nerve root and of Cauda Equina.

Symptoms of Cauda Equina Syndrome

The 5 Five Red Flags of CES are SPINE:

- **S- Saddle Anaesthesia**
  - Weakness, tingling or numbness in your ‘saddle region’- the parts of your body that would touch the saddle if you were sitting on a horse. Upper inner thighs, groin, buttocks, and genitals
- **P- Pain**
  - Pain, weakness tingling or numbness in your lower back, backs of thighs, lower legs, and feet.
- **I - Incontinence**
  - Loss of control, urgency or finding it hard to pee or poo. Any strange sensation or numbness when you pee or poo.

- **N- Numbness**
    - Numbness in your lower back, backs of thighs, lower legs and feet. Loss of feeling around your groin during sex. This is also known as **saddle anaesthesia.**
- **E- Emergency**
    - Any of these can be a warning sign of CES. Go to your GP or Emergency Department without delay.

Illustration of saddle anesthesia;
- The S5, S4, and S3 nerves provide sensory innervation to the rectum, perineum, and inner thigh.

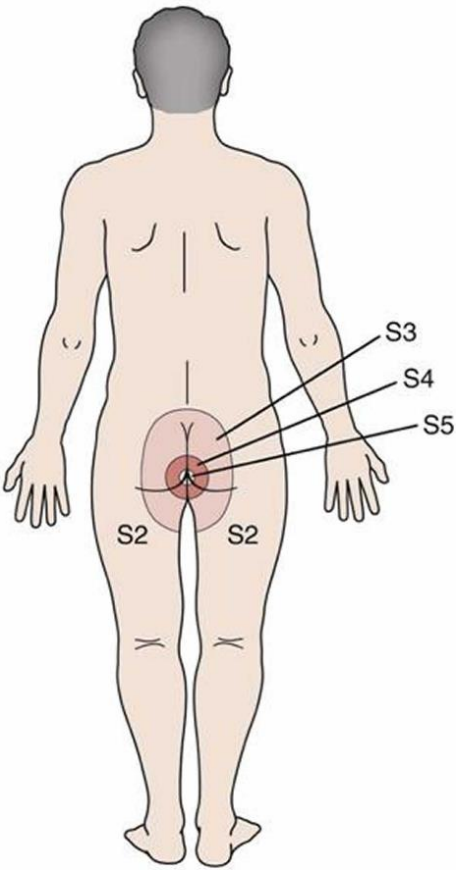


Figure 91 Diagram showing distribution of symptoms with Cauda Equina Syndrome

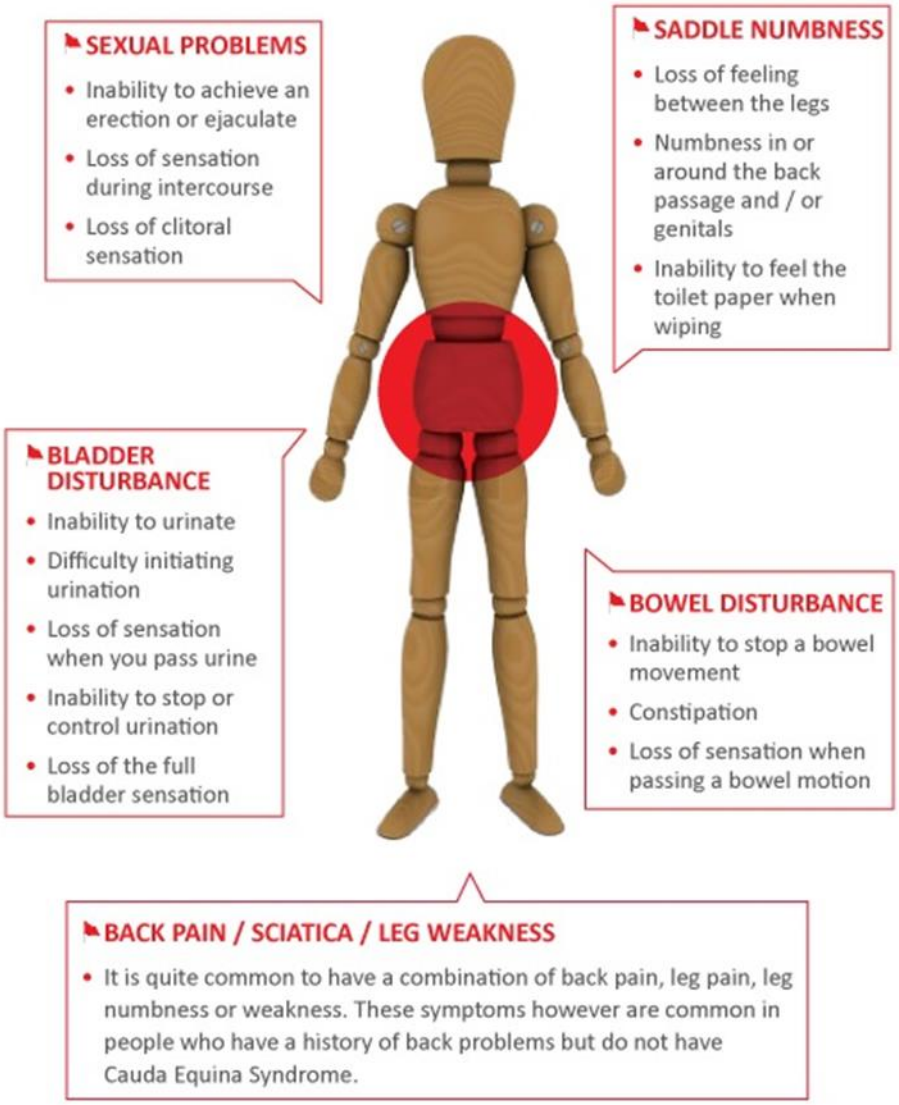


Figure 92 Diagram showing symptoms of cauda equina syndrome symptoms.

Low back pain can be divided into local and radicular pain.

- Local pain is generally a deep, aching pain resulting from soft-tissue and vertebral body irritation.
- Radicular pain is generally a sharp, stabbing pain resulting from compression of the dorsal nerve roots. Radicular pain projects in dermatomal distributions.
- Low back pain in cauda equina syndrome may have some characteristic that suggests something different from the far more common lumbar strain. Patients may report severity or a trigger, such as head turning, that seems unusual.
- Severe pain is an early finding in 96% of patients with cauda equina syndrome secondary to spinal neoplasm. Later findings include lower extremity weakness due to involvement of the ventral roots. Patients generally develop hypotonia and hyporeflexia.
- Sensory loss and sphincter dysfunction are also common.

Urinary manifestations of cauda equina syndrome include the following:

- Retention
- Difficulty initiating micturition.
- Decreased urethral sensation.
- Typically, urinary manifestations begin with urinary retention and are later followed by an overflow urinary incontinence.

