The Journal of Turkish Spinal Surgery 2003; 14 (3-4): 27-36

SPINAL STENOSIS-MECHANICS

Prof. Dr. Tarık YAZAR, MD

Institution(s):
Ankara University, School of Medicine, Ibn-i Sina Hospital, Department of orthopaedics and Traumatology

Intial Symptoms

Patients with spinal stenosis always commence with sensory problems long before motor symptoms appear. They feel unnatural heaviness or deadness of the limbs and a sense that their legs are going to give way. Most cases describe these symptoms as commencing in the feet spreading up the legs though some describe the opposite. Severe cases describe the onset of perineal numbness as they walk or even the development of priapism. In some cases the sensory symptoms may ascend to a clinical level significantly higher than the radiological level, almost certainly due to a vascular effect on the cauda equina. In some cases, as the patients walk they will become aware of weakness of the dorsiflexion of the ankle and describe that their feet slap on the ground as they walk further or that they start to trip up.

In all cases these neurologic spymptoms are promptly relieved by sitting down or by leaning forward, whereas merely standing still affords no relief at all. Typically, they will develop the symptoms at a regular distance (the threshold) but be able to continue walking for a further similar distance before having to flex forward for relief. Symptoms of sphincteric disturbance are rare, but some patients may describe a sensation of urgency of micturition. At rest, the patients usually complain of little other than backache on prolonged sitting, though some will complain of cramp or a sensation of “restless legs” especially at night.

Neurologic examination of a patient with lumbar spinal stenosis often is remarkably normal.

Loss of ankle jerk and distal vibration sense may be present, but in any case are common in the age group of affected patients. A voluntary decrease in the range of lumbar extension often is seen as it may precipitate symptoms. Straight-leg raising is usually normal.

Dermatomal sensory loss and muscle weakness are uncommon at rest, although they may appear if the patient is reexamined after walking to their tolerance limit. In view of the age range of the typical patient, diminished peripheral pulses or limitation of hip movement may be found.

The Development of Spinal Stenosis

Spinal stenosis is an anatomical term used to describe a small vertebral canal. Its cause is usually developmental. There may be added degenerative change that further restricts the space for the cauda equina, but in the absence of developmental stenosis this does not usually compromise the nerve roots. There is normally

adequate spare capacity inthe extradural space in both the central and the root canals.

The Causes of Developmental Stenosis

The vertebral canal reaches maturity very early in life. By 1 year of age L1 to L4 has reached the size of the adult cross-sectional area, and L5 is mature by 5 years of age. The canal's most rapid growing period is between 18 and 36 weeks of intrauterine life. Impaired nutrition at that time may permanently stunt the canal.

Biomechanical Factors That May Influence the Development of the Trefoil Shape

The shape of the canal is as important as its size, and about 15% of canals are trefoil at L5. Trefoilness is less common at L4 and rare at more proximal levels. The trefoil configuration and a small mid-sagittal diameter is an unpleasant combination, because the nerve roots can then be very tight in the lateral recess.

Dynamic Changes That Affect the Canal's Size

The vertebral canal is not a fixed bony tube. It is an anatomical space in a segmental structure the size of which is influenced by posture and motion. In addition, the extradural soft tissues within the canal are similarly affected by creep and the vascular enlargement that accompanies changing posture. Furthermore, the size of the neural intramural contents changes with exercise.

The Upright Posture

Axial compression of cadaver spines decreased the cross-sectional area by an average of 50 mm2 This has been confirmed in vivo by measuring the axially loaded spines during magnetic resonance imaging. We have yet to disvover how much of this space reduction is caused by buckling of the ligamentum flavum, and how much is the result of creep.

Spinal flexion and Extension

Knutsson used functional myelography in a stenotic patient in 1942, and showed that the continuity of a completely interrupted contrast column at L4/5 could be restored by flexion. Anatomical studies have confirmed that between flexion and extension, there is an avegage change in cross-section area of 40 mm2. This is the result of bulging of the soft tissues anteriorly and posterolateraly. The more a canal is stenotic, the greater will be the relative narrowing by changing posture. In the severe grades of stenosis, even the slightest degree of extension may compress the neural elements.

Posture and Root Canal

In flexion the root has a generous degree of freedom, but in extension there is a pincer action compressing the nerve between the superior articular process posteriorly, and the disc and inferior rim of the vertebral body anteriorly. More distally in the root canal, extension will similarly compress a tight root trapping it between the disc in front and the ligamentum flavum behind.

The Effect of Walking on the Stenotic Canal

Clinical studies suggest that the space in a stenotic canal is reduced further during the activity of walking. The epidural pressure measured by pressure transducer in stenotic patients increased by about 20 mmHg with each step. The pressure was significantly less when a stenotic patient walked in flexion. However, there is not a marked increase in lumbar lordosis du-

ring gait and therefore some of the explanation for increased pressure must be sought elsewhere.

There are two possibilities. First the activity of walking produces a segmental motion not only in one plane, but in all of the three planes of rotation. There is a combination of rotation, lateral bend, and sagittal motion all of which affect the space within a stenotic canal. Secondly, the contents of the canal increase in volume during the activity of walking, both from increased extradural venous pressure caused by an increased venous return, and also from vasodilatation of the vessels of the cauda equina. Patients with spinal stenosis not only flex forward after the onset of claudication symptoms, but the center of gravity moves laterally with an increased sway. This suggests that more space is created by movement in more than one plane.

The Clinical Significance of Spinal Stenosis

A small canal does not necessarily cause problems. It is often symptomless, and has been reported in 21% of asymptomatic subjects over 60 years of age. However, stenosis is a factor in the symptomatology of a number of clinical conditions when the canal is compromised by other pathology. It can be important in these conditions. [(1)] Symptomatic disc protrusion, when a nerve root is compressed by a disc in a small canal[(2)]. Root entrapment syndrome, when in the presence of degenerative change, a nerve root can be Symptomatic Disc Protrusion.

Pathology of the Protrusion

When a disc protrusion is symptomatic, it has been preceded by a longstanding degenerative pathological process. The biomechanics of this degeneration are not understood. In vivo, when an axial spinal load is applied to a healthy spine, the vertebral bodies fracture before the disc is damaged. Similarly in vitro, the bone fails before the healthy disc. Poor nutrition may be responsible for an unhealthy disc developing fissures in response to load, and with multiple fissures, fragments develop. The mechanics of the disc change considerably once a fragment forms, and with a fairly minimal load the fragment displaces posterior, causing the back of the annulus to bulge and sometimes to rupture.

The Mechanics of Root Symptoms

Recent studies suggest that two components are responsible for the root pain in a symptomatic disc protrusion, compression and inflammation. Provided the canal is sufficiently large, patients with a protrusion may experience some back pain, but they are spared root symptoms because the root is not compressed. Patients having disc surgery can have root pressure over 100 mmHg. This is reduced to zero after operation. The root pressure is not related to the degree of reduction in straight-leg raising, suggesting that this root tension sign is probably more related to inflammation than to pressure.

Root Entrapment Syndrome From Degenerative Change

These patients with root entrapment syndrome have constant and severe root pain as a result of nerve root compression in the root canal. Unlike the patient with disc protrusion, they do not have abnormal root tension signs or a trunk list. The pain is present at rest. It is insidious in onset, and it frequently resolves over several weeks or months.

Pathology of Root Compromise

In many patients there is a gradual increase in degenerative change that slowly reduces the size of the root canal. It is a combination of bony change and soft tissue thickening (Fig), which involves the facet joint capsule, the posterior annulus, and the ligamentum flavum.

Biomechanics of Root Entrapment

Many patients with root entrapment syndrome have some degree of vertebral displacement. If the root canal is already small, segmental displacement will reduce the avail-able space further. This can be a particular problem in patients with degenerative spondylolisthesis. If the 5th lumbar root is already tight beneath the superior facet of L5, as the body of L4 displaces forward, the root becomes critically affected (Fig). There is frequently a degree of rotational displacement, which will then give asymmetrical symptoms. When lumbar scoliosis is associated with a stenotic canal, the root entrapment symptoms are particularly troublesome and progressive.

- Figure-

Neurogenic Claudication

The Symptoms of Neurogenic Claudication

Neurogenic claudication is a clinical condition causing discomfort, numbness, and pain or heaviness in one or both legs after the patient has walked a short distance. It is relieved by rest. There is no leg pain at rest. There is often a long history of back pain. Neurogenic claudication is sometimes called spinal stenosis, but stenosis is really an anatomical term. Stenotic spines can be symptomless. There is a biomechanical mechanism to explain why stenotic spines can produce symptoms with walking. This becomes apparent as we examine the abnormal signs.

Abnormal Signs and Characteristics of Patients With Neurogenic Claudication

Patients with neurogenic claudication tend to stoop as they walk, and at the limit of walking tolerance they stop further and then rest (the Stop Test). Some patients can walk up a hill more comfortably than walking down a hill; and some can lean forwards and cycle for a long distance without leg symptoms (the Cycle Test7, although the Cycle Test is not a good discriminator between neurogenic claudication and intermittent claudication (peripheral vascular disease). Patients with neurogenic claudication are generally over years of age, with men affected more frequently than women. Most patients with neurogenic claudication have multiple-level spinal stenosis. Half the patients with bilateral claudication have a degenarative spondylolisthesis, which usually affects men rather than women (although degenerative spondylolisthesis is more common in women than men). Half of the patients with unilateral claudication have a degenerative lumbar scoliosis, and this combination is more common in women than in men. Peripheral vascular disease and spinal stenosis often co-exist. Calcitonin can relieve claudication symptoms in a proportion of patients.

Figure

A single Level of Spinal Stenosis

A single level of stenosis does not usually produce claudication symptoms. For example, a large disc protrusion may almost occlude the canal at one level and produce back and/or leg pain but not claudication. Similarly, a large spinal tumor in the lumbar canal may produce bizarre symptoms but not claudication. Compres-

sion of a nerve root in the central or root canal will cause root entrapment pain but not claudication. In canine studies a single-level, experimental stenosis constricting the cauda equina by 25% did not cause a neurological deficit.

Multiple Levels of Stenosis

Most patients with neurogenic claudication have two or more levels of stenosis. There may be two levels of central stenosis, or one of central stenosis and one of a more distant root canal stenosis. Animal studies have shown that two levels of cadua equina compression at just above venous pressure can produce major changes in nerve conduction, axon transport, and blood flow.

The Venous Anatomy of the Nerve Roots of the Cauda Equina

The venous anatomy of the cauda equina is highly specialized, with centrifugal venous drainage from the conus down the nerve roots to the foramen. By contrast, the arterial flow is centripetal. There is a physiological valve in the radicular veins at the level of the nerve root seleeve. This prevents a back flow of venous blood from the veins in the extradural nerve root, protecting the cauda equina from high venous pressure.

If there is a single level of stenosis the veins of the peripheral part of the nerve root drain to the intervertebral foramen, whereas the veins proximally drain back to the conus. These anastomose with other root veins and then drain distally to the foramen of the respective roots. There is no significant venous congestion. However, in the presence of two levels of stenosis (above the venous pressure) there will be venous enlargement of the root veins in the segment between the two stenoses to a pressure equal to the occlusion pressure (Fig). This might be as great as 100 mm Hg, and it will probably affect nerve root function in a similar way to that shown in animal studies.

Although this hypothesis accounts for some of the features of neurogenic claudicatioın, it is necessary to explain the absence of symptoms at rest, the age and sex characteristics, and the biomechanical affect of posture and of walking.

The Effect of Exercise on Claudication Symptoms.

Electrical stimulation of the cauda equina in a porcine model is associated with electromyclography activity in the tail muscles and an increase in cauda equina blood flow to 300% of the resting level. This is maintained if the stimulation continues for more than 30 minutes. However, if a double level of occlusion is applied to the cauda equine, and the proximal region is then stimulated electrically, the increase in blood flow is less marked and of shorter duration (Fig). This model suggests that in the presence of venous congestion, the arterial vasodilatation associated with exercise is inadequate for prolonged activity. Is suggests that there is an arterial explanation for the claudication symptoms, and that with lower limb activity, arterial vasodilatation of the cauda equina fails and nerve conduction may be impaired. An arterial component of the pathology is compatible with these patients being in the arteriosclerotic age group, often having coexistent peripheral vascular disease, and sometimes responding to calcitonin, which is a potent arterial vasodilator.

The Affect to Posture on claudication Symptoms

A stooping posture can help to relieve caludication symptoms in some patients. As they le-

an forward on a bicycle or when walking up a hill, they may have less discomfort.

It has been shown that flexing the lumbar spine can increase the cross sectional area of the

Figure

Blood flow in a porcine cauda equina. (A) After stimulation of the cauda equina proximally. (B) When producing a two-level block just above venous pressure. (C) Stimulation of the cauda equina in the presence of a two-level block. Central canal by reducing the posterior bulge o fthe annulus, and stretching a buckling ligamentum flavum. Similarly flexion will increase the crosssectional area o fthe root canal.

It is probable that some patients have a block pressure at one of the stenotic levels just above venous pressure in extension, but just below venous pressure in flexion. These patients are able to walk in flexion without the roots being congested; but with extension and rotation, venous enlargement causes problems. However, patients with a very stiff anklyosed spine do not have sufficient segmental motion for symptoms to be influenced by posture.

Symptoms of lumbar spinal origin in and complaints due to vascular disease sometimes may be confused.

The most common differential diagnosis of neurogenic claudication is intermittent ischemic claudication due to peripheral vascular disease. This originally was described in horses and then in humans by Charcot.

The nature and mechanisms of lumbar spinal stenosis and vascular disease are completely different. With vascular problems, pain and malfunction are initiated in tissues (internal organs, muscles, skin) inadequately irrigated or drained by the defective vessels, with an exception for the acute aortic dissection and the rupturing aneurysm where nociceptive signals also arise from the vessel wall itself and from the possible effects of acute exansion in the surrounding retropertioneal space.

The clinical picture is likely to be blurred in patients suffering from both vascular and spinal conditions. Reaching a precise and complete diagnosis in such circumstances can be more challenging.

ACUTE CONDITIONS:

Ruptured abdominal Aortic or Iliac Aneurysm, Acute Aortic Dissection, and Acute Leg Ischemia

This situation is the result of acute arterial occlusion by thrombosis, embolism, dissection, trauma, or extrinsic compression. The history may yield immediate clues to the diagnosis (risk factors such as smoking, previous arterial disease, cardiac disease, trauma). The signs and symptoms are obvious: decreased temperature and capillary fill, absent pulses, and pale or marmore-like skin.

Presentation

Arterial claudication involves the posterior leg muscles only, sometimes the buttocks, perhaps the thigh, always the calf, never the anterior muscles, and never the groin (Fig.). It is most likely to be confused with S1 root suffering. Internittent numbness of the sole of the foot may occur after exercise. Numbness (hypoesthesia) must not be confused with paresthesia (pins and needles).

In spinal claudication, elements other than the leg pain alone often are present: sensorimotor disturbances (pain, paresthesia, numbness) in the related nerve root area and low back pain. The pain may appear or be worsened by lying supine, sitting, or walking downs-

tairs. Bending forward often will alleviate the pain. These factors would never be seen in arterial disease. However, both arterial and spinal claudication may be absent when riding a bicycle and may be present on climbing stairs (the latter as a rule in arterial claudication).

Diagnosis

The diagnosis is to be oriented by taking a careful history (smoking, previous arterial disease, cold feet, previous lumbar problems, postural and occupational pain factors, walking distance, walking stairs) and giving a thorough physical examination, including appropriate orthopedic and neurological tests (Table-17A) quick run through the pedal pulses before and after a simple tip-toe exercise test also should be performed. Immediately after the exercise test, remember to look at the color of the soles. In many cases it will be possible to exclude one of the two conditions on clinical grounds alone. In the troublesome case doubt will persist. Sometimes people develop both arterial and lumbar disease. In such a case the orthopedic and vascular surgeons must cooperate in evaluating the patient.

Conclusion

Spinal stenosis does not usually cause syptoms unless there is an added pathology. The clinical syndromes are now clearly defined. An apperciation of the affect o fload and motion on the stenotic spine and its contents should help us to understand more about the pathophysiology of these conditions and how best they can be managed.

Total Laminectomy

The primary aim of decompression should be the relief of leg pain and neurogenic intermittent claudication, and not the treatment of low back pain.

In the last decade, new advances in diagnostic imaging techniques have allowed better localization of the offending areas of neural compression. More conservative surgical approaches have been recommended with hemilaminectomies, partial laminotomies, and even multilevel interiaminar decompression to avoid postoperative instability. These usually are used in selective patients with predominantly lateral stenosis.

In central canal stenosis and mixed centrolateral stenosis, as in thi trefoil-shaped central canal, wide decompression by total laminectomy with facet foint sparing technique is a relatively safe operation that has high success in the medium to long term

Age is not a limitation for this type of surgery, although co-morbidity (diabetes mellitus, hip osteoarthritis, cardiovascular and pulmonary disease) contribute to poor outcome.

Preoperative instability should be evaluated as well as possible by standing and flexion extension radiographs.

SURGICAL TECHNIQUE: IMPORTANT POINTS

Proper positioning of the patient to avoid abdominal pressure will minimize blood loss during operation.

If there is no containdication due to hip or knee osteoarthritis, the kneeling position is preferred, as it allows the abdomen to hang free, decreases lumbar lordosis, and releases tension of the distal nerve roots.

In special circumstances, as with patients who are obese or who have respiratory problems, the lateral position with a pad between the flexed knees and a pelvic restraining strap may be chosen.

Magnification with a loop and use of a fiberoptic head light afford better visualization of neural and vascular structures. Bipolar coagulation is desirable.

The appropriate level should be marked carefully and checked against the patient's x-ray films for anatomic marks or anomalies that can be easily identified. If in doubt, the appropriate level should be checked by radiograph o image intensifier. We must remember that surgical failure can occur by performing the right operation at the wrong vertebral level.

The paraspinous ligament often can be preserved to increase posterior stability. Careful segmental paraspinal muscle separation may decrease blood loss with good exposure of the posterior arch. In developmental stenosis, the laminae may be thicker and shorter than normal, whereas in degenerative stenosis the osteophytes and overgrowth of the posterior facets may give the laminae a shorter appearance. Overlapping of the laminae may make access to the spinal canal more difficult. When excising the ligamentum flavum, we may find that its consistency varies between normal elasticity and partial ossification (Fig.).

In developmental stenosis, the convex laminae may produce considerable narrowing of the central canal, which carries a great risk of neural damage during surgical decompression. In degenerative stenosis, constriction of the central portion of the canal is produced by osteophytes and overgrowth of the inferior facet of the cephalad vertebra. Degenerative changes of the superior facet of the caudal vertebra produce narrowing of the lateral recess and the foramen.

Excision of the medial half of the facets often gives good decompression of the central and lateral recess, which allows dural re-expension and good boliziation of the compressed root; however, complete decompression is the main object of the surgical procedure and total facetectomy should be done if it is needed. For adequate decompression, all stenosed levels must be decompressed.

As Verbiest pointed out, with mixed stenosis there is a question of whether a part of the canal showing relative stenosis in the absence of an additional compressive agent should be decompressed prophylactically. With pure relative stenosis, the problem is how far to extend decompression beyond the level of any additional compressive agent. Bulging discs should be left undisturbed, and disc protrusions inside an area of stenosis should never bu removed without previous posterior decompression. In developmental stenosis, it is a surgical dilemma whether the decompressive laminectomy should be performed over the entire area to avoid recurrence of symptoms of stenosis at other non-decompressed levels.

Any dural laceration should be repaired carefully. Safe and accurate interoperative and postoperative bleeding control is mandatory. The exposed dura should be covered by a free or pedicle fat graft, Gelfoam, or other synthetic membranes or products to isolate it from the paraspinal musculature to decrease epidural scarring.

The dorsolumbar fascia should be sutured are fully to the paraspinous ligament to maintain lordosis and to increase posterior stability. A suction drain may be placed over the fascia, but never proximal to the exposed dura.

Extensive decompression increases the risk of instability of the correspondent vertebral level. Preoperative instability and postoperative hypermobility following decompression is an indication for concomitant arthrodesis of the decompressed segments. Discectomy and especially preoperative lumbar scoliosis are known

to increase the risk of postoperative instability and may be indications for adjunctie fixation.

With spinal instability, the best chance to regain permanent stability is a solid instrumented posterolateral arthrodesis with pedicular screws and rods. Fusion indications and techniques will be discussed extensively in the following chapters.

The problem with fusion is that it considerably increases the operative time, blood loss, and morbidity of the surgical procedure, especially in elderly patients with degenerative stenosis. There is extensive agreement that the dangers of postoperative instability and vertebral slippage due to extensive, careful decompression are much less than the consequences of insufficient decompression of the neural structures in the stenosed lumbar spinal canal. Therefore, in the absence of obvious segmental instability in elderly patients, no fusion is necessary after decompression surgery (Fig.).

The risk of postoperative slipping is assumed to be low in older patients with advanced degenerative changes of the disc. The results of local decompression fo sciatica and neurogenic claudication in the elderly are good, and fusion is not indicated in older patients with degenerative stenosis.

Neural compression at multiple levels is a relative contraindication to surgical decompression in the elderly. The results of surgery tend to be disappointing when two or more spinal levels are involved.

Figure

Diabetic patients had high rates of postoperative wound complications and prolonged hospitalization. The poorer results may have been related to coexisting diabetic neuropathy or to failure of the nerve roots to recover after decompressive procedures.

Spivak stated that, in his experience, relief of activity-related symptoms after decompression has been as reliable in patients who have diabetes as in those who do not. The relief of constant pain and abnormal sensations in the lower extremity has been less reliable in patients who have diabetes, presumably because of residual symptoms of underlying diabetic neuropathy.

Postacehini and Cinotti reviewed 40 patients treated surgically for spinal stenosis, 5 to 19 years (mean 8) after operation, and evaluated bone growth after total laminectomy and bilateral laminotomy. Their findings indicate that growth of the posterior arch is stimulated by abnormal vertebral motion and represents an attempt to increase vertebral stability. Growth of posterior facet joints may deteriorate the quality of the results with increasing time from surgery, reproducing previous pathologic conditions. On the other hand, growth of the laminar arch does not cause significant compression, except in degenerative spondylolisthesis. The proportion of satisfactory clinical results progressively decreased from the group with mild bone growth to the group with marked growth. Growth was more likely to occur after a narrow laminectomy (Fig.) and if the operated vertebral level was unstable.

Chen et al. also described varying degrees of bone growth in surgical laminar defects as a natural postoperative repair process, with increased association with instability and in levels adjacent to spinal fusion. This association was related with poor clinical outcome in the middle and late follow-up periods.

Current trends toward more limited operative decompression with retention of the stabilizing elements and a decrease in short-term morbidity may lead to a higher rate of long-term failure due to recurrent stenosis or

the development of stenosis at an adjacent level.

Another more immediate consequence of connective tissue repair following laminectomy is epidural scarring and heterotopic bone formation.

Peridural fibrosis is a natural consequence of laminectomy and surgical invasion of the spinal canal. The extent of fibrosis depends primarily on the extent of the surgical procedure and the degree of hemostasis. Peridural fibrosis can be well visualized by magnetic resonance imaging with or without gadolinium enhancement.

Scar fixation of the dura and nerve roots interferes with normal physiological movement of these structures within the spinal canal, leading to pain and limitation of activities and simulating spinal instability ("instability catch").

Experimental studies of LaRocca and MacNab and Langensidold and Kiviluoto showed that the principal source of the scar was from the erector spine muscle mass, which was covering the dura and extending into the canal to adhere to the dura and nerve roots. They advocated covering the dura with Gelfoam and free or pedicule fat transplants to prevent epidural scar formation.

Other synthetic membranes have bene used, including Silastic, Dacron, and Gelfoam impregnated with methylprednisolone, with inconsistent results. None of these synthetic membranes covers the nerve roots and ventral areas adequately.