# Handbook of Neurosurgery

Eighth Edition

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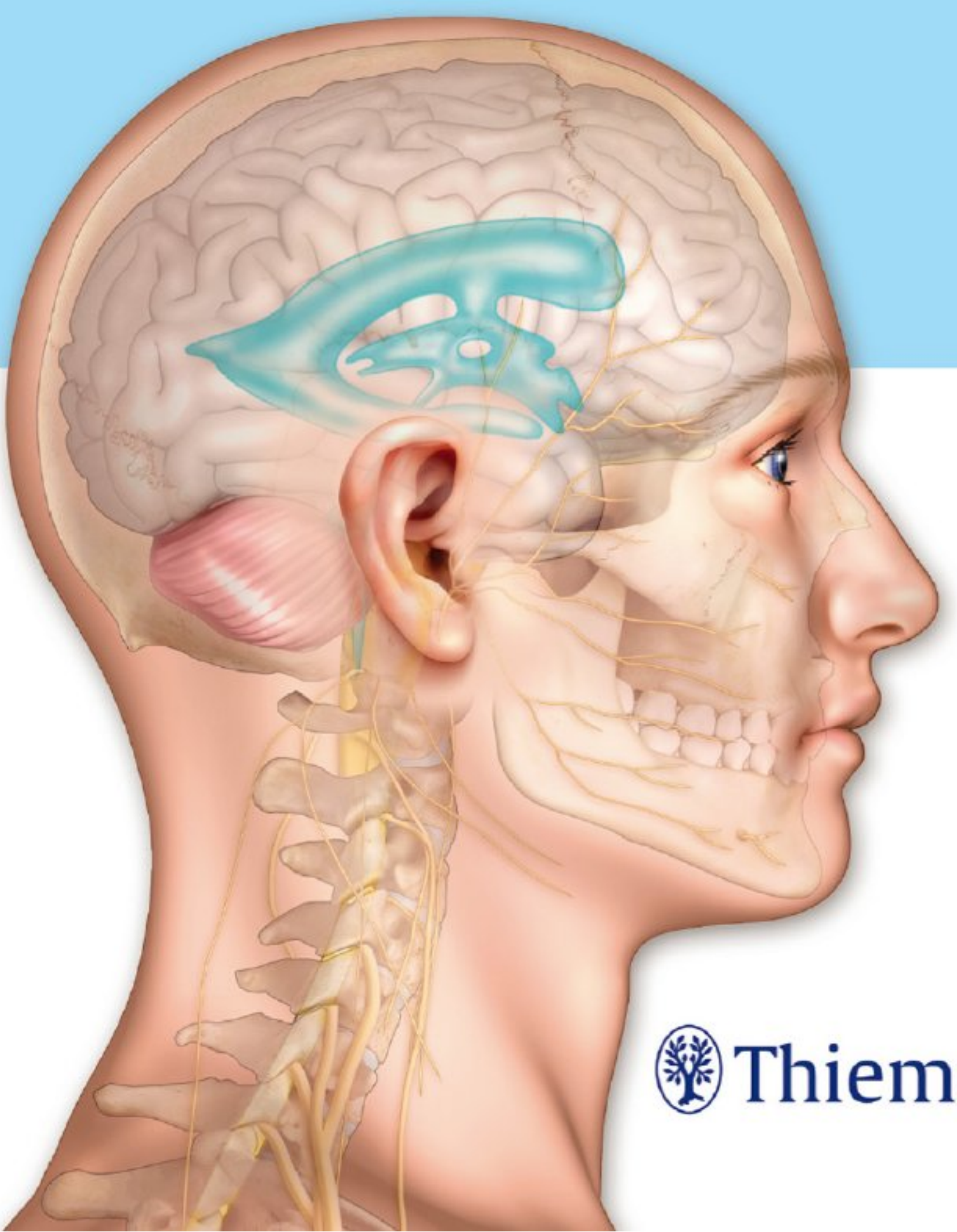

Thieme

# Part XVI

## Spine and Spinal Cord



XVI

# 68 Low Back Pain and Radiculopathy

## 68.1 General information

### Key concepts

See reference.[1]

- low back pain is common, and in ≈ 85%of cases no specific diagnosis can be made
- initial assessment is geared to detecting "red flags" (indicating potentially serious pathology), and in the absence of these, imaging studies and further testing of patients is usually not helpful during the first 4 weeks of low back symptoms
- relief of discomfort is usually best achieved with nonprescription pain meds and/or spinal manipulation
- while activities may need to be modified, bed rest beyond 4 days may be more harmful than helpful, and patients are encouraged to return to work or their normal daily activities as soon as possible
- 89–90%of patients with low back problems will improve within 1 month even without treatment (including patients with sciatica from disc herniation)



Low back pain (LBP) is extremely prevalent, and is the second most common reason for people to seek medical attention.[2] After the common cold, it is the number two cause for loss of time at work. LBP accounts for ≈ 15%of all sick leave from work, and is the most common cause of disability for persons <45 yrs age.[3] Estimates of lifetime prevalence range from 60–90%, and the annual incidence is 5%[4] Only 1%of patients will have nerve-root symptoms, and only 1–3%have lumbar disc herniation. The prognosis for most cases of LBP is good, and improvement usually occurs with little or no medical intervention.

## 68.2 Intervertebral disc

### 68.2.1 General information

The function of the intervertebral disc is to permit stable motion of the spine while supporting and distributing loads under movement. The intervertebral disc has been characterized as the largest nonvascularized structure in the human body, which imparts some unique attributes to it.

### 68.2.2 Anatomy

Anulus fibrosus (anulus may alternatively be spelled annulus, but fibrosus is the only correct spelling and is distinct from fibrosis)[5]: the multilaminated ligament that encompasses the periphery of the disc space. Attaches to the end-plate cartilage and ring apophyseal bone. Blends centrally with the nucleus pulposus.

Nucleus pulposus: the central portion of the disc. A remnant of the notocord.

Capsule[5]: combined fibers of the anulus fibrosus and the posterior longitudinal ligament (this term is useful because these 2 structures may not be distinguishable on imaging studies).

## 68.3 Nomenclature for disc pathology

Historically, the terminology for lumbar disc pathology has been contentious and nonstandardized. A committee tasked to standardize the nomenclature has issued version 2.0 of their recommendations.[6] Some of these standardizations are useful primarily for consistency related to radiographic reports and for research, and may not be as useful for day-to-day clinical practice A subset of the recommendations is shown in □ Table 68.1.

Degenerated disc: (see □ Table 68.1 for definition) some reports indicate that these can cause radicular pain possibly by an inflammatory mechanism,[7] but this is not universally accepted.

Vacuum disc: gas in the disc space (empty space on imaging), usually indicates disc degeneration, not infection.

Table 68.1 Nomenclature for lumbar disc pathology[6]

| Term | Description |
|---|---|
| anular tears AKA anular fissures | separations between anular fibers, avulsions of fibers from their VB insertions, or breaks through fibers that extend radially, transversely, or concentrically |
| degeneration | desiccation, fibrosis, narrowing of the disc space, diffuse bulging of the anulus beyond the disc space, extensive fissuring (numerous anular tears), mucinous degeneration of the anulus, defects & sclerosis of endplates, & osteophytes at the vertebral apophyses |
| degenerative disc disease | clinical syndrome of symptoms related to degenerative changes in the intervertebral disc (described above), also often considered to encompass degenerative changes outside the disc as well |
| bulging disc | generalized displacement of disc material (arbitrarily defined as >50% or 180°) beyond the peripheral limits of the disc space[a]. Not considered a form of herniation. May be a normal finding, not usually symptomatic |
| herniation | localized displacement of disc material (<50% or 180°) beyond the limits of the intervertebral disc space[a] |
| | focal: <25% of the disc circumference |
| | broad-based: 25–50% of the disc circumference |
| | protrusion: the fragment does not have a "neck" that is narrower than the fragment in any dimension |
| | extrusion: the fragment has a "neck" that is narrower than the fragment in at least 1 dimension. 2 subtypes<br>a) sequestration: the fragment has lost continuity with the disc of origin (AKA free fragment)<br>b) migration: the fragment is displaced away from the site of extrusion, regardless of whether sequestered or not |
| | intravertebral herniation (AKA Schmorl's node (p. 1060): disc herniates in the cranio-caudal direction through the cartilaginous end-plate into the VB |

Table 68.2 Modic's classification

| Modic Type | Intensity changes | | Description | |
|---|---|---|---|---|
| | T1WI | T2WI | | |
| 1[a] | ↓ | ↑ | bone marrow edema associated with acute or subacute inflammation | |
| 2 | ↑ | iso or ↑ | chronic changes | replacement of bone marrow by fat |
| 3 | ↓ | ↑ | | reactive osteosclerosis |

[a]Type 1 Modic changes disappear on STIR images (the signal characteristics mimic CSF/water). Back pain in this group may respond to fusion (p. 1036).

## 68.4 Vertebral body marrow changes

Associated with degenerative or inflammatory changes. Modic's classification[8] of MRI characteristics is shown in □ Table 68.2.

## 68.5 Clinical terms

□ Radiculopathy. Dysfunction of a nerve root; signs and symptoms may include: pain in the distribution of that nerve root, dermatomal sensory disturbances, weakness of muscles innervated by that nerve root, and hypoactive muscle stretch reflexes of the same muscles

 Mechanical low back pain (p.1024) . AKA "musculoskeletal" back pain (both non-specific terms). The most common form of low back pain. May result from strain of the paraspinal muscles and/or ligaments, irritation of facet joints... Excludes anatomically identifiable causes (e.g. tumor, disc herniation...)

 Sciatica. Pain along the course of the sciatic nerve, usually resulting from nerve root compromise (the sciatic nerve is comprised of nerve roots L1 through L5)

## 68.6 Disability, pain and outcome determinations

Disability scales for low back pain have been developed to assess outcomes for research purposes. Some widely used measures include:

1. visual analogue scale: used for any type of pain. The patient is asked to mark their pain level on a line divided into segments with sequential labels 0 (no pain) to 10 (the worst pain)
2. Oswestry disability index (ODI)[9]: a categorical ordinal scale that is used for low back pain. There are 4 English versions in wide use,[10] version 2.0[11] is recommended.[10]
   It consists 10 questions related to activities of daily living. Each item is scored 0–5 (5 being the most disability) and the total is multiplied by 2%to obtain the final score (range: 0–100%). The interpretation of the final score is shown in  Table 68.3. A score >45%is essentially completely disabled. A score in the teens is very functional
3. Roland–Morris disability questionnaire[12]
4. Short Form 36 (SF36)[13]



## 68.7 Differential diagnosis of low back pain

The differential diagnosis of low back pain (p.1024) overlaps with that of myelopathy. In ≈ 85%of cases of LBP no specific diagnosis can be made,[14] however, serious and/or dangerous conditions can usually be reliably ruled out.

## 68.8 Initial assessment of the patient with back pain

### 68.8.1 Background

Initial assessment consists of a history and physical exam focused on identifying serious underlying conditions such as: fracture, tumor, infection or cauda equina syndrome (p.1050). Serious conditions presenting as low back problems are relatively rare.

### 68.8.2 History

The following information has been found to be helpful in identifying patients with serious underlying conditions such as cancer and spinal infection.[1]  Table 68.4 shows the sensitivity and specificity of some features of the history for various conditions.

1. age
2. history of cancer (especially malignancies that are prone to skeletal metastases: prostate, breast, kidney, thyroid, lung, lymphoma/myeloma)
3. unexplained weight loss

Table 68.3 Oswestry disability index score

| Score | Interpretation |
|---|---|
| 0–20% | minimal disability: can cope with most daily activities |
| 21–40% | moderate disability: pain and difficulty with sitting, lifting & standing. The patient may be disabled from work |
| 41–60% | severe disability: pain is the main problem, but other areas are affected |
| 61–80% | crippled: back pain impinges on all aspects of the patient's life |
| 81–100% | these patients are either bed-bound or else are exaggerating their symptoms |

Table 68.4 Sensitivity and specificity of historical findings in patients with low back problems[1]

| Condition | History | Sensitivity | Specificity |
|---|---|---|---|
| cancer | age ≥ 50 yrs | 0.77 | 0.71 |
| | previous Ca | 0.31 | 0.98 |
| | unexplained weight loss | 0.15 | 0.94 |
| | failure to improve after conservative therapy × 1 month | 0.31 | 0.90 |
| | any of the above | 1.00 | 0.60 |
| | pain > 1 month | 0.50 | 0.81 |
| spinal osteomyelitis | IV drug abuse, UTI, or skin infection | 0.40 | NA |
| compression fracture | age ≥ 50 yrs | 0.84 | 0.61 |
| | age ≥ 70yrs | 0.22 | 0.96 |
| | trauma | 0.30 | 0.85 |
| | steroid use | 0.06 | 0.995 |
| HLD | sciatica | 0.95 | 0.88 |
| spinal stenosis | pseudoclaudication | 0.60 | NA |
| | age ≥ 50 yrs | 0.90[a] | 0.70 |
| ankylosing spondylitis | positive response to 4 out of 5 of the following | 0.23 | 0.82 |
| | age at onset ≤ 40 yrs | 1.00 | 0.07 |
| | pain not relieved when supine | 0.80 | 0.49 |
| | AM back stiffness | 0.64 | 0.59 |
| | pain ≥ 3 mos duration | 0.71 | 0.54 |

[a]estimate



4. immunosuppression: from steroids, organ transplant medication, or HIV
5. prolonged use of steroids
6. duration of symptoms
7. responsiveness to previous therapy
8. pain that is worse at rest
9. history of skin infection: especially furuncle
10. history of IV drug abuse
11. UTI or other infection
12. pain radiating below the knee
13. persistent numbness or weakness in the legs
14. history of significant trauma. In a young patient: usually involves MVA, a fall from a height, or a direct blow to the back. In an older patient: minor falls, heavy lifting or even a severe coughing episode can cause a fracture especially in the presence of with osteoporosis
15. findings consistent with cauda equina syndrome (p.1050):
    a) bladder dysfunction (usually urinary retention, or overflow incontinence) or fecal incontinence
    b) saddle anesthesia (p.1050)
    c) unilateral or bilateral leg weakness or pain
16. psychological and socioeconomic factors may influence the patient's report of symptoms (p.1033), and one should inquire about:
    a) work status
    b) typical job tasks
    c) educational level

d) pending litigation
e) worker's compensation or disability issues
f) failed previous treatments
g) substance abuse
h) depression

### 68.8.3 Physical examination

Less helpful than the history in identifying patients who may be harboring conditions such as cancer, but may be more helpful in detecting spinal infections.

1. spinal infection (p.349): findings that suggest this as a possibility (but are also common in patients without infection)
   a) fever: common in epidural abscess and vertebral osteomyelitis, less common in discitis
   b) vertebral tenderness
   c) very limited range of spinal motion
2. findings of possible neurologic compromise: the following physical findings will identify most cases of clinically significant nerve root compromise due to L4–5 or L5-S1 HLD which comprise > 90% of cases of radiculopathy due to HLD; limiting the exam to the following might not detect the much less common upper lumbar disc herniations, which may be difficult to detect on PE (p.1057)



   a) dorsiflexion strength of ankle and great toe: weakness suggests L5 and some L4 dysfunction
   b) achilles reflex: diminished reflex suggests S1 root dysfunction
   c) light touch sensation of the foot:
      - diminished over medial malleolus and medial foot: suggests L4 nerve root involvement
      - diminished over dorsum of foot: suggests L5
      - diminished over lateral malleolus and lateral foot: suggests S1
   d) straight leg raising (SLR); also check for crossed SLR (p.1048)

### 68.8.4 "Red flags" in the history and physical exam for low back problems

Based upon the above history and physical exam, the findings in Table 68.5 would suggest the possibility of a serious underlying condition as the cause of the low back problem. Also, thoracic region pain is relatively uncommon and should raise the index of suspicion.

### 68.8.5 Special diagnostic tests

For patients without features suggesting a serious underlying condition, special diagnostic tests are not needed during the first month of symptoms. This covers approximately 95% of patients with low back problems.[1]

Table 68.5 "Red flags" for patients with low back problems

| Condition | Red flags |
|---|---|
| cancer or infection | 1. age >50 or <20 yrs<br>2. history of cancer<br>3. unexplained weight loss<br>4. immunosuppression (see text)<br>5. UTI, IV drug abuse, fever or chills<br>6. back pain not improved with rest |
| spinal fracture | 1. history of significant trauma (see text)<br>2. prolonged use of steroids<br>3. age >70 yrs |
| cauda equina syndrome or severe neurologic compromise | 1. acute onset of urinary retention or overflow incontinence<br>2. fecal incontinence or loss of anal sphincter tone<br>3. saddle anesthesia<br>4. global or progressive weakness in the LEs |

## 68.9 Radiographic evaluation

### 68.9.1 General information

Diagnosing lumbar spinal stenosis or herniated intervertebral disc is usually helpful only in potential surgical candidates.[15] This includes patients with appropriate clinical syndromes who have not responded satisfactorily to adequate non-surgical treatment over a sufficient period of time, and who have no medical contraindications to surgery. Radiologic confirmation of these diagnoses usually requires CT, myelography, MRI, or some combination (see below). NB: myelography,[16] CT,[17] or MRI[18] may also show bulging or herniated lumbar discs (HLD) or spinal stenosis in asymptomatic patients (e.g. 24% of asymptomatic patients have herniated discs on MRI and 4% have spinal stenosis; these numbers become 36% and 21% respectively in patients 60–80 years old).[19] Thus, these tests must be interpreted in light of clinical findings, and the anatomic level and side should correspond to the history, examination, and/or other physiologic data. Diagnostic radiology is of limited benefit as the initial evaluation in the majority of spinal disorders.[20]

In the absence of red flags for serious conditions, imaging studies are not recommended in the first month of symptoms.[1] For patients who have had previous back surgery, MRI with contrast is probably the best test. Myelography (with or without CT) is invasive and has increased risk of complications, and is therefore indicated only in situations where MRI cannot be done or is inadequate, and the possibility of surgery is anticipated.



**Σ**

Patients for whom radiographic imaging is recommended are those with:
1. suspected benign conditions with symptoms persisting >4 weeks of great enough severity to consider surgery, including:
   a) back related leg symptoms and clinically specific signs of nerve root compromise
   b) a history of neurogenic claudication (p. 1100) or other finding suggestive of lumbar spinal stenosis
   c) symptoms related to spinal deformity/imbalance, especially positional back pain that increases with time spent upright
2. red flags: physical examination or other test results suggesting other serious conditions affecting the spine (e.g. cauda equina syndrome, fracture, infection, tumor, or other mass lesions or defects)

Recommendations for use of MRI and discography to select patients for fusion are shown in the Practice Guideline (p. 1029).

**Practice guideline: MRI and discography for patient selection for lumbar fusion***

1. Level II[21]:
   a) MRI is recommended as the initial diagnostic test
   b) normal appearing discs on MRI should not be considered for discography or treatment
   c) lumbar discography should not be used as a stand-alone test
   d) to consider a disc level for treatment, if discography is used, there should be a concordant pain response[a] and associated abnormalities on MRI[b]
2. Level III[21]: discography should be reserved for equivocal MRI findings, especially at levels adjacent to unequivocally abnormal levels

Notes:
* see also recommendations on use of facet injections (p. 1036)
[a] concordant pain response: pain identical or very similar to the patient's usual pain complaints (NB: discography can produce severe LBP in patients with no prior complaints[22,23])
[b] abnormal disc morphology on MRI: loss of T2WI signal intensity ("black disc"), disc space collapse, Modic changes (see □ Table 68.2), and high-intensity zones (these findings also frequently occur in asymptomatic patients[24])

## 68.9.2 Plain lumbosacral x-rays

### General information

Unexpected findings occurred in only 1 in 2500 adults <50 years age.[25] Diagnosis of surgical conditions of disc herniation and spinal stenosis cannot be made from plain films (although they may be inferred, further study would be required). Various congenital abnormalities of uncertain significance may be identified (e.g. spina bifida occulta), and evidence of degenerative changes (including osteophytes) are as frequent in symptomatic as in asymptomatic patients. Gonadal radiation is significant. Seldom indicated during pregnancy.

### Recommendation

Not recommended for routine evaluation of patients with acute low back problems during the first month of symptoms unless a "red flag" is present (see below). Reserve LS x-rays for patients with a likelihood of having spinal malignancy, infection, inflammatory spondylitis, or clinically significant fracture. In these cases, plain x-rays are often just a starting point, and further study (CT, MRI...) may be indicated even if the plain x-rays are normal. "Red flags" for these conditions include the following:



- age > 70 years, or < 20 yrs
- systemically ill patients
- temp > 100°F (or > 38° C)
- history of malignancy
- recent infection
- patients with neurologic deficits suggesting possible cauda equina syndrome (saddle anesthesia, urinary incontinence or retention, LE weakness) (p. 1050)
- heavy alcohol or IV drug abusers
- diabetics
- immunosuppressed patients (including prolonged treatment with corticosteroids)
- recent urinary tract or spinal surgery
- recent trauma: any age with significant trauma, or > 50 yrs old with mild trauma
- unrelenting pain at rest
- persistent pain for more than ≈ 4 weeks
- unexplained weight loss

When spine x-rays are indicated, AP and lateral views are usually adequate.[26] Obliques and coned-down L5-S1 views more than double the radiation exposure, and add information in only 4–8% of cases,[27] and can be obtained in specific instances where warranted (e.g. to diagnose spondylolysis when spondylolisthesis is found on the lateral film).

## 68.9.3 MRI

Unless contraindicated, noncontrast MRI is the initial diagnostic test of choice for diagnosing most cases of disc herniation and spinal stenosis. Specificity and sensitivity for HLD are on the same order as CT/myelography, which is better than myelography alone.[1,28,29]

Advantages:

- Provides the most information about soft tissues (intervertebral discs, spinal cord, inflammation...) of any available diagnostic test
- provides information regarding tissue outside of the spinal canal, e.g. extreme lateral disc herniation (p. 1058), tumors...
- non-invasive and does not utilize ionizing radiation

Disadvantages:

- patients in severe pain or with claustrophobia may have difficulty holding still
- dose not visualize bone well
- poor for studying blood early (e.g. spinal epidural hematoma)
- expensive
- interpretation with scoliosis is more difficult, may be partially compensated by contouring visualization plane through center of canal
- a number of contraindications: see Contraindications to MRI (p. 230)

Findings:

In addition to demonstrating herniated lumbar disc (HLD) outside of the disc interspace compressing nerve root or thecal sac, MRI can demonstrate signal changes within the interspace suggestive of disc degeneration[30] (loss of signal intensity on T2WI, loss of disc space height) and is useful in diagnosing infections and tumors.

## 68.9.4 Lumbosacral CT

If technically adequate images can be obtained (e.g. good quality scanner, images not obscured by artifact from patient movement or obesity), CT can demonstrate most spine pathology. For HLD, sensitivity is 80–95% and specificity is 68–88%[31,32] However, even some large disc herniations will be missed with plain CT. CT studies for HLD tend to be less satisfactory in the elderly. When MRI is an option, the main utility of CT is for imaging bone to assess fractures or to demonstrate details of bony anatomy for surgery.

Disc material has density (Hounsfield units) ≈ twice that of the thecal sac. Associated findings with herniated disc include:

- loss of epidural fat (normally seen as low density in the anterolateral canal)
- loss of normal "convexity" of thecal sac (indentation by herniated disc)

Advantages:

- excellent bony detail
- non-invasive
- outpatient evaluation
- evaluates paraspinal soft tissue (e.g. to rule out tumor, paraspinal abscess…)
- advantages over MRI: faster scanning (significant in patients who have difficulty laying still for long time), less expensive, less claustrophobic, fewer contraindications, see Contraindications to MRI (p.230)



Disadvantages:

- involves ionizing radiation (x-rays)
- sensitivity is significantly lower than MRI or myelogram/CT

## 68.9.5 Myelography

With water soluble intrathecal contrast introduced via lumbar puncture, sensitivity (62–100%) and specificity (83–94%)[33,34,35,36] are similar to CT for detection of HLD. Usually combined with post-myelographic CT scan (myelogram/CT), which increases the sensitivity and especially the specificity.[37] A herniated disk in the large space between thecal sac and posterior border of vertebral bodies at L5-S1 (insensitive space) may not be seen on myelography alone (CT or MRI are usually better at demonstrating this).

Advantages:

1. evaluates cauda equina better than noncontrast CT
2. provides "functional" information about degree of stenosis (a high-degree block will allow flow of dye only after certain position changes)
3. when combined with CT, may demonstrate some anatomy obscured by metal artifact on MRI in patients with prior instrumentation

Disadvantages:

1. may miss pathology outside of the dura (including far laterally herniated disc), sensitivity is improved with post-myelographic CT
2. invasive
   a) drugs e.g. warfarin must be stopped, and sometimes bridged to heparin
   b) with occasional side effects (post LP H/A, N/V, rare seizures)
3. iodine allergic patients
   a) requires iodine allergy prep
   b) may still be risky (especially in severely iodine allergic patients)

Findings:

HLD produces extradural filling defect at the level of the intervertebral disc. Massive disc herniation or severe lumbar stenosis may produce a total or near total block. In some cases of HLD, the finding may be very subtle and may consist of a cut-off of the filling (with contrast) of the nerve root sleeve (compared to normal nerve(s) on contralateral side or at other levels). Another subtle finding may be a "dual shadow" on lateral view.

### 68.9.6 Bone scan

See Bone scan for low back problems (p.1032).

### 68.9.7 Discography

Injection of water-soluble contrast agent directly into the nucleus pulposus of the intervertebral disc being studied by percutaneous needle access through Kambin's triangle in an awake patient. Results of the test depend on volume of dye accepted into the disc, the pressure needed to inject the dye, the configuration of the dye (including leakage from the confines of the disc space) on radiographic imaging (plain x-rays produce the so-called "discogram", CT scan is often also be utilized), and reproduction of the patient's pain on injection. Some of the basis for performing a discogram is to identify levels that may produce "discogenic pain" or "painful disc syndrome" (p.1032), a controversial point. When the pain produced mimics the patient's presenting pain, the pain is said to be "concordant."



Critique:

Invasive. Interpretation is equivocal, and complications may occur (disc space infection, disc herniation, and significant radiation exposure with CT-discography). May be abnormal in asymptomatic patients[22,23] (as any of the above tests may be) although the false positive rate may not be quite this high.[38] See Practice guideline: MRI and discography for patient selection for lumbar fusion (p.1029) for recommendations.

## 68.10 Electrodiagnostics for low back problems

If the diagnosis of radiculopathy seems likely on clinical grounds, electrophysiologic testing is not recommended.[1]

1. needle EMG (p.242): can assess acute and chronic nerve root dysfunction, myelopathy and myopathy, and may be useful for patients with suspicion of other conditions (e.g. neuropathy) or when a reliable strength exam is not possible. Reduced recruitment may be seen within the first several days of onset, however, spontaneous activity takes 10–21 days to develop (p.242) ( less helpful in the first ≈ 3 weeks). Also, not usually helpful with normal muscle strength exam. Accuracy is highly operator dependent and improves with knowledge about imaging studies and clinical information.[39] See findings in radiculopathy (p.243).
2. H-reflex (p.243): measures sensory conduction through nerve roots. Use is limited to assessing S1 radiculopathy.[40] Correlates with achilles reflex.
3. SSEPs (p.239): assesses afferent fibers which travel in peripheral nerve and the posterior column of the spinal cord. May be abnormal in conditions affecting the dorsal columns with impaired joint position and proprioception (e.g. cervical spondylotic spinal myelopathy)
4. nerve conduction studies (including NCVs): helps identify acute and chronic entrapment neuropathies that may mimic radiculopathy
5.  not recommended for assessing acute low back problems[1]
   a) F-wave response (p.243): measures motor conduction through nerve roots, used to assess proximal neuropathies
   b) surface EMG: assesses acute and chronic recruitment patterns during static or dynamic tasks using surface (instead of needle) electrodes

## 68.11 Bone scan for low back problems

Description: injection of a radiolabeled compound (usually technetium-99m) that is taken up by metabolically active bone. A gamma camera localizes regions of uptake. Total radiation dose is ≈ to a set of lumbar spine x-rays.[1] Contraindicated during pregnancy. Breast feeding must be briefly suspended following a bone scan due to presence of radiotracer in the breast milk.

A moderately sensitive test which may be used in evaluating low back pain when spinal tumor,[41] infection,[42] or occult fracture is suspected from "red flags" (see  Table 68.5) on history or examination, or results of lab tests or plain x-rays. Not very specific, but may locate occult lesions and help

differentiate these conditions from degenerative changes. A positive bone scan suggesting one of these conditions usually must be confirmed by other diagnostic tests or procedures (no studies have compared bone scans to CT or MRI).

Low yield in patients with longstanding low back problems and normal plain x-rays and laboratory tests (especially ESR or CRP).[41]

SPECT scans may provide additional information to a bone scan.

## 68.12 Thermography for low back problems

□ Not recommended.[1] Did not accurately predict absence or presence of nerve root compression seen at surgery,[43] and may be positive in a significant percentage of asymptomatic patients.[44]

## 68.13 Psychosocial factors

Although some patients with chronic LPB (>3 months duration) may have started off with a diagnosable condition, psychological and socioeconomic factors (such as depression, secondary gain…) may come to play a significant role in perpetuating or amplifying pain. Psychological factors, especially elevated hysteria or hypochondriasis scales on the Minnesota Multiphasic Personality Inventory (MMPI) were found to be a better predictor of outcome than findings on radiographic imaging in one study.[39] A screening scale of 5 factors has been proposed[45] (positive findings in any 3 suggests psychological distress):

1. These items are potentially reliable[46]
   a) pain on simulated axial loading: press on top of head
   b) inconsistent performance: e.g. difficulty tolerating straight leg raising (SLR) while supine, but no difficulty when sitting
   c) overreaction during the physical exam
2. These items may not be reliable[46]
   a) inappropriate tenderness that is superficial or widespread
   b) motor or sensory abnormalities not corresponding to anatomic boundaries (e.g. for sensation: dermatomes, peripheral nerve distribution…)



However, the usefulness of this information is limited, and no effective interventions have been identified to address these factors. Therefore the AHCPR panel was unable to recommend specific assessment tools or interventions.[1]

## 68.14 Treatment

### 68.14.1 General information

An initial period of nonsurgical management (below) is indicated except in the following circumstances where urgent surgery is indicated:

Situations where conservative treatment is not indicated:

- symptoms of cauda equina syndrome: urinary retention, saddle anesthesia... (p.1050)
- progressive neurologic deficit, or profound motor weakness
- a relative indication for proceeding to urgent surgery without conservative management is severe pain that cannot be sufficiently controlled with adequate pain medication (rare)

If specific diagnoses such as herniated intervertebral lumbar disc or symptomatic lumbar stenosis are made, surgical treatment for these conditions may be considered if the patient fails to improve satisfactorily. In cases where no specific diagnosis can be made, management consists of conservative treatment and following the patient to rule out the possible development of symptoms suggestive of a more serious diagnosis that may not have initially been evident.

### 68.14.2 "Conservative" treatment

This term has regrettably come to be used for non-surgical management. With minor modification, similar approaches can be used for mechanical low back pain, as well as for acute radiculopathy from disc herniation.

Recommendations (based on AHCPR findings[1] in the absence of "red flags"; note: some key literature citations are given here, primarily those from the better studies that support the Agency for

Health Care Policy and Research [AHCPR] panel recommendations. However, refer to Bigos et al.[1] for full analysis and list of references):

1. activity modifications: no studies were found that met the panels review criteria for adequate evidence. However, the following information was felt to be useful:
   a) bed rest: for 2–3 days maximum
      - the theoretical objective is to reduce symptoms by reducing pressure on the nerve roots and/or intradiscal pressures which is lowest in the supine semi-Fowler position,[47] and also to reduce movements which are experienced as painful by the patient
      - deactivation from prolonged bed rest (>4 days) appears to be worse for patients (producing weakness, stiffness, and increased pain) than a gradual return to normal activities[48]
      - recommendations: the majority of patients with low back problems will not require bed rest. Bed rest for 2–4 days may be an option for those with severe initial radicular symptoms, however, this may be no better than watchful waiting[49] and may be harmful[50]
   b) activity modification
      - the goal is to achieve a tolerable level of discomfort while continuing sufficient physical activity to minimize disruption of daily activities
      - risk factors: although there is not agreement on their exact role, the following were identified as having an increased incidence of low back problems. Jobs requiring heavy or repetitive lifting, total body vibration (from vehicles or industrial machinery), asymmetric postures, or postures sustained for long periods (including prolonged sitting)
      - recommendations: temporarily limit heavy lifting, prolonged sitting, and bending or twisting of the back. Establish activity goals to help focus attention on expected return to full functional status
   c) exercise (may be part of a physical therapy program):
      - during the 1st month of symptoms, low-stress aerobic exercise can minimize debility due to inactivity. In the first 2 weeks, utilize exercises that minimally stress the back: walking, bicycling, or swimming
      - conditioning exercises for trunk muscles (especially back extensors, and possibly abdominal muscles) are helpful if symptoms persist (during the first 2 weeks, these exercises may aggravate symptoms)
      - there is no evidence to support stretching of back muscles, or to recommend back-specific exercise machines over traditional exercise
      - recommended exercise quotas that are gradually escalated results in better outcome than having patients simply stop when pain occurs[51]
2. analgesics:
   a) for the initial short-term period, acetaminophen (APAP) or NSAIDs (p.137) may be used. In one study[52] of acute LBP, NSAIDs did not add any benefit to APAP+standard education (see below)
   b) stronger analgesics – mostly opioids (p.138) – may be required for severe pain, usually severe radicular pain. For non-specific back pain, there was no earlier return to full activity than with NSAIDs or APAP.[1] Opioids should not be used >2–3 weeks, at which time NSAIDs should be instituted unless contraindicated
3. muscle relaxants
   a) the therapeutic objective is to reduce pain by relieving muscle spasm. However, muscle spasms have not been proven to cause pain, and the most commonly used muscle relaxants have no peripheral effect on muscle spasm
   b) probably more effective than placebo, but have not been shown to be more effective than NSAIDs, and their use in combination with NSAIDs has not been shown to be more effective than use of NSAIDs alone
   c) potential for side effects: drowsiness (in up to 30%). Most manufacturers recommend use for < 2–3 weeks. Agents such as chlorzoxazone (Parafon Forte® and others) may be associated with risk of serious and potentially fatal hepatotoxicity[53]
4. education: (may be provided as part of a physical therapy program)
   a) explanation of the condition to the patient[54] in understandable terms, and positive reassurance that the condition will almost certainly subside[55] have been shown to be more effective than many other forms of treatment
   b) proper posture, sleeping positions, lifting techniques… should be conveyed to the patient. Formal "back school" seems to be marginally effective.[56] There may be some early benefit, but long-term efficacy could not be shown.[57] The quality and expense of such programs varies widely[1]
5. spinal manipulation therapy (SMT): defined as manual therapy in which loads are applied to the spine using long or short lever methods with the selected joint being taken to its end range of

voluntary motion, followed by application of an impulse loading (may be part of a physical therapy program)
   a) may be helpful for patients with acute low back problems without radiculopathy when used in the first month of symptoms (efficacy after 1 month is unproven) for a period not to exceed 1 month. One study[52] found no added benefit to APA+standard education
   b) there is insufficient evidence to recommend SMT in the presence of radiculopathy
   c) SMT should not be used in the face of severe or progressive neurologic deficit until serious conditions have been ruled out
   d) □ reports of arterial dissection: especially vertebral artery (p.1325) and stroke, myelopathy & subdural hematoma with cervical SMT and cauda equina syndrome with lumbar SMT[58,59,60] and the uncertainty of benefits have led to the questioning of the use of SMT[58] (especially cervical)
6. epidural injections:
   a) epidural (cortico)steroid injections (ESI): there is no evidence that this is effective in treating acute radiculopathy.[61] Most studies that show benefit are retrospective and noncontrolled. Prospective studies yield varied results.[62] Some improvement at 3 & 6 weeks may occur (but no functional benefit, and no change in the need for surgery), with no benefit at 3 months.[63] The response in chronic back pain is poor in comparison to acute pain. ESI may be an option for short-term relief of radicular pain when control on oral medications is inadequate or for patients who are not surgical candidates
   b) there is no evidence to support the use of epidural injections of steroids, local anesthetics and/or opioids for LBP without radiculopathy
   c) reports on efficacy with conditions such as lumbar spinal stenosis are conflicting,[62] relief is almost uniformly temporary (4–6 weeks with initial injection, shorter times with subsequent ones)



□ Not recommended by the AHCPR panel[1] for treatment of acute low back problems in the absence of "red flags" (□ Table 68.5):
1. medications
   a) oral steroids: no difference was found at one week and 1 year after randomization to receive 1 week therapy with oral dexamethasone or placebo[64]
   b) colchicine: conflicting evidence shows either some[65] or no[66] therapeutic benefit. Side effects of N/V and diarrhea were common[1]
   c) antidepressant medications: most studies of these medications were for chronic back pain. Some methodologically flawed studies failed to show benefits when compared to placebo for chronic (not acute) LBP[67]
2. physical treatments
   a) TENS (transcutaneous electrical nerve stimulation): not statistically significantly better than placebo, and added no benefit to exercise alone[68]
   b) traction (including pelvic traction): not demonstrated to be effective.[69] One possible explanation for lack of benefit is that due to the sizable paraspinal muscles and ligaments (as compared to the cervical spine) the amount of weight required to distract the intervertebral disc space is approximately ≥2/3 of the patient's body weight, which is painful and/or pulls the patient to the foot of the bed
   c) physical agents and modalities: including heat (including diathermy), ice, ultrasound. Benefit is insufficiently proven to justify their cost, however, self-administered home programs for application of heat or cold may be considered. Ultrasound and diathermy should not be used in pregnancy
   d) lumbar corsets and support belts: not proven beneficial for acute back problems. Prophylactic use has been advocated to reduce time lost from work by individuals doing frequent lifting as part of their job, but this is controversial[70]
   e) biofeedback: has not been studied for acute back problems. Primarily advocated for chronic LBP, where effectiveness is controversial[71]
3. injection therapy
   a) trigger point and ligamentous injections: the theory that trigger points cause or perpetuate LBP is controversial and disputed by many experts. Injections of local anesthetic are of equivocal efficacy (saline may be as effective[72]) and are mildly invasive
   b) (zygapophyseal) facet joint injections: theoretical basis is that there exists a "facet syndrome" producing LBP which is aggravated by spine extension, with no nerve root tension signs (p.1047). No studies have adequately investigated injections for pain <3 months duration. For chronic LBP, neither the agent nor the location (intrafacet or pericapsular) made a significant difference in outcomes[73,74]

c) epidural injections in the absence of radiculopathy: see above
d) acupuncture: no studies were found that evaluated the use in acute back problems. All randomized clinical trials found were for patients with chronic LBP, and even the best studies were felt to be mediocre and contradictory. Meta-analysis found acupuncture was more effective in relieving chronic LBP than sham or no treatment,[75] but there was no comparison to other therapies

## Practice guideline: Injection therapy for low-back pain

### Therapeutic recommendations

Level III[76]: lumbar epidural injections or trigger point injections are not recommended for long-term relief of chronic LBP. These techniques or facet injections may be used to provide temporary relief in select patients

### Diagnostic recommendations

Level III[76]: lumbar facet injections

- may predict the response to radiofrequency facet ablation
- □ not recommended as a diagnostic tool to predict the response to lumbar fusion



## 68.14.3 Surgical treatment

### Indications for surgery for herniated lumbar disc

See the section on herniated lumbar discs (p.1049).

### Indications for fusion for chronic LBP without stenosis or spondylolisthesis

Very controversial.

## Practice guideline: Lumbar fusion for LBP without stenosis or spondylolisthesis

Level I[77]: lumbar fusion is recommended for carefully selected patients with disabling LBP due to one- or two-level degenerative disease without stenosis or spondylolisthesis (in the primary quoted study[78] patients had chronic LBP for ≥ 2 years and had radiologic evidence of disc degeneration at L4-L5, L5-S1, or both, and had failed best medical management)

Level III[77,79]: an intensive course of PT and cognitive therapy is recommended as an option for patients with LBP in whom conventional medical management has failed

## Practice guideline: Choice of fusion technique

Level II[80]: for ALIF or ALIF + instrumentation, the addition of a posterolateral fusion is not recommended (the demonstrated benefit does not outweigh the additional time and blood loss involved)

Level III[80]:

- either a posterolateral fusion or an interbody fusion (PLIF, TLIF or ALIF) are options for patients with LBP due to DDD at 1 or 2 levels
- an interbody graft is an option to improve fusion rates and functional outcome (caution: the improvement in fusion rate and outcome is marginal, and interbody fusion is associated with an increased complication rate, especially with combined approaches, e.g. 360° fusion)

□ the use of multiple approaches (anterior + posterior) is not recommended as a routine option for LBP without deformity

## Surgical treatment options

The type of surgical procedure chosen is tailored to the specific condition identified. Examples are shown in □ Table 68.6. Discussion of some options is also provided below.

### Lumbar spinal fusion

Although there is no consensus on the indications,[81] lumbar spinal fusion (LSF) is accepted treatment for fracture/dislocation or instability resulting from tumor or infection.

For degenerative spine disease, practice parameters have been developed and are included herein. Pain associated with Modic type 1 changes (see □ Table 68.2) may respond to stabilization procedures, the other Modic types do not exhibit this association.

#### Practice guideline: Lumbar fusion for disc herniation

Level III[82]:

1. lumbar fusion is not routinely recommended following disc excision in patients with HLD or 1st time recurrent HLD causing radiculopathy
2. lumbar fusion is a potential adjunct to disc excision in cases of a HLD or recurrent HLD:
   a) with evidence of preoperative lumbar spinal deformity or instability
   b) in patients with chronic axial LBP associated with radiculopathy



### Instrumentation as an adjunct to fusion

#### Practice guideline: Pedicle screw fixation

Level III[83]: pedicle screw fixation is recommended as a treatment option for patients with LBP treated with posterolateral fusion who are at high risk for fusion failure (routine use of pedicle screws is discouraged because of conflicting evidence of benefit, together with considerable evidence of increased cost and complications)

The use of instrumentation increases the fusion rate.[84] Hardware used in the absence of fusion will eventually fatigue, especially in the region of the lumbar lordosis. Therefore, instrumentation must be viewed as a temporary internal stabilizing measure while awaiting the fusion process to complete.

## 68.15 Chronic low back pain

Rarely can an anatomic diagnosis be made in patients with chronic LBP ≥3 months duration.[85] Also, see Psychosocial factors (p.1033). Patients with chronic pain syndromes (CPS) refer to their problems with affective or emotional terms with a higher frequency than those with acute pain.[86] The

Table 68.6 Surgical options for low back problems

| Condition | Surgical treatment options |
|---|---|
| "routine" HLD or initial recurrence of HLD | • standard discectomy and microdiscectomy are of similar efficacy<br>• □ intradiscal procedures: nucleotome, laser disc decompression are not recommended (p.1052) |
| foraminal or far lateral HLD | • partial or total facetectomy (p.1059)<br>• extracanal approach (p.1059)<br>• endoscopic techniques |
| lumbar spinal stenosis | • simple decompressive laminectomy<br>• laminectomy plus fusion: may be indicated for patients with degenerative spondylolisthesis, stenosis and radiculopathy, adult degenerative scoliosis (ADS), or instability |

Table 68.7 Chances of patients going back to work

| Time out of work | Chances of getting back to work |
|---|---|
| <6 mos | 50% |
| 1 yr | 20% |
| 2 yrs | <5% |

amount of time that a patient has been out of work due to low back problems is related to the chances of the patient getting back to work as shown in Table 68.7.

## 68.16 Coccydynia

### 68.16.1 General information

Pain and tenderness around the coccyx. A symptom, not a diagnosis. Typically, discomfort is experienced on sitting or on rising from sitting. More common in females, possibly due to a more prominent coccyx. The condition is unusual enough in males that in the absence of local trauma, strong consideration should be given to an underlying condition.



### 68.16.2 Etiologies

For differential diagnosis, see Acute low back pain (p.1416). Better accepted etiologies include[87]:

1. local trauma (may be associated with fracture or dislocation):
   a) 25% of patients give a history of a fall
   b) 12% had repetitive trauma (rowing machine, prolonged bicycle riding...)
   c) 12% started with parturition
   d) 5% started following a surgical procedure (half of which were in the lithotomy position)
2. idiopathic: excluding traumatic cases, no etiology can be identified in most cases
3. neoplasms
   a) chordoma
   b) giant cell tumor
   c) intradural schwannoma
   d) perineural cyst
   e) intra-osseous lipoma
   f) carcinoma of the rectum
   g) sacral hemangioma[88]
   h) pelvic metastases (e.g. from prostate cancer)
4. prostatitis

Controversial etiologies include[87,89]:

1. local pressure over a prominent coccyx
2. referred pain:
   a) spinal disease
      - herniated lumbosacral disc
      - cauda equina syndrome
      - arachnoiditis
   b) pelvic/visceral disease
      - pelvic inflammatory disease (PID)
      - perirectal abscess
      - perirectal fistula
      - pilonidal cyst
3. inflammation of the various ligaments attached to the coccyx
4. neurosis or frank hysteria

Histological evaluation of the coccyx has not helped delineate the cause, even though avascular necrosis has been suggested.[90]

### 68.16.3 Evaluation

MRI: effective for detecting soft tissue masses, including presacral masses

CT scan: no characteristic finding in coccydynia findings. Very sensitive for detecting bony pathology (fracture, destructive lesion…).

Sacrococcygeal films are often performed to rule-out a bony destructive lesion. Often, the question of a fracture will be raised, and many times cannot be definitely ruled-in or out based on this study. There may or may not be any significance of such a fracture.

Nuclear bone scans were not helpful in 50 patients with coccydynia.[87]

### 68.16.4 Treatment

Numerous treatments have been proposed, and some are offered here for historical purposes[87] (and to dissuade casual attempts to effect a "new" cure that in reality has already been tried):

1. plaster jackets
2. hot baths (sitz baths), heating pads
3. massage therapy
4. XRT
5. psychotherapy

Most cases resolve within ≈ 3 months of conservative management consisting of NSAIDs, mild analgesics, and measures to reduce pressure on the coccyx (e.g. a rubber ring ("doughnut") sitting cushion, lumbar supports to maintain sitting lumbar lordosis to shift weight from coccyx to posterior thighs).[91]

Management recommendations for refractory cases[87,91]

1. local injection: 60% respond to corticosteroid + local anesthetic (40 mg Depo-Medrol® in 10 cc of 0.25% bipuvicaine). Recommended as initial treatment; response should be achieved by 2 injections
2. manipulation of the coccyx: usually under general anesthesia. ≈ 85% successful when combined with local injection
3. ± physiotherapy (diathermy & ultrasound): found to be of benefit only in ≈ 16% (may be more effective with the addition of gentle manipulation of the coccyx without general anesthesia[92])
4. caudal epidural steroid injection
5. blockade or neurolysis (with chemicals or by cryoablation[93]) of the ganglion impar (AKA ganglion of Walther, the lowest ganglion of the paired paravertebral sympathetic chain, located just anterior to the sacrococcygeal junction): some success has been described with this technique (traditionally used for intractable sympathetic perineal pain of neoplastic etiology[94])
6. neurolytic techniques directed to S4, S5 and coccygeal nerves
7. coccygectomy (surgical removal of the mobile portion of the coccyx, followed by smoothening of the residual bony prominence on the sacrum): was required in ≈ 20% of patients in one series,[87] with a reported success rate of 90% However, many practitioners do not view this as a highly effective treatment and feel that great restraint should be used in considering this form of therapy

### 68.16.5 Recurrence

Occurs in ≈ 20% of conservatively treated cases, usually within the first year. Repeat therapy was often successful in providing permanent relief. More aggressive treatment may be considered for refractory cases.

## 68.17 Failed back surgery syndrome

### 68.17.1 General information

Definition: failure to satisfactorily improve low back pain or radiculopathy following back surgery. These patients often require analgesics and are unable to return to work. The failure rate for lumbar discectomy to provide satisfactory long-term pain relief is ≈ 8–25%[95] Pending legal or worker's compensation claims were the most frequent deterrents to a good outcome.[96]

## 68.17.2 Etiologies

Factors that may cause or contribute to the failed back syndrome:

1. incorrect initial diagnosis
   a) inadequate pre-op imaging
   b) clinical findings not correlated with abnormality demonstrated on imaging
   c) other causes of symptoms (sometimes in the presence of what was considered to be an appropriate lesion on imaging studies which may have been asymptomatic): e.g. trochanteric bursitis, diabetic amyotrophy…
2. continued nerve root or cauda equina compression caused by:
   a) residual compression (retained disc material, osteophytes…)
   b) recurrent pathology at same level: disc reherniation at the same level, usually have pain-free interval >6 mos post-op (p.1061); or restenosis (over many years[97] – was more common with midline fusions)
   c) adjacent level pathology: disc herniation or stenosis[97]
   d) compression of nerve root by peridural scar (granulation) tissue (see below)
   e) pseudomeningocele
   f) epidural hematoma
   g) conjoined nerve roots with compression at another level or in atypical location
   h) segmental instability: 3 patterns,[98] 1) lateral rotational instability, 2) post-op spondylolisthesis, 3) post-op scoliosis
3. permanent nerve root injury from the original disc herniation or from surgery, includes deafferentation pain which is usually constant and burning or ice cold
4. adhesive arachnoiditis: responsible for 6–16%of persistent symptoms in post-op patients[99] (see below)
5. discitis (p.356): usually produces exquisite back pain 2–4 weeks post-op
6. spondylosis
7. other causes of back pain unrelated to the original condition: paraspinal muscle spasm, myofascial syndrome… Look for trigger points, evidence of spasm
8. post-op reflex sympathetic dystrophy, RSD (p.1054)
9. "non-anatomic factors": poor patient motivation, secondary gains, drug addiction, psychological problems (p.1053)…

## 68.17.3 Arachnoiditis (AKA adhesive arachnoiditis)

### General information

Inflammatory condition of the lumbar nerve roots. Actually a misnomer, since adhesive arachnoiditis is really an inflammatory process or fibrosis that involves all three meningeal layers (pia, arachnoid, and dura).

### Etiologies/risk factors

Many putative "risk factors" have been described for the development of arachnoiditis, including[100]:

1. spinal anesthesia: either due to the anesthetic agents or to detergent contaminants on the syringes used for same
2. spinal meningitis: pyogenic, syphilitic, tuberculous
3. neoplasms
4. myelographic contrast agents: less common with currently available nonionic water soluble contrast agents
5. trauma
   a) post-surgical: especially after multiple operations
   b) external trauma
6. hemorrhage
7. idiopathic

### Radiographic findings in arachnoiditis

NB: Radiographic evidence of arachnoiditis may also be found in asymptomatic patients.[100] Arachnoiditis must be differentiated from tumor: the central adhesive type (see below) may resemble CSF seeding of tumor, and myelographic block may mimic intrathecal tumor.

Table 68.8 Myelographic classification of arachnoiditis

| Type | Description |
|---|---|
| 1 | unilateral focal filling defect centered on the nerve root sleeve adjacent to disc space |
| 2 | circumferential constriction around thecal sac |
| 3 | complete obstruction with "stalactites" or "candle guttering", "candle-dripping", or "paint-brush" filling defects |
| 4 | infundibular cul-de-sac with loss of radicular striations |

### MRI

3 patterns on MRI[101,102]:

1. central adhesion of the nerve roots into 1 or 2 central "cords"
2. "empty thecal sac" pattern: roots adhere to meninges around periphery, only CSF signal is visible intrathecally
3. thecal sac filled with inflammatory tissue, no CSF signal. Corresponds with myelographic block and candle-dripping appearance

Enhancement: acute arachnoiditis may enhance. Chronic arachnoiditis usually does not enhance with gadolinium as much as e.g. tumor.



### Myelogram

May demonstrate complete block, or clumping of nerve roots. One of many myelographic classification systems[103] for arachnoiditis is shown in Table 68.8.

## 68.17.4 Peridural scar

### General information

Although peridural scar tissue is frequently blamed for causing recurrent symptoms,[104,105] there has been no proof of correlation between the two.[106] Peridural fibrosis is an inevitable sequelae to lumbar disc surgery just as post-op fibrosis is a consequence of any surgical procedure. Even patients who are relieved of their pain following discectomy develop some scar tissue post-op.[107] Although it has been shown that if a patient has recurrent radicular pain following a lumbar discectomy there is a 70% chance that extensive peridural scar will be found on MRI,[106] this study also showed that on post-op MRIs at 6 months, 43% of patients will have extensive scar, but 84% of the time this will be asymptomatic.[108] Thus, one must use clinical grounds to determine if a patient with extensive scar on MRI is in the 16% minority of patients with radicular symptoms attributable to scar.[108]

See a discussion of measures to reduce peridural scarring (p. 1053).

### Radiologic evaluation

#### General information

Patients with only persistent low back or hip pain without a strong radicular component, with a neurologic exam that is normal or unchanged from pre-op, should be treated symptomatically. Patients with signs or symptoms of recurrent radiculopathy (positive SLR is a sensitive test for nerve root compression), especially if these follow a period of apparent recovery, should undergo further evaluation.

It is critical to differentiate residual/recurrent disc herniation from scar tissue and adhesive arachnoiditis as surgical treatment has generally poor results with the latter two (below).

#### MRI without and with IV gadolinium

Diagnostic test of choice. The best exam for detecting residual or recurrent disc herniation, and to reliably differentiate disc from scar tissue. Pre-contrast studies with T1WI and T2WI yields an accuracy of ≈ 83%, comparable to IV enhanced CT.[109,110] With the addition of gadolinium, using the protocol below yields 100% sensitivity, 71% specificity, and 89% accuracy.[111] May also detect adhesive arachnoiditis (see above). As scar becomes more fibrotic and calcified with time, the differential enhancement with respect to disc material attenuates and may become undetectable at some point, ≈ 1–2 years post-op[110] (some scar continues to enhance for > 20 yrs).

### Recommended MRI protocol

See reference.[111]

Get pre-contrast T1WI and T2WI. Give 0.1 mmol/kg gadolinium IV. Obtain T1WI images within 10 minutes (early post-contrast). No benefit from post-contrast T2WI.

### Findings on unenhanced MRI

Signal from a HLD becomes more intense as the sequence is varied from T1WI → T2WI, whereas scar tissue becomes less intense with this transition. Indirect signs (also applicable to CT):

1. mass effect: a nerve root is displaced away from disc material, whereas it may be retracted toward scar tissue by adherence to it
2. location: disc material tends to be in contiguity with the disc interspace (best seen on sagittal MRI)

### Findings on enhanced MRI

On early (≤10 mins post-contrast) T1WI images: scar enhances inhomogeneously, whereas disc does not enhance at all. A nonenhancing central area surrounded by irregular enhancing material probably represents disc wrapped in scar. Venous plexus also enhances, and may be more pronounced when it is distorted by disc material, but the morphology is easily differentiated from scar tissue in these cases.

On late (>30 mins post-contrast) T1WI: scar enhances homogeneously, disc had variable or no enhancement. Normal nerve roots do not enhance even on late images.



#### CT scan without and with IV (iodinated) contrast

Unenhanced CT scan density measurements are unreliable in the postoperative back.[112] Enhanced CT is only fairly good in differentiating scar (enhancing) from disc (unenhancing with possible rim enhancement). Accuracy is about equal to unenhanced MRI.

#### Myelography, with post-myelographic CT

Postoperative myelographic criteria alone are unreliable for distinguishing disc material from scar.[100,113] With the addition of CT scan, neural compression is clearly demonstrated, but scar still cannot be reliably distinguished from disc.

Myelography (especially with post-myelographic CT) is very capable of demonstrating arachnoiditis[113] (see above).

#### Plain LS x-rays

Generally helpful only in cases of instability, malalignment, or spondylosis.[113] Flexion/extension views are most helpful when trying to demonstrate instability.

## 68.17.5 Treatment of failed back surgery syndrome

### Post-operative discitis

For treatment of intervertebral disc-space infection, see Discitis (p.356).

### Symptomatic treatment

Recommended for patients who do not have radicular signs and symptoms, or for most patients demonstrated to have scar tissue or adhesive arachnoiditis on imaging. As in other cases of non-specific LBP treatment includes: short-term bed rest, analgesics (non-narcotic in most cases), anti-inflammatory medication (non-steroidal, and occasionally a short course of steroids), and physical therapy.

### Surgery

Generally reserved for those with recurrent or residual disc herniation, segmental instability, or patients with a pseudomeningocele. Patients with post-op spinal instability should be considered for spinal fusion (p.1037).[98]

In most series with sufficient follow-up, success rates after reoperation are lower in patients with only epidural scar (as low as 1%) compared to those patients with disc and scar (still only ≈ 37%).[95]

An overall success rate (>50% pain relief for >2 yrs) of ≈ 34% was seen in one series,[105] with better results in patients that were young, female, with good results following previous surgery, a small number of previous operations, employment prior to surgery, predominantly radicular (cf axial) pain, and absence of scar requiring lysis.

In addition to the absence of disc material, factors associated with poor outcome were: sensory loss involving more than one dermatome, and patients with past or pending compensation claims.[95,114]

Arachnoiditis:

Surgery for carefully selected patients with arachnoiditis (those with mild radiographic involvement (Types 1 & 2 in □ Table 68.8), and <3 previous back operations)[103] has met with moderate success (although in this series, no patient returned to work). Approximate success rate in other series[115,116]: 50% failure, 20% able to work but with symptoms, 10–19% with no symptoms. Surgery consists of removal of extradural scar enveloping the thecal sac, removing any herniated disc fragments, and performing foraminotomies when indicated. Intradural lysis of adhesions is not indicated since no means for preventing reformation of scar has been identified.[116]

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# 69 Lumbar and Thoracic Intervertebral Disk Herniation / Radiculopathy

## 69.1 Lumbar disc herniation and lumbar radiculopathy

### 69.1.1 General information

**Key concepts**

- Radiculopathy: pain and/or subjective sensory changes (numbness, tingling…) in the distribution of a nerve root dermatome, possibly accompanied by weakness and reflex changes of muscles innervated by that nerve root
- typical disc herniation → radiculopathy in the nerve exiting at the level below
- massive disc herniations can → cauda equina syndrome (a medical emergency). Typical symptoms: saddle anesthesia, urinary retention, LE weakness (p. 1050).
- most patients do as well with conservative treatment as with surgery, □ initial nonsurgical (conservative) treatment should be attempted for the vast majority
- surgery indications: cauda equina syndrome, progressive symptoms or neurologic deficits despite conservative treatment, or severe radicular pain > ≈ 6 weeks



### 69.1.2 Pathophysiology

Intervertebral discs may undergo degenerative changes (p. 1096); see □ Table 68.1 for description: this includes desiccation and fibrosis which further leads to fissuring and tearing which in turn increases the risk of herniation of disc material outside the normal confines of the disc space (see same table for definitions).

Disc herniations may compress one or more nerve roots, producing lumbar radiculopathy or less frequently cauda equina syndrome.

### 69.1.3 Herniation zones

#### Central and paramedial disc herniations

The posterior longitudinal ligament is strongest in the midline, and the posterolateral annulus may bear a disproportionate portion of the load exerted by the weight from above. This may explain why most herniated lumbar discs (HLD) occur posteriorly, slightly off to one side within the central canal zone or in the subarticular zone as illustrated in □ Fig. 69.1. In the lumbar spine this characteristically compresses the nerve root en passage (that is, the nerve entering the lateral recess just before it exits through the neural foramen of the level below).

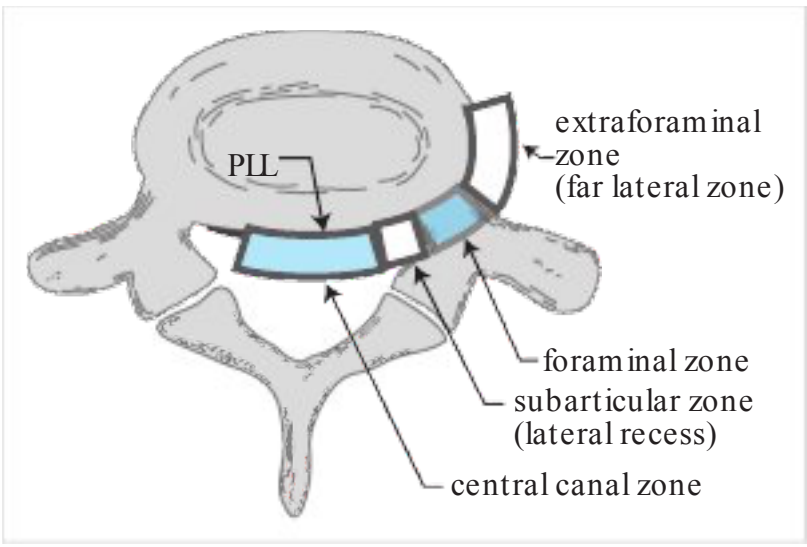


Fig. 69.1 Zones of lumbar disc herniation

### Extreme lateral disc herniation

Disc herniations may also occur in the foraminal zone, which typically involves the nerve root exiting at that level.

Disc herniations in the extraforaminal zone occasionally involve the nerve root exiting at that level, however disc herniation here and those herniating anterior to the spine may not result in any nerve root involvement.

## 69.1.4 Other disc herniation variants

1. intravertebral disc herniation: AKA Schmorl's node. See Herniation through the endplate into the vertebral body (p.1060)
2. intradural disc herniation (p.1060)
3. limbus fracture: traumatic separation of a segment of bone from the edge of the vertebral ring apophysis at the site of anular attachment. May accompany HLD

## 69.1.5 Characteristic findings on the history

- symptoms may start off with back pain, which after days or weeks gradually or sometimes suddenly yields to radicular pain often with reduction of the back pain
- precipitating factors: various factors are often blamed, but are rarely identified[1] with certainty
- pain relief upon flexing the knee and thigh (e.g. lying supine with a pillow under the knees)
- patients generally avoid excessive movements, however, remaining in any one position (sitting, standing, or lying) too long may also exacerbate the pain, sometimes necessitating position changes at intervals that range from every few minutes to 10–20 minutes. This is distinct from constant writhing in pain e.g. with ureteral obstruction
- "cough effect": ↑ pain with coughing, sneezing, or straining at the stool. Occurred in 87%of patients with HLD in one series[2]
- bladder symptoms: the incidence of voiding dysfunction is 1–18%[3 (p 966)] Most common: difficulty voiding, straining, or urinary retention. Reduced bladder sensation may be the earliest finding. The possible causes of this are sensory loss, or incomplete interruption of the preganglionic parasympathetic fibers. Later it is not unusual to see "irritative" symptoms including urinary urgency, frequency (including nocturia), increased post-void residual. Less common: enuresis, and dribbling incontinence[4]; NB: frank urinary retention may indicate cauda equina syndrome (p.1050). Occasionally a HLD may present only with bladder symptoms which may improve after surgery.[5] Discectomy may improve bladder function, but this cannot be assured



Back pain per se is usually a minor component (only 1%of patients with acute low back pain have sciatica[6]), and when it is the only presenting symptom, other causes should be sought; see Low back pain (p.1024). Sciatica has such a high sensitivity for disc herniation, that the likelihood of a clinically significant disc herniation[7] in the absence of sciatica is ≈ 1 in 1000. Exceptions include a central disc herniation which may cause symptoms of lumbar stenosis (i.e. neurogenic claudication) or a cauda equina syndrome.

## 69.1.6 Physical findings in radiculopathy

### General information

Nerve root impingement gives rise to a set of signs and symptoms present to variable degrees. Characteristic syndromes are described for the most common nerve roots involved; see Nerve root syndromes (p.1049).

In a series of patients referred to neurosurgical outpatient clinics for radiating leg pain, 28%had motor loss (yet only 12%listed motor weakness as a presenting complaint), 45%had sensory disturbance, and 51%had reflex changes.[8]

Findings suggestive of nerve root impingement include the following. □ Table 69.1 shows the sensitivity and specificity of some findings on the exam among patients with sciatica.

1. signs/symptoms of radiculopathy (□ Table 69.1)
   a) pain radiating down LE
   b) motor weakness
   c) dermatomal sensory changes
   d) reflex changes: mental factors may influence symmetry[9]
2. positive nerve root tension sign(s): including Lasègue's sign (see below)
3. tenderness over the sciatic notch

Table 69.1 Sensitivity and specificity of physical findings for HLD in patients with sciatica[10]

| Test | Comment | Sensitivity | Specificity |
|---|---|---|---|
| ipsilateral SLR | positive result: pain at <60° elevation | 0.80 | 0.40 |
| crossed SLR | reproduction of contralateral pain | 0.25 | 0.90 |
| ↓ ankle jerk | HLD usually at L5-S1 (total absence increases specificity) | 0.50 | 0.60 |
| sensory loss | area of loss is poor in localizing level of HLD | 0.50 | 0.50 |
| ↓ patellar reflex | suggests upper HLD | 0.50 | NA |
| Weakness | | | |
| knee extension (quadriceps) | HLD usually at L3–4 | <0.01 | 0.99 |
| ankle dorsiflexion (anterior tibialis) | HLD usually at L4–5 | 0.35 | 0.70 |
| ankle plantarflexion (gastrocs) | HLD usually at L5-S1 | 0.06 | 0.95 |
| great toe extension (EHL) | HLD at L5-S1 in 60%, at L4–5 in 30% | 0.50 | 0.70 |



## Nerve root tension signs

Includes[11]:

1. Lasègue's sign: AKA straight leg raising (SLR) test. Helps differentiate sciatica from pain due to hip pathology. Test: with patient supine, raise afflicted limb by the ankle until pain is elicited[12] (should occur at <60°, tension in nerve increases little above this angle). A positive test consists of leg pain or paresthesias in the distribution of pain (back pain alone does not qualify). The patient may also extend the hip (by lifting it off table) to reduce the angle. Although not part of Lasègue's sign, ankle dorsiflexion with SLR usually augments pain due to nerve root compression. SLR primarily tenses L5 and S1, L4 less so, and more proximal roots very little. Nerve-root compression produces a positive Lasègue's sign in ≈ 83% of cases[2] (more likely to be positive in patients <30 yrs age with HLD[13]). May be positive in lumbosacral plexopathy (p.544). Note: flexing both thighs with the knees extended ("long-sitting" or sitting knee extension) may be tolerated further than flexing the single symptomatic side alone
2. Cram test: with patient supine, raise the symptomatic leg with the knee slightly flexed. Then, extend the knee. Results similar to SLR
3. crossed straight leg-raising test AKA Fajersztajn's sign: SLR on the painless leg causes contralateral limb pain (a greater degree of elevation is usually required than the painful side). More specific but less sensitive than SLR (97% of patients undergoing surgery with this sign have confirmed HLD[14]). May correlate with a more central disc herniation
4. femoral stretch test,[15] AKA reverse straight leg raising: patient prone, examiner's palm at popliteal fossa, knee is maximally dorsiflexed. Often positive with L2, L3, or L4 nerve root compression (e.g. in upper lumbar disc herniation), or with extreme lateral lumbar disc herniation (may also be positive in diabetic femoral neuropathy or psoas hematoma); in these situations SLR (Lasègue's sign) is frequently negative (since L5 & S1 not involved)
5. "bowstring sign": once pain occurs with SLR, lower the foot to the bed by flexing knee, keeping the hip flexed. Sciatic pain ceases with this maneuver, but hip pain persists
6. sitting knee extension test: with patient seated and both hips and knees flexed 90°, slowly extend one knee. Stretches nerve roots as much as a moderate degree of SLR

## Other signs useful in evaluation for lumbar radiculopathy

1. FABER: an acronym for Flexion ABduction External-Rotation, AKA FABERE test (the trailing "e" is for extension), AKA Patrick's-test (after Hugh Talbot Patrick). A test of hip motion. Method: the hip and knee are flexed and the lateral malleolus is placed on the contralateral knee. The ipsilateral knee is gently displaced downward towards the exam table. This stresses the hip joint and does not usually exacerbate true nerve-root compression. Often markedly positive in the presence of hip joint disease – e.g. trochanteric bursitis (p.1101) – sacroiliitis or mechanical low-back pain

2. Trendelenburg sign: examiner observes pelvis from behind while patient raises one leg while standing. Normally the pelvis remains horizontal. A positive sign occurs when the pelvis tilts down toward the side of the lifted leg indicating weakness of the contralateral thigh adductors (primarily L5 innervated)
3. crossed adductors: in eliciting patellar reflex (knee jerk (KJ)), the contralateral thigh adductors contract. In the presence of a hyperactive ipsilateral KJ it may indicate an upper motor neuron lesion, in the presence of a hypoactive ipsilateral KJ it may be a form of pathological spread, indicating nerve root irritability
4. Hoover sign[16]: to distinguish unilateral functional weakness of iliopsoas from organic weakness using synergistic contraction of the contralateral gluteus medius. The supine patient is asked to lift one leg off the bed against resistance from the examiner's hand. The examiner simultaneously places the palm of his/her other hand under the heel of the unlifted leg and gently lifts. Test 1: when the patient lifts the normal leg, if the paretic leg pushes down with more force than was exhibited on manual testing of the limb beforehand, the weakness is judged functional, if the force is equally weak the weakness is judged organic. Test 1 cannot be used if the hip extensor was normal beforehand. Test 2: (the better known test) the patient is asked to lift the weak leg. If the heel on the normal side lifts passively by the examiner, it suggests the weakness is functional (i.e. the patient is not trying). Not totally reliable[17,18]
5. abductor sign: an alternative to the Hoover test, to differentiate functional from organic weakness in the thigh abductors using synergistic contraction of the contralateral thigh abductors.[18] With the patient supine, the examiner places a hand on the lateral aspect of both legs. The patient is asked to abduct one leg, and then the other while the examiner applies resistance with his/her hand. The examiner mentally notes the response of the non-abducting LE. The results are as noted in □ Table 69.2



### Nerve root syndromes

Due to the facts listed below, a herniated lumbar disc (HLD) usually spares the nerve root exiting at that interspace, and impinges on the nerve exiting from the neural foramen one level below (e.g. a L5-S1 HLD usually causes S1 radiculopathy). This gives rise to the characteristic lumbar nerve root syndromes shown in □ Table 69.3.

Important applied anatomy in lumbar disc disease:

1. in the lumbar region, the nerve root exits below and in close proximity to the pedicle of its like-numbered vertebra
2. the intervertebral disc space is located well below the pedicle
3. not all patients have 5 lumbar vertebrae; see Localizing levels in spine surgery (p.1436)

## 69.1.7 Radiographic evaluation

See Radiographic evaluation under Low back pain (p.1416). 70% of herniated discs that migrate do so inferiorly.

## 69.1.8 Nonsurgical treatment

See nonsurgical treatment measures (p.1033).

## 69.1.9 Surgical treatment

### Indications for surgery

No predictive factors have been identified that can determine which patients are likely to improve on their own and which would be better served with surgery.

Table 69.2 Abductor sign

| Abducting LE | Contralateral (nonabducting) LE | |
|---|---|---|
| | Organic weakness | Functional weakness |
| weak LE | maintains position | hyperadducts |
| normal LE | hyperadducts | maintains position |

Table 69.3 Lumbar disc syndromes

| Syndrome | Level of herniated lumbar disc | | |
|---|---|---|---|
| | L3–4 | L4–5 | L5-S1 |
| root usually compressed | L4 | L5 | S1 |
| % of lumbar discs | 3–10% (5% average) | 40–45% | 45–50% |
| reflex diminished | knee jerk[a] (Westphal's sign) | medial hamstring[b] | Achilles[a] (ankle jerk) |
| motor weakness | quadriceps femoris (knee extension) | tibialis anterior (foot drop) & EHL[c] | gastrocnemius (plantarflexion), ± EHL[c] |
| decreased sensation[d] | medial malleolus & medial foot | large toe web & dorsum of foot | lateral malleolus & lateral foot |
| pain distribution | anterior thigh | posterior LE | posterior LE, often to ankle |

[a] Jendrassik maneuver may reinforce (see □ Table 29.2)
[b] medial hamstring reflex is unreliable (not always pure L5), may also stimulate adductors when eliciting
[c] see WEAKNESS in □ Table 69.1 for breakdown
[d] sensory impairment is most common in the distal extremes of the dermatome[19]



Surgical indications in patients with a radiographically identified herniated disc that correlates with findings on the history and physical exam:

1. failure of non-surgical management to control pain after 5–8 weeks: over 85% of patients with acute disc herniation will improve without surgical intervention in an average of 6 weeks[20] (70% within 4 weeks[21]). Most clinicians advocate waiting somewhere between 5–8 weeks from the onset of radiculopathy before considering surgery (assuming none of the items listed below applies)
2. " EMERGENT SURGERY": (i.e. before the 5–8 weeks of symptoms have lapsed). Indications:
   a) cauda equina syndrome (CES): (see below)
   b) progressive motor deficit (e.g. foot drop). NB: paresis of unknown duration is a doubtful indication for surgery[1,22,23] (no study has documented that there is less motor deficit in surgically treated patients with this finding[24]). However, the acute development or progression of motor weakness is considered an indication for rapid surgical decompression
   c) "urgent" surgery may be indicated for patients whose pain remains intolerable in spite of adequate narcotic pain medication
3. ± patients who do not want to invest the time in a trial of non-surgical treatment if it is possible that they will still require surgery at the end of the trial

## Cauda equina syndrome

The clinical condition arising from dysfunction of multiple lumbar and sacral nerve roots within the lumbar spinal canal. Usually due to compression of the cauda equina (the bundle of nerve roots below the conus medullaris arising from the lumbar enlargement and conus). See □ Table 53.2 for features to help differentiate CES from a conus lesion.

Possible findings in CES:

1. sphincter disturbance:
   a) urinary retention: the most consistent finding. Sensitivity ≈ 90% (at some point in time during course).[25,26] To evaluate acutely: have patient empty bladder and check post-void residual (by catheterization or with bladder ultrasound). In a patient without retention, only 1 in 1000 will have a CES. Cystometrogram (when done) shows a hypotonic bladder with decreased sensation and increased capacity
   b) urinary and/or fecal incontinence[27]: some patients with urinary retention will present with overflow incontinence
   c) anal sphincter tone: diminished in 60–80%
2. "saddle anesthesia": the most common sensory deficit. Distribution: region of the anus, lower genitals, perineum, over the buttocks, posterior-superior thighs. Sensitivity ≈ 75% Once total perineal anesthesia develops, patients tend to have permanent bladder paralysis[28]

3. significant motor weakness: usually involves more than a single nerve root (if untreated, may progress to paraplegia)
4. low back pain and/or sciatica (sciatica is usually bilateral, but may be unilateral or entirely absent, prognosis may be worse when absent or bilateral[26])
5. bilateral absence of Achilles reflex has been noted[29]
6. sexual dysfunction (usually not detected until a later time)

Etiologies of CES includes:
1. compression of cauda equina
   a) massive herniated lumbar disc: see below
   b) tumor
      - from compression: e.g. with metastatic disease to the spine with epidural extension
      - intravascular lymphomatosis (B-cell lymphoma) (p.711): a circulating lymphoma without solid mass. Often presents with CNS findings: dementia, enhancing meninges on MRI, lymphoma cells in CSF, and CES
   c) free fat graft following discectomy[30]
   d) trauma: fracture fragments compressing cauda equina
   e) spinal epidural hematoma
2. infection: may cause neurologic deficit from
   a) compression: typically from spinal epidural abscess complicating discitis or vertebral osteomyelitis
   b) a significant number of cases of CES from infection may be due to vascular compromise resulting from local septic thrombophlebitis. This may carry a worse prognosis as surgical decompression cannot correct this mechanism
3. neuropathy:
   a) ischemic
   b) inflammatory
4. ankylosing spondylitis: etiology is often obscure (p.1123)



CES from HLD: May be due to massive herniated disc, usually midline, most common at L4–5, often superimposed on a preexisting condition (spinal stenosis, tethered cord...).[27]

Prevalence of CES
1. 0.0004 in all patients with LBP[7]
2. only ≈ 1–2% of HLD that come to surgery[7]

Time course: CES tends to develop either acutely, or (less typically) slowly (prognosis is worse in the acute onset group, especially for return of bladder function, which occurred in only ≈ 50%).[25] 3 patterns[31]:
- Group I – sudden onset of CES symptoms with no previous low back symptoms
- Group II – previous history of recurrent backache and sciatica, the latest episode combined with CES
- Group III – presentation with backache & bilateral sciatica that later develop CES

Surgical issues: some advise a bilateral laminectomy[27] (but this is not mandatory). Occasionally, when it is difficult to remove a very tense midline disc, transdural removal may be helpful.[29]

Timing of discectomy in CES: controversial, and the point of contention in numerous lawsuits. In spite of early reports emphasizing rapid decompression,[29] other reports found no correlation between the time to surgery after presentation and the return of function.[25,26] Some evidence supports the goal of performing surgery within 48 hours (although performing surgery within 24 hours is desirable if possible, there is no statistically significant proof that delaying up to 48 hrs is detrimental).[32,33]

## Booking the case: Lumbar discectomy

Also see defaults & disclaimers (p.27).
1. position: prone
2. equipment: microscope (if used), minimally invasive retractors (if used)
3. consent (in lay terms for the patient – not all-inclusive):
   a) procedure: through the back to go between the bones and remove the piece of disc that is pressing on the nerve(s)

b) alternatives: nonsurgical management
c) complications: usual spine surgery complications (p. 1018) plus the disc can herniate again in the same place in ≈ 6% of cases, it is possible that a fragment of disc can be missed at the time of surgery, there might not be the amount of pain relief desired (back pain does not respond as well to surgery as nerve-root pain)

## Surgical options for lumbar radiculopathy

Once it is decided to treat surgically, options include:

1. trans-canal approaches
   a) standard open lumbar laminectomy and discectomy: 65–85% reported no sciatica one year post-op compared to 36% for conservative treatment.[34] Long-term results (>1 year) were similar. 10% of patients underwent further back surgery during the first year[34]
   b) "microdiscectomy"[35,36]: similar to standard procedure, however smaller incision is utilized. Advantages may be cosmetic, shortened hospital stay, lower blood loss. May be more difficult to retrieve some fragments.[37 (p 1319),38] Overall efficacy is similar to standard discectomy[39]
   c) sequestrectomy: removal of only the herniated portion of the disc without entering the disc space to remove disc material from there
2. intradiscal procedures (see below): a number of procedures have been devised over the years to percutaneously treat HLD by creating a cavity within the disc. Some have been abandoned for various reasons, not the least of which is controversy regarding the validity of the underlying premise that this can work
   a) chemonucleolysis: using chymopapain to enzymatically dissolve the disc (no longer used)
   b) automated percutaneous lumbar discectomy: utilizes a nucleotome
   c) percutaneous endoscopic intradiscal discectomy: see below
   d) intradiscal endothermal therapy (IDET or IDTA): see below
   e) laser disc decompression



## Intradiscal surgical procedures (ISP)

ISPs (see below for specific procedures) are among the most controversial procedures for lumbar spine surgery. The theoretical advantage is that epidural scarring is avoided, and that a smaller incision or even just a puncture site is used. This is also purported to reduce postoperative pain and hospital stay (often performed as an outpatient procedure). The conceptual problem with ISPs is that they are directed at removing disc material from the center of the disc space (which is not producing symptoms) and rely on the reduced intradiscal pressure to decompress the herniated portion of the disc from the nerve root. Only ≈ 10–15% of patients considered for surgical treatment of disc disease are candidates for an ISP. ISPs are usually done under local anesthetic in order to permit the patient to report nerve root pain to identify impingement on a nerve root by the surgical instrument or needle. Overall, ISPs are not recommended until rigorous controlled trials prove the efficacy.[10]

Indications utilized by proponents of intradiscal procedures:

1. type of disc herniation: appropriate only for "contained" disc herniation (i.e. outer margin of anulus fibrosus intact)
2. appropriate level: best for L4–5 HLD. May also be used at L3–4. Difficult but often workable (utilizing angled instruments or other techniques) at L5-S1 because of the angle required and interference by iliac crest
3. not recommended in presence of severe neurologic deficit[40]

Results:

"Success" rate (≈ pain free and return to work when appropriate) reported ranges from 37–75%[41,42,43]

Automated percutaneous lumbar discectomy: AKA nucleoplasty. Utilizes a nucleotome[44] to remove disc material from the center of the intervertebral disc space. 1 year success rate of 37% Complications include cauda equina syndrome from improper nucleotome placement.[45] In another study, nucleoplasty (with or without IDET (see below)) for HLD showed only modest reduction in pain at 9 months.[46]

Laser disc decompression: Insertion of a needle into the disc, and introduction of a laser fiber-optic cable through the needle to allow a laser to burn a hole in the center of the disc[47,48] (with or without endoscopic visualization).

The 2104 North American Spine Society Coverage Committee[49] position statement:

"Laser spine surgery in the cervical or lumbar spine is NOT indicated at this time. Due to lack of high quality clinical trials concerning laser spine surgery with the cervical or lumbar spine, it cannot be endorsed as an adjunct to open, minimally invasive, or percutaneous surgical techniques."

Percutaneous endoscopic lumbar discectomy (PELD) : This term refers to an essentially intradiscal procedure indicated primarily for contained disc herniations, although some small "noncontained" fragments may be treatable.[50] No large randomized study has been done to compare the technique to the accepted standard, open discectomy (with or without microscope). In one report[51] of 326 patients with L4–5 HLD, only 8 (2.4%) met study criteria (no previous operation, failure of conservative treatment, imaging study proving disc protrusion followed by discography to R/O "disc perforation") for PELD. Of these 8, only 3 were reported as having a good result. This study is not adequate for evaluating the technique.

Intradiscal endothermal therapy (IDET): AKA intradiscal (electro)thermal anuloplasty (IDTA). Efficacy: 23–60%at 1 year for treating "internal disc disruption"[52] (radial fissures in the nucleus pulposus extending into the anulus fibrosus) which is purported to account for 40%of patients with chronic low back pain of unknown etiology.[53]

## Adjunctive treatment in lumbar laminectomy

### Epidural steroids following discectomy

Perioperative epidural steroids after routine surgery for lumbar degenerative disease may result in a small reduction of post-op pain, length of stay, and the risk of not returning to work at 1 year, but most of the evidence originates from studies not using validated outcome assessment that favor positive results, and further study is recommended (various agents, dosages, co-administered drugs, and delivery methods were reported).[54] However, the combination of systemic steroids at the start of the case (Depo-Medrol® 160 mg IM and methylprednisolone sodium succinate (Solu-Medrol®) 250 mg IV) combined with infiltration of 30 ml of 0.25%bupivacaine (Marcaine®) into the paraspinal muscles at incision and closure, may reduce hospital stay and post-op narcotic requirements.[55]



## Methods to reduce scar formation

### Epidural free fat graft

The use of an autogenous free fat graft in the epidural space has been employed in an attempt to reduce post-op epidural scar formation. Opinion varies widely as to the effectiveness, some feel it is helpful, others feel it actually exacerbates scarring.[56] In some patients, no evidence of the graft will be found on reoperation years later. The fat graft can very rarely be a cause of nerve root compression[57] or cauda equina syndrome[30] within the first few days post-op, and there is a case report of compression 6 years following surgery.[58]

### Other measures

Other measures include the placement of barrier films or gels. There are numerous products available, none has been shown to have reproducible benefit.

## Risks of lumbar laminectomy

### General information

Overall risk of mortality in large series[59,60]: 6 per 10,000 (i.e. 0.06%), most often due to septicemia, MI, or PE. Complication rates are very difficult to determine accurately,[34] but the following is included as a guideline.

### Common complications

1. infection:
   a) superficial wound infection: 0.9–5%[61] (risk is increased with age, long term steroids, obesity, ? DM): most are caused by S. aureus; see Laminectomy wound infection (p.345) for management
   b) deep infection: <1%(see below under Uncommon complications)
2. increased motor deficit: 1–8%(some transient)
3. unintended "incidental" durotomy (the term "unintended durotomy" has been recommended in preference to "dural tear", see below): incidence is 0.3–13%(risk increases to ≈ 18%in re-do operations).[62] Possible sequelae include those listed in □ Table 69.4

Table 69.4 Possible sequelae of dural opening

| Well documented |
|---|
| 1. CSF leak<br>a) contained: pseudomeningocele<br>b) external: CSF fistula<br>2. herniation of nerve roots thorough opening<br>3. associated nerve root contusion, laceration or injury to the cauda equina<br>4. CSF leak collapses the thecal sac and may increase blood loss from epidural bleeding |
| **Less well documented** |
| 1. arachnoiditis<br>2. chronic pain<br>3. bladder, bowel and/or sexual dysfunction |

a) CSF fistula (external CSF leak): the risk of a CSF fistula requiring operative repair is ≈ 10 per 10,000[59]
b) pseudomeningocele: 0.7–2%[62] (may appear similar radiographically to spinal epidural abscess (SEA), but post-op SEA often enhances, is more irregular, and is associated with muscle edema)

4. recurrent herniated lumbar disc (same level either side) (p.1061): 4%(with 10 year follow-up)[63]
5. Post-operative urinary retention (POUR): usually temporary, but may delay hospital discharge

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## Uncommon complications

1. direct injury to neural structures. For large disc herniations, consider a bilateral exposure to reduce risk
2. injury to structures anterior to the vertebral bodies (VB): injured by breaching the anterior longitudinal ligament (ALL) through the disc space, e.g. with pituitary rongeur. The depth of disc space penetration with instruments should be kept ≤ 3 cm, since 5%of lumbar discs had diameters as small as 3.3 cm.[64] Asymptomatic perforations of the ALL occur in up to 12%of discectomies. Breach of the ALL risks potential injuries to:
   a) great vessels[65]: risks include potentially fatal hemorrhage, and arteriovenous fistula which may present years later. Most such injuries occur with L4–5 discectomies. Only ≈ 50%bleed into the disc space intraoperatively, the rest bleed into the retroperitoneum. Emergent laparotomy or endovascular treatment[66] is indicated, preferably by a surgeon with vascular surgical experience, if available. Mortality rate is 37–67%
      - aorta: the aortic bifurcation is on the left side of the lower part of the L4 VB, and so the aorta may be injured above this level
      - below L4, the common iliac arteries may be injured
      - veins (more common than arterial injuries): vena cava at and above L4, common iliac veins below L4
   b) ureters
   c) bowel: at L5-S1 the ileum is the most likely viscus to be injured
   d) sympathetic trunk
3. wrong site surgery: incidence in self-reporting survey was 4.5 occurrences per 10,000 lumbar spine operations.[67] Factors identified as potential contributors to the error: unusual patient anatomy, not performing localizing radiograph. 32%of responding neurosurgeons indicated that they removed disc material from the wrong level at some time in their career
4. rare infections:
   a) meningitis
   b) deep infection: < 1% Including:
      - discitis (p.356): 0.5%
      - spinal epidural abscess (SEA) (p.349): 0.67%
5. cauda equina syndrome: may be caused by post-op spinal epidural hematoma (see below). Incidence was 0.21%in one series of 2842 lumbar discectomies[68] and 0.14%in a series of 12,000 spine operations.[69] Red flags: urinary retention, anesthesia that may be saddle or bilateral LE
6. postoperative visual loss (POVL)[70]: (see below)
7. complications of positioning:
   a) compression neuropathies: ulnar, peroneal nerves. Use padding over elbows and avoid pressure on posterior popliteal fossa

b) anterior tibial compartment syndrome: due to pressure on anterior compartment of leg (reported with Andrew's frame). An orthopedic emergency that may require emergent fasciotomy
c) pressure on the eye: corneal abrasions, damage to the anterior chamber
d) cervical spine injuries during positioning due to relaxed muscles under anesthesia

8. post-op arachnoiditis (p.1040): risk factors include epidural hematoma, patients who tend to develop hypertrophic scar, post-op discitis, and intrathecal injection anesthetic agents or steroids. Surgical treatment for this is disappointing. Intrathecal depo-medrol may provide short-term relief (in spite of the fact that steroids are a risk factor for the development of arachnoiditis).
9. thrombophlebitis and deep-vein thrombosis with risk of pulmonary embolism (PE)[59]: 0.1%; see Thromboembolism in neurosurgery (p.167)
10. complex regional pain syndrome AKA reflex sympathetic dystrophy (RSD) (p.497): reported in up to 1.2% of cases, usually after posterior decompression with fusion, often following reoperations[71] with onset 4 days to 20 weeks post-op. See also critique of RSD (p.497). Treatment includes some or all of: PT, sympathetic blocks, oral methylprednisolone, removal of hardware if any
11. very rare: Ogilvie's syndrome (pseudo-obstruction ("ileus") of the colon). Usually seen in hospitalized/debilitated patients. May be related to narcotics, electrolyte deficiencies, possibly from chronic constipation. Also reported following spinal surgery/trauma, spinal/epidural anesthesia, spinal metastases, & myelography[72]

## Unintended durotomy



Unintentional opening of the dura during spinal surgery has an incidence of 0–14%[73]

Terminology: The terms "unintended durotomy", "incidental durotomy",[73] or even just "dural opening", have been recommended in preference to "dural tear" which may imply carelessness[62] when none was present. Dural openings have been associated with one or more alleged complications or sequelae in medical malpractice suits involving surgery on the lumbar spine.

The injury: By itself, opening the dura intentionally or otherwise is not expected to have a deleterious effect on the patient.[62,74] In fact, dural opening is often a standard part of the operation for intradural disc herniation,[75] tumors, etc. Although not frequent, (for incidence, see above) unintended durotomy is not an unusual occurrence, and alone, is not considered an act of malpractice. However, it may result from an event or events that produce more serious injuries. These events and injuries should be dealt with on their own merits.

In the SPORT study, there was a 9% incidence of unintended durotomy in patients undergoing first-time open laminectomy.[76] There were no long-term differences in nerve root injuries, mortality, additional operations, or outcome measures. Short-term differences included longer inpatient stay, increased blood loss, and duration of surgery.[76]

Possible sequelae include those listed in □ Table 69.4. A CSF leak may produce "spinal headache" (p.1508) with its associated symptoms and if it breaches the skin it may be a risk factor for meningitis. Pain or sensory/motor deficits may be associated with injuries to nerve roots or delayed herniation of nerve roots through the dural opening.

Etiologies: Potential causes are many, and include[62]: unanticipated anatomic variations, adhesion of the dura to removed bone, slippage of an instrument, an obscured fold of dura caught in a rongeur or curette, thinning of the dura in cases of longstanding stenosis, and the possibility of a delayed CSF leak caused by perforation of the dura when it expands onto a surgically created spicule of bone.[77] The risk may be increased with anterior decompression for OPLL, with revision surgery, and with the use of high-speed drills.[73]

Treatment: If the opening is recognized at the time of surgery, watertight primary closure (with or without patch graft) should be attempted with nonabsorbable suture if at all possible to prevent pseudomeningocele and/or CSF fistula. A cottonoid placed over the opening prevents aspiration of nerve roots.[78] Care must be taken to avoid incorporating a nerve root into the closure. Most repairs will be accomplished with no complication or sequelae to the patient. When the opening is in the far (anterior) side of the dura, consideration may be given to intradural repair accessed through a posterior durotomy which is subsequently closed (this may risk additional injury to the nerve roots). Biocompatible fixatives (e.g. fibrin glue[73]) may be used to supplement primary closure.

Primary repair may be impossible in some situations (e.g. when the opening cannot be found or accessed, as is sometimes the case when it occurs on the nerve root sleeve) and alternatives here include placement of a fat or muscle graft over the suspected leak site, use of a the patient's own blood for a "blood patch" (one technique is to have the anesthesiologist draw ≈ 5–10 ml of the patient's blood from an arm vein, keeping it in the syringe for several minutes until it starts to coagulate, and then to have the anesthesiologist inject the blood onto the dura), use of gelfoam, fibrin

glue… Some recommend that the wound not be drained post-op, with a water-tight closure of fascia, fat, and skin to add to the barrier. Others use a subcutaneous drain or epidural catheter. CSF diversionary procedures (e.g. through a drain inserted 1 or more levels away) may also be used.

Although bed rest ×4–7 days is often advocated to reduce symptoms and facilitate healing, when watertight closure has been achieved, normal post-op mobilization is not associated with a high failure rate (bed rest is recommended if symptoms develop).[73]

In one report of 8 patients with leaks that appeared post-op, re-operation was avoided when treated by resuturing the skin under local anesthesia, followed by bed rest in slight Trendelenburg position (to reduce pressure on the leakage site), broad spectrum antibiotics and antibiotic ointment over the skin incision, and daily puncture and drainage of the subcutaneous collection.[79]

See other treatment measures for H/A associated with CSF leak (p.1049).

### Postoperative visual loss

1. ischemic optic neuropathy[80]: the most common cause of the very uncommon post-operative loss of vision. Often bilateral. Usually associated with significant blood loss (median: 2 L), and/or prolonged operative time (≥6 hrs). All cases had anesthetic time >5 hrs or blood loss >1 L. Blood loss can cause hypotension (may cause release of endogenous vasoconstrictors in addition to reduced blood flow due to low hemodynamic pressure) and increased platelet aggregation. Is not due to direct pressure on the globe in most cases, and can occur at any age and even in otherwise healthy patients. No association with age, HTN, atherosclerosis, smoking or DM.
   The blindness can be extensive and is often permanent. Prevention is critical since there is no known effective treatment.[81]
   a) posterior ischemic optic neuropathy (PION)[80]: may follow surgery (surgical PION). Risk factors as above, plus:
      - surgery in the prone position (can cause periorbital edema, and rarely, direct pressure on the orbit)
      - lack of tight glycemic control
      - use of Trendelenburg position
      - hemodilution or overuse of crystalloid vs. colloid (blood) fluid replacement
      - prolonged hypotension
      - cellular hypoxia
      - decreased renal perfusion
   b) 6 independent risk factors for POVL[81]
      - male gender: odds ratio (OR) = 2.53
      - obesity: by clinical assessment or BMI ≥ 30 OR = 2.83
      - use of Wilson frame: OR = 4.30
      - length of anesthesia: OR = 1.39 per hour
      - EBL: OR = 1.34 per Liter
      - use of colloid as a percentage of nonblood replacement: less certain (small difference). OR = 0.67 per 5%colloid
   c) anterior ischemic optic neuropathy (AION): divided into arteritic (as with GCA) and nonarteritic (common with DM)
2. central retinal artery occlusion
3. cortical blindness: from occipital lobe infarction possibly due to embolis

## Post-op care

### Post-op orders

The following are guidelines for post-operative orders for a lumbar laminectomy without intra-operative complications; variations between surgeons and institutions must be taken into consideration:

1. admit post-anesthesia care unit (PACU)
2. vital signs on the nursing unit: q 2° ×4 hrs, q 4° ×24°, then q 8°
3. activity: up with assist, advance as tolerated
4. nursing care
   - I's & O's
   - intermittent catheterization q 4–6° PRN no void
   - optional: TED hose (may reduce risk of DVT) or PCB
   - optional (if drain used): empty drain q 8° and PRN
5. diet: clear liquids, advance as tolerated
6. IV: D5 1/2 NS + 20 mEq KCl/l @75 ml/hr, D/C when tolerating PO well (after antibiotics D/C'd if prophylactic antibiotics are used)

7. meds
   - laxative of choice (LOC) PRN
   - sodium docusate (e.g. Colace®) 100 mg PO BID when tolerating PO (stool softener, does not substitute for LOC)
   - optional: prophylactic antibiotics if used at your institution
   - acetaminophen (Tylenol®) 325 mg 1–2 PO or PR q 6° PRN
   - narcotic analgesic
   - optional: steroids are used by some surgeons to reduce nerve-root irritation from surgical manipulation
8. labs
   - optional (if significant blood loss during surgery): CBC

### Post-op check

In addition to routine, the following should be checked:

1. strength of lower extremities, especially muscles relevant to nerve root, e.g. gastrocnemius for L5-S1 surgery, EHL for L4–5 surgery…
2. appearance of dressing: look for signs of excessive bleeding, CSF leak…
3. signs of cauda equina syndrome (p.1050), e.g. by post-op spinal epidural hematoma
   a) loss of perineal sensation (“saddle anesthesia”)
   b) inability to void: may not be not unusual after lumbar laminectomy, more concerning if accompanied by loss of perineal sensation
   c) pain out of the ordinary for the post-op period
   d) weakness of multiple muscle groups



Any new neurologic deficit should prompt rapid evaluation for spinal epidural hematoma[69] (EDH). Delayed deficits may be due to EDH or epidural abscess. Post-op films in the recovery room can rule out graft or hardware malposition for fusions or instrumentation procedures, or changes in alignment. The diagnostic test of choice is MRI. If contraindicated or not available, CT/myelography may be indicated. An extradural defect immediately post-op suggests EDH.

## Outcome of surgical treatment

In a series of 100 patients undergoing discectomy, at 1 year post-op 73%had complete relief of leg pain and 63%had complete relief of back pain; at 5–10 years the numbers were 62%for each category.[2] At 5–10 years post-op, only 14%felt that the pain was the same or worse than pre-op (i.e. 86% felt improved), and 5%qualified as having a failed back surgery syndrome (a heterogeneous not-precisely defined term, here meaning not returned to work, requiring analgesics, receiving worker’s compensation, see Failed back surgery syndrome (p.1039).

Attempts to measure relative merits of conservative treatment vs. surgery have failed. The recent SPORT study[82,83] suffered from significant selection bias since patients were allowed to crossover to the other arm of the study and thus more nearly approximated the current methodology of surgical selection than an actual RCT.[84] Earlier attempts at randomized trials also suffered from methodological flaws.[1] Conclusions that can be drawn from these studies[84]: most patients with manageable or improving pain and less disability typically choose conservative treatment and most have improvement in symptoms, whereas patients with severe, persistent or worsening pain and/or neurologic deficit are more likely to choose surgery with a resultant excellent outcome.

In patients with a diminished knee-jerk or ankle-jerk pre-op, 35%and 43%(respectively) still had reduced reflexes 1 year post-op[8]; reflexes were lost post-op in 3%and 10%respectively. The same study found that motor loss was improved in 80%, aggravated in 3%, and was newly present in 5% post-op; and that sensory loss was improved in 69%and was worsened in 15%post-op.

Foot drop: severe or complete paralysis of ankle dorsiflexion occurs in 5–10%of HLD, and about 50%of cases recover with or without treatment. Discectomy does not improve the outcome, especially in cases of painless foot drop.[24]

See Recurrent disc herniation (p.1061).

# 69.1.10 Herniated upper lumbar discs (levels L1–2, L2–3, and L3–4)

## General information

L4–5 & L5-S1 herniated lumbar discs (HLD) account for most cases of HLD (realistically ≈ 90%, possibly as high as 98%). 24%of patients with HLD at L3–4 have a past history of a HLD at L4–5 or L5-S1, suggesting a generalized tendency towards disc herniation. In a series of 1,395 HLDs, there were 4 at L1–2 (0.28%incidence), 18 at L2–3 (1.3%), and 51 at L3–4 (3.6%).[85]

## Presentation

Typically presents with LBP, onset following trauma or strain in 51% With progression, paresthesias and pain in the anterior thigh occur, with complaints of leg weakness (especially on ascending stairs).

## Signs

Quadriceps femoris was the most common muscle involved, demonstrating weakness and sometimes atrophy.

Straight leg raising was positive in only 40% Psoas stretch test was positive in 27% Femoral stretch test may be positive (p.1048).

50%had reduced or absent knee jerk; 18%had ankle jerk abnormalities; reflex changes were more common with L3–4 HLD (81%) than L1–2 (none) or L2–3 (44%).

# 69.1.11 Extreme lateral lumbar disc herniations

## General information

Definition: herniation of a disc at (foraminal disc herniation) or distal to (extraforaminal disc herniation) the facet (some authors do not consider foraminal disc herniation to be "extreme lateral"). See □ Fig. 69.1.

Incidence (□ Table 69.5): 3–10%of herniated lumbar discs (HLD) (series with higher numbers[86] include some HLD that are not truly extreme lateral).



Occurs most commonly at L4–5 and next at L3–4 (see □ Table 69.5), thus L4 is the most common nerve involved and L3 is next. With a clinical picture of an upper lumbar nerve root compression (i.e. radiculopathy with negative SLR), chances are ≈ 3 to 1 that it is an extremely lateral HLD rather than an upper lumbar disc herniation.

Differs from the more common central and subarticular HLD in that:

- the nerve root involved is usually the one exiting at that level (c.f. the root exiting at the level below)
- straight leg raising (SLR) (p.1048) is negative in 85–90%of cases ≥ 1 week after onset (excluding double herniations; ≈ 65%will be negative if double herniations are included); may have positive femoral stretch test
- pain is reproduced by lateral bending to the side of herniation in 75%
- higher incidence of extruded fragments (60%)
- higher incidence of double herniations on the same side at the same level (15%)
- pain tends to be more severe (may be due to fact that the dorsal root ganglion may be compressed directly) and often has more of a burning dysesthetic quality

## Presentation

Quadriceps weakness, reduction of patellar reflex, and diminished sensation in the L3 or L4 dermatome are the most common findings.

## Differential diagnosis

1. lateral recess stenosis or superior articular facet hypertrophy
2. retroperitoneal hematoma or tumor

Table 69.5 Incidence of extreme lateral HLD by level[a]

| Disc level | No. | % |
|---|---|---|
| L1–2 | 1 | 1% |
| L2–3 | 11 | 8% |
| L3–4 | 35 | 24% |
| L4–5 | 82 | 60% |
| L5-S1 | 9 | 7% |

[a]series of 138 cases[86]

3. diabetic neuropathy (amyotrophy) (p.545)
4. spinal tumor
   a) benign (schwannoma or neurofibroma)
   b) malignant tumors
   c) lymphoma
5. infection
   a) localized (spinal epidural abscess)
   b) psoas muscle abscess
   c) granulomatous disease
6. spondylolisthesis (with pars defect)
7. compression of conjoined nerve root
8. on MRI, enlarged foraminal veins may mimic extreme lateral disc herniation

## Radiographic diagnosis

NB: if actively sought, many asymptomatic far-lateral disc herniations may be demonstrated on MRI or CT. Careful clinical correlation is usually required.

MRI: diagnostic test of choice. Sagittal views through the neural foramen may help demonstrate the disc herniation.[87] MRI may have ≈ 8% false positive rate due to presence of enlarged foraminal veins that mimic extreme lateral HLD.[88]

Myelography: myelography alone is rarely diagnostic (usually requires post myelo CT.[89,90] Fails to disclose the pathology in 87% of cases due to the fact that the nerve root compression occurs distal to the nerve root sleeve (and therefore beyond the reach of the dye).[91]

CT scan[90]: reveals a mass displacing epidural fat and encroaching on the intervertebral foramen or lateral recess, compromising the emerging root. Or, may be lateral to foramen. Sensitivity is ≈ 50% and is similar with post-myelographic CT.[91] Post-discography CT[91,92] has also been able to demonstrate.



## Surgical treatment

NB: compression of the dorsal root ganglion may result in a slower recovery from discectomy and overall less satisfying outcome than with the more commonplace paramedian disc herniation.

### Foraminal discs

Usually requires mesial facetectomy to gain access to the region lateral to the dural sac without undue retraction on nerve root or cauda equina. Caution: total facetectomy combined with discectomy may result in a high incidence of instability (total facetectomy alone causes ≈ 10% rate of slippage), although other series found this risk to be lower (≈ 1 in 33[93,94]). An alternative technique is to remove just the lateral portion of the superior articular facet below.[95] Endoscopic techniques may be well suited for herniated discs in this location.[96]

## Discs herniated beyond (lateral to) the foramen

Numerous approaches are used, including:

1. traditional midline hemilaminectomy: the ipsilateral facet must be partially or completely removed. The safest way to find the exiting nerve root is take the laminectomy of the inferior portion of the upper vertebral level (e.g. L4 for a L4–5 HLD) high enough to expose the nerve root axilla, and then follow the nerve laterally through the neural foramen by removing facet until the HLD is identified
2. lateral approach (i.e. extra-canal) through a paramedian incision.[97] Advantages: the facet joint is preserved (facet removal combined with discectomy may lead to instability), muscle retraction is easier. Disadvantages: unfamiliar approach for most surgeons and the nerve cannot be followed medial to lateral. A localizing x-ray is taken with a spinal needle. A 4–5 cm vertical skin incision is made 3–4 cm lateral to the midline on the side of the disc herniation. The incision is taken down to the thoracolumbar fascia and the subcutaneous tissue is dissected off the fascia. Above L4, one may palpate the groove between multifidus (medial) and longissimus (lateral), where the fascia is incised. The facet joint is palpated, and blunt dissection is used to gain access to the lateral facet joint and transverse processes above and below the level of the disc herniation. The correct level is confirmed on x-ray using a probe as a marker. The intertransversarius muscle and fascia are divided. Care must be taken to avoid mechanical and electrocautery injury to the nerve and dorsal root ganglion (which lies immediately beneath the intertransverse ligament). The radicular artery, vein, and nerve root are located just beneath the transverse processes, usually slightly

medial to this position. The nerve root hugs the pedicle of the level above as it exits the neural foramen (palpating this e.g. with a dental dissector helps locate it), and it may be splayed over the herniated disc fragment. If more medial exposure is necessary, the lateral facet joint may be resected. The HLD is removed. Additional removal of disc material from the disc space may be performed with down-biting pituitary rongeurs. Extracanal approach to L5-S1 requires removal of part of the sacral ala in order to access the space caudal to the L5 transverse process

## 69.1.12 Lumbar disc herniations in pediatrics

Less than one percent of surgery for herniated lumbar disc is performed on patients between the ages of 10 and 20 yrs (one series at Mayo found 0.4% of operated HLD in patients <17 yrs age[98]). These patients often have few neurologic findings except for a consistently positive straight leg raising test.[99] Herniated disc material in youths tends to be firm, fibrous and strongly attached to the cartilaginous end-plate unlike the degenerated material usually extruded in adult disc herniation. Plain radiographs disclosed an unusually high frequency of congenital spine anomalies (transitional vertebra, hyperlordosis, spondylolisthesis, spina bifida…). 78% did well after their first operation.[98]

## 69.1.13 Intradural disc herniation

Herniation of a fragment of disc into the thecal sac, or into the nerve root sleeve (the latter sometimes referred to as "intraradicular" disc herniation) has been recognized with a reported incidence of 0.04–1.1% of disc herniations.[75,100] Although it may be suspected on the basis of pre-op MRI or myelography, the diagnosis is rarely made preoperatively.[100] Intraoperatively, it may be suggested by the appreciation of a tense firm mass within the nerve root sleeve or by the negative exploration of a level with obvious clinical signs and clear cut radiographic abnormalities (after verifying that the correct level is exposed).



Surgical treatment:

Although a surgical dural opening may be utilized,[75] others have found this to be necessary in a minority of cases.[101]

## 69.1.14 Intravertebral disc herniation

### General information

AKA Schmorl's node or nodule. Named for German pathologist Christian Georg Schmorl (1861–1932) the term was first used in 1971.[102] AKA Schmor's (no "l") nodule AKA Geipel hernia.[103] Disc herniation through the cartilaginous end plate into the cancellous bone of the vertebral body (VB) (AKA intraspongious disc herniation). Often an incidental finding on x-ray or MRI. Clinical significance is controversial. May produce low back pain initially that lasts ≈ 3–4 months after onset. Diffuse displacement (as may be seen in osteoporosis) is sometimes referred to as a balloon disc.[104]

### Clinical findings

During the acute (symptomatic) phase, patients may exhibit LBP that is aggravated by weight bearing and movement. There may be tenderness to percussion or manual compression over the involved segment.

### Radiographic findings

MRI: the extrusion of disc material into the VB is easily appreciated on sagittal images. It has been suggested[105] that acute (symptomatic) lesions may appear differentiated from chronic (asymptomatic) lesions by the presence of MRI findings of inflammation in the bone marrow immediately surrounding the node as outlined in □ Table 69.6.

CT: demonstrates defect in endplate and vertebral body since disc material has a significantly lower density than bone.

Plain x-ray: ≤33% may be seen on plain x-rays.[106] They may not be detectable acutely until sclerotic osseous bone casting develops.

### Treatment

Conservative treatment is indicated, usually consisting of non-steroidal anti-inflammatory drugs (NSAIDs). Occasionally stronger pain medication and/or lumbar bracing may be required. Surgery is rarely indicated.

Table 69.6 MRI signal intensity in Schmorl's nodes[a]

| Lesion | T1WI | T2WI |
|---|---|---|
| symptomatic (acute) | low | high |
| asymptomatic (chronic) | high[b] | low[b] |

[a]signal intensity in surrounding marrow
[b]the same as normal marrow

### Outcome

With conservative treatment, symptoms generally resolve within 3–4 months of onset (as with most vertebral body fractures).

## 69.1.15 Recurrent herniated lumbar disc

### General information

Rates quoted in the literature range from 3–19%with the higher rates usually in series with longer follow-up.[107] In an individual series with 10 year mean F/U, the rate of recurrent disc herniation was 4%(same level, either side), one third of which occurred during the 1st year post-op (mean: 4.3 yrs).[63] A second recurrence at the same site occurred in 1%in another series[107] with mean F/U of 4.5 yrs. In this series,[107] patients presenting for a second time with disc herniation had a recurrence at the same level in 74%, but 26%had a HLD at another level. Recurrent HLD occurred at L4–5 more than twice as often as L5-S1.[107]

It is often possible for a smaller amount of recurrent herniated disc to cause symptoms than in a "virgin back", due to the fact that the nerve root is often fixated by scar tissue and has little ability to deviate away from the fragment.[56]



### Treatment

Initial recommended treatment is as with a first time HLD. Nonsurgical treatment should be utilized in the absence of progressive neurologic deficit, cauda equina syndrome (CES) or intractable pain.

## 69.1.16 Surgical treatment

Disagreement occurs regarding optimal treatment. See Practice guideline: Lumbar fusion for disc herniation (p.1037).

Surgical outcome:

As with first time HLD, the outcome from surgical treatment is worse in worker's compensation cases and in patients undertaking litigation, only ≈ 40%of these patients benefit.[107,108] A worse prognosis is also associated with: patients with <6 mos relief after their first operation, cases where fibrosis without recurrent HLD is found at operation.

## 69.1.17 Spinal cord stimulation

One study actually showed a better response rate to spinal cord stimulation than to reoperation.[109] Since surgery for recurrent HLD carries a higher risk of dural and nerve root injury, and a lower success rate than first time operations, this may be a viable option for some patients.

# 69.2 Thoracic disc herniation

## 69.2.1 General information

### Key concepts

- comprise only 0.25%of herniated discs, and <4%of operations for herniated disc
- usually occur at or below T8 (the more mobile portion of the thoracic spine)
- frequently calcified □get CT through disc (may affect choice of surgical approach)

- primary indications for surgery: refractory pain, progressive myelopathy
- surgical treatment: laminectomy is usually not appropriate

Account for 0.25–0.75% of all protruded discs.[110] 80% occur between the 3rd and 5th decades. 75% are below T8 (the more mobile portion of the thoracic spine), with a peak of 26% at T11–12. 94% were centrolateral and 6% were lateral.[111] A history of trauma may be elicited in 25% of cases.

Most common symptoms: pain (60%), sensory changes (23%), motor changes (18%). With thoracic radiculopathy, pain and sensory disturbance is in a band-like distribution radiating anteriorly and inferiorly along the involved root's dermatome. Motor involvement is difficult to document.

## 69.2.2 Evaluation

MRI is the mainstay of diagnosis. However, it is almost mandatory to also obtain a CT scan to determine if the disc is soft or calcified ("hard disc") which can have a profound effect on the approach. The CT is also useful to demonstrate bony detail if instrumentation is needed.

## 69.2.3 Indications for surgery

Herniated thoracic discs requiring surgery are rare.[111] Indications: refractory pain (usually radicular, bandlike) or progressive myelopathy. Uncommon: symptomatic syringomyelia originating at level of disc herniation.



## 69.2.4 Surgical approaches

Surgery for thoracic disc disease is problematic because of: the difficulty of anterior approaches, the proportionately tighter space between cord and canal compared to the cervical and lumbar regions, and the watershed blood supply which creates a significant risk of cord injury with attempts to manipulate the cord when trying to work anteriorly to it from a posterior approach. Herniated thoracic discs are calcified in 65% of patients considered for surgery[111] (more difficult to remove from a posterior or lateral approach than non-calcified discs).

Open surgical approaches[111,112]:

1. posterior (midline laminectomy): primary indication is for decompression of posteriorly situated intracanalicular pathology (e.g. metastatic tumor) especially over multiple levels. There is a high failure and complication rate when used for single-level anterior pathology (e.g. midline disc herniation)
2. posterolateral
   a) lateral gutter: laminectomy plus removal of pedicle.
   b) transpedicular approach[113]
   c) costotransversectomy (see below)
   d) transfacet pedicle sparing
3. anterolateral (transthoracic): usually through the pleural space
4. lateral extracavitary (retrocolic)[114] staying external to the pleural space

Thoracoscopic surgery is an alternative to open surgery.

## 69.2.5 Choosing the approach

### General information

See anterior approaches to the thoracic spine (p.1489).

Intraoperative SSEPs and MEPs may be helpful for patients with myelopathy.

For a laterally herniated thoracic disc without myelopathy: posterolateral approach with medial facetectomy is technically simple, and has generally good results. For a central disc herniation, or when myelopathy is present: transthoracic approach has the lowest incidence of cord injury with the best operative results (□ Table 69.7). For anterior access, unless pathology is predominantly left-sided, a right-sided thoracotomy is preferred because the heart does not impede access.

### Costotransversectomy

Indications: in the past this was often used to drain tuberculous spine abscess. It may be used for lateral disc herniation, biopsy of VB or pedicle, limited unilateral decompression of spinal cord from

Table 69.7 Results with various approaches for thoracic spine pathology[115]

| Approach | Indication | Total no. | Outcome | | | |
|---|---|---|---|---|---|---|
| | | | Normal | Improved | Same | Worse |
| laminectomy | posteriorly located tumor | 129 | 15% | 42% | 11% | 32% |
| posterolateral (transpedicular) | radicular pain with lateral disc herniation; biopsy of tumor | 27 | 37% | 45% | 11% | 7% |
| lateral (costotransversectomy) | fair for midline disc; good ipsilateral access, poor access to opposite side | 43 | 35% | 53% | 12% | 0 |
| transthoracic | best for midline lesions, especially for reaching both sides of cord | 12 | 67% | 33% | 0 | 0 |

tumor or bone fragments, or sympathectomy. Can be used at ≈ any T-spine level. Limitations: difficult to visualize anterior canal to access midline anterior pathology. Better for soft disc than for calcified central disc.

Involves resection of the transverse process and at least ≈ 4–5 cm of the posterior rib. A serious risk of this approach is interruption of a significant radicular artery which may compromise spinal cord blood supply; see Spinal cord vasculature (p. 87). There is also a risk of pneumothorax which is less grave.



## Booking the case: Costotransversectomy

Also see defaults & disclaimers (p. 27).

1. position: prone, usually on chest rolls
2. equipment:
   a) microscope (not used for all cases)
   b) C-arm
3. implants: if post-op instability is anticipated, thoracic pedicle screws and possibly a cage (e.g. for fracture or tumor, not typically for disc herniation)
4. neuromonitoring: SSEP/MEP
5. blood availability: type and cross 2 U PRBC
6. consent (in lay terms for the patient – not all-inclusive):
   a) procedure: surgery through the back of the chest to remove a small piece of rib to permit removal of the herniated/calcified disc
   b) alternatives: nonsurgical management, surgery from the side through the chest
   c) complications: spinal cord injury with paralysis, lung complications including pneumothorax or hemothorax (blood or air outside lungs), possible seizures with MEPs

### 69.2.6 Surgical technique

The approach can be somewhat difficult due to the infrequent encounter with the anatomy by most neurosurgeons. Be prepared for a "deep, red hole, where everything initially looks the same and the bony anatomy is not easy to define." With patience and persistence and the help of an anatomic model in the O.R., the surgeon can get his/her bearings. One of the most helpful landmarks is following the NVB (or just the nerve root) medially to the neural foramen.

In the O.R., before the prep and skin incision, localizing x-rays are obtained; a spinal needle inserted between 2 spinous processes may be used as a marker.

Patient position: the approach is from the side of the pathology/symptoms; for central disc herniations a right-sided approach reduces risk of injury to artery of Adamkiewicz (located on the left in 80%(p. 87)). Options:

1. lateral oblique, ≈ 30° elevated from straight prone, a "bean-bag" is good for stabilization. For a thin patient, the surgeon may stand in front of the patient (gives more horizontal angle of view – does not work as well with heavier patients due to mass of skin/muscle in the way laterally)

2. prone on chest rolls: the chest roll on the side of the pathology should be more medial to allow the shoulder and scapula to fall forward out of the way

Skin incision options:

1. curved paramedian skin incision: apex oriented away from the midline along the slight depression demarcating the junction of the lateral border of the paraspinal muscles with the ribs (≈ 6–7 cm lateral to midline) centered over the interspace of interest extending ≈ 3 vertebral bodies (VB) above and below. The incision is carried through the skin, subcutaneous fat, trapezius, and (for lower 6 thoracic levels, where most thoracic disc herniations occur) the latissimus dorsi, down to the ribs, and this musculocutaneous flap can be reflected medially as a unit
2. midline incision: need to extend 3–4 levels above and below the level of pathology to get an angle low enough to visualize posterior to the facet in order to access the posterior vertebral body. The inferior aspect can be curved laterally towards the side of pathology. Advantage: a laminectomy can more easily be performed if needed (if the angle does not provide adequate visualization, as a "bail-out" contingency, a facetectomy may be performed, and pedicle may even be removed to access inferior to the disc space. This usually permits easy decompression of the entire thecal sac. In the thoracic spine, stabilization is optional, and if chosen, unilateral pedicle screws and fusion are usually adequate)

Rib removal and thoracic exposure: for a simple biopsy or drainage of a small abscess, removal of only 1 rib may suffice.  The rib to be removed is from the level inferior to the disc space to be accessed[116] (e.g. remove the T5 rib to access T4–5 disc space). For most other pathologies, 2 or 3 ribs are often removed.[117] To access a VB, the like-numbered rib and the rib below are removed.

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There are a number of ligaments attached to the rib: the intercostal neurovascular bundle (NVB) courses medial to the superior costotransverse ligament which extends from the superior aspect of the rib to the transverse process of the level above. This ligament and the lateral costotransverse ligament are divided and the transverse process is rongeured off (the base of which lies on the lamina directly posterior to the pedicle). This exposes the rib anterior to the transverse process. The periosteum is incised on the rib from the angle of the rib to the costovertebral articulation, and by subperiosteal dissection around its circumference the pleura is dissected off the anterior surface of the rib. The NVB is dissected from the deep-inferior surface along with the periosteum. The rib is then transected laterally at the angle (≈ 5 cm lateral to the rib head) with rib shears, it is gripped with a clamp, and is rotated while the ligaments (including the radiate ligaments which attach the rib to the both the VB above and the VB below the disc space at the superior and inferior costal facet, respectively, except T1, 11 & 12 which only articulate with their like-numbered VB) are sharply dissected off the rib which is then removed. The removed rib material may be used for fusion substrate except in cases of tumor or infection. The pleura is then dissected from the deep surface of the adjacent ribs and VB (taking care not to injure the segmental vessels and to dissect the sympathetic trunk off the VB with the pleura). The pleura is then retracted laterally with a malleable ribbon or Deaver retractor.

The intervertebral foramen of interest may be located by following the NVB of the rib above proximally, the intercostal nerve (the ventral ramus of the nerve root at that level) enters between the two pedicles. The dura may then be exposed by enlarging the neural foramen by removing part of the pedicles with a high-speed drill and Kerrison rongeurs.

Instrumentation/fusion are rarely required for simple discectomy. Instability due to fracture, tumor, or extensive resection (e.g. with total facet takedown) necessitates surgical stabilization, typically with pedicle screws/rods extending 2 levels above and 2 levels below. Prior to closure, check for air leak by filling the opening with saline and having the anesthesiologist apply a valsalva maneuver. If an air leak is identified, a Cook catheter may be placed into the pleural space through the surgical exposure, or alternatively a chest tube is placed through a separate intercostal incision after the laminectomy wound is closed. A post-op CXR is obtained regardless of whether an air leak is identified.

## Transpedicular approach

Drilling down the pedicle and removing a small amount of bone from the vertebral body, then pushing material from the epidural space into the defect created and removing it. Requires removal of just the rib head. Advantages: minimal risk of pneumothorax, more familiar anatomy. Disadvantages: requires instrumentation especially if done bilaterally, the angle is not very obliques so visualization of epidural space is minimal, may need to be done bilaterally if there are extensive bilateral components to the pathology.

## Booking the case: Transpedicular approach

Same as for costotransversectomy (p. 1062).

### Transthoracic approach

Indications: thoracic disc disease with central fragment or calcified disc, burst fractures of the thoracic spine, etc.

Advantages[118]:

- excellent anterior exposure (especially advantageous for multiple levels)
- little compromise of stability (due to supporting effect of rib cage)
- low risk of mechanical cord injury

Disadvantages:

- requires thoracic surgeon (or familiarity with thoracic surgery)
- some risk of vascular cord injury (due to sacrifice of intercostal arteries)
- definitive diagnosis may not be possible if it is uncertain prior to procedure

Possible complications:

- pulmonary complications: pleural effusion, atelectasis, pneumonia, empyema, hypoventilation
- CSF-pleural fistula



## Booking the case: Transthoracic spine surgery

Also see defaults & disclaimers (p. 27).

1. position: typically on the side, often on a beanbag
2. equipment:
   a) microscope (not used for all cases)
   b) C-arm
3. anesthesia: double lumen tube
4. implants: if post-op instability is anticipated, thoracic pedicle screws and possibly a cage (e.g. for fracture or tumor, not typically for disc herniation)
5. neuromonitoring: SSEP/MEP
6. blood availability: type and cross 2 U PRBC
7. some surgeons use chest surgeon for the approach, closure and for follow-up
8. consent (in lay terms for the patient – not all-inclusive):
   a) procedure: surgery through the chest with removal of a small piece of rib to permit removal of the herniated/calcified disc
   b) alternatives: nonsurgical management, surgery from the side or through the back
   c) complications: spinal cord injury with paralysis, pneumothorax, possible seizures with MEPs

### Key technical points

1. the services of an experienced thoracic surgeon are usually engaged
2. position: true lateral (facilitates intra-op localizing x-rays); approached from the more involved side. For the upper thoracic midline region, some prefer right-side-up to eliminate thoracic aorta from obstructing exposure and to reduce the possibility of encountering the artery of Adamkiewicz,[119] others prefer left-side-up to use the aorta as a landmark[118] (for levels below the cardiophrenic angle, a left-sided approach is preferred because the inferior vena cava is difficult to mobilize)
3. usually one rib is resected; most often the rib of the vertebra immediately above the disc space desired (facilitates exposure). Multiple ribs may be resected to increase exposure
4. when removing the vertebral body (VB) (corpectomy, e.g. for osteomyelitis, especially Pott's disease or for kyphoscoliosis)

a) the posterior cortex of the VB must be pulled anteriorly (e.g. with angled curettes) to avoid mechanical cord trauma
b) anterior fusion may be performed using the removed rib. If inadequate, fibula or iliac crest may be used

5. sizeable radicular arteries are spared. The intercostal nerve is used as a guide to the intervertebral foramen (nerve enters foramen superiorly and posteriorly)
6. the disc space is situated off the caudal aspect of the intervertebral foramen for most thoracic levels
7. one or two intervertebral arteries and veins usually have to be sacrificed; to minimize the risk of ischemic cord injury, cut them as close to the midline of the spine as possible (collaterals tend to lie on the lateral aspect of the spine)
8. the sympathetic chain is dissected off the VBs and is pushed posteriorly

## Lateral (retrocolic) approach)

See reference.[114]

The same instrumentation used for lateral lumbar interbody fusion (p.1498) may be used to access the lateral thoracic bodies for thoracic disc herniation.

Check the pre-op MRI for the location of the aorta and to rule-out aortic aneurysm. Above T11, enter on the right side. The access retractor is "reversed" so that the center blade is positioned anteriorly and the shim is place so that as the retractor is expanded in the AP direction, the lateral blades move posteriorly, giving more access to the posterior disc space. In general, do not penetrate the contralateral anulus because of proximity of aorta. At or just below L12-L1, the diaphragm attaches to the VB. A dual lumen endotracheal tube is not required. A chest tube is mandatory if there is an air leak, otherwise it is optional (a pigtail catheter may suffice).



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# 70 Cervical Disc Herniation

## 70.1 General information

Important applied anatomy in herniated cervical disc (HCD):

1. in the cervical region, the nerve root exits above the pedicle of its like-numbered vertebra (opposite to the situation in the lumbar spine, due to the fact that there are 8 cervical nerve roots and only 7 cervical vertebrae)
2. each root exits passes through its neural foramen in close relation to the undersurface of the pedicle
3. the intervertebral disc space is located close to the inferior portion of the pedicle (unlike the lumbar region)

## 70.2 Cervical nerve root syndromes (cervical radiculopathy)

### 70.2.1 General information

Due to the facts listed above, a HCD usually impinges on the nerve exiting from the neural foramen at the level of the herniation (e.g. a C6–7 HCD usually causes C7 radiculopathy). This gives rise to the characteristic cervical nerve root syndromes shown in Table 70.1.

### 70.2.2 Miscellaneous clinical facts

C4 radiculopathy is not common, and may produce nonradiating axial neck pain.

Left C6 radiculopathy (e.g. from C5–6 HCD) occasionally presents with pain simulating an MI (pseudo-angina).

C8 and T1 nerve root involvement may produce a partial Horner's syndrome.

The most common scenario for patients with herniated cervical disc is that the symptoms were present upon awakening in the morning, without identifiable trauma or stress.[1]



## 70.3 Cervical myelopathy and SCI due to cervical disc herniation

Acute cord compression presenting with myelopathy or spinal cord injury (SCI) (including complete SCI and incomplete syndromes, especially central cord syndrome (p.944) and sometimes Brown-Sequard syndrome (p.947) [2] is well described in association with traumatic cervical disc herniation.[3] Less commonly, these findings may occur in non-traumatic cervical disc herniation.

Table 70.1 Cervical disc syndromes

| Syndrome | Cervical disc syndromes | | | |
|---|---|---|---|---|
| | C4–5 | C5–6 | C6–7 | C7-T1 |
| % of cervical discs | 2% | 19% | 69% | 10% |
| compressed root | C5 | C6 | C7 | C8 |
| reflex diminished | deltoid & pectoralis | biceps & brachioradialis | triceps | finger-jerk[a] |
| motor weakness | deltoid | forearm flexion | forearm ext (wrist drop) | hand intrinsics |
| paresthesia & hypesthesia | shoulder | upper arm, thumb, radial forearm | fingers 2 & 3, all fingertips | fingers 4 & 5 |

[a]not everyone has a finger flexor reflex. Description: gently lift the fingertips of the patient's pronated hand and tap the underside of the fingers with a reflex hammer. When present, fingers flex in response

## 70.4 Differential diagnosis

See Differential diagnosis (p.1420).

## 70.5 Physical exam for cervical disc herniation

### 70.5.1 Overview

1. evaluation for radiculopathy
   a) lower motor neuron findings
      - weakness usually in one myotome group on one side
      - muscle bulk and tone: atrophy and fasciculations, may be present
   b) sensation: with nerve root compression, sensory loss will follow a dermatomal pattern and will be in the same nerve root distribution as the weakness
   c) muscle stretch reflexes
   d) mechanical signs: reproduction of radicular symptoms with axial loading of the head
2. evidence of spinal cord involvement (myelopathy)
   a) upper motor neuron findings, usually in the lower extremities
      - weakness may occur without atrophy or fasciculations
      - spasticity: poor control of the Legs when walking, scissoring of the legs
   b) sensation: any loss below the level of involvement will follow spinal cord patterns
      - complete loss
      - Brown-Sequard pattern: unilateral loss of pinprick with contralateral vibratory and position sense loss
      - Central cord syndrome: suspended sensory loss in upper extremities, less impaired below
      - Pathologic reflexes: Hoffmann's reflex, Babinski sign, ankle clonus



### 70.5.2 Signs useful in evaluating cervical radiculopathy

#### General information

Almost all herniated cervical discs cause painful limitation of neck motion. Neck extension usually aggravates pain when cervical disc disease is present (a minority of patients instead exhibit pain with flexion). Some patients find relief in elevating the arm and cupping the back or the top of the head with the hand (abduction relief sign, see below for shoulder abduction test). Lhermitte's sign (electrical shock-like sensation radiating down the spine) may be present; see DDx (p.1421).

#### Miscellaneous

The following tests were found to be specific, but not particularly sensitive in detecting cervical root compression[4]:

1. Spurling's sign[5]: radicular pain reproduced when the examiner exerts downward pressure on vertex while tilting head towards symptomatic side (sometimes adding neck extension). Causes narrowing of the intervertebral foramen and possibly increases disc bulge. Used as a "mechanical sign" analogous to SLR for lumbar disc herniation
2. axial manual traction: 10–15 kg of axial traction is applied to a supine patient with radicular symptoms (pull up on patient's mandible and occiput). The reduction or disappearance of radicular symptoms is a positive finding
3. shoulder abduction test[6]: a sitting patient with radicular symptoms lifts their hand above their head. The reduction or disappearance of radicular symptoms is a positive finding. Moderately sensitive, fairly specific[7]

## 70.6 Radiologic evaluation

### 70.6.1 MRI

The study of choice for initial evaluation for herniated cervical disc (HCD). Accuracy is less than water soluble contrast myelogram/CT (≈ 85–90% accuracy for MRI because of only fair to good imaging of neural foramen), but is non-invasive. For myelopathy, MRI is >95% effective in diagnosing.

Protocol:

1. sagittal T1WI
2. multiple echo cardiac gated sagittal images (Tr = 1560, Te = 25, 4th echo)

3. GRASS image: axial partial flip-angle fast scan (Tr = 25, Te = 13, angle = 8°). Dark material adjacent to disc space is bone, disc is higher signal, CSF and flowing blood are high signal.

### 70.6.2 CT and myelogram/CT

Indications: when MRI cannot be done or when more bony detail than what MRI provides is required. Evaluates for ossification of the posterior longitudinal ligament (OPLL) when suspected.

Plain CT: is usually good at C5–6, is variable at C6–7 (due to artifact from patient's shoulders, depending on body habitus), and is usually poor at C7-T1.

Myelogram/CT (water soluble intrathecal contrast): invasive, on rare occasions requires overnight hospitalization. Accuracy is ≈ 98% for cervical disc disease.

### 70.6.3 Electrodiagnostics (EMG and NCV)

Compression may occur at the level of the dorsal (pre-ganglionic) sensory root (which, if occurs alone, produces a sensory-only radiculopathy) and/or at the ventral (motor) root. When motor exam is normal, EMG is unlikely to show abnormality. The AANEM practice parameter for cervical radiculopathy[8,9,10] reports sensitivity of 50-71% for the needle EMG examination and correlation between positive needle EMG and radiologic findings of 65-85%

EMG can also be normal in sensory only radiculopathy, which occasionally occurs in cervical spine, but not in lumbar spine. Since most muscles have at least dual innervation this poses a particular challenge for proximal cervical radiculopathies in which many muscles have the same shared innervation, e.g. biceps, deltoid, brachioradialis, infraspinatus and supraspinatus are all innervated by C5-C6.

For both cervical and lumbosacral radiculopathy, screening 6 muscles representing all root levels to include paraspinal muscles yield consistently high identification rates.[11]

For muscles to demonstrate fibrillations and positive waves there must be axonal loss in the motor nerve axons which innervates a muscle. Muscle demonstrates fibs and positive waves within 1 to 2 weeks following loss of innervation depending on the distance from the nerve to the muscle.

NCV is helpful to assess for peripheral neuropathies which may have symptoms similar to radiculopathy (e.g. carpal tunnel syndrome vs. C6 radiculopathy; ulnar neuropathy vs. C8 radiculopathy). A good physical exam can differentiate these entities in most cases.



#### Practice guideline: EDX guidelines for cervical radiculopathy

See reference.[10]

1. Guideline: EMG needle examination:
   a) needle examination of at least 1 muscle innervated by C5, C6, C7, C8 and T1 spinal roots in a symptomatic limb
   b) cervical paraspinal muscles at 1 or more levels (except in patients with prior posterior approach cervical surgery
   c) if abnormalities are identified, perform studies of 1 or 2 additional muscles innervated by the suspected root and different peripheral nerve
2. Guideline: At least 1 motor and 1 sensory nerve conduction study (NCS) in the clinically involved limb to determine if there is concomitant polyneuropathy or nerve entrapment. Motor and sensory NCS of median and ulnar nerves if symptoms and signs suggest CTS or ulnar neuropathy. If 1 or more NCS are abnormal or if clinical features suggest polyneuropathy, further evaluation may include NCS of other nerves in the ipsilateral and contralateral limb

## 70.7 Treatment

### 70.7.1 General information

Over 90% of patients with acute cervical radiculopathy due to cervical disc herniation can improve without surgery,[12] and regression of an extruded cervical disc has been demonstrated radiographically by CT and MRI.[13,14,15] The recovery period may be made more tolerable by adequate pain medication, anti-inflammatory medication (NSAIDs or short-course tapering steroids) and intermittent cervical traction (e.g. gradually escalating up to 10–15 lbs for 10–15 minutes, 2–3 × daily).

Surgery is indicated for those that fail to improve or those with progressive neurologic deficit while undergoing non-surgical management.

Management of myelopathy/central cord syndrome associated with acute cervical disc herniation is controversial, since the natural history is favorable in most cases. However, some patients have poor recovery and experience permanent deficits even with emergency surgery.[16]

## 70.7.2 Conservative management

Modalities include:

1. physical therapy, which may also include cervical traction.
2. Interventional pain management
   a) Trigger point injections
   b) Facet blocks
   c) Epidural steroid injection: not used as often and with lumbar spine

## 70.7.3 Surgery

### Surgical options

1. anterior cervical discectomy: see below
   a) without any prosthesis or fusion: rarely used today
   b) combined with interbody fusion: the most common approach
      - without anterior cervical plating
      - with anterior cervical plating or with zero profile
   c) with artificial disc AKA cervical disc arthroplasty
2. posterior approaches
   a) cervical laminectomy: not typically used for a herniated cervical disc, more common for cervical spinal stenosis, OPLL
      - without posterior fusion
      - with lateral mass fusion
   b) keyhole laminotomy: sometimes permits removal of disc fragment



For practice guidelines regarding intra-op electrophysiologic, see monitoring for surgery for cervical radiculopathy (p.1090).

### Anterior cervical discectomy with fusion (ACDF)

Without special modifications, a routine anterior approach is usually able to access levels C3–7. In patients with short thick necks, access may be even more limited. In some cases, with long thin necks, up to C2–3 or as low as C7-T1 can be approached anteriorly.

Advantages over posterior (nonfused) approach:

1. safe removal of anterior osteophytes
2. fusion of disc space affords immobility (up to 10%incidence of subluxation with extensive posterior approach)
3. only viable means of directly dealing with centrally herniated disc

Disadvantages over posterior approach: immobility at fused level may increase stress on adjacent disc spaces. If a fusion is performed, some surgeons prescribe a rigid collar (e.g. Philadelphia collar) for 6–12 weeks. Multiple level ACDF can devascularize the vertebral body (or bodies) between discectomies.

### Booking the case: ACDF

Also see defaults & disclaimers (p.27).

1. position: supine, some use halter traction with this
2. equipment:
   a) microscope (not used by all surgeons)
   b) C-arm
3. implants: graft (e.g. PEEK, cadaver bone, titanium cage…) and anterior cervical plate (optional, especially on single level ACDF)

4. neuromonitoring: (optional) some surgeons used SSEP/MEP
5. consent (in lay terms for the patient – not all-inclusive):
   a) procedure: surgery through the front of the neck to remove the degenerated disc and bone spurs, and to place a graft where the disc was, and possibly place a metal plate on the front of the spine. Some surgeons take bone from the hip to replace the removed disc
   b) alternatives: nonsurgical management, surgery from the back of the neck, artificial disc (in some cases)
   c) complications: swallowing difficulties are common but usually resolve, hoarseness of the voice (<4%chance of it being permanent), injury to: foodpipe (esophagus), windpipe (trachea), arteries to the brain (carotid), spinal cord with paralysis, nerve root with paralysis, possible seizures with MEPs

## Technique

A summary of the steps involved is included here. For C5–6, the skin incision is made at level of cricoid cartilage, for other levels, appropriate adjustments up or down may be made, sometimes with the assistance of fluoroscopy. The incision is approximately 4–5 cm horizontally, centered on the SCM. Many right handed surgeons prefer operating from the right side of the neck, although the risk to the recurrent laryngeal nerve (RLN) is lower with a left sided approach (the RLN lies in a groove between the esophagus and trachea). The skin may be undermined off the platysma to permit a vertical incision in the platysma in the same orientation as its muscle fibers. Alternatively, some incise the platysma horizontally with scissors horizontally.

Dissect in tissue plane medial to SCM. For the C5–6 interspace, angle slightly cranially during dissection. For the C6–7 disc, proceed almost straight down to spine. Sweep omohyoid medially (to stay out of it and to protect the RLN). The trachea + esophagus are retracted medially. The carotid sheath + SCM are retracted laterally.



After verification of level with lateral C-spine x-ray with spinal needle in the interspace, bipolar the prevertebral fascia and medial edges of the longus coli muscles longitudinally in the midline. Self-retaining retractor blades are inserted underneath the fascia to retract the longus coli muscles laterally. The anesthesiologist is asked to deflate the cuff on the endotracheal tube and then to re-inflate it using minimal leak technique to reduce the risk of compression injury from the retractor. The disc space is incised with a 15 scalpel blade. The discectomy is performed with curettes and pituitary rongeurs; a vertebral body spreader aids the exposure. The posterior longitudinal ligament is incised, one technique is to elevate it with a sharp nerve hook and then incise it with a #11 scalpel. The subligamentous space is probed with a blunt nerve hook. The posterior lip of the VB above and below are removed with a Kerrison rongeur with a small foot-plate. Decompression of the roots is verified with the blunt nerve hook. Fusion is performed at this time if desired by placing the graft in the interspace.

For redo operations (same or different levels): approach is usually from the same side as previous operation(s) since many patients have swallowing issues post-op, and some may be due to partial recurrent laryngeal nerve injury (which can be subclinical) and which could result in a permanent need for a feeding tube if a contralateral injury occurs. If for some reason it is desired to go to the opposite side, an evaluation by an ENT physician is recommended, and should include scoping the patient to rule-out subclinical problems that could turn into major difficulties if bilateral.

## Choice of graft material

Autologous bone (usually from iliac crest), non-autologous bone (cadaveric), bone substitutes (e.g. hydroxylapatite[17]) or synthetics (e.g. PEEK or titanium cage) filled with osteogenic material. Substitutes for autologous bone eliminate problems with the donor site (p.1075), but may have a higher rate of absorption. There were also cases of HIV transmission from cadaveric bone grafts in 1985, however, as a result of the heightened awareness of AIDS since that time together with significant improvements in antibody testing and careful screening of donors, no further cases have been reported.

## Anterior cervical plating

Recommendations for plating following ACDF are shown in Practice guideline: Anterior cervical plating (p.1074).

## Practice guideline: Anterior cervical plating

1 level ADCF: The addition of an anterior plate to an ACDF is recommended to reduce the pseudarthrosis rate and graft problems (Level D Class III) and to maintain lordosis (Level C Class II) but it does not improve clinical outcome alone (Level B Class II)[18]

2 level ADCF: Plating is recommended to improve arm pain. Plating does not improve other outcome parameters (Level C Class II)[18]

Use of bone morphogenic proteins (BMP)

## Practice guideline: Use of BMP in cervical interbody grafting

Current evidence does not support the routine use of rhBMP-2 for cervical arthrodesis (Level C Class II)[19] (note: italics added. Use with precautions (see text) may be indicated in cases with high risk of nonunion).

Use of BMP in anterior cervical discectomies is not FDA approved, but has been used off-label. Complication rates as high as 23–27% have been reported (including post-op swallowing or respiratory difficulties as a result of edema which is usually temporary) compared to 3% without BMP.[19] If used, it is recommended that a smaller dose be employed than in the lumbar spine (25% has been advocated) and to avoid contact of BMP with soft tissues in the neck.



### Post-op check

In addition to routine, the following should be checked

1. evidence of airway obstruction – post-op wound hematoma: should be first consideration. Wound may need to be emergently opened at bedside (before getting to the OR) if airway is compromised, see Carotid endarterectomy, disruption of arteriotomy closure, management (p. 1294). Also consider swelling from IJV thrombosis (rare) in differential diagnosis (see below)
   a) respiratory distress
   b) extreme difficulty swallowing: alternatively may indicate anterior extrusion of bone graft impinging upon esophagus (check lateral C-spine x-ray)
   c) tracheal deviation: may be visible or may be seen on AP C-spine x-ray
2. weakness of nerve root of level operated: e.g. biceps for C5–6, triceps for C6–7
3. long tract signs (Babinski sign…) which may indicate cord compression by spinal epidural hematoma
4. hoarseness: may indicate vocal cord paresis from recurrent laryngeal nerve injury: hold oral feeding until this can be further assessed

### ACDF complications

#### General information

Common ones listed below, see references[20,21] for more details. The most common complication following ACDFs: swallowing difficulties (may be multifactorial).

1. exposure injuries
   a) perforation of viscus: minimize risk by blunt retraction until longus colli is separated from its attachment to vertebrae
      - pharynx
      - esophagus: difficult to manage, and require multidisciplinary effort including ENT involvement.[22] The incidence may be higher with use of anterior cervical plate, and injury may not manifest until years after the fusion (may be due to repetitive motion of esophagus over the plate). Treatment of esophageal perforation is usually facilitated by plate removal
      - trachea
   b) vocal cord paresis: due to injury of the recurrent laryngeal nerve (RLN) or vagus. Incidence: 11% temporary, 4% permanent paresis. Symptoms include: hoarseness, breathiness, cough, aspiration, mass sensation, dysphagia, and vocal cord fatigue.[23] Avoid sharp dissection in paratracheal muscles. Some cases may be due to prolonged retraction against trachea and not to nerve division; to reduce this risk, after the self-retaining retractor is placed, have the

anesthesiologist deflate the cuff on the ET tube and then inflate it to minimal leak pressure. More common with right sided approaches, primarily in the lower cervical spine (C5–6 and below) where the RLN is more vulnerable[23]

c) vertebral artery injury: thrombosis or laceration. 0.3%incidence.[21] Treatment alternatives include: packing, direct repair by temporary clipping with aneurysms clips and repair with 8–0 prolene[24] and endovascular trapping. Risks of treating hemorrhagic complications with packing include: recurrent bleeding, AV fistula, pseudoaneurysm, arterial thrombosis,[21] distal embolic stroke (primarily in cerebellum)
d) carotid injury: thrombosis, occlusion, or laceration (usually by retraction)
e) CSF fistula: usually difficult to repair directly. Place fascial graft beneath bone plug. Keep HOB elevated post-op. Consider: dural sealant (fibrin glue, DuraSeal®…), lumbar drain
f) Horner's syndrome: sympathetic plexus lies within longus coli, thus do not extend dissection far laterally into these muscles
g) thoracic duct injury: in exposing lower cervical spine, primarily on left
h) thrombosis of internal jugular vein[25]: rare. Carries 2–3%risk of PE.[26] Treatment options: anticoagulation (oral or IV) may lower the mortality,[27] SVC filter if anticoagulation is contraindicated,[28] percutaneous thrombectomy[29]

2. spinal cord or nerve root injuries
   a) spinal cord injury: especially risky in myelopathy due to narrowed canal. Minimize risk by penetrating the osteophyte at the lateral margin of interspace (however, this increases risk to nerve root)
   b) avoid hyperextension during intubation: anesthesiologist may need to determine patient's tolerance pre-op. Consider fiberoptic guided or awake nasotracheal intubation in extreme stenosis
   c) bone graft must be shorter than interspace depth. Exercise caution in tapping graft into position
   d) sleep induced apnea: rare but serious complications of C3–4 level operations.[30] May be associated with bradycardia & cardiorespiratory instability. Possibly due to disruption of the afferent component of the central respiratory control mechanism
3. bone fusion problems
   a) failure of fusion (pseudarthrosis): see below
   b) anterior (kyphotic) angulation deformity: may be as high as 60%with Cloward technique (may be reduced by collar immobilization). May develop in Hirsch technique with excessive bone removal
   c) graft extrusion: 2%incidence (rarely requires re-operation unless compression of cord posteriorly, or esophagus or trachea anteriorly occurs)
   d) donor site complications: hematoma/seroma, infection, fracture of ilium, injury to lateral femoral cutaneous nerve, persistent pain due to scar, bowel perforation
4. miscellaneous
   a) wound infection: incidence < 1%
   b) post-op hematoma: see above. Placing cervical collar in O.R. may delay recognition
   c) dysphagia and hoarseness: common. Usually transient (see below)
   d) adjacent level degeneration: controversial whether this represents a sequelae to altered biomechanics from surgery, or a predisposition to cervical spondylosis.[31] Many (≈ 70%) are asymptomatic[32]
   e) postoperative discomfort:
      - globus: the sensation of a lump in throat (see below)
      - nagging discomfort in neck, shoulder, and very commonly in interscapular regions (may last several months). May correlate with amount of distraction of the disc space
   f) complex regional pain syndrome (CRPS) AKA reflex sympathetic dystrophy (RSD): rarely described in the literature,[33] possibly due to stellate ganglion injury; see discussion of RSD (p.497)
   g) angioedema: massive edema of the tongue and neck.[34] A dramatic hypersensitivity reaction (not really a direct complication of ACDF, but superficially can mimic some findings of post-op hematoma). If limited to the tongue, the airway is not compromised. See treatment (p.222)
   h) pneumothorax or hemothorax[35]: accessing C7-T1 or lower may expose the pleural apex



## Dysphagia following ACDF

Symptoms: Include: difficulty swallowing (solids, liquids including saliva), pain with swallowing (odynophagia), globus (sensation of a lump in throat) and compromise of ability to protect against aspiration. Food may stick in the throat (or feel as if it is) and there may be coughing or choking.

Incidence: Early dysphagia is common. Incidence: 60%[36] in retrospective survey after noninstrumented fusion (dysphagia occurred in 23% in a control group undergoing unrelated lumbar spine surgery[36]), 50% in prospective study.[37] At 6 months, only ≈ 5% reported moderate or severe dysphagia.[37] Surgery at multiple levels increased the risk at 1 & 2 months.[37] Decreases significantly in most cases by 6 months.[37]

Etiologies: Etiologies of post-op dysphagia include:

1. post-op hematoma. If severe, may cause tracheal obstruction (see above)
2. post-op edema, due in part to retraction of esophagus
3. effects of general anesthesia: e.g. irritation from ET tube. Accounts for up to 23% of early symptoms (dysphagia occurred in 23% in a control group undergoing unrelated lumbar spine surgery[36]). Usually subsides within ≈ 24–72 hours
4. recurrent laryngeal nerve dysfunction:
   a) temporary: usually due to traction on the nerve
   b) permanent: 1.3% at 12 months[37]
5. esophageal injury
   a) at time of surgery
   b) delayed: possibly from repetitive abrasion on surgical site/hardware or from unrecognized esophageal injury at time of surgery[22]
6. cervical collar
   a) prevents patient from lowering jaw during swallow phase, which compromises effective glottic closure of airway
   b) may be too tight thereby directly squeezing the throat
7. protrusion of graft/hardware anterior to the vertebral bodies
   a) some protrusion is present with most anterior hardware. This may be minimized with "zero profile" instrumentation
   b) hardware failure (screw-pullout/backout/breakage, plate pullout)
   c) interbody graft migration: without anterior plate, or in conjunction with anterior plate displacement
8. excessive adhesions[38]
9. denervation of the pharyngeal plexus[38]
10. rare conditions: swelling from IJV thrombosis, angioedema

Management:

1. initial management: rule-out emergent/serious conditions (severe edema, hematoma with airway compromise, risk of aspiration)
   a) if there is significant stridor or dysphonia, especially if tracheal deviation is obvious, someone must stay with the patient as efforts are made to emergently take the patient to the O.R. for wound exploration evacuation. Consider opening the wound at the bedside if delays occur or if symptoms are severe; see Carotid endarterectomy, disruption of arteriotomy closure, management (p. 1294). Emergent anesthesia consultation for airway protection – alert them to the likelihood of deviated trachea which challenges even the most expert at intubating
2. once emergent conditions are ruled out, early management is geared towards amelioration of symptoms
   a) advise patient to eat softer foods (temporarily avoiding steak or bread), to chew food well, to wash down dry foods with a drink. Reassure patient that most cases largely resolve with 6 months[37]
   b) if significant symptoms persist >2 weeks
      - refer patient to ENT for laryngoscopy to rule out vocal cord paralysis (from RLN injury) or other etiologies. For RLN, see below
      - modified barium swallow
3. persistent symptoms may be amenable to surgical intervention, including hardware removal and lysing of adhesions,[38] management of esophageal perforation usually requires consultation with ENT

Management of esophageal perforation:

There is no consensus on optimal management. A multidisciplinary approach with head and neck surgeons together with the spine surgeon is suggested with the following[22]:

- Closure with a sternocleidomastoid muscle flap, or possibly a pedicle flap
- Removal of all anterior hardware. If there is evidence that the fusion is not solid, posterior instrumentation may be necessary

### Recurrent laryngeal nerve paralysis

Heralded by breathy voice, hoarseness, or aspiration. Refer to ENT to have patient scoped to determine if cord is paralyzed and its position. 4 possible positions: 1) median, 2) paramedian, 3) intermediate, 4) lateral (cadaveric). Many patients can compensate for median or paramedian position. Patients requiring intervention usually are treated with medialization technique, either 1) injection, or 2) medialization thyroplasty using an implant. For injections, different materials may be selected for desired duration of effect (Teflon used to be the only available agent, and was essentially permanent) and so early intervention may be employed with temporary materials (instead of waiting 1 year, which was the previous paradigm).

### Pseudarthrosis (or pseudoarthrosis) following ACDF

Pseudarthrosis may occur with or without supplemental anterior cervical plating.

## Practice guideline: Assessment of subaxial fusion

>2 mm movement between spinous processes on dynamic (flexion-extension) cervical spine x-rays is recommended as a criteria for pseudarthrosis (Level B Class II), this measurement is unreliable when performed by the treating surgeon (Level C Class II).[39]

Visualization of bone trabeculation across the fusion on static films is a less reliable marker for fusion (Level D Class III) (2D reformatted CT increases the accuracy (Level D Class III))[39]



Incidence: Difficult to assess because of lack of validated criteria. Estimate: 2–20% Higher with dowel technique (Cloward) than with keystone technique of Bailey & Badgley or with interbody method of Smith-Robinson (10%) or with non-fusion advocated by Hirsch. One criteria: motion >2 mm between the tips of the spinous processes on lateral flexion/extension x-rays.[40,41] Other criteria: lucencies around the screws of an anterior plate, toggling of the screws on flexion/extension x-rays.

Presentation: Not uniformly associated with symptoms or problems.[40,42] Some patients may have chronic or recurrent neck pain, some may present with radicular symptoms. (NB: when DePalma's data is analyzed with patients reclassified as failures if neck and/or arm symptoms persist, the success rate of surgery is lower with pseudarthrosis[43]).

Management: Guidelines are shown in Practice guideline: Management of anterior cervical pseudarthrosis (p.1077). No treatment is required for asymptomatic pseudarthrosis. Options for symptomatic patients include re-resection of the bone graft with repeat fusion[44] (some recommend using autologous bone if allograft was used, a plate may be considered if one was not used previously), cervical corpectomy with fusion,[44] or posterior cervical fusion.

## Practice guideline: Management of anterior cervical pseudarthrosis

Revision of symptomatic pseudarthrosis should be considered (Level D Class III).[45] Posterior approaches may be associated with higher fusion rates on revision than anterior approaches (Level D Class III)[45]

### Cervical disc arthroplasty

An alternative to fusion. Uses an artificial disc to preserve motion at the level of the discectomy. Some of the available cervical disc replacement (CDR) models are shown in Table 70.2.[46]

Contraindications described by the FDA have included: isolated axial neck pain, ankylosing spondylitis or pregnancy, rheumatoid arthritis, autoimmune disease, diffuse idiopathic skeletal hyperostosis, severe spondylosis with bridging osteophytes or ossification of the posterior longitudinal ligament, disc height loss >50%, spinal infection, metal allergy to components of the prosthesis, severe osteoporosis/osteopenia, active malignancy, metabolic bone disease, trauma, segmental instability, 3 or more levels requiring treatment, insulin dependent diabetes mellitus, human immunodeficiency virus, hepatitis B/C, morbid obesity, absence of motion (<2 degrees), and posterior facet arthrosis.

Table 70.2 Artificial cervical discs

| Trade name | Manufacturer | Material | IAR[a] | Comment |
|---|---|---|---|---|
| Prestige® | Medtronic | MOM[a] (chrome cobalt stainless steel) | variable ball in trough | 1st FDA approved CDR; lots of MRI artifact |
| Bryan® | Medtronic | lubricated elastic nucleus sealed in a flexible membrane | variable in center of disc space | |
| Advent® | Blackstone (Orthofix) | flexible elastomer core | | withdrawn from market |
| ProDisc-C | Synthes | metal-on-polyethylene | posterior part of inferior VB | midline keel inserts into VB; lots of MRI artifact |
| Mobi-C® | LDR Spine | metal-on-polyethylene | variable in center of disc space | approved for 1 or 2 levels |
| PCM® | Cervitech | metal-on-polyethylene | gliding motion | contoured to endplates |

[a]Abbreviations: IAR = instantaneous axis of rotation, MOM = metal-on-metal, PCM = Porous-coated Motion

## Practice guideline: Cervical disc arthroplasty

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Cervical arthroplasty is a recommended alternative to ACDF in selected patients for control of arm and neck pain (Level B Class II)[18]

## Booking the case: Cervical disc arthroplasty

Also see defaults & disclaimers and/or more significant (p. 27).

1. position: supine, some use halter traction with this
2. equipment:
   a) microscope (not used by all surgeons)
   b) C-arm
3. implants: schedule vendor to provide desired artificial disc
4. neuromonitoring: (optional) some surgeons used SSEP/MEP
5. consent (in lay terms for the patient – not all-inclusive):
   a) procedure: surgery through the front of the neck to remove the degenerated disc and bone spurs, and to place an artificial disc
   b) alternatives: nonsurgical management, surgical fusion (from the front or the back of the neck)
   c) complications: swallowing difficulties are common but usually resolve, hoarseness of the voice (<4%chance of it being permanent), injury to: foodpipe (esophagus), windpipe (trachea), arteries to the brain (carotid) with stroke, spinal cord with paralysis, nerve root with paralysis, possible seizures with MEPs (if used). The disc may eventually wear out and further surgery may be needed

Post-op orders:

1. no cervical collar (the goal is to preserve motion at the operated level)
2. NSAIDs around the clock for ≈ 2 weeks (this inhibits bone growth which theoretically helps avoid undesirable fusion at the operated level)

## Posterior cervical decompression (cervical laminectomy)

Not necessary for unilateral radiculopathy (use either ACD or keyhole laminotomy). Consists of removal of cervical lamina (laminectomy) and spinous processes in order to convert the spinal canal from a “tube” to a “trough.”

Usually reserved for the following conditions:

1. multiple cervical discs or osteophytes (anterior cervical discectomy (ACD) is usually used to treat only 2, or possibly 3, levels without) with myelopathy
2. where the anterior pathology is superimposed on cervical stenosis, and the latter is more diffuse and/or more significant (p.1083)
3. in professional speakers or singers where the 4%risk of permanent voice change due to recurrent laryngeal nerve injury with ACD may be unacceptable

## Booking the case: Cervical laminectomy

Also see defaults & disclaimers (p.27).

1. position: prone, some use pin headholder
2. equipment:
   a) C-arm
   b) high-speed drill
3. implants: cervical lateral mass screws and rods if fusion is being done
4. neuromonitoring: some surgeons used SSEP/MEP
5. consent (in lay terms for the patient – not all-inclusive):
   a) procedure: surgery through the back of the neck to remove the bone over the compressed spinal cord and nerves and possibly to place screws and rods to fuse the boned together
   b) alternatives: nonsurgical management, surgery from the front of the neck, posterior surgery without fusion, laminoplasty
   c) complications: nerve root injury (C5 nerve root is the most common), may not relieve symptoms necessitating further surgery, possible seizures with MEPs. If fusion is not done, risk of progressive bone slippage which would require further surgery



### Posterior keyhole laminotomy

AKA "keyhole foraminotomy." First described in 1951.[47] A technique to decompress only individual nerve roots (but not the spinal cord) by creating a small "keyhole" in the lamina to access the nerve root.

## Practice guideline: Cervical laminoforaminotomy

Cervical laminoforaminotomy is recommended as a surgical treatment option for symptomatic cervical radiculopathy caused by disc herniation or lateral recess narrowing (Level D Class III)[48]

Indications for keyhole approach (as opposed to anterior discectomy):

1. monoradiculopathy with posterolateral soft disc sequestration (small lateral osteophytic spurs may also be addressed). This approach does not provide adequate decompression with central or broad-based disc herniation or with stenosis of the spinal canal
2. radiculopathy in patients who are professional speakers or singers where the risk of recurrent laryngeal nerve injury is untenable (see above)
3. for lower (e.g. C7, C8 or T1) or upper (e.g. C3 or C4) cervical nerve root compression, especially in a patient with a short thick neck, making an anterior approach more difficult
4. in patients with a herniated disc when it is desired to avoid a fusion (as would be done with an anterior approach)

## Booking the case: Cervical keyhole laminectomy

Also see defaults & disclaimers (p.27).

1. position: prone, some use pin headholder
2. equipment:
   a) microscope (not used by all surgeons)
   b) C-arm

3. instrumentation: some surgeons use a tube retractor system
4. neuromonitoring: some surgeons used SSEP/MEP
5. consent (in lay terms for the patient – not all-inclusive):
   a) procedure: surgery through the back of the neck to remove the bone over the compressed nerve root and possibly remove fragment of herniated disc
   b) alternatives: nonsurgical management, surgery from the front of the neck, posterior surgery with fusion
   c) complications: nerve root injury, may not relieve symptoms necessitating further surgery, possible seizures with MEPs

## Technique

See references.[49,50,51]

Position:

a) prone, on chest rolls. Adhesive tape is used to retract shoulders down for any level below about C4–5. The head is stabilized on a horse-shoe headrest or in a Mayfield head holder.
b) sitting position: generally abandoned. However, may be used with proper precautions (p.1445)

## "Open" keyhole foraminotomy



The desired level is localized with intra-op fluoroscopy before making the skin incision, a 2–3 cm midline incision is adequate. A unilateral exposure suffices. Periosteal elevators are used to dissect muscles off the lamina and facet joint in the sub-periosteal plane. A Kocher clamp may be placed on the spinous process to permit confirmation of the correct level on intra-operative x-ray. A Scoville retractor or equivalent is employed.

A high-speed drill (e.g. with diamond burr) is used to make an opening in the medial one-third to one-half of the inferior facet of the vertebra above the desired disc space, extending slightly medially into the junction with the lamina. Once the inferior facet is penetrated, the superior facet of the inferior vertebral level will be visualized. This is also thinned with the drill (it is critical to remove the bone of the superior facet of the level below caudally to where it meets the pedicle). A small Kerrison rongeur may be used to slightly enlarge the laminectomy. An opening is made in the ligamentum flavum overlying the lateral aspect of the spinal cord dura. The nerve root can be identified as it exits from the thecal sac, and can be followed as it travels between the pedicles of the vertebrae above and below. Soft tissues (including ligamentum flavum) form fibrous bands across the dorsum of the nerve, and are removed to further expose the dura of the nerve root. The venous plexus around the nerve root is coagulated with bipolar cautery and then divided to mobilize the nerve. The nerve may then be gently moved a few millimeters rostrally using a micro nerve hook. The dura overlying the spinal cord should not be manipulated, and the disc space need not be entered. Inspection for free disc fragments should begin in the nerve root axilla using a probe (e.g. blunt nerve hook). Next, the space anterior to the root (the region of the disc) may be palpated. Any disc fragments that are dislodged are removed with a small pituitary rongeur. If the disc fragment is contained anterior to the posterior longitudinal ligament (PLL), the PLL may be incised in the region of the nerve root axilla with a #11 scalpel blade in a motion that is directed downward and laterally, away from the nerve root and spinal cord. The foraminotomy may be extended slightly laterally if the foramen still feels tight when probed. Small osteophytes can potentially be reduced using a small reversed-angled curette, although some surgeons believe that the need for this is obviated by the decompression provided by the keyhole opening. In some cases, simple posterior decompression of the nerve root (without removing a disc fragment) may be adequate to relieve compression. Spinal stability is usually preserved if less than half the facet joint is removed.

## MIS keyhole foraminotomy

Positioning as described above.

1. skin incision
   a) use fluoro to locate the correct level for the incision
   b) incision 1 cm off midline on the side of the pathology at the level of the disc space
   c) remove adhesive plastic barrier (e.g. Ioban®) from around the opening to prevent pieces from being dragged into the incision

2. avoid using a guidewire to reduce the risk of penetrating the interlaminar space. STAY LATERAL and insert the thinnest dilator. Dock the dilator on the lateral mass and insert progressively sized dilators
3. use Bovie to expose lateral lamina and medial facet joint. Start laterally where bone is more easily felt and there is little danger of penetrating the interlaminar space and injuring the spinal cord
4. use a straight curette to expose the inferior edge of the superior lateral lamina and the medial facet joint
5. drill off the medial inferior facet, to expose the superior facet of the level below
6. drill the medial superior facet until you are flush with the superior aspect of the pedicle below
7. this completes the bony work, the soft tissue work proceeds as above under open keyhole foraminotomy

## Outcome

A number of large series have reported good or excellent outcome in the range of 90–96%[50]

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# 71 Degenerative Cervical Disc Disease and Cervical Myelopathy

## 71.1 General information

Cervical degenerative disc disease is generally discussed in terms of "cervical spondylosis", a term which is sometimes used synonymously with "cervical spinal stenosis." Spondylosis usually implies a more widespread age-related degenerative condition of the cervical spine including various combinations of the following:

1. congenital spinal stenosis (the "shallow cervical canal"[1])
2. degeneration of the intervertebral disc producing a focal stenosis due to a "cervical bar" which is usually a combination of:
   a) osteophytic spurs ("hard disc" in neurosurgical jargon)
   b) and/or protrusion of intervertebral disc material ("soft disc")
3. hypertrophy of any of the following (which also contributes to canal stenosis):
   a) lamina
   b) dura
   c) articular facets
   d) ligaments, including
      - increased stenosis in extension is more common than with flexion (based on MRI studies[2] and cadaver studies), largely due to posterior inbuckling of ligamentum flavum[3]
      - posterior longitudinal ligament: may include ossification of the posterior longitudinal ligament (OPLL) (p. 1127).[4] May be segmental or diffuse. Often adherent to dura
      - ossification of the ligamentum flavum[5] (yellow ligament)
4. subluxation: due to disc and facet joint degeneration
5. altered mobility: severely spondylotic levels may be fused and are usually stable, however there is often hypermobility at adjacent or other segments
6. telescoping of the spine due to loss of height of VBs → "shingling" of laminae
7. alteration of the normal lordotic curvature[6] (NB: the amount of abnormal curvature did not correlate with the degree of myelopathy)
   a) reduction of lordosis: including
      - straightening
      - reversal of the curvature (kyphosis): may cause "bowstringing" of the spinal cord across osteophytes
   b) exaggerated lordosis (hyperlordosis): the least common variant (may also cause bowstringing)

Although the majority of individuals > 50 yrs old have radiologic evidence of significant degenerative disease of the cervical spine, only a small percentage will experience neurologic symptoms.[7]



## 71.2 Pathophysiology

Pathogenesis is controversial. Theories include the following alone or in combination:

1. direct cord compression between osteophytic bars and hypertrophy or infolding of the ligamentum flavum, especially if superimposed on congenital narrowing or cervical subluxations
2. ischemia due to compression of vascular structures[8] (arterial deprivation[9] and/or venous stasis[10])
3. repeated local cord trauma by normal movements in the presence of protruded discs and/or osteophytic (spondylotic) bars (cord and root injuries[11])
   a) cephalad/caudad movement with flexion extension[12]
   b) anterior/posterior traction on the cord by dentate ligaments[13] & nerve roots
   c) diameter of spinal canal varies during flexion and extension
      - increased stenosis is more common in extension (see above)
      - unstable segments may sublux (so-called pincer mechanism)[14]

Histologically,[15] there is degeneration of the central grey matter at the level of compression, degeneration of the posterior columns above the lesion (particularly in the anteromedial portion), and demyelination in the lateral columns (especially the corticospinal tracts) below the lesion. Anterior spinal tracts are relatively spared. There may be atrophic changes in the ventral and dorsal roots and neurophagia of anterior horn cells.

## 71.3 Clinical

### 71.3.1 General information

Cervical spondylosis condition may produce several types of clinical problems[16]:

1. Myeloradiculopathy: some combination of
   a) Radiculopathy: nerve root compression may cause nerve-root (radicular) complaints
   b) spinal cord compression may cause myelopathy. Some stereotypical syndromes are presented below (see Cervical spondylotic myelopathy (CSM) below)
2. pain and paresthesias in the head, neck and shoulders with little or no suggestion of radiculopathy nor abnormal physical findings. This group is the most difficult to diagnose and treat, and often requires a good physician-patient relationship to decide if surgical treatment should be undertaken in an attempt to provide relief

Cervical spondylosis is the most common cause of myelopathy in patients >55 yrs of age.[17] CSM is rare in patients <40 years of age.

Cervical spondylotic myelopathy (CSM) develops in almost all patients with ≥30% narrowing of the cross-sectional area of the cervical spinal canal[18] (although some patients with more severe cord compression do not have myelopathy[19,20]).

Gait disturbance, often with LE weakness or stiffness, is a common early finding in CSM.[21] Ataxia may result from spinocerebellar tract compression. Early on, patients may experience difficulty running. Cervical pain and mechanical signs are uncommon in cases of pure myelopathy. See Table 71.1 for the frequency of symptoms in CSM in one series. In most cases the disability is mild, and the prognosis for these is good.

### 71.3.2 Motor

Findings can be due to cord (UMN) and/or root (LMN) compression. The earliest motor findings are typically weakness in the triceps and hand intrinsics.[23] There may be wasting of the hand muscles.[24] Slow, stiff opening and closing of the fists may occur.[25] Clumsiness with fine motor skills (writing, buttoning buttons...) is common.

There is often proximal weakness of the lower extremities (mild to moderate iliopsoas weakness occurs in 54%) and spasticity of the LEs.

### 71.3.3 Sensory

Sensory disturbance may be minimal, and when present are often not radicular in distribution. There may be a glove-distribution sensory loss in the hands.[26] A sensory level may occur a number of levels below the area of cord compression.

LEs often exhibit loss of vibratory sense (in as many as 82%), and occasionally have reduced pinprick sensation (9%) (almost always restricted to below the ankle). Compression of the spinocerebellar tract may cause difficulty running. Lhermitte's sign was present in only 2 of 37 cases. Some patients may present with a prominence of posterior column dysfunction (impaired joint position sense and 2 point discrimination).[27]

### 71.3.4 Reflexes

In 72–87% reflexes are hyperactive at a varying distance below the level of stenosis. Clonus, Babinski's sign (p.90) or Hoffman's sign (p.90) may also be present. Dynamic Hoffman's sign[28] may be more sensitive: test for Hoffman's sign during multiple cervical flexion and extension movements as tolerated by the patient. 94% of asymptomatic individuals with Hoffman's reflex will have significant spinal cord compression on MRI.[29] Inverted radial reflex: flexion of the fingers in response to eliciting the brachioradialis reflex, said to be pathognomonic of CSM.[30]

A hyperactive jaw jerk indicates upper motor neuron lesion above the midpons, and distinguishes long tract findings due to pathology above the foramen magnum from those below (e.g. cervical myelopathy): not helpful if absent (a normal variant). Primitive reflexes (grasp, snout, rooting) are not reliable localizing signs (except perhaps the grasp reflex) of frontal lobe pathology.

### 71.3.5 Sphincter

Urinary urgency and frequency are common in CSM, however these complaints are also protean in the aging population. Urinary incontinence is rare. Anal sphincter disturbances are uncommon.

Table 71.1 Frequency of symptoms in CSM (37 cases[22])

| Finding | % |
|---|---|
| pure myelopathy | 59% |
| myelopathy + radiculopathy | 41% |
| reflexes | |
| • hyperreflexia | 87% |
| • Babinski | 54% |
| • Hoffman | 13% |
| sensory deficits | |
| • sensory level | 41% |
| • posterior column | 39% |
| • dermatomal arm | 33% |
| • paresthesias | 21% |
| • positive Romberg | 15% |
| motor deficits | |
| • arm weakness | 31% |
| • paraparesis | 21% |
| • hemiparesis | 18% |
| • quadriparesis | 10% |
| • Brown-Séquard | 10% |
| • muscle atrophy | 13% |
| • fasciculations | 13% |
| pain | |
| • radicular arm | 41% |
| • radicular leg | 13% |
| • cervical | 8% |
| spasticity | 54% |
| sphincter disturbance | 49% |
| cervical mechanical signs | 26% |



## 71.3.6 Syndromes

Clustering of CSM into these 5 clinical syndromes has been described[25]:

1. transverse lesion syndrome: involvement of corticospinal and spinothalamic tracts and posterior columns, with anterior horn cells segmentally involved. Most frequent syndrome, possibly an "end-stage" of the disease process
2. motor system syndrome: primarily corticospinal tract and anterior horn involvement with minimal or no sensory deficit. This creates a mixture of lower motor neuron findings in the upper extremities and upper motor neuron findings (myelopathy) in the lower extremities which can mimic ALS (see below). Reflexes may be hyperactive below the area of maximal stenosis (including the upper extremities), occasionally beginning several levels below the stenosis
3. central cord syndrome: motor and sensory deficit affecting the UEs more than the LEs. This syndrome is characterized by dysfunction of the watershed areas located centrally within the cord,

which may be responsible for prominence of hand symptoms[31] (results in "numb-clumsy hands"[32]). Lhermitte's sign may be more common in this group
4. Brown-Séquard syndrome: often with asymmetric narrowing of the canal with the side of greater narrowing producing ipsilateral corticospinal tract (upper motor neuron weakness) and posterior column dysfunction with contralateral loss of pain and temperature sensation
5. brachialgia and cord syndrome: radicular UE pain with lower motor neuron weakness, and some associated long tract involvement (motor and/or sensory)

### 71.3.7 Grading

1. the modified Japanese Orthopaedic Association scale (mJOA) (□ Table 71.2) is a valid and reliable grading system, although it is non-specific
2. Neck Disability Index[33]: a 10 question survey similar to the Oswestry Disability Index for the lumbar spine (see □ Table 68.3). Mild disability is defined as a score of 10–28%, moderate = 30–48%, severe = 50–68%, complete ≥ 72%
3. other commonly used scales (not tested for validity or reliability):
   a) Nurick[34] (see □ Table 74.2)
   b) Harsh

### 71.3.8 Natural history

The time course of symptoms is highly variable and unpredictable. In ≈ 75% of cases of CSM, there is progression either in a stepwise fashion (in one–third) or gradually progressive (two–thirds).[35] In some series, the most common pattern was that of an initial phase of deterioration followed by a stabilization that typically lasts for years and may not change thereafter.[36,37] In these cases, the degree of disability may be established early in the course of CSM. Others disagree with such a "benign" outlook and cite that over 50% of cases continue to deteriorate with conservative treatment.[7] Sustained spontaneous improvement is probably rare.[17]

In patients < 75 years age and mJOA score > 12 (mild myelopathy), the clinical condition remained stable in 3-years of follow-up (Class I)[38] (however, these patients can still have significant disability that can respond to surgery). Patients with stenosis without myelopathy who have electrodiagnostic abnormalities or clinical radiculopathy are at risk of developing myelopathy (Class I).[38] Longstanding severe stenosis over many years may cause irreversible deficit due to necrosis in gray and white matter (Class III).[38]

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## 71.4 Differential diagnosis

### 71.4.1 General information

See Myelopathy (p.1407) for other possible causes. Some of these (e.g. spinal cord tumor, OPLL) may be demonstrated radiographically. Asymptomatic cervical spondylosis is very common, and ≈ 12% of cases of cervical myelopathy attributed to spondylosis are later found to be due to another disease process including:

1. ALS: see below
2. multiple sclerosis (MS): spinal cord demyelination may mimic CSM. With MS, remissions and exacerbations are common, and patients tend to be younger
3. herniated cervical disc (soft disc): patients tend to be younger than with CSM. Course is more rapid
4. subacute combined system disease: abnormal vitamin B12 level and possibly macrocytic anemia (p.1409)
5. hereditary spastic paraplegia: family history is key. Diagnosis of exclusion[39]
6. (spontaneous) intracranial hypotension (p.178)

### 71.4.2 Amyotrophic lateral sclerosis (ALS)

AKA (anterior horn) motor neuron disease; also see Amyotrophic lateral sclerosis (p.183). Can mimic the motor system syndrome of CSM (see above), and spinal cord compression may be seen on MRI in > 60% of patients with ALS.[40]

"Triad" of ALS:

1. atrophic weakness of hands and forearms (early) – LMN finding
2. mild LE spasticity – UMN finding
3. diffuse hyperreflexia – UMN finding

Table 71.2 Modified JOA score for cervical myelopathy[23a]

| Score | Description | |
|---|---|---|
| Upper extremity (UE) motor dysfunction | | |
| 0 | unable to feed self | |
| 1 | unable to use knife & fork; can eat with spoon | |
| 2 | can use knife & fork with much difficulty | |
| 3 | can use knife & fork with slight difficulty | |
| 4 | none (normal) | |
| Lower extremity (LE) motor dysfunction | | |
| 0 | unable to walk | |
| 1 | can walk on flat surface with walking aid | |
| 2 | can walk up and/or down stairs with handrail | |
| 3 | lack of smooth and stable gait | |
| 4 | none (normal) | |
| Sensory deficit | | |
| 0 | UE | severe sensory loss or pain |
| 1 | | mild sensory loss |
| 2 | | none (normal) |
| 0 | LE | severe sensory loss or pain |
| 1 | | mild sensory loss |
| 2 | | none (normal) |
| 0 | trunk | severe sensory loss or pain |
| 1 | | mild sensory loss |
| 2 | | none (normal) |
| Sphincter dysfunction | | |
| 0 | unable to void | |
| 1 | marked voiding difficulty (retention) | |
| 2 | some voiding difficulty (urgency or hesitation) | |
| 3 | none (normal) | |

[a]total score ranges from 0 to 17 (normal)



Inevitably, some cases of demyelinating disease will be misdiagnosed initially as CSM until some features suggestive of ALS occur (in one series of 1500 ALS patients, 4%underwent spine surgery (56% cervical, 42%lumbar, 2%thoracic)[40] before ALS was correctly diagnosed).

Features that may help differentiate ALS from CSM:

1. ALS: sensory changes are conspicuously absent
2. ALS: bulbar symptoms (dysarthria, hyperactive jaw-jerk…)[41]

3. ALS: extensive weakness/muscle atrophy of hands, usually with fasciculations[42]
4. ALS: lower-motor neuron (LMN) findings in the tongue (visible fasciculations, or positive sharp waves on EMG) or in the LEs (e.g. fasciculations and atrophy) favors the diagnosis of ALS over CSM (however, LMN findings in the LEs may occur in CSM if there is coincidental lumbar radiculopathy)
5. CSM or herniated cervical disc: usually includes neck and shoulder pain, limitation of neck movement, sensory changes, and LMN findings restricted to 1 or 2 spinal cord segments

# 71.5 Evaluation

## 71.5.1 Plain x-rays

### General information

Minimum evaluation consists of AP, lateral (neutral position), and open-mouth odontoid views. If desired, flexion-extension views and/or oblique views may be obtained but requires specific orders.

When MRI is available, the additional information provided by plain cervical spine x-rays in patients with CSM is limited. In this setting, x-rays may be best for:

1. Demonstrating dynamic instability with flexion-extension views (see below)
2. Sagittal balance measured on standing lateral cervical spine x-rays may provide prognostic information[43]
3. X-rays may be able to compensate for the following MRI deficiencies, but cervical CT is much better
   a) Differentiating calcified discs or bone spurs from "soft discs"
   b) Differentiating OPLL form a thickened posterior longitudinal ligament
   c) Bone abnormalities: fractures, bony lytic lesions

### Cervical spinal stenosis



Cervical spinal stenosis can be inferred from plain x-rays. □ NB: Canal diameters measured on x-rays are actually surrogate markers for the entity of interest: viz. spinal canal narrowing sufficient to produce spinal cord compression and thereby spinal cord symptoms. This can be directly demonstrated on MRI or CT/myelo, and MRI can also detect intrinsic spinal cord signal abnormalities.

See Canal diameter (p.214) for normal dimensions and measurement techniques. Patients with CSM have an average minimal AP canal diameter of 11.8 mm,[44] and values ≤ 10 mm were likely to be associated with myelopathy.[45] Patients with an AP diameter < 14 mm may be at increased risk,[46] and CSM is rare in patients with a diameter > 16 mm, even with significant spurs.[17]

Cervical spinal stenosis is also suggested on plain films when the spinolaminar line is close to the posterior margin of the lateral masses.

Pavlov ratio (AKA Torg ratio[47,48]): the ratio of the AP diameter of the spinal canal at the mid VB level to the VB at the same location. A ratio < 0.8 is sensitive for transient neuropraxia, but has been shown to have poor positive predictive value for CSM.

### Oblique views

Oblique views can delineate foraminal compromise caused by osteophytic spurs.

### Flexion-extension views

Lateral flexion/extension x-rays may provide valuable information by detecting dynamic instability (abnormalities that manifest with movement) that cannot be appreciated on (static) CT or MRI, including widening of the atlanto-dental interval on flexion (p.213).

## 71.5.2 MRI

MRI provides information about the spinal canal, and can also show intrinsic cord abnormalities (demyelination, syringomyelia, spinal cord atrophy, edema...). MRI also rules out other diagnostic possibilities (Chiari malformation, spinal cord tumor...).

Bony structures and calcified ligaments are poorly imaged. These shortcoming and the difficulties in differentiating osteophytes from herniated discs on MRI are overcome with the addition of plain cervical spine films[49] or to better advantage with thin-section CT bone windows.

Findings that correlate with poor outcome (Class III)[50]:

1. multilevel T2WI hyperintensity within the spinal cord parenchyma

2. single level T2WI hyperintensity with corresponding T1WI hypointensity (single level T2WI hyperintensity without T1WI changes are of uncertain prognostic significance)
3. spinal cord atrophy (transverse area < 45 $mm^2$)

Other MRI findings seen with CSM:

1. reduced transverse area of the spinal cord (TASC) at the level of maximum compression. A "banana" shaped cord on axial images has a high correlation with the presence of CSM.[46] There is conflicting evidence whether the degree of canal stenosis predicts outcome.[50] Sagittal T2WIs tend to exaggerate the magnitude of spinal cord compression by osteophytes and/or discs, and therefore axial images and T1WIs also need to be considered in the evaluation. Narrowing is not specific for CSM: ≈ 26% of asymptomatic individuals > 64 years of age have spinal cord compression on MRI[51]
2. "snake eyes" (AKA "owl's eyes") within the spinal cord on axial T2WI (□ Fig. 71.1) may be related to cystic necrosis of the cord[52] and may correlate with poor outcome (Class III)[50]

## 71.5.3 CT and CT/myelogram

Plain CT scans may demonstrate a narrow canal, but do not provide adequate information regarding soft tissues (discs, ligaments, spinal cord and nerve roots). However, bony detail may prove invaluable in the surgical treatment of CSM.

Cervical myelography followed by high-resolution CT scanning provides sagittal and axial information (including spinal cord atrophy), and delineates bony detail better than MRI.[49] Unlike MRI, CT/myelogram is invasive (requires LP) and involves ionizing radiation and does not provide information about changes within the spinal cord parenchyma.

## 71.5.4 EMG

Not routinely useful in CSM. EMG has poor sensitivity in cervical radiculopathy and is not reliable in predicting outcome from surgery for CSM (Class III).[50] EMG is most helpful in suspicious cases to eliminate etiologies such as peripheral neuropathy or ALS.

## 71.5.5 Sensory evoked potentials (SEPs)

SSEPs are of limited usefulness, although a normal pre-op SEP or normalization of SEPs in the early post-op period are associated with better outcome.[53]

### Practice guideline: Pre-op SEPs in CSM

Pre-op SEPs should be considered if the additional prognostic information would help treatment decisions (Level B Class II)[53]

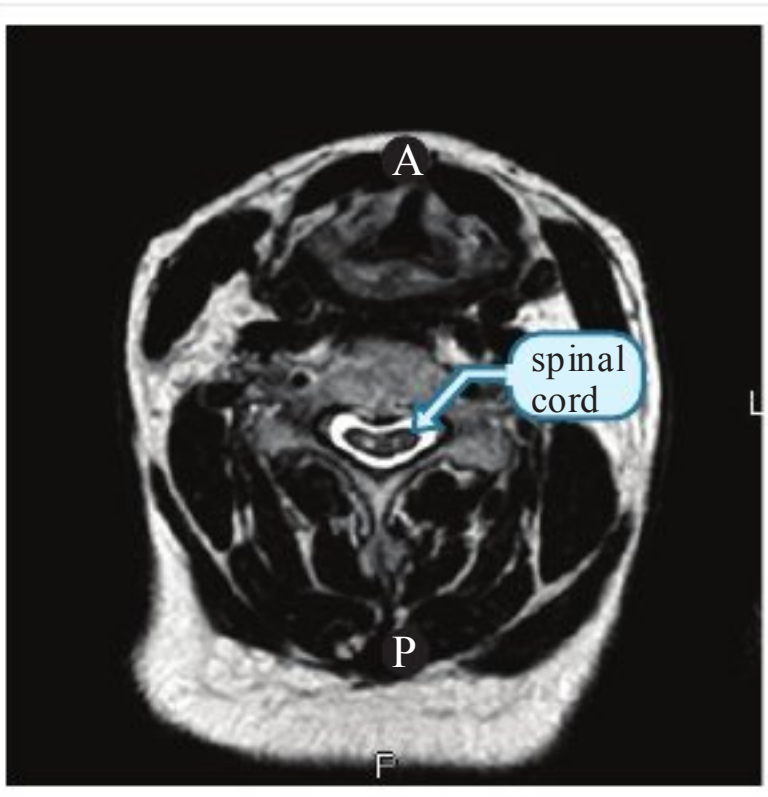


Fig. 71.1 Snake eyes (two foci of high signal) within a slightly flattened and mildly atrophic spinal cord on axial T2WI MRI

## 71.6 Treatment

### 71.6.1 Nonoperative management

Measures include: prolonged immobilization with rigid cervical bracing in an attempt to reduce motion and hence the cumulative effects of trauma on the spinal cord, modified activity to eliminate "high-risk" activities or bed rest, and anti-inflammatory medications.[54]

### 71.6.2 Surgical treatment

#### Indications for surgery

See Practice guideline: Surgical vs. nonsurgical management (p.1090).

#### Practice guideline: Surgical vs. nonsurgical management

Mild myelopathy (mJOA score > 12): in the short-term (3 years) patients may be offered the option of surgical decompression or nonoperative management (prolonged immobilization in a rigid cervical collar, anti-inflammatory medications, and "low-risk" activities or bed rest (Level C Class II)).[55] Note: patients with mJOA scores > 12 (see □ Table 71.2) may not always be mildly impaired, they may derive significant improvement from surgery, and deterioration from this point may be ominous.

More severe myelopathy: should be treated with surgical decompression with benefits maintained at 5 and 15 years post-op (Level D Class III)[55]

Level B Class I[56] Degenerative cervical radiculopathy: patients do better with anterior decompression ± fusion (compared to conservative management) for

- rapid relief (within 3–4 months) of arm & neck pain and sensory loss
- relief of longer term (≥ 12 months) symptoms of weakness of wrist extension, elbow extension, shoulder abduction and internal rotation



#### Intraoperative electrophysiologic monitoring

#### Practice guideline: Intra-operative electrophysiologic monitoring during surgery for CSM or radiculopathy

Use of intra-op EP monitoring during routine surgery for CSM or cervical radiculopathy is not recommended as an indication to alter the surgical plan or administer steroids since this paradigm has not been observed to reduce the incidence of neurologic injury (Level D Class III)[57]

#### Choice of approach

##### General information

The debate between anterior approaches (anterior cervical discectomy or corpectomy) and posterior approaches (decompressive cervical laminectomy or laminoplasty) dates back to the time that both became widely practiced.[16] General sentiment is to treat anterior disease at the disc level (e.g. osteophytic bar, herniated disc...) usually limited to ≤ 3 levels (or occasionally 4) with an anterior approach, and to use a posterior approach as the initial procedure in the situations outlined below. Considerations of spinal curvature may need to enter into the decision process.

#### Practice guideline: Choice of surgical approach for CSM

There was not enough evidence to recommend any of the following techniques over the other (in terms of short-term success in treating CSM): ACDF, anterior corpectomy and fusion, laminectomy (with or without fusion) and laminoplasty (Level D Class III)[58]

Laminectomy without fusion, however, is associated with a higher incidence of late kyphotic deformity (Level D Class III; incidence 14–47%, not all cases are symptomatic, not all cases need treatment: see text)[58]

## Posterior approach

Options include:

1. laminectomy alone laminectomy/arthrodesis (i.e. laminectomy + lateral mass fusion): Class III (this procedure was found to be effective, the class shows the strength of the evidence)[59])
2. laminoplasty (Class III; this procedure was found to be effective, the class shows the strength of the evidence[60]): methods include unilateral ("open door") and midline enlargement ("French door")
3. multilevel foraminotomies: usually not adequate for central canal stenosis

Situations where a posterior approach would generally be the initial approach:

1. congenital cervical stenosis where removing osteophytes will still not provide at least ≈ 12 mm of AP canal diameter
2. disease over ≥ 3 levels (although up to 4 may occasionally be dealt with anteriorly)
3. primary posterior pathology (e.g. infolding of ligamentum flavum)
4. some cases of OPLL (anterior approach has higher risk of dural tear)

Disadvantages of the posterior approach:

1. laminectomy without fusion
   a) degeneration and osteophytes continue to progress following surgery
   b) risk of subsequent subluxation or progressive kyphotic angulation ("swan neck" deformity) (facetiously dubbed "spina bifida neurosurgica")
      - quoted incidence: 14–47%[61,62,63] (risk may be minimized by careful preservation of facet joints)
      - not all cases need to be treated: in one series, 31%(18/58) developed post-op kyphosis, and 16%of these (3/18) required surgical stabilization[64]
      - the development of kyphotic deformity does not appear to diminish the clinical outcome[63] and does not correlate with neurologic deterioration when deterioration occurs[65]
2. more painful initially post-op and sometimes more prolonged rehabilitation
3. long-term complaints of a heaviness of the head possibly associated with atrophy of the paraspinal muscles
4. □ contraindicated with pre-existing swan neck deformity, and not recommended in the presence of reversal of the normal cervical lordosis (i.e. kyphotic curve)[66] where the spinal cord won't tend to move away from the anterior compression or in the presence of ≥ 3.5 mm subluxation or > 20° rotation in the sagittal plane[46] and caution must be exercised in hyperlordosis (see below)



## Anterior approach

Also shown to be effective (Class III[55]).

Instrumentation options: in terms of fusion rates for 2-level anterior operations (i.e. 2 disc spaces) (Class III)[58]:

2-level ACDF with anterior plate ¼ 1-level corpectomy with plate > 1-level corpectomy without plate* > 2-level ACDF without plate

* however; the graft extrusion rate is higher for corpectomy than ACDF

Worsening of myelopathy has been reported in 2–5% of patients after anterior decompression[67,68] (intraoperative SSEP monitoring may reduce this rate[68]) and C5 radiculopathy may occur (see below).

Anterior cervical plating

Many systems are available, with more similarities than differences. All include some method of preventing screw back out. Some general pointers:

1. for single level fusion, typical plate length is 22–24 mm
2. screw length: rule of thumb is 12 mm for females, 14 mm for males
3. do not completely tighten a single screw (to avoid kicking up plate) until the diagonally opposite screws are placed and loosely tightened
4. most systems have fixed and variable angle screws. Variable angle screws allow for load sharing with the graft (here is where a derivative of Wolff's law is often invoked: the weight sharing helps stimulate fusion). Avoid over-angling screws which may prevent the locking mechanism from properly engaging

5. optimal plate placement allows for contact of the plate with the VB at the screw locations. This may require
   a) contouring of plate to follow the lordosis of the c-spine
   b) reduction of anterior osteophytes

## Posterior approach

For decompression, some recommend cervical laminectomy extending one or two levels beyond the stenosis above and below.[69,70] A C3–6 laminectomy is often considered a "standard" laminectomy. An "extended laminectomy" includes C7 and/or C2.

Curvature considerations: extending the laminectomy to include C2 and sometimes C1 has been recommended for patients with straightening of the cervical curvature.[6] In cases of hyperlordosis, posterior migration of the spinal cord following an extensive laminectomy may put increased tension on the nerve roots and blood vessels (with possible neurologic worsening), and a limited laminectomy just where the cord is compressed is often recommended (below).

"Keyhole foraminotomies" or medial facetectomy with undercutting of the facets may be performed at levels involved with radiculopathy.

Position: choices are primarily: prone, lateral oblique, or sitting. The prone position has a major disadvantage of difficulty elevating the head above the heart, resulting in venous engorgement with significant operative bleeding. The sitting position has a number of inherent risks (p.1445), including cord hypoperfusion[68] and air embolism. The lateral oblique position may introduce some distortion to the anatomy due to asymmetrical positioning.

The reported rate of post-op spinal deformity is 25–42% Neurologic worsening has been reported in 2%in some series, higher in others. C5 radiculopathy may occur (see below).

To avoid significant destabilization of the cervical spine:

1. during the dissection, do not remove soft tissue overlying the facet joints (to preserve their blood supply)
2. take the laminectomy only as far lateral as the extent of the spinal canal, carefully preserving the facet joints[7] (use keyhole laminotomies where necessary)
3. avoid removing a total of one facet at any given level

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## Outcome

### General information

Even excluding cases that are later proven to have demyelinating disease, the outcome from surgery for CSM is often disappointing. Once CSM is clinically apparent, complete remission almost never occurs. The prognosis with surgery is worse with increasing severity of involvement at the time of presentation[69] and with longer duration of symptoms (48%showed clinical improvement or cure if operated within 1 yr of onset, whereas only 16%responded after 1 yr[7]). The success of surgery is also lower in patients with other degenerative diseases of the CNS (ALS, MS...).

Progression of myelopathy may be arrested by surgical decompression. This is not always borne out, and some early series[34,37] showed similar results with conservative treatment as with laminectomy which yielded improvement in 56%, no change in 25%, and worsening in 19% Also as discussed earlier some cases of CSM develop most of the deficit early and then stabilize (p.1086).

Some series show good results, with ≈ 64–75%patients having improvement in CSM post-op.[22] However, other authors remain less enthusiastic. Utilizing a questionnaire in 32 post-op patients operated anteriorly, 66%had relief from radicular pain, while only 33%had improvement in sensory or motor complaints.[22] In one series, half the patients had improvement in fine motor function of the hands, but the other half worsened postoperatively.[71] Spinal cord atrophy as a result of continued pressure or ischemia may be partly responsible for poor recovery. Bedridden patients with severe myelopathy rarely recover useful function.

### Post-op C5 palsy

Criteria: weakness of deltoid and/or biceps with no worsening of myelopathy. Follows ≈ 3–5% of extensive anterior or posterior decompression (including laminoplasty).[67,72] 50% have motor involvement only (deltoid > biceps), 50%also have C5 dermatomal sensory loss and/or C5 dermatomal pain (shoulder). Most occur < 1 week post-op.[72] 92%are unilateral.[72] No pre-op risk factors have been identified.[73] Etiology: unproven, may be related to traction on the nerve root from posterior migration of the cord after decompression or to bone graft displacement. Prognosis for spontaneous recovery is generally good; more severe deficits take longer to recover.[72]

### Late developments

Some patients who show early improvement will develop late deterioration (7–12 yrs after reaching a plateau),[46] with no radiographically apparent explanation in up to 20%of these cases.[74] In others, degeneration at levels adjacent to the operated segments may be demonstrated.

Adjacent segment disease (ASD): degeneration that develops at a motion segment adjacent to a previous fusion. Findings include: disc degeneration, stenosis, facet hypertrophy, scoliosis, listhesis and instability. After ACDF, ASD occurred at a rate of 2.9%per year over 10 years observation.[75] Estimate: 25%of patients will develop symptomatic adjacent level changes within 10 years of surgery.[75] This rate was higher with single level fusion at C5–6 or C6–7 than it was with multilevel fusion, and natural progression of the disease was felt to be a significant contributor[75] (i.e. it was not all attributable to the fusion). Most cases of ASD observed radiographically are asymptomatic.

## 71.7 Coincident cervical and lumbar spinal stenosis

In 5% lumbar and cervical stenoses are symptomatic simultaneously.[76]

Coincident symptomatic lumbar and cervical spinal stenosis is usually managed by first decompressing the cervical region, and later operating on the lumbar region (unless severe neurogenic claudication dominates the picture). It is also possible, in selected cases, to operate on both in a single sitting.[76,77]

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# 72 Thoracic and Lumbar Degenerative Disc Disease

## 72.1 General information about degenerative disc disease (DDD)

Since structures outside of the disc are usually also involved, the term degenerative spine disease (DSD) may be preferable to degenerative disc disease. Spondylosis is a non-specific term which may include degenerative spine disease. "Cervical spondylosis" is occasionally used synonymously with cervical stenosis (p.1083).

Symptomatic spinal stenosis in the thoracic region is rare,[1] and is generally seen in the setting of a calcified disc. The majority of this chapter deals with lumbar DSD. For the cervical region, see that chapter (p.1083).

## 72.2 Anatomic substrate

### 72.2.1 General information

DSD is a progressive deterioration of the structures of the spine including:

1. disc abnormalities:
   a) the proteoglycan content of the disc nucleus decreases with age
   b) disc desiccation (loss of hydration) occurs
   c) tears develop in the disc annulus and progress to internal disruption of the lamellar architecture. Herniation of the nucleus may occur from increased nuclear pressure under mechanical loads
   d) mucoid degeneration and ingrowth of fibrous tissue ensues (disc fibrosis)
   e) subsequently disc resorption occurs
   f) there is a loss of disc space height and increased susceptibility to injury
2. facet joint abnormalities: hypertrophy and capsular laxity
3. osteophytes often form on the edges of the VB bordering the degenerated disc
4. spondylolisthesis: subluxation of one VB on another (see Spondylolisthesis below)
5. hypertrophy of the ligamentum flavum



Neurologic involvement in lumbar spinal stenosis may occur in one or more of the following 3 sites:

1. central canal stenosis: narrowing of the AP dimension of the spinal canal below a critical value. The reduction in canal size may cause local neural compression and/or compromise of the blood supply to the spinal cord (cervical) or the cauda equina (lumbar)
   a) Congenital (as in the achondroplastic dwarf)
   b) Acquired: as with hypertrophy of facets and ligamentum flavum
   c) Most commonly – acquired superimposed on congenital narrowing.
2. foraminal stenosis: narrowing of the neural foramen. May be the result of any combination of: foraminal disc protrusion, spondylolisthesis, facet hypertrophy, disc space collapse, hypertrophy of uncovertebral joints (cervical), synovial cyst
3. lateral recess stenosis; lumbar spine only (p.1097)

### 72.2.2 Lumbar spinal stenosis

#### Key concepts

- caused by hypertrophy of facets and ligamentum flavum, may be exacerbated by disc bulging or spondylolisthesis, may be superimposed on congenital narrowing
- most common at L4–5 and then at L3–4
- symptomatic stenosis produces gradually progressive back and leg pain with standing and walking that is relieved by sitting or lying (neurogenic claudication)
- symptoms differentiated from vascular claudication which is usually relieved at rest regardless of position
- usually responds to decompressive surgery (sometimes with fusion) or interspinous spacer

Symptomatic lumbar stenosis is most common at L4–5, then L3–4, L2–3 and lastly L5-S1.[2] It is rare at L1–2. Generally occurs in patients with congenitally shallow lumbar canal – see Normal LS spine measurements (p.1102) – with superimposed acquired degeneration in the form of some combination of facet hypertrophy, hypertrophy of the ligamentum flavum, protruding (and often calcified) intervertebral discs, and spondylolisthesis. First recognized as a distinct clinical entity producing characteristic symptoms in the 1950's and 60's.[3,4]

May be classified as[5]:

1. stable form of lumbar spinal stenosis: hypertrophy of facets and ligamentum flavum accompanied by disc degeneration and collapse
2. unstable: have the above with superimposed
   a) degenerative spondylolisthesis (p.1098), the unisegmental form
   b) degenerative scoliosis: the multisegmental form

## 72.2.3 Central canal stenosis

The central canal may be narrowed by a combination of any of the following:

- Hypertrophy of the ligamentum flavum
- Hypertrophy of the facet joints
- Congenitally shot pedicles
- Bulging intervertebral discs
- Osteophytes arising from the posterior VB
- Juxtafacet cysts
- Spondylolisthesis

## 72.2.4 Lateral recess syndrome

The lateral recess is the "gutter" alongside the pedicle which the nerve root enters just proximal to its exit through the neural foramen (□ Fig. 72.1). It is bordered anteriorly by the vertebral body, laterally by the pedicle, and posteriorly by the superior articular facet of the inferior vertebral body. Hypertrophy of this superior articular facet compresses the nerve root. Narrowing of the lateral recess is present in essentially all cases of central canal stenosis, but it can be symptomatic by itself.[6] L4–5 is the most commonly involved facet.



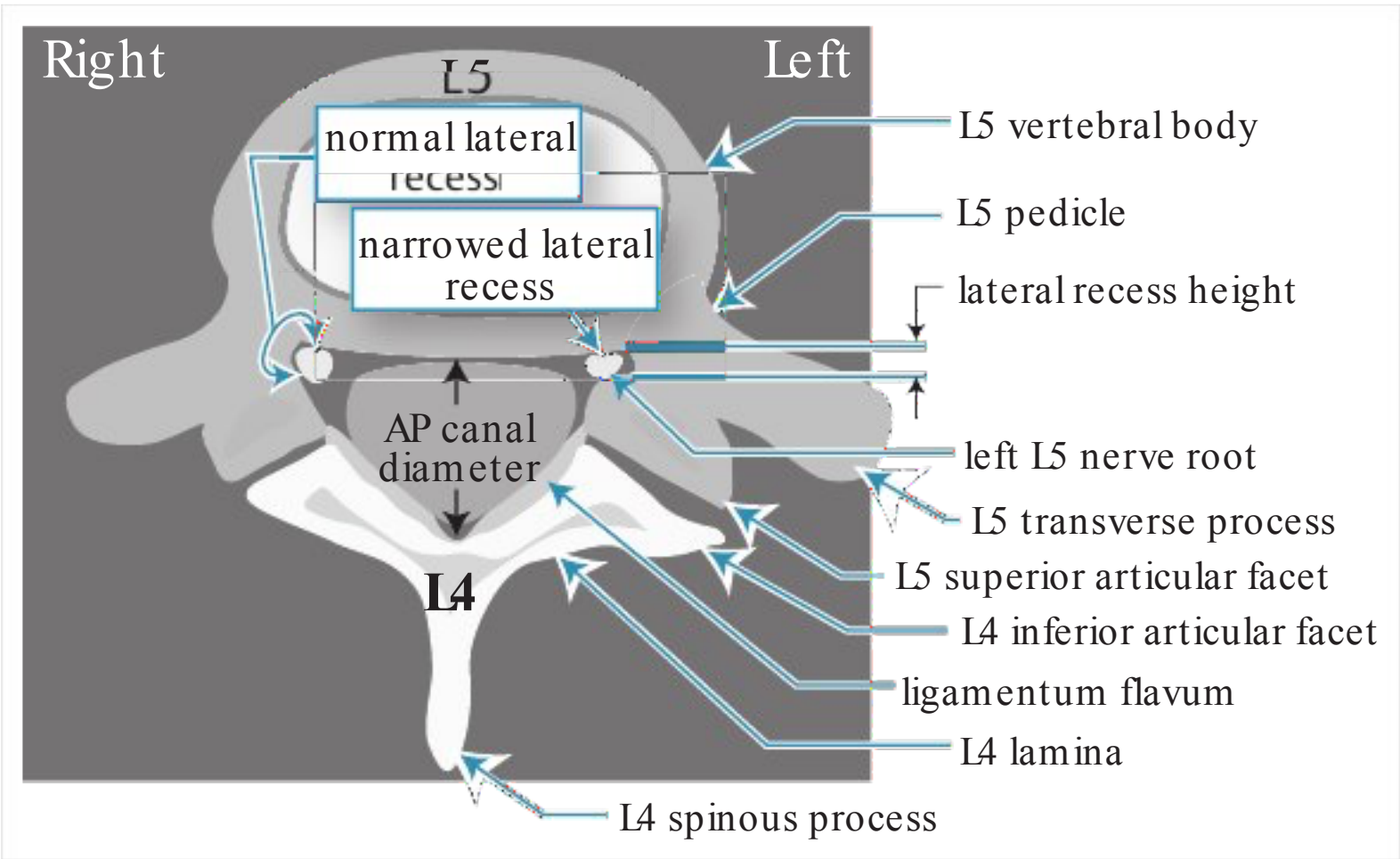


Fig. 72.1 Schematic axial CT through the L4-5 facet joint showing the lateral recesses (normal on patient's right, stenotic on left)

## 72.2.5 Spondylolisthesis

### General information

Anterior subluxation of one vertebral body (VB) on another, most commonly the upper VB is anterior to the inferior one. Usually L5 on S1, the next most common is L4 on L5.

Disc herniation and nerve root compression with spondylolisthesis: It is rare for a herniated lumbar disc to occur at the level of the listhesis, however the disc may "roll" out as it is uncovered and produce findings on MRI that may resemble a herniated disc which has been termed a "pseudodisc." It is more common to see a herniated disc at the level above the listhesis.

If the listhesis does cause nerve root compression, it tends to involve the nerve exiting below the pedicle of the anteriorly subluxed upper vertebra (e.g. if an L4–5 spondylolisthesis causes nerve root compression, it will generally involve the L4 root). The compression is usually due to upward displacement of the superior articular facet of the level below together with disc material, and symptoms typically resemble neurogenic claudication, although true radiculopathy may sometimes occur. There also may be a contribution from a fibrous/inflammatory mass from the nonunion.

Isthmic spondylolisthesis rarely produces central canal stenosis since only the anterior part of the vertebral body shifts forward. May present with radiculopathy or neurogenic claudication from compression in the neural foramen, with the nerve exiting under the pedicle at that level being the most vulnerable. May also present with low back pain. Many cases are asymptomatic.

### Adolescent spondylolisthesis

In adolescents and teens, spondylolisthesis usually occurs in athletes subject to repetitive hyperextension of the lumbar spine. In girls, this is frequently encountered in gymnasts and softball pitching. In boys, football is common.

In these youngsters, cessation of sports for several months usually produces resolution.

Surgery is sometimes performed for patients who are unwilling to discontinue athletics.

### Grading spondylolisthesis

The Meyerding[7,8] grading of subluxation in the sagittal plane is shown in Table 72.1.



### Types of spondylolisthesis

1. Type 1: dysplastic: congenital. Upper sacrum or arch of L5 permits the spondylolisthesis. No pars defect. 94%are associated with spinal bifida occulta. Some of these may progress (no way to accurately identify those that will progress)
2. Type 2: isthmic spondylolisthesis AKA spondylolysis: a failure of the neural arch as a result of a defect in the pars interarticularis (identifiable as a discontinuity in the neck of the "Scotty dog" on oblique LS-spine x-ray). May be seen in 5–20%of spine x-rays.[9] Rarely produces central canal stenosis since only the anterior part of the vertebral body shifts forward. May cause narrowing of the neural foramen. Three subtypes:
   a) lytic: fatigue fracture or insufficiency fracture of pars. In the pediatric age group may occur in athletes (especially gymnasts or football players); in some this may be an exacerbation of a pre-existing defect, in others it may be a result of repetitive trauma
   b) elongated but intact pars: possibly due to repetitive fractures and healing
   c) acute fracture of pars

Table 72.1 Spondylolisthesis grading

| Grade | % subluxation[a] |
|---|---|
| I | <25% |
| II | 25–50% |
| III | 50–75% |
| IV | 75%-complete |
| spondyloptosis | >100% |

[a]% of the AP diameter of the VB

3. Type 3: degenerative: due to long-standing intersegmental instability. Usually at L4–5. No break in the pars. Found in 5.8%of men and 9.1%of women (many of whom are asymptomatic)[9]
4. Type 4: traumatic: due to fractures usually in areas other than the pars
5. Type 5: pathologic: generalized or local bone disease, e.g. osteogenesis imperfecta

### Natural history

Progression of spondylolisthesis may occur without surgical intervention, but is more common following surgery.[10]

## 72.2.6 Degenerative scoliosis

One of the main differentiating features from juvenile scoliosis is that in degenerative scoliosis, the disc spaces are asymmetrically narrowed in the coronal plane and the vertebral bodies tend to maintain a more normal configuration.

# 72.3 Risk factors

1. The risk of developing DSD is multifactorial and includes:
2.  The most powerful determinant in developing DSD in a study of twins was genetic influence, and possibly other unidentified factors.[11] Environmental factors studied (including sedentary vs. active lifestyles, occupation, cigarette smoking…) exerted only a modest influence, which may explain why conflicting findings for these have been reported
3. cumulative effects of microtrauma and macrotrauma to the spine
4. osteoporosis
5. cigarette smoking: several epidemiologic studies have shown that the incidence of back pain, sciatica and spinal degenerative disease is higher among cigarette smokers than among non-smokers[12,13]
6. in the lumbar spine:
   a) stresses on the spine including effects of excess body weight
   b) loss of muscle tone (primarily abdominals and paraspinals) resulting in increased dependence on the bony spine for structural support



# 72.4 Associated conditions

1. congenital:
   a) achondroplasia
   b) congenitally narrowed canal
2. acquired:
   a) spondylolisthesis
   b) acromegaly
   c) post-traumatic
   d) Paget's disease (p.1120)
   e) ankylosing spondylitis (p.1123)
   f) ossification of the ligamentum flavum: more common in East Asians, rare in Caucasians.[14] Often, but not always, associated with OPLL[15]

# 72.5 Clinical presentation

## 72.5.1 General information

1. the degenerative abnormalities may produce spinal stenosis which can lead to neural compromise producing the following symptoms
   a) radicular symptoms (more common in cervical spine than lumbar)
   b) neurogenic claudication (lumbar) or spinal myelopathy (cervical)
2. Sagittal imbalance and scoliosis as a result of degenerative changes can place focal stress on specific spine structures which may be painful. Also, muscles used to compensate for the imbalance can cause pain from overuse fatigue
3. discogenic pain (controversial) may be less prevalent in the late stages of DSD. May contribute to "musculoskeletal low back pain" but the actual pain generators here are not definitively identified

4. Many cases of DSD (including spinal stenosis and spondylolisthesis) are asymptomatic, and the degenerative changes are discovered incidentally

## 72.5.2 Neurogenic claudication

Lumbar spinal stenosis often presents as neurogenic claudication (NC), (claudicate: from Latin, claudico, to limp) AKA pseudoclaudication. To be differentiated from vascular claudication (AKA intermittent claudication) which results from ischemia of exercising muscles (see □ Table 72.2 for distinguishing characteristics).

NC characteristics: unilateral or bilateral buttock, hip, thigh or leg discomfort that is precipitated by standing or walking and characteristically relieved by a change in posture (usually sitting with the waist flexed, squatting, or lying in the fetal position). Painful burning paresthesias of the lower extremities are also described. Valsalva maneuvers usually do not exacerbate the pain. Many patients report increased pain first thing in the morning that improves once they have been out of bed for varying periods (usually an hour more or less).

The time course is usually gradually progressive over many months to years. As the condition progresses, the ability to get relief from position changes tends to decrease. However, presenting with acute, unrelenting pain is not characteristic and other causes should be sought.

In comparison, a HLD usually causes increased pain on sitting, has a more abrupt onset, has pain on straight leg raising, and is worsened by Valsalva maneuvers.

NC is thought to arise from ischemia of lumbosacral nerve roots, as a result of increased metabolic demand from exercise together with vascular compromise of the nerve root due to pressure from surrounding structures. NC is only moderately sensitive (≈ 60%) but is highly specific for spinal stenosis.[17] Pain may not be the major complaint, instead, some patients may develop paresthesias or LE weakness with walking. Some may complain of muscle cramping, especially in the calves.

Relief from symptoms: occurs with positions that decrease the lumbar lordosis which increases the diameter of the central canal (by reducing inward buckling of the ligamentum flavum) and distracts the facet joints (which enlarges the neural foramina. Favored positions include sitting, squatting and recumbency. Patients may develop "anthropoid posture" (exaggerated waist flexion). "Shopping cart sign" patients often can walk farther if they can lean forward e.g. as on a grocery cart. Riding a bicycle is also often well tolerated.



Table 72.2 Clinical features distinguishing neurogenic from vascular claudication[16]

| Feature | Neurogenic claudication | Vascular claudication |
|---|---|---|
| distribution of pain | in distribution of nerve (dermatomal) | in distribution of muscle group with common vascular supply (sclerotomal) |
| sensory loss | dermatomal distribution | stocking distribution |
| inciting factors | variable amounts of exercise, also with prolonged maintenance of a given posture (65% have pain on standing at rest); coughing produces pain in 38% | reliably reproduced with fixed amount of exercise (e.g. distance ambulated) that decreases as disease progresses; rare at rest (27% have pain on standing at rest) |
| relief with rest | slow (often > 30 min), variable, usually positional (stooped posture or sitting often required, □ standing and resting is usually not sufficient) | almost immediate; not dependent on posture (relief of walking induced symptoms with standing is a key differentiating feature) |
| claudicating distance | variable day-to-day in 62% | constant day-to-day in 88% |
| discomfort on lifting or bending | common (67%) | infrequent (15%) |
| foot pallor on elevation | none | marked |
| peripheral pulses | normal; or if ↓ usually reduced only unilaterally | ↓ or absent; femoral bruits are common |
| skin temp of feet | normal | decreased |

### 72.5.3 Neurologic exam

The neurologic exam is normal in ≈ 18%of cases (including normal muscle stretch reflexes and negative straight leg raising). Weakness in the anterior tibialis and/or extensor hallicus longus may occur in some cases of central canal stenosis at L4–5, or with foraminal stenosis of L5-S1. Absent or reduced ankle jerks and diminished knee jerks are common,[17] however this is also prevalent in the aged population. Pain may be reproduced by lumbar extension.

## 72.6 Differential diagnosis

### 72.6.1 General considerations

1. vascular insufficiency: (AKA vascular or intermittent claudication) see above
2. hip disease: trochanteric bursitis (see below), degenerative joint disease
3. disc herniation (lumbar or thoracic)
4. facet joint pain (controversial): may respond to medial branch block (therapeutic & diagnostic)
5. Baastrup's syndrome[18]: AKA arthrosis interspinosa. Radiographically: contact of adjacent spinous processes ("kissing spines") with enlargement, flattening and reactive sclerosis of apposing interspinous surfaces. Produces localized midline lumbar pain & tenderness on back extension relieved by flexion, local anesthetic injection or partial excision of the involved spinous processes
6. juxtafacet cyst: (p.1143)
7. arachnoiditis
8. intraspinal tumor
9. Type I spinal AVM (spinal dural AVM) (p.1140)
10. diabetic neuritis: with this, the sole of the foot is usually very tender to pressure from the examiner's thumb
11. delayed onset muscle soreness (DOMS): onset usually 12–48 hours after beginning a new activity or changing activities (NC occurs during the activity). Symptoms typically peak within 2 days and subside over several days
12. inguinal hernia: typically produces groin pain
13. functional etiologies



#### Degenerative hip disease

Trochanteric bursitis (TBS) and degenerative arthritis of the hip are also included in the differential diagnosis of NC.[19,20] Although TBS may be primary, it can also be secondary to other conditions including lumbar stenosis, degenerative arthritis of the lumbar spine or knee, and leg length discrepancy. TBS produces intermittent aching pain over the lateral aspect of the hip. Although usually chronic, it occasionally may have acute or subacute onset. Pain radiates to lateral aspect of thigh in 20–40%(so called "pseudoradiculopathy"), but rarely extends to the posterior thigh or as far distally as the knee. There may be numbness and paresthesia-like symptoms in the upper thigh which are usually not dermatomal in distribution. Like NC, the pain may be triggered by prolonged standing, walking and climbing, but unlike NC it is also painful to lie on the affected side. Localized tenderness over the greater trochanter can be elicited in virtually all patients, with maximal tenderness at the junction of the upper thigh and greater trochanter. Pain increases with weight bearing (and is often present from the very first step, unlike NC) and with certain hip movements, especially external rotation (over half the patients have a positive Patrick-FABERE test (p.1048), and rarely with hip flexion/extension. Treatment includes NSAIDs, local injection of glucocorticoid (usually with local anesthetic), physical therapy (with stretching and muscle strengthening exercises) and local application of ice. No controlled studies have compared these modalities.

## 72.7 Diagnostic evaluation

### 72.7.1 Radiographic evaluation

#### Comparison of modalities

MRI: demonstrates impingement on neural structures and loss of CSF signal on T2WI due to central canal stenosis, lateral recess stenosis, foraminal stenosis as well as juxtafacet cysts. Increased fluid in the facet joint and vacuum disc MRI is poor for visualizing bone which contributes significantly to the pathology. Asymptomatic abnormalities are demonstrated in up to 33%of patients 50–70 years old without back-related symptoms.[2]

Lumbosacral spine x-rays: may disclose spondylolisthesis. AP diameter of canal is usually narrowed (congenitally or acquired) (below) whereas the interpediculate distance (IPD) may be normal.[16] Oblique films may demonstrate pars defects. Adding flexion/extension views can assess "dynamic" instability.

Standing scoliosis x-rays: provide information about scoliosis and sagittal balance. See Adult degenerative scoliosis for technique and measurements.

CT scan (either routine, or following water-soluble myelography): classically shows "trefoil" canal (cloverleaf shaped, with 3 leaflets). CT also demonstrates AP canal diameter, hypertrophied ligaments, facet arthropathy, pars fractures and occasionally may show bulging annulus or herniated disc

Myelogram: lateral films often show "washboard pattern" (multiple anterior defects), AP films often show "wasp-waisting" (narrowing of dye column), may also show partial or complete (especially in prone position) block. May be difficult to perform LP if stenosis is severe (poor CSF flow and difficulty avoiding nerve roots the LP needle).

## Normal LS spine measurements

Normal dimensions of the lumbar spine are shown in Table 72.3 for plain film and Table 72.4 for CT.

## 72.7.2 Adjuncts to radiographic evaluation

"Bicycle test": patients with NC can usually tolerate longer periods of exercise on a bicycle than patients with intermittent (vascular) claudication because the position in bicycling flexes the waist.

Noninvasive studies to rule-out vascular insufficiency: Ratio of ankle to brachial blood pressure (A:B ratio): >1.0 is normal; mean of 0.59 in patients with intermittent claudication; 0.26 in patients with rest pain; <0.05 indicates impending gangrene.

EMG with NCV may show multiple nerve-root abnormalities bilaterally.



Table 72.3 Normal AP diameter of lumbar spinal canal on lateral plain film (from spinolaminar line to posterior vertebral body)[21]

| | |
|---|---|
| average (normal) | 22–25 mm |
| lower limits of normal | 15 mm |
| severe lumbar stenosis | <11 mm |

Table 72.4 Normal lumbar spine measurements on CT[22]

| | |
|---|---|
| AP diameter | ≥11.5 mm |
| interpediculate distance (IPD) | ≥16 mm |
| canal cross-sectional area | ≥1.45 $cm^2$ |
| ligamentum flavum thickness[23] | ≤4–5 mm |
| height of lateral recess (see below) | ≥3 mm |

Table 72.5 Dimensions of lateral recess on CT (bone windows)

| Lateral recess height | Degree of lateral recess stenosis |
|---|---|
| 3–4 mm | borderline (symptomatic if other lesion co-exists, e.g. disc bulging) |
| <3 mm | suggestive of lateral recess syndrome |
| <2 mm | diagnostic of lateral recess syndrome |

## 72.8 Treatment

### 72.8.1 General information

In one study of 27 unoperated patients, 19 remain unchanged, 4 improved, and 4 worsened (mean follow-up: 49 months; range: 10–103 months).[24]

NSAIDs (acetaminophen may be as effective) and physical therapy are usually the initial measures of nonsurgical management. Unlike the cervical spine, traction is not usually helpful.

Brace therapy, e.g. with an LSO may be attempted. Guidelines are shown below.

#### Practice guideline: Brace therapy

Level II[25]:

- short-term use (1–3 weeks) of a rigid lumbar support is recommended for treatment of LBP of relatively short duration (<6 months)
- bracing in patients with LBP>6 months duration is not recommended because it has not been shown to have long-term benefit

Level III[25]:

- lumbar braces may reduce the number of sick days due to LBP among workers with a previous lumbar injury. Braces are not recommended for LBP in the general working population
- the use of pre-op bracing or transpedicular external fixation as tools to predict outcome for lumbar fusion is not recommended

Interventional pain management s an option for persistent pain. Epidural steroids may provide temporary relief (usually days to weeks at most). Facet blocks and, if helpful, rhizotomies (for longer relief), may be employed.

### 72.8.2 Management of isthmic spondylolisthesis



See reference.[9]

Some special management considerations for isthmic spondylolisthesis as a subset of spinal stenosis.

1. lesions with sclerotic borders are usually well established with little chance of healing.
2. Surgery is reserved for patients with neurologic deficit or incapacitating symptoms or progression of spondylolisthesis
3. lesions without sclerosis that show increased uptake on bone scan (indicating active lesion with potential for healing) or MRI high signal changes on T2WI[26] or STIR may heal in a rigid orthosis such as the Boston brace for ≥3 months
4. management of symptoms:
   a) LBP only: treat with NSAIDs, PT
   b) LBP with myelopathy, radiculopathy, or neurogenic claudication: surgical treatment[27] (see □ Table 72.6 for surgical options)

Table 72.6 Surgical recommendations for spondylolisthesis

| Nature of spondylolisthesis | Nature of problem | Type of procedure needed |
|---|---|---|
| degenerative | nerve root compression within confines of spinal canal | decompression (preserving facets) |
| | spinal stenosis at the level of spondylolisthesis | decompression; some advocate with intertransverse-process fusion[29] |
| | nerve root compression far lateral, outside confines of spinal canal | radical decompression (Gill procedure, see below) plus fusion |
| traumatic | (does not matter) | decompression plus fusion |

5. in pediatrics: may be managed with TLSO and long course of PT (e.g. 6–9 months) for symptoms. Resumption of sports may be considered when symptoms subside, but recurrence should prompt elimination of athletics or consideration of surgery

## 72.8.3 Indications for surgery

Surgical intervention is undertaken when symptoms become severe in spite of conservative management. The goals of surgery are pain relief, halting progression of symptoms, and possibly reversal of some existing neurologic deficit. Most authors do not consider surgery unless the symptoms have been present >3 months, and most patients who have surgery for this have symptoms of >1 year duration.

## 72.8.4 Surgery

### Surgical options

1. laminectomy: posterior (direct) decompression of central canal and neural foramina without or with fusion. Fusion options:
   a) posterolateral fusion ± pedicle screw/rod fixation
   b) interbody fusion: generally not done as a "stand-alone" (i.e. usually requires additional stabilization, options here include: pedicle screws, facet screws, facet dowels, spinous process clamp…)
      - posterior lumbar interbody fusion (PLIF) (p. 1497): usually bilateral graft placement
      - transforaminal lumbar interbody fusion (TLIF) (p. 1497): unilateral graft placement though a facet take-down on that side
2. procedures to increase disc space height and thereby indirectly decompress neural foramina without direct decompression
   a) anterior lumbar interbody fusion (ALIF) (p. 1493): through laparotomy
   b) lateral lumbar interbody fusion (p. 1498): some techniques trademarked as extreme lateral interbody fusion (XLIF™) or direct-lateral (DLIF™)
   c) axial lumbar interbody fusion (Ax-LIF): L5-S1 only
3. limitation of extension by interspinous spacer: e.g. X-Stop® (see below)



### Choosing which procedure to use

Items that factor into consideration when choosing which procedure to use include:

1. consider indirect decompression (lateral interbody fusion (e.g. XLIF® or DLIF®), ALIF, interspinous decompression (e.g. X-Stop):
   a) when foraminal stenosis appears to be the dominant problem (e.g. with loss of disc space height, facet hypertrophy, on the concave side of a scoliotic curve)
   b) previous spine surgery that might make exposure of the nerves more difficult or risky
   c) when the disc space is compressed (more difficult to distract an already tall disc space with normal disc)
2. consider direct decompression (e.g. laminectomy)
   a) "pinpoint" central canal stenosis especially when disc height and neural foramina are well preserved
   b) where a significant contributor to the compression is a focal, correctable lesion, e.g. herniated disc, synovial cyst, intraspinal tumor
   c) to avoid a fusion (in select cases)
3. consider motion-preservation surgery when a fusion is undertaken at a level and the adjacent level is already starting to show some degenerative changes that have not yet reached a surgical magnitude. Motion preservation at this adjacent segment theoretically shields it from some of the transmitted stresses from the fused level
4. situations where a fusion should be considered in addition to direct or indirect decompression of the nerves:
   a) spondylolisthesis (especially > Grade I)
   b) symptomatic sagittal imbalance or degenerative scoliosis
   c) dynamic instability on flexion/extension lateral lumbar spine x-rays
   d) expectation that the decompression will destabilize the spine (e.g. facet takedown for a TLIF)
   e) multiply recurrent herniated disc (when this is the third or more operation for the same disc)
   f) controversial: "black disc" on MRI with positive concordant discogram at this level: fusion without decompression has been advocated when there is no neural compression

## When spondylolisthesis is present

May occur without decompression, but is more common following surgery.[10] However, lumbar instability following decompressive laminectomy is rare (only ≈ 1% of all laminectomies for stenosis will develop progressive subluxation). Fusion is rarely required to prevent progression of subluxation with degenerative stenosis.[28]

For Grade I and low Grade II spondylolisthesis, laminectomy without fusion may be considered. Stability (without need for instrumentation) is thought to be maintained if >50–66% of the facets are preserved during surgery and the disc space is not violated (maintains integrity of anterior and middle column). Younger or more active patients are at higher risk of subluxing. Patients with a tall (normal) disc space are at higher risk of subluxing than those with collapsed disc space.

One approach is to obtain flexion/extension x-rays pre-op, and follow patients after decompression. Those who develop symptomatic slippage post-op are treated by fusion, possibly in conjunction with spinal instrumentation.

When surgery is indicated, □ Table 72.6 serves as a guide to the type of procedure.

## Laminectomy/laminotomy – surgical technique

Posterior approach with removal of the spines and lamina of affected levels (surgical "unroofing"), along with the associated ligamentum flavum. Individual nerve roots are palpated for compression within their neural foramen, with foraminotomies performed at appropriate levels. Doing a total L4 laminectomy for stenosis allows access to the L4–5 foramen, and the upper part of the L5-S1 foramen. If, in addition, the lower part of L3 is also removed, access is gained to the inferior pedicle of L3 and thus the L3–4 neural foramen. Undercutting the superior articular facet is often necessary to decompress the nerves in the lateral recess and neural foramen (p.1097). Treatment of moderate stenosis at adjacent levels appears warranted as these levels have been shown to have a significant likelihood of becoming symptomatic later.[30]

Alternatively, laminotomies (as opposed to laminectomies) may be performed in cases where the central canal has a normal AP diameter, but the lateral canal gutter is stenotic.[31,32] Multilevel subarticular fenestrations are another slight variation on this theme.[33]

Position: either of the following is acceptable

1. prone: on a frame or chest rolls or knee-chest position to decompress the abdomen to decrease venous pressure and thus reduce bleeding
2. lateral decubitus position: if there is no laterality to symptoms, right lateral decubitus (left-side-up) is easier for most right-handed surgeons to use angled Kerrison rongeur parallel to nerve roots



## Booking the case: Lumbar laminectomy

Also see defaults & disclaimers (p.27).

1. position: prone
2. implants: for fusions, schedule with the vendor for the desired implants and associated instrumentation
3. consent (in lay terms for the patient – not all-inclusive):
   a) procedure: through the back to remove bone, ligament and any other tissue that is pressing on the nerve(s). If a fusion is to be done, then typically this will be accomplished using screws, rods and small cages, as required
   b) alternatives: nonsurgical management
   c) complications: usual spine surgery complications (p.28), plus there might not be the amount of pain relief desired (back pain does not respond as well to surgery as nerve-root pain. The surgery slightly weaken the spine, and as a result, about 15% people will need a fusion at a later date

## Minimally invasive spine surgery (MISS) decompression

Usually using ≈ 1" incisions and expandable retractors.

1. options include bilateral laminotomies (see above)
2. bilateral decompression through a unilateral laminotomy
   a) entry site: 3.5–4 cm off the midline to permit the needed angle

b) when using a retractor with an "open side" orient the retractor with the open side facing laterally (e.g. with the Nuvasive Maxcess® place the handles medially) to permit the angle needed for contralateral decompression
c) the laminectomy and facet takedown (usually for a TLIF) are done
d) open the ligamentum flavum on the side you're working on, to visualize the posterior extent of the spinal canal, to permit finding the plane between the posterior part of the ligamentum flavum and the undersurface of the bone
e) the ligamentum flavum is left in place on the contralateral side to protect the dura during drilling
f) complete the decompression and disc removal on the side you're working on
g) the undersurface of the bone (spinous process and contralateral lamina) are then drilled to decompress the contralateral side
h) once the undersurface of the contralateral posterior canal has been drilled, the ligamentum flavum is removed with pituitary rongeurs. It is possible to even do a contralateral foraminotomy at this point (curved Kerrison rongeurs are very helpful for this)
i) pedicle screws are placed through the open side, and then percutaneously through the contralateral side
j) this is generally followed by a transforaminal lumber interbody fusion (TLIF)

## Interspinous process decompression/stabilization/fusion

Interspinous spacers (e.g. X-Stop™ (Medtronic)) limit extension at 1 or 2 levels (without fusion), preventing narrowing of the associated neural foramen, and may also off-load the facet joints and even the disc. "Success rate": 63%at 2 years. This device may be used as a standalone.

Interspinous plates (e.g. Aspen® (Lanx), Affix™ (Nuvasive), Spire® (Medtronic)) clamp across two spinous processes to fixate them (unlike X-Stop™ which just limits extension). The Aspen® clamps have a space for a graft which may optionally be used to promote fusion between the spinous processes. Interspinous plates may be used to augment other constructs e.g. lateral interbody fusion,[34] but are not intended for standalone use. Biomechanical stability is reported to be similar to bilateral pedicle screws in flexion, and unilateral pedicle screws in lateral bending.[35]

Contraindications (includes exclusionary criteria from the IDE study):

1. instability at level considered for procedure: spondylolisthesis > Grade 1 or scoliosis with Cobb angle ≥ 25°
2. cauda equina syndrome
3. acute fracture of the spinous process
4. bilateral pars defects (disconnects spinous process from the anterior elements)
5. osteoporosis. Contraindications per the IDE: DEXA scan (p.1009) with spine or hip T-score < –2.5 (i.e. more than 2.5 SD below the mean for normal adults) in the presence of ≥ 1 fragility fractures. Concerns: spinous process fracture at the time of insertion, or late subsidence due to microfractures. However, Kondrashov[36] interprets a T-score < –2.5 anywhere as indicative of osteoporosis (even without fragility fractures). Options here include:
   a) augmenting the spinous processes by injecting ≈ 0.5–1 cc of PMMA into each spinous process (SP) with a 13 Ga needle inserted ≈ halfway into the SP on lateral fluoro[36] prior to dilating the interspace or placing the X-Stop. Verify central position within SP on AP fluoro, and monitor injection on fluoro
   b) X-Stop$^{PK}$® made of titanium and PEEK (the modulus of elasticity of PEEK is closer to bone than titanium is)
6. ankylosed level (i.e. already fused)
7. L5-S1 level: the spinous process of S1 is usually too small (not usually an issue since symptomatic stenosis at L5-S1 is rare)
8. age < 50 years: not studied in IDE investigation

Surgical pointers:

1. it is critical that the spacer sit in the anterior third of the spinous process
2. results may be better with the patient awake, under local anesthesia, lying on their side in a position that they feel is relieving their pain (thus opening up the critical levels). This may reduce the risk of undersizing the prosthesis

Post-op (based on manufacturer's recommendations):
1. to avoid spinous process stress fracture: build-up physical activity gradually
2. 1st 6 weeks post-op: no spine hyperextension, no heavy lifting. Minimize stair climbing
3. initially, walking (for <1 hour) is recommended as long as it is comfortable
4. at 2 weeks post-op: cycling (stationary or bicycle) may be added
5. 6 months post-op: may add sports such as swimming, golf, racquetball, tennis, running or jogging

## Gill procedure

This procedure, and its modifications,[37] consist of radical decompression of nerve roots including removal of the loose posterior elements and total facetectomy. This is most usually followed by fusion (posterolateral or interbody). Fusion rate may be enhanced with the use of internal fixation (e.g. transpedicular screw-rod fixation).[38]

## Reduction of spondylolisthesis

Reduction of spondylolistheseis can be accomplished with instrumentation, and requires a fusion.

The risk of nerve root injury with reduction of grade I or II spondylolisthesis is low.

Reduction of high-grade (grade III or IV) spondylolisthesis carries a risk of radiculopathy (e.g. L5 radiculopathy in cases of L5-S1 spondylolisthesis) in 50%of cases (some permanent) and may produce a cauda equina syndrome, probably from stretching nerve roots by distraction. Some have recommended intraoperative stimulation of nerve while performing EMG recording as the listhesis is gradually reduced, and stopping if the current required for stimulation increases 50%above baseline.

## Isthmic spondylolisthesis (spondylolysis) – pars interarticularis defect

Instrumentation and/or fusion

### Practice guideline: Fusion in patients with lumbar stenosis without spondylolisthesis

Level III[39]:
- in situ posterolateral fusion is not recommended following decompression in patients with lumbar stenosis in whom there is no evidence of preexisting spinal instability or likely iatrogenic instability due to facetectomy
- in situ posterolateral fusion is recommended in patients with lumbar stenosis in whom there is evidence of spinal instability
- the addition of pedicle-screw instrumentation is not recommended in conjunction with posterolateral fusion following decompression



### Practice guideline: Fusion in patients with lumbar stenosis and spondylolisthesis

Level II[40]: posterolateral fusion is recommended for patients with stenosis and associated degenerative spondylolisthesis who require decompression

Level III[40]: pedicle screw fixation as an adjunct to posterolateral fusion should be considered in patients with stenosis and spondylolisthesis in cases where there is pre-op evidence of spinal instability or kyphosis at the level of the spondylolisthesis or when iatrogenic instability is anticipated (note: the definition of "instability" and "kyphosis" varies, and has not been standardized).

Fusion may accelerate degenerative changes at adjacent levels. Some surgeons recommend fusion at levels of spondylolisthestic stenosis.[5,30] Patients with combined degenerative spondylolisthesis, stenosis, and radiculopathy may be reasonable candidates for fusion.[41]

## Booking the case: Lumbar lami ± fusion for stenosis

Also see defaults & disclaimers (p. 27).

1. position: prone
2. implants: for fusions, schedule with the vendor for the desired implants and associated instrumentation
3. consent (in lay terms for the patient – not all-inclusive):
   a) procedure: through the back to remove bone, ligament and any other tissue that is pressing on the nerve(s). If a fusion is to be done, then typically this will be accomplished using screws, rods and small cages, as required
   b) alternatives: nonsurgical management
   c) complications: usual spine surgery complications (p. 28), plus there might not be the amount of pain relief desired (back pain does not respond as well to surgery as nerve-root pain). There can be problems with the implants, including breakage, migration (slippage), or undesirable positioning which may require further surgery.

# 72.9 Outcome

## 72.9.1 Morbidity/mortality

Risk of in-hospital mortality is 0.32%[17] Other risks include: unintended durotomy (p. 1055) (0.32%[17] to ≈ 13%[28,42]), deep infection (5.9%), superficial infection (2.3%), and DVT (2.8%); see also Risks of lumbar laminectomy (p. 1053).

## 72.9.2 Nonunion

Risk factors for nonunion in fusion operations (does not necessarily correlate with success of operation):

1. cigarette smoking delays bone healing and increases the risk of pseudoarthrosis following spinal fusion procedures, especially in the lumbar spine[12]
2. number of levels: in lumbar fusions, fusing 2 levels resulted in increased nonunion rates compared to fusing 1 level[43]
3. NSAIDs: controversial
   a) short-term (≤5 days) post-op use: high-dose ketorolac (120–240 mg/d) was associated with increased risk of nonunion, but low-dose ketorolac (≤110 mg/d), and celecoxib (200–600 mg/d) or rofecoxib (50 mg/d) were not[43]
   b) some feel that long-term NSAID use does lower fusion rate[44]



## 72.9.3 Success of operation

### General information

Patients with a postural component to their pain had much better results (96% good result) than those without a postural component (50% good results), and the relief of leg pain was much more successful than relief of back pain.[45] Surgery is most likely to reduce LE pain and improve walking tolerance.[41]

### Outcome studies

#### SPORT study

There have been many attempts to ascertain the benefit of surgery, including the $13.5 million SPORT study. Shortcomings of the study include: patients were allowed to decline randomization and were then entered into an observational cohort which may introduce bias into the groups, cross-overs were allowed between patients randomized to surgery and those randomized to nonsurgical treatment (degrading the "intention to treat" analysis), no standardized surgical or nonsurgical technique, relatively low long-term follow-up (52% at 8 years), change in paradigm from analyzing intention-to-treat to an as-treated analysis.

Results indicated a strong benefit of surgery at 4-years follow-up[46] that appeared to diminish by 8-years in the randomized cohort but persisted in the observational cohort.[47]

### Other long-term outcome studies

Literature review[17] with long-term follow-up found good or excellent outcome after surgery with a mean of 64% (range: 26–100%). A patient satisfaction survey indicated that 37% were much improved and 29%somewhat improved (total: 66%) post-op.[48] A prospective study found a success rate of 78–88%at 6 wks and 6 months, which dropped to ≈ 70%at 1 year and 5 yrs.[49] Success rates were slightly lower for lateral recess syndrome.

## Reasons for surgical failure

Surgical failure may be divided into two groups:

1. patients with initial improvement who develop recurrent difficulties. Although short-term improvement after surgery is common,[46] many patients progressively deteriorate over time.[47,50] One study found a 27%recurrence of symptoms after 5 years follow-up[30] (30%due to restenosis at the operated level, 30%due to stenosis at a new level ("adjacent segment failure"); 75%of these patients respond to further surgery). Other etiologies include: development of herniated lumbar disc, development of late instability including kyphosis ("proximal junction kyphosis" – PJK), coexisting medical conditions
2. patients who fail to have any post-op pain relief (early treatment failures). In one series of 45 such patients[51]:
   a) the most common finding was a lack of solid clinical and radiographic indications for surgery (e.g. non-radicular LBP coupled with modest stenosis)
   b) technical factors of surgery had less influence on outcome, with the most common finding being failure to decompress the lateral recess (which, in non-fusion cases, requires judicious medial facet resection or undercutting the superior articular facet)
   c) other diagnoses (e.g. arachnoiditis), missed diagnosis (e.g. spinal AVM)

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# 73 Adult Spinal Deformity and Degenerative Scoliosis

## 73.1 General information

### Key concepts

- Adult spinal deformity (ASD) encompasses scoliosis and sagittal imbalance
- Sagittal balance correlates with quality of life measures
- Basic spine measurements needed: LL, PI, PT, ± SVA
- Major alignment objectives: LL = PI ± 9°, PT < 20°, SVA < 5 cm

Adult spinal deformity (ASD) is a broad term that refers to a wide spectrum of structural abnormalities of a mature spine. ASD encompass abnormalities in the coronal plane (scoliosis) as well as abnormalities in the sagittal plane.

The term "adult degenerative scoliosis" (ADS) (as distinguished from idiopathic juvenile scoliosis (IJS)) is often used interchangeably with ASD. Definition of adult degenerative scoliosis: spinal deformity with Cobb angle[1] > 10° in a skeletally mature individual.[2] ADS may be the result of childhood idiopathic scoliosis persisting into adulthood, or may be de novo.

Deformity in ASD can be primarily due to asymmetric disc degeneration or secondary to hip pathology, osteoporosis and asymmetric loads.[3] It subsequently involves posterior elements (including facet joints) and thereafter axial rotation, lateral olisthesis and ligamentous laxity.[2,4] Progressive facet and discogenic degeneration may lead to segmental instability and subsequent central/foraminal stenosis secondary to ligamentum flavum hypertrophy and osteophyte formation[5] and well as spondylolisthesis.

While treatment goals include reduction of pain, symptomatic neural compression and disability due to deformity, the methodology and biomechanics of ADS treatment differ greatly from treating IJS in the adolescent.

ADS tends to progress at an average rate of 3° per year (range: 1–6°).[4] Factors associated with higher rates of progression: Cobb angle > 30°, apical rotation > Grade II (on the Nash-Moe system,[6] which is falling into disuse), lateral listhesis > 6 mm, and an intercrest line through L5.[4] Factors not correlated with rate of progression: age and gender. Controversial associations: osteopenia.



## 73.2 Epidemiology

ASD is more prevalent in patients age > 60 years, however true prevalence is not well defined. > 50% of adults hospitalized with spinal deformity are > 65 years.[7] Incidence of asymptomatic scoliosis ranges from 1.4%-32% and up to 68% in patients > 60.[8]

## 73.3 Clinical evaluation

Location, time and duration pain (leg vs. axial back) are important factors in evaluation of a patient with ASD. These patients may also have symptoms of spinal stenosis (central or radicular), which may require concomitant decompression. The patient's ability to perform activities of daily living and medical co-morbidities (e.g. cardiac, osteoporosis etc.) need be taken into consideration for treatment planning.

Some patients present with obvious spinal deformity (scoliosis, forward flexion at the waist, walking with knees bent).

As with neurogenic claudication, patients tend to be more symptomatic when up on their feet. A significant amount of pain may be generated by attempting to correct for spinal imbalance by using paraspinal muscles as well as retroverting the pelvis (rotating it backward at the hips) and not fully extending the knees. All this extra muscle activity is fatiguing and begins to produce muscle pain in the back and thighs. Patients with ASD tends to be better in the morning when they are rested.

Unlike lumbar spinal stenosis in the absence of scoliosis, symptoms may not relieved by flexion.[2] There may be some relief when supporting the trunk with the arms.

## 73.4 Diagnostic testing

- CT / MRI. Both are recommended for the evaluation of symptomatic spondylosis and ASD to determine extent of neural compression.

 DEXA (dual-energy x-ray absorptiometry). Patients should be evaluated for osteopenia/osteoporosis prior to surgical planning. Medical treatment may be beneficial in the peri-operative period.

Some surgeons use Forteo for 3 months (off-label use – controversial) in an effort to try and quickly increase osteoporotic bone strength for surgery.

 Standing scoliosis x-rays. Recommended for the evaluation of global and regional spinal balance. Pre and postoperative plain films help confirm that alignment objectives are achieved.

Measurements related to sagittal balance are taken from standing scoliosis x-rays (CT & MRI are obtained supine and are not equivalent). Technical requirements for the lateral image:

- x-ray must image from C7 down to the femoral heads
- the patient needs to try to keep the knees straight (extended)
- arms should be folded in front of the chest (and they should not lean or hold on to anything)

Dynamic scoliosis x-rays ("lateral bending films") help determine the degree of curve rigidity preoperatively.

## 73.5 Pertinent spine measurements

### 73.5.1 General information

Quantification of severity of spinal deformity and classification helps guide appropriate treatment paradigms.[9,10]

### 73.5.2 Scoliosis nomenclature

Scoliosis is measured using Cobb angles. On an AP x-ray, the "end vertebrae" are identified at the top and bottom of the scoliotic curve and are defined as the vertebrae with the greatest angle relative to the horizontal plane. One horizontal line is drawn through the superior endplate of the superior "end vertebra", and a second is drawn through the inferior endplate of the inferior "end vertebra." The Cobb angle is the angle between these 2 lines. Curves are named for the convex side (dextroscoliosis = convex to right, levoscoliosis = convex to left).

A non-structural curve can correct on side bending. A structural curve is not flexible.

The major curve is the largest structural curve. A fractional curveis the curve below the major curve.



### 73.5.3 Spino-pelvic parameters

Measurement methodology and pertinent information are shown in  Table 73.1 and illustrated in  Fig. 73.1 and  Fig. 73.2. Basic measurements that can be correlated with pain reduction and quality of life measures:

- LL (lumbar lordosis)
- PI (pelvic incidence)
- PT (pelvic tilt)
- ± SVA (sagittal vertical alignment): while this can be helpful at times, it also appears to be subject to variability depending on how much pain the patient has with standing up straight

With the exception of CSVL, the measurements shown in  Table 73.1 are all taken from a lateral standing x-ray ( Fig. 73.1 and  Fig. 73.2).

## 73.6 SRS-Schwab classification of adult spinal deformity

Adult scoliosis has been classified by the Scoliosis Research Society (SRS)[16] based on its regional/global radiographic features (a modification of previously established adolescent King/Moe and Lenke classifications) and most recently by spino-pelvic parameters as it relates to health-related quality of life[17,18,19] is shown here.

- Coronal Curve types
  - T: Thoracic only (with lumbar curve <30°)
  - L: Thoracolumbar/Lumbar only (with thoracic curve <30°)
  - D: Double curve (both T and T/L curves >30°)
  - N: No major coronal deformity (all coronal curves <30°)
- Sagittal Modifiers
  - Pelvic harmony (PI minus LL)
    - 0: non pathologic (PI–LL<10°)
    - +: moderate deformity (10°<PI–LL<20°)
    - ++: marked deformity ( PI–LL>20°)

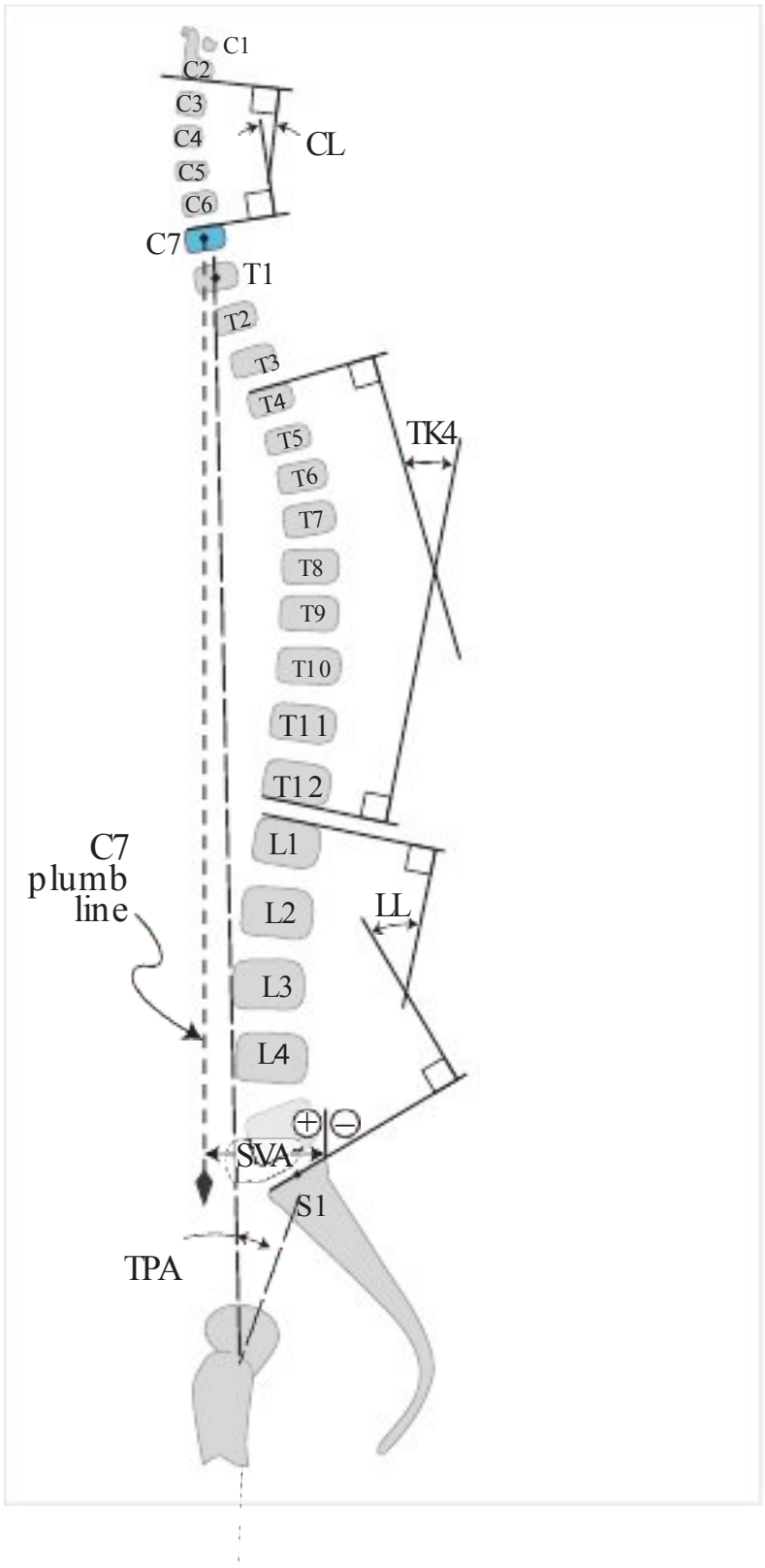



Fig. 73.1 Schematic lateral spine diagram showing method for measuring CL, TK, LL SVA and TPA.



- Global alignment (SVA)
  - 0: non pathologic (SVA<4 cm)
  - +: moderate deformity (4 cm <SVA<9.5 cm)
  - ++: marked deformity (SVA>9.5 cm)
- Pelvic Tilt (PT)
  - 0: non pathologic (PT<20°)
  - +: moderate deformity (20°<PT<30°)
  - ++: marked deformity (PT>30°)

## 73.7 Treatment/management

### 73.7.1 Options

1. observation
2. focal decompression
3. surgical correction of deformity
   a) MIS (minimally invasive (spine) surgery)
   b) Hybrid (MIS+open)
   c) Traditional open surgery (transforaminal lumbar interbody fusion (TLIF), posterior lumbar interbody fusion (PLIF)…)

Treatment options are based on clinical symptoms (axial back pain ± radiculopathy vs. radiculopathy alone) and degree of abnormalities in sagittal (balance need for open osteotomies or anterior column release ACR). Neuropathic symptoms most often originate from foraminal compromised on the

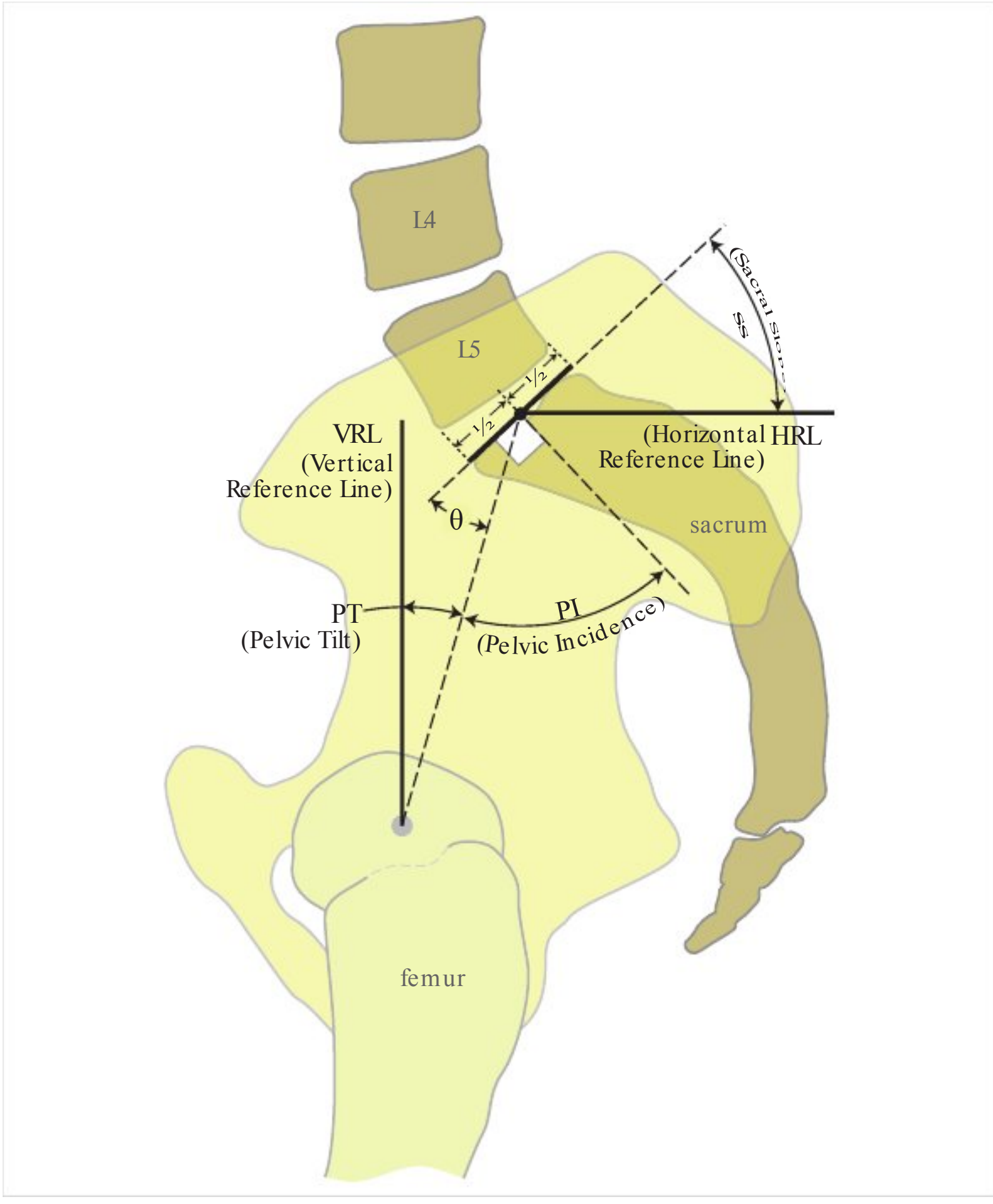
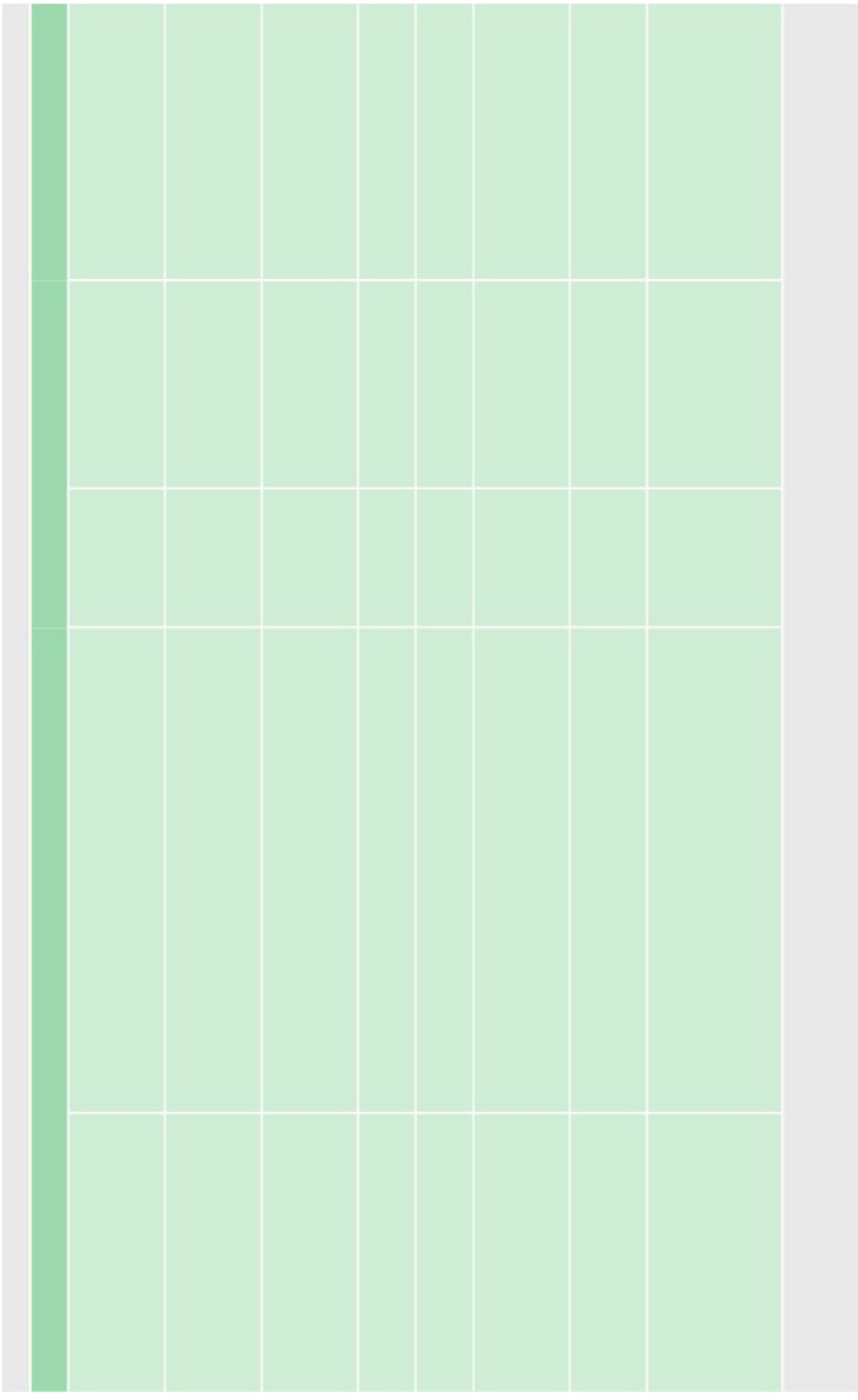



Fig. 73.2 Schematic lateral spine diagram showing method for measuring PT, PI and SS.

concavity of the curve, but can be seen on the convexity in the setting of facet hypertrophy and may improve with indirect decompression and correction in the coronal plane. Significant central stenosis (neurogenic claudication) may require concomitant direct decompression in addition of deformity correction.

Goal of surgery: to improve quality of life measures, neuropathic and axial pain. A surgeon's armamentarium includes traditional open, MIS and hybrid procedures, which is patient and deformity specific. More recently, the surgical decision making paradigms have included MIS techniques to limit approach related morbidity. MIS techniques include lateral interbody fusion, ALLR, MIS-TLIF and percutaneous pedicle screw fixation, which can be used with posterior osteotomies to enhance corrective power.



## 73.7.2 Correction of global spinal balance

Indications for surgery

- Axial back pain ± neuropathic symptoms (deleterious to ADLs)
  - Abnormal SVA
  - ± CSVL (central sacral vertical line) abnormality
  - or derangement of spino-pelvic parameters.
- Patient age and co-morbidities must be taken in to consideration (osteopenia, anesthetic risk and medical co-morbidities can limit correction goal and amount of surgery that is safe)

Summary of spino-pelvic objectives

- LL = PI ± 9°
- PT < 20°
- SVA < 5 cm

In most instances, correction of sagittal imbalance is due to a shortfall of lumbar lordosis (LL) relative to the pelvic incidence (PI) – which can be considered flat back syndrome. That is, LL is usually more than 9° below PI.

Pelvic tilt > 20° suggests the patient is trying to compensate by retroverting the pelvis (some authors accept up to 25° as normal).

The minimum amount of correction that the surgeon tries to achieve is therefore that amount that LL needs to increase to bring it within 9° of PI, and typically also adds in the amount that the patient is compensating (i.e. the amount that PT is greater than 20°) which yields the following approximation (applies when LL is more than 9° lower than PI, and PT is greater than 20°); see Eq (73.1):

$$\text{Increase in LL needed} = (\text{PI} - \text{LL} - 9^\circ) + (\text{PT} - 20^\circ) \quad (73.1)$$

□ Coronal balance. Measured on AP standing scoliosis x-ray. A plumb line is dropped from the center of the C7 VB. If it falls > 4 cm from the midline of the sacrum (where the CSVL is located), there is coronal imbalance (if the plumb line falls to the right of the CSVL it is positive, to the left it is negative).

## 73.7.3 Surgical options for increasing lumbar lordosis

Various surgical techniques may be used to increase the lumbar lordosis to bring it within the desired specifications. If necessary, these techniques can be combined with procedures to decompress the neural elements (e.g. laminectomy). A comparison of the approximate amount of lordosis than can be achieved with different techniques is shown in □ Table 73.2.

□ Transforaminal lumbar interbody fusion (TLIF) and posterior lumbar interbody fusion (PLIF). Traditional operation. May be done open or MIS.

□ Lateral lumbar interbody fusion (LLIF). E.g. XLIF™, DLIF™, OLIF™. Approach through psoas muscle (XLIF, DLIF) or anterior to psoas muscle (OLIF) through a lateral or anterolateral approach. Can distract the vertebral bodies by increasing the height of the disc space and thereby indirectly decompressing the neural elements. If bone quality is good, and there is no instability nor spondylolisthesis > Grade I, a standalone procedure (i.e. without screw instrumentation) may be an option if cage width of at least 22 mm (or preferably 26 mm) in the AP dimension is used.

Table 73.2 Comparison of the amount of lumbar lordosis that can be obtained from various surgical techniques.

| Technique | TLIF/PLIF | LLIF | ALIF | SPO + ACR | PSO |
|---|---|---|---|---|---|
| Degrees of lumbar lordosis | < 0 (i.e. kyphosis) up to 2°[20] | 1°[21] | 6°[20] | 16°[22] | 30–40°[22,23] |

Abbreviations: TLIF = transforaminal lumbar interbody fusion; PLIF = posterior lumbar interbody fusion; LLIF = lateral lumbar interbody fusion; ALIF = anterior lumbar interbody fusion; SPO = Smith-Petersen osteotomy; ACR = anterior column release; PSO = pedicle subtraction osteotomy

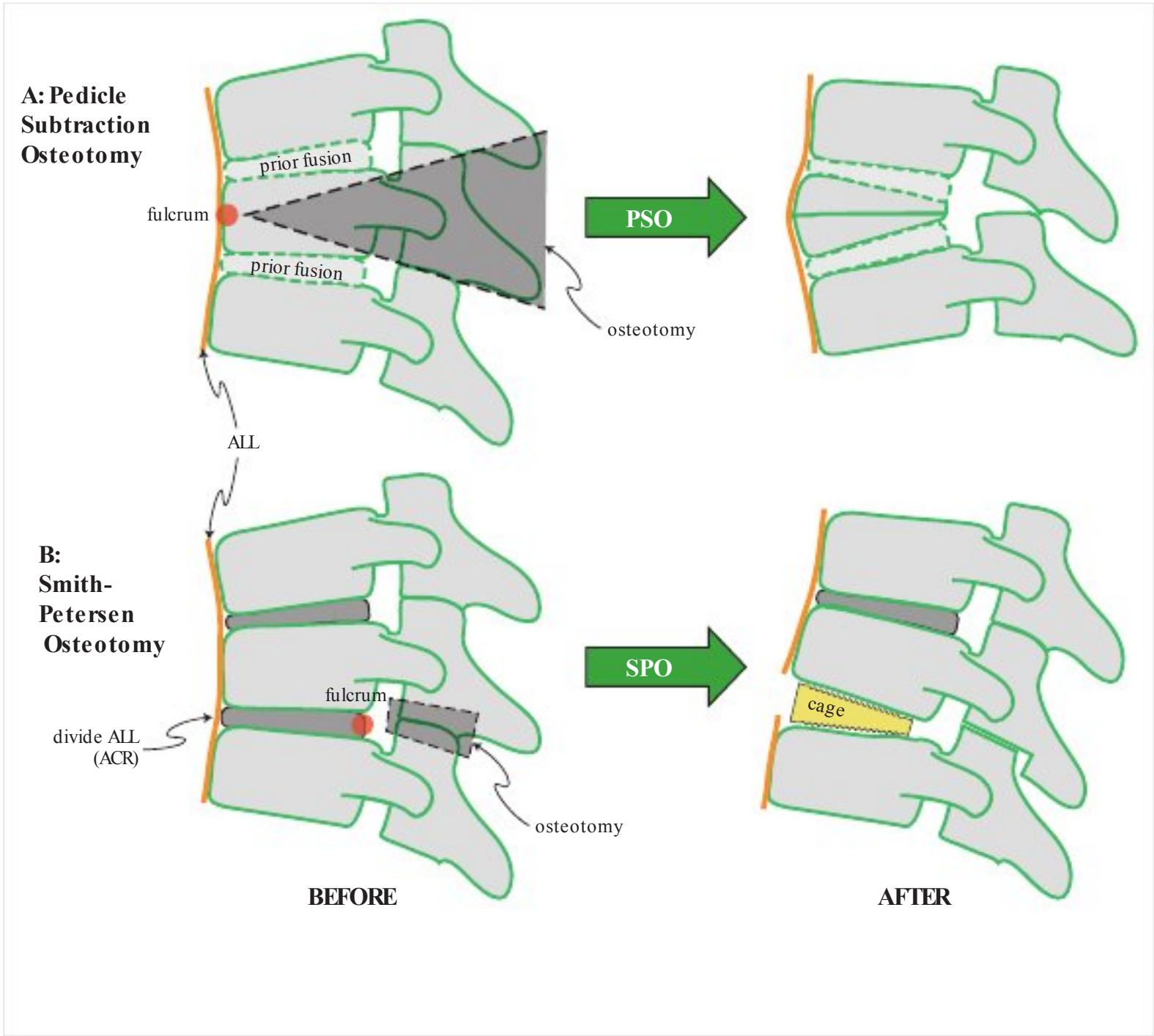



Fig. 73.3 Comparison of A: Pedicle subtraction osteotomy and B: Smith Petersen osteotomy plus ACR. Abbreviations: ALL = anterior longitudinal ligament, ACR = anterior column release.



 Anterior column release (ACR). Involves division of the anterior longitudinal ligament (ALL release (ALLR)) typically with placement of a "hyperlordotic" cage (20–30° lordosis) from an anterior approach (e.g. lateral interbody technique). This is followed by posterior fixation, often with a Smith-Petersen osteotomy (especially for 30° cages) and compression. It can increase LL up to 12° per ACR level and improvement of SVA up to 3 cm (depending on the level at which it is performed).[22,24]

Risk of injury to great vessels, either directly (when cutting the ALL) or indirectly by elongating the anterior column. It is critical to evaluate the great vessels on axial MRI or CT or on angiogram and to not do the procedure if the vessels appear tightly approximated to the bodies or to osteophytes, at that level.

 Smith-Petersen osteotomy (SPO). AKA "chevron or extension osteotomy" can increase lordosis up to 10° per level. Approximately one degree for each millimeter of bone resected.[25,26] Involves removal of bilateral superior and inferior facets along with the ligamentum flavum and portion of the lamina above and below. The created gap is then closed with compression of the posterior elements to give lordosis (resecting posterior elements and using the middle column as a fulcrum to elongate the anterior column,  Fig. 73.3 A).[26,27] LL is increased approximately 1° for every 1 mm of bone removed.

Table 73.3 MIS management recommendations based on severity of ASD[9] with approximate SRS-Schwab Class[17] equivalence.

| | Mild (balanced) | | Moderate (compensated) | | Severe (uncompensated) | |
|---|---|---|---|---|---|---|
| | Deukmedjian at al.[9] | SRS-Schwab[17] | Deukmedjian at al.[9] | SRS-Schwab[17] | Deukmedjian et al.[9] | SRS-Schwab[17] |
| CCA | <30° | N | >30° | T, L or D | >30° | T, L or D |
| PI – LL | <20° | 0 or + | 20–30° | ++ | >30° | ++ |
| SVA | <5 cm | 0 | 5–9 cm | + | >10 cm | ++ |
| PT[a] | <25° | 0 | 25–30° | + | >30° | ++ |
| Recommendations for anterior procedure | MIS LLIF | | MIS-LLIF to neutral vertebrae + ACR | | MIS-LLIF to neutral vertebrae ± ACR | |
| Recommendations for posterior procedure | If PT<20° consider standalone[b], otherwise percutaneous fixation | | Percutaneous fixation to S1 ± facetectomy(ies) | | Open fixation to S2 or iliac + osteotomy(ies) | |

To use this table, determine which category the patient fits into (Mild, Moderate or Severe) using either the Deukmedjian parameters (on which this reference is based) or the roughly equivalent SRS-Schwab parameters shown

Abbreviations: CCA = Cobb coronal angle; LLIF = lateral lumbar interbody fusion (e.g. XLIF, DLIF, OLIF..); ACR = anterior column release
[a] in the SRS-Schwab classification, PT<20° is considered normal
[b] standalone meaning no posterior fixation, assumes that bone quality is not osteoporotic and that a cage width of at least 22 mm is used (to reduce the risk of subsidence)

The term "Ponte osteotomy" is often used interchangeably with SPO, but the Ponte was originally described for treating Scheuermann's kyphoscoliosis.

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□ Pedicle subtraction osteotomy (PSO). Involves removal of the posterior elements including the ligmanetum flavum, lamina and facets widely, followed by isolation and resection of pedicles bilaterally and wedge shaped removal of the vertebral body just barely up to the ventral cortex. The created gap is then closed by compression of the posterior elements and subsequent greenstick fracture of the isolated ventral cortex (□ Fig. 73.3 B).[23] Can increase LL by 30°- 40° per level, improvement of SVA 5.5 – 13 cm per level.[22,23]

This procedure is technically challenging and is associated with high blood loss (3 L in 1 series[28]) and increased risk of complications (including proximal junctional kyphosis (PJK) in 23%[28]) compared to SPO. Generally reserved for spines that have previously been fused where it is not possible to get the amount of lordosis needed from the unfused levels. The PSO is a spine "shortening" procedure.

Uses the anterior column as a fulcrum. Usually limited to levels below the conus medullaris (i.e. L1–2) due to inward buckling of the dura (L3 is the most common level). Intraoperative electrophysiologic monitoring is required. Relative contraindication: poor bone quality.

□ Anterior lumbar interbody fusion (ALIF). Best for L5-S1 (where the great vessels tend not to interfere with the access, and where every degree of correction produces a more significant amount of improvement in SVA than at other levels as a result of being at the lowest point in the spine).

## 73.7.4 Guidelines for MIS treatment for ASD

A simple algorithm for MIS management of sagittal imbalance based on spino-pelvic parameters and the approximate SRS-Schwab class is shown in □ Table 73.3.[9] See references[10,29] for a more detailed protocol.

Currently under investigation by the SRS: when, and to what degree, does improvement in sagittal balance occur with simple decompression (possibly with a minimal fusion) as a result of pain relief permitting the patient to stand up straighter with less pain.

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# 74 Special Conditions Affecting the Spine

## 74.1 Paget's disease of the spine

### 74.1.1 Pathophysiology

Paget's disease (PD) (AKA osteitis