Symptoms and Signs of Conus Medullaris and Cauda Equina Syndromes		
	Conus Medullaris Syndrome	Cauda Equina Syndrome
Presentation	Sudden and bilateral	Gradual and unilateral
Reflexes	Knee jerks preserved but ankle jerks affected	Both ankle and knee jerks affected
Radicular pain	Less severe	More severe
Low back pain	More	Less
Sensory symptoms and signs	Numbness tends to be more localized to perianal area; symmetrical and bilateral; sensory dissociation occurs	Numbness tends to be more localized to saddle area; asymmetrical, may be unilateral; no sensory dissociation; loss of sensation in specific dermatomes in lower extremities with numbness and paraesthesias; possible numbness in pubic area, including glans penis or clitoris
Motor strength	Typically, symmetric, hyperreflexia distal paresis of lower limbs that is less marked; fasciculations may be present	Asymmetric areflexic paraplegia that is more marked; fasciculations rare; atrophy more common
Impotence	Frequent	Less frequent; erectile dysfunction that includes inability to have erection, inability to maintain erection, lack of sensation in pubic area (including glans penis or clitoris), and inability to ejaculate
Sphincter dysfunction	Urinary retention and atonic anal sphincter cause overflow urinary incontinence and faecal incontinence; tend to present early in course of disease	Urinary retention; tends to present late in course of disease

Delay in diagnosis and treatment can equal **disability** so, the **timeframe** is especially important.

Sequence of symptoms in patients:

Patients can present with:

- 97% have back pain and sciatica.
- Weakness and changes in sensation in the lower extremities
- 92% of patients have bladder dysfunction (disruption of autonomic fibers results in either retention or incontinence)
- 72% patients have bowel dysfunction (retention or incontinence)

- 93% of patients have saddle anaesthesia or decreased sensation in the perineum.
- Sexual dysfunction (impotence in men)

It can be difficult for the practitioner to ask ‘intimate’ questions and care is required here.

- When you wipe your bottom, can you feel it and can you feel it equally on both sides?
- Can you feel it when you are passing urine?
- Can you feel your bladder when it is full or empty?
- Can you feel it when you are passing a motion?
- Do you have a loss of sensation with activities like sex?
- Do you experience erectile dysfunction?
- Do you achieve orgasm?

Diagnosing Cauda Equina Syndrome

A doctor can diagnose cauda equina syndrome. Here is what may be needed to confirm a diagnosis:

- A medical history, in which answers to questions about health, symptoms, and activity
- A physical exam to assess strength, reflexes, sensation, stability, alignment, and motion. Blood tests may also be required.
- Magnetic resonance imaging (MRI) scan, which uses magnetic fields and computers to produce three-dimensional images of the spine.
- A myelogram -- an X-ray of the spinal canal after injection of contrast material -- which can pinpoint pressure on the spinal cord or nerves.
- A computed tomography (CT) scan

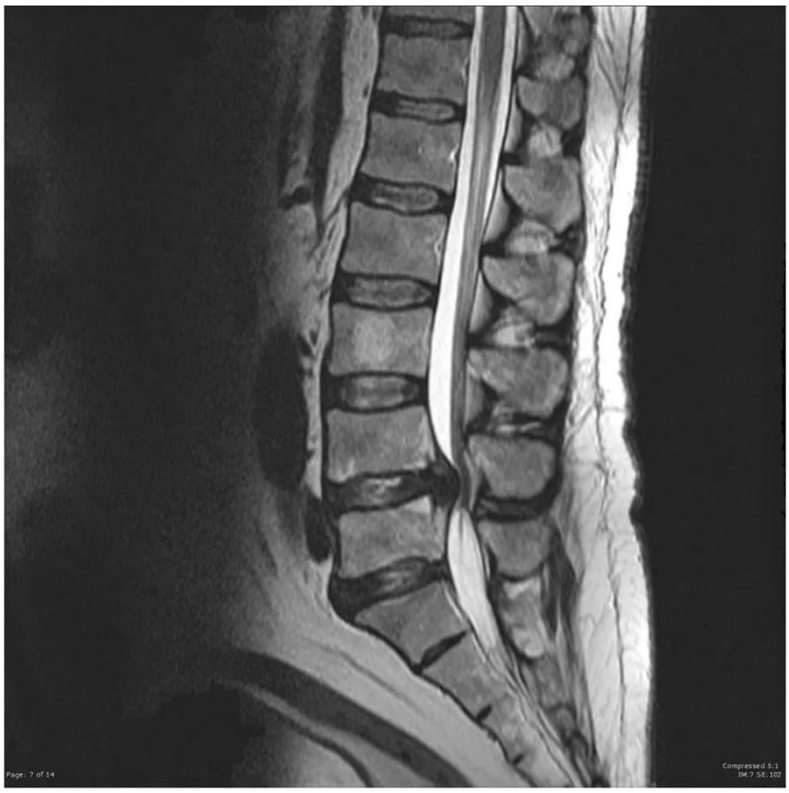


Figure 93 MRI scan of L-Spine showing compression of cauda equina from L4/5 disc prolapse

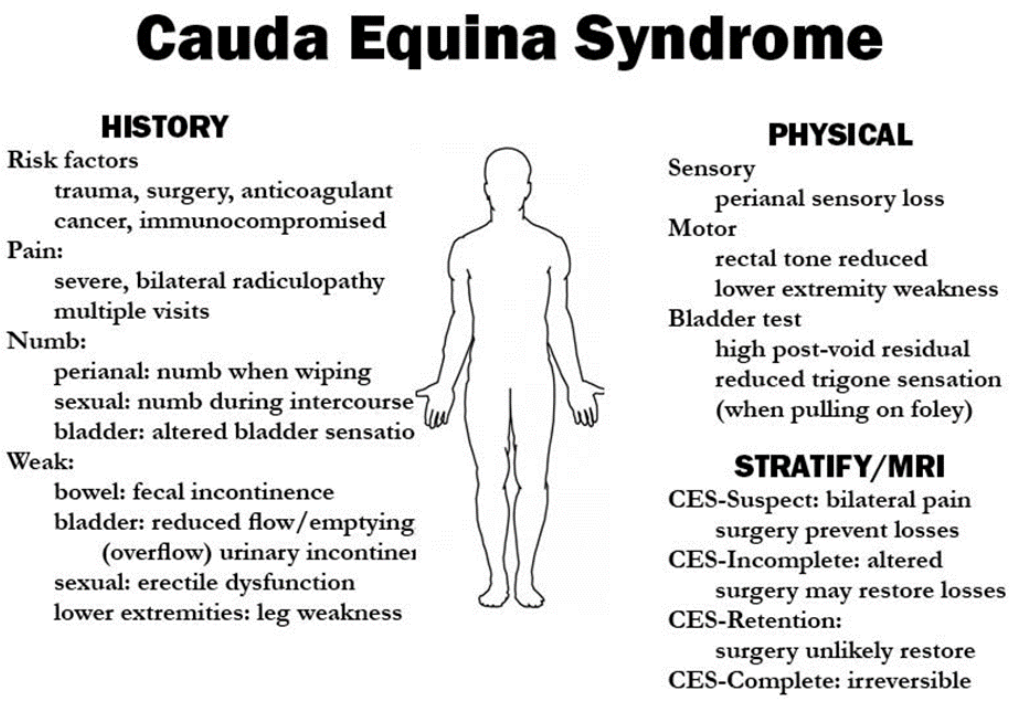


Figure 94 Schematic diagram of history and symptoms of cauda equina syndrome

Pain and Deficits Associated with Specific Nerve Roots				
Nerve Root	**Pain**	**Sensory Deficit**	**Motor Deficit**	**Reflex Deficit**
L2	Anterior medial thigh	Upper thigh	Slight quadriceps weakness; hip flexion; thigh adduction	Slightly diminished suprapatellar
L3	Anterior lateral thigh	Lower thigh	Quadriceps weakness; knee extension; thigh adduction	Patellar or suprapatellar
L4	Posterolateral thigh, anterior tibia	Medial leg	Knee and foot extension	Patellar
L5	Dorsum of foot	Dorsum of foot	Dorsiflexion of foot and toes	Hamstrings
S1-2	Lateral foot	Lateral foot	Plantar flexion of foot and toes	Achilles
S3-5	Perineum	Saddle	Sphincters	Bulbocavernosus; anal

Treatment of Cauda Equina Syndrome

If cauda equina syndrome is diagnosed, it will require prompt treatment to relieve pressure on nerves.

Surgery must be done quickly to prevent permanent damage, such as paralysis of the legs, loss of bladder and bowel control, sexual function, or other problems.

In acute compression of the conus medullaris or cauda equina, surgical decompression as soon as possible becomes mandatory. The goal is to relieve the pressure on the nerves of the cauda equina by removing the compressing agent and increasing the space in the spinal canal. Traditionally, cauda equina syndrome has been considered a surgical emergency,

with surgical decompression considered necessary within **48 hours** after the onset of symptoms, and preferably performed within **6 hours** of injury.

Depending on the cause of the CES, high doses of corticosteroids may be required. These can reduce swelling. Antibiotics may be required if an infection is diagnosed. If a tumour is responsible, radiation or chemotherapy may be needed after surgery.

Surgery, however, is controversial. The timing of decompression is controversial, with immediate, early, and late surgical decompression showing varying results. For mechanical compression of the cauda due to disk herniation, surgical intervention may be indicated. Removal of a large central disc prolapse can be considerably more difficult than routine discectomy and may require an extensive exposure. When performed under less than optimal conditions, as often exists in the emergency setting, surgery may even add to rather than alleviate morbidity.

Even with treatment, full function may not fully return. It depends on how much damage has occurred. If surgery is successful, bladder and bowel function may continue to recover over a period of years.

Living with Cauda Equina Syndrome

If permanent damage has occurred, surgery cannot always repair it. Then the cauda equina syndrome is chronic. This will require adaptations to the changes in the body's functions. Here, physical and emotional support is essential as well.

It is possible that the whole family will be involved here. Many professionals can also provide support. Depending on limitations, help can be sought from:

- An occupational or physical therapist
- A social worker
- A continence advisor and continence physiotherapists
- A sex therapist
- And, as with many conditions, there may be nothing quite as helpful as support from those who really understand what the person is going through. That is why joining a cauda equina support group may be a good idea.

If there is loss of bladder or bowel function, the following tips may help:

- Use a catheter to completely empty your bladder three or four times a day.
- Drink plenty of fluids and use good personal hygiene to prevent urinary tract infections.
- Check for waste and clear the bowels with gloved hands. If needed, use glycerine suppositories or enemas.
- Wear protective pads and pants to prevent leaks.
- Also, doctors can provide medication for help with pain, as well as bladder and bowel problems.

Differential Diagnosis

Diagnostic Considerations

Conus medullaris infarction should be considered in the differential diagnosis, and a source of emboli should be sought by ultrasound to rule out an abdominal aortic aneurysm. Heterotopic ossification should be ruled out by triple-bone scan in a patient with pain and

swelling of the lower extremity in whom deep venous thrombosis (DVT) has been ruled out. In other words, heterotopic ossification should always be considered as a differential diagnosis of DVT in these patients.

Metastatic malignant neoplasms of the spine should be ruled out and the primary source sought as part of the workup in any patient presenting with any of the symptoms listed in Clinical.

Other problems to be considered include the following:

- Amyloidosis with deposits in the spinal cord
- Ankylosing spondylitis and other spondyloarthropathy
- Charcot-Marie-Tooth disease (types 1 and 3)
- Intravascular lymphomatosis
- Lipomas within the spine
- Lumbar stenosis (multilevel)
- Paget disease of the spine
- Spinal infection/abscess and meningitis
- Tethered cord syndrome/short filum terminale
- Vascular intermittent claudication
- Differential Diagnoses
- Acute Inflammatory Demyelinating Polyradiculoneuropathy
- Amyotrophic Lateral Sclerosis in Physical Medicine and Rehabilitation
- Diabetic Neuropathy
- Guillain-Barré Syndrome
- Multiple Sclerosis
- Neoplasms, Spinal Cord
- Neuromuscular and Myopathic Complications of HIV
- Neurosarcoidosis
- Spinal Cord Infections
- Traumatic Peripheral Nerve Lesions

Neoplasms

Cauda equina syndrome can be caused by primary or metastatic spinal neoplasms. Among the primary tumours able to cause CES include myxopapillary ependymoma, schwannoma, and paraganglioma.

Metastatic lesions of the spine are being reported with increasing frequency because of earlier diagnosis, better imaging, and more effective treatment modalities. Although metastasis accounts for most tumours in the spine in general, metastatic tumours in the cauda equina are relatively rare compared with primary tumours.

For the spine in general, sources of spinal metastases are as follows:

- Lung cancer (40-85%)
- Breast cancer (11%)
- Renal cell carcinoma (4%)
- Lymphatic cancer (3%)
- Colorectal cancer (3%)

Prognosis

Several studies have looked at prognosis and outcomes based on the timing of surgical decompression. Early intervention by surgical decompression in patients with conus medullaris and cauda equina syndromes is associated with a better prognosis, particularly when surgery occurs within 48 hours of initial presentation.

The longer the compression continues, the worse the permanent structural and functional impairment, and the poorer the prognosis. It is important to note that the presence of bladder dysfunction prior to surgery has been linked to poorer outcomes regardless of the timing of decompression, although early decompression is still the recommendation for a better prognosis irrespective of clinical status at initial presentation.

Complications

Complications in cauda equina syndrome and conus medullaris syndrome occur in a large percentage of those diagnosed. One study looked at 63-day outcomes on micturition, defecation, saddle anaesthesia, sexual function, and sciatica in cauda equina syndrome.

The data indicate that a large percentage of patients still experience residual symptoms irrespective of their time to surgical decompression.

- Micturition deficits such as retention requiring self-catheterization or presence of suprapubic or indwelling catheters and incontinence still presented in 47.7% of patients.
- Dysfunction with defecation decreased post-operatively significantly, but 41.8% of patients still had problems at 63 days post-operatively.
- Sexual dysfunction persisted in 53.3% of patients, and saddle anaesthesia in 56.6%.
- Sciatica was present in 47.5% of patients.
- The best predictors of outcome are neurological status at presentation and degree of injury. Incomplete injuries tend to have better outcomes.

ASIA impairment scale

In defining impairments associated with a spinal cord lesion, the American Spinal Cord Injury Association (ASIA) impairment scale is used in determining the level and extent of injury.

This scale should also be used in defining the extent of conus medullaris syndrome/cauda equina syndrome. The scale is as follows:

A - Complete; no sensory or motor function preserved in sacral segments S4-S5

B - Incomplete; sensory, but not motor, function preserved below the neurologic level and extends through sacral segments S4-S5

C - Incomplete; motor function preserved below the neurologic level, and the majority of key muscles below the neurologic level have a muscle grade less than 3

D - Incomplete; motor function preserved below the neurologic level, and the majority of key muscles below the neurologic level have a muscle grade greater than or equal to 3

E - Normal; sensory and motor function normal

The injury should be described using this scale, for example, ASIA class A. Most patients with cauda equina/conus medullaris syndrome are in ASIA class A or B initially and gradually improve to class C, D, or E.

Deterrence and Patient Education

- Patients presenting with sciatica and no other evidence of CES/CMS in the history or exam should receive counsel on the possible development of other related symptoms such as bladder or bowel dysfunction, impotence, and saddle anaesthesia. These patients must be given strict return precautions in the event they develop other symptoms pointing to CES/CMS.
- Patients who are suspected to have CES/CMS and are undergoing evaluation for these syndromes need to remain informed and updated on their investigations (MRI, bladder scan, etc.) and pending surgical consultations.
- Patients who receive a definitive diagnosis need counselling on complication rates and must receive a realistic prognosis based on their degree of injury.
- Due to the nature of sensitive lifelong sequelae resulting from these syndromes, cases with CES/CMS have a high involvement in medicolegal litigation.
- For physicians, it is vital to document the history and physical exam thoroughly and accurately and reach the diagnosis as promptly as possible. In court cases involving CES, a positive association exists between time to surgical decompression greater than 48 hours and an adverse decision for the physician involved.
- The degree of functional loss did not affect court rulings in the cases studies.

Other affecting factors:

Age:

Increased likelihood in under 50-year-olds, with the condition having a peak onset between **40-50 years of age.**

Decreased likelihood in over 50-year-olds (because of reduced intervertebral disc space)

Older people will experience increased low back pain, possibly with neurological symptoms.

Coccydynia

Coccydynia is a medical term meaning pain in the coccyx or tailbone area. Coccydynia is also known as coccygodynia, coccygeal pain, coccyx pain, or coccalgia.

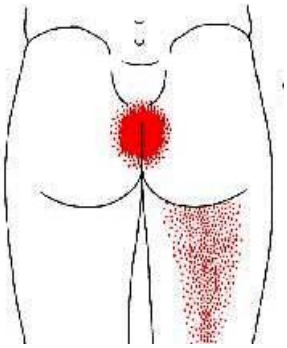


Figure 95 Coccygeal pain

Structure

Coccydynia occurs in the lowest part of the spine, the coccyx, which represents a vestigial tail, or in other words the “tail bone”. The name coccyx is derived from the Greek word for cuckoo due to its beak like appearance. The coccyx itself is made up of 3 to 5 fused vertebrae. The ventral side of the coccyx is slightly concave whereas the dorsal aspect is slightly convex. Both of these sides have transverse grooves that show where the vestigial coccygeal units had previously fused. The coccyx attaches the sacrum, with the attachment being either a symphysis or synchondrosis, and also to the gluteus maximus muscle, the coccygeal muscle, and the anococcygeal ligament.

Causative factors could be local trauma, like falling on it, but this is not always the case.

Activities that put pressure on the affected area are bicycling, horseback riding, and other activities such as increased sitting that put direct stress on the coccyx. The medical condition is often characterized by pain that worsens with constipation and may be relieved with bowel movement. Rarely, even sexual intercourse can aggravate symptoms.

Treatment

This can take the form of local steroid injection, to relieve symptoms. If it is caused by local trauma, there may be local spasm, damage or fibrosis of the local musculature and this can be treated by physical therapy. It can also be treated by ‘balancing’ the perineal muscles (those of the pelvic diaphragm) by a pelvic diaphragm release (as in craniosacral therapy) or functionally releasing the coccyx through holding it directly (one finger inside the anal canal and the out outside).

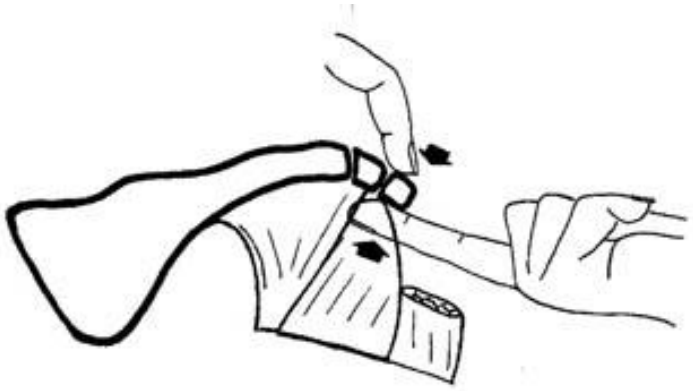


Figure 96 Coccygeal correction

Referred symptoms

Referred symptoms is pain perceived at a location other than the site of the painful stimulus. An example is the case of ischemia brought on by a myocardial infarction (heart attack), where pain is often felt in the neck, shoulders, and back rather than in the chest, the site of the injury. The International Association for the Study of Pain, as of 2001, has not officially defined the term; hence several authors have defined the term differently.

Radiation is different from referred pain. The pain related to a myocardial infarction could either be referred pain or pain radiating from the chest. Classically the pain associated with a myocardial infarction is located in the mid or left side of the chest where the heart is actually located. The pain can radiate to the left side of the jaw and into the left arm. Referred pain is when the pain is located away from or adjacent to the organ involved. Referred pain would be when a person has pain only in their jaw or left arm, but not in the chest. Myocardial infarction can rarely present as referred pain and this usually occurs in people with diabetes or older age.

Physicians and scientists have known about referred pain since the late 1880s. Despite an increasing amount of literature on the subject, the mechanism of referred pain is unknown, although there are several hypotheses.

Characteristics of referral

- The size of referred pain is related to the intensity and duration of ongoing/evoked pain.
- Temporal summation is a potent mechanism for generation of referred muscle pain.
- Central hyper-excitability is important for the extent of referred pain.
- Patients with chronic musculoskeletal pains have enlarged referred pain areas to experimental stimuli. The proximal spread of referred muscle pain is seen in patients with chronic musculoskeletal pain and very seldom is it seen in healthy individuals.
- Modality-specific somatosensory changes occur in referred areas, which emphasize the importance of using a multimodal sensory test regime for assessment.
- Referred pain can be difficult to appreciate as they have origins via ‘shared’ neurological origins, or shared embryological origins.

Mechanism of referral

There are several proposed mechanisms for referred pain. Currently there is no definitive consensus regarding which is correct. The cardiac general visceral sensory pain fibres follow the sympathetics back to the spinal cord and have their cell bodies located in thoracic dorsal root ganglia 1-4(5). As a general rule, in the thorax and abdomen, general visceral afferent (GVA) pain fibres follow sympathetic fibres back to the same spinal cord segments that gave rise to the preganglionic sympathetic fibres. The central nervous system (CNS) perceives pain from the heart as coming from the somatic portion of the body supplied by the thoracic spinal cord segments 1-4(5). Also, the dermatomes of this region of the body wall and upper limb have their neuronal cell bodies in the same dorsal root ganglia (T1-5) and synapse in

the same second order neurons in the spinal cord segments (T1-5) as the general visceral sensory fibres from the heart. The CNS does not clearly discern whether the pain is coming from the body wall or from the viscera, but it perceives the pain as coming from somewhere on the body wall, i.e., substernal pain, left arm/hand pain, jaw pain.

Chart of referred symptoms

Lungs **Liver, stomach, oesophagus, ribs, sternum, costal cartilage** **Rib 1 – stellate ganglion** **T 1-4** **Cervical spine**	Large Intestine **Acute or chronic low back pain** **Sciatica left (venous circulation problems)** **Sciatica right (caecum)** **Varicose veins – left** **Joint pains in lower limbs** **Glenohumeral periarthritis** **Sacroiliac problems**
Liver **C 4-5 right of bilateral** **Right scapula** **Right glenohumeral joint** **Cervical/brachial plexus and fascia** **T 7-10, Ribs 7-10 right, costovertebral joints** **Cranial base restriction – right** **Sciatica left – venous hepatic origin** **Sciatica right – related to hepatic fascia, right kidney, ascending colon**	Kidneys **T 6-7** **T 10-12** **T11-12 costovertebral** **Inferior navicular (K2 acupuncture point)**
Stomach **Cervical spine – left with left sternoclavicular joint** **Glenohumeral periarthritis – left** **T 6-11** **T 6 – left costovertebral (stomach dermatome)** **Rib 7 – right** **T12 – L3 (crura)** **Sacroiliac – left (related to L1)**	Jejunoileum **T10-12** **Acute of chronic low back pain** **Sciatica left (venous circulation problems)** **Joint pains in lower limbs**

Duodenum **T12-L1 (right > left)**	Bladder **L 2-3 – associated with incontinence** **Sacrococcygeal – associated with feet**
Gallbladder **C 4-6 left** **C4 transverse process** **T 7-9 right costovertebral joint**	Female reproductive system **Lumbosacral – urogenital problems** **Knee – genitocrural nerve** **C 2-4 – hormone problems, via hypothalamic-pituitary axis**
	Coccyx **Refers to bladder and uterus**

Convergent-projection

Convergent projection proposes that afferent nerve fibres from tissues converge onto the same spinal neuron. This large or chronic sensory input can precipitate an ‘overflow’ to other nerves sharing that spinal segment, causing pain in other areas of the body that share that spinal segment. This explains why referred pain is believed to be segmented in much the same way as the spinal cord. Additionally, experimental evidence shows that when local pain (pain at the site of stimulation) is intensified the referred pain is intensified as well.

Criticism of this model arises from its inability to explain why there is a delay between the onset of referred pain after local pain stimulation. Experimental evidence also shows that referred pain is often unidirectional. For example, stimulated local pain in the anterior tibial muscle causes referred pain in the ventral portion of the ankle; however, referred pain moving in the opposite direction has not been shown experimentally. Lastly, the threshold for the local pain stimulation and the referred pain stimulation are different, but according to this model they should both be the same.

Convergence-facilitation

Convergence facilitation believes that the internal organs are insensitive to stimuli and that non-nociceptive afferent inputs to the spinal cord created what he termed "an irritable focus". This focus caused some stimuli to be perceived as referred pain. However, these ideas did not gain widespread acceptance from critics due to its dismissal of visceral pain.

Recently this idea has regained some credibility under a new term, central sensitization. Central sensitization occurs when neurons in the spinal cord's dorsal horn or brainstem become more responsive after repeated stimulation by peripheral neurons, so that weaker signals can trigger them. The delay in appearance of referred pain shown in laboratory experiments can be explained due to the time required to create the central sensitization.

Axon-reflex

Axon reflex suggests that the afferent fibre is bifurcated before connecting to the dorsal horn. Bifurcated fibres do exist in muscle, skin, and intervertebral discs. Yet these particular neurons are rare and are not representative of the whole body. Axon-Reflex also does not

explain the time delay before the appearance of referred pain, threshold differences for stimulating local and referred pain, and somatosensory sensibility changes in the area of referred pain.

Hyper-excitability

Hyper-excitability hypothesizes that referred pain has no central mechanism. However, it does say that there is one central characteristic that predominates. Experiments involving noxious stimuli and recordings from the dorsal horn of animals revealed that referred pain sensations began minutes after muscle stimulation. Pain was felt in a receptive field that was some distance away from the original receptive field. According to hyper-excitability, new receptive fields are created as a result of the opening of latent convergent afferent fibres in the dorsal horn. This signal could then be perceived as referred pain.

Several characteristics are in line with this mechanism of referred pain, such as dependency on stimulus and the time delay in the appearance of referred pain as compared to local pain. However, the appearance of new receptive fields, which is interpreted to be referred pain, conflicts with the majority of experimental evidence from studies including studies of healthy individuals. Furthermore, referred pain generally appears within seconds in humans as opposed to minutes in animal models. Some scientists attribute this to a mechanism or influence downstream in the supraspinal pathways. Neuroimaging techniques such as PET scans or fMRI may visualize the underlying neural processing pathways responsible in future testing.

Thalamic-convergence

Thalamic convergence suggests that referred pain is perceived as such due to the summation of neural inputs in the brain, as opposed to the spinal cord, from the injured area and the referred area. Experimental evidence on thalamic convergence is lacking. However, pain studies performed on monkeys revealed several pathways converging on both subcortical and cortical neurons.

Referred pain can be indicative of nerve damage. A case study done on a 63-year-old man with a sustained injury during his childhood developed referred pain symptoms after his face or back was touched. After even a light touch, there was shooting pain in his arm. The study concluded that the reason for this man's pain was possibly due to a neural reorganization which sensitized regions of his face and back after the nerve damage occurred. It is mentioned that this case is very similar to what phantom limb syndrome patients suffer. This conclusion was based on experimental evidence gathered by V. Ramachandran in 1993, with the difference being that the arm that is in pain is still attached to the body.

Orthopaedic diagnosis

From the above examples one can see why understanding of referred pain can lead to better diagnoses of various conditions and diseases. In 1981 physiotherapist Robin McKenzie described what he termed *centralization*. He concluded that centralization occurs when referred pain moves from a distal to a more proximal location. Observations in support of this idea were seen when patients would bend backward and forward during an examination.

Studies have reported that the majority of patients that centralized were able to avoid spinal surgery via isolating the area of local pain. However, the patients that did not centralize had to undergo surgery to diagnose and correct problems. As a result of this study there has been a lot of research into the elimination of referred pain through certain body movements.

One example of this is referred pain in the calf. McKenzie showed that the referred pain would move closer to the spine when the patient bent backwards in full extension a few times. More importantly, the referred pain would dissipate even after the movements were stopped.

General diagnosis

As with myocardial ischaemia referred pain in a certain portion of the body can lead to a diagnosis of the correct local centre. Somatic mapping of referred pain and the corresponding local centres has led to various topographic maps being produced to aid in pinpointing the location of pain based on the referred areas. For example, local pain stimulated in the oesophagus is capable of producing referred pain in the upper abdomen, the oblique muscles, and the throat. Local pain in the prostate can radiate referred pain to the abdomen, lower back, and calf muscles. Kidney stones can cause visceral pain in the ureter as the stone is slowly passed into the excretory system. This can cause immense referred pain in the lower abdominal wall.

Further, recent research has found that ketamine, a sedative, is capable of blocking referred pain. The study was conducted on patients suffering from fibromyalgia, a disease characterized by joint and muscle pain and fatigue. These patients were looked at specifically due to their increased sensitivity to nociceptive stimuli. Furthermore, referred pain appears in a different pattern in fibromyalgic patients than non-fibromyalgic patients. Often this difference manifests as a difference in terms of the area that the referred pain is found (distal vs. proximal) as compared to the local pain. The area is also much more exaggerated owing to the increased sensitivity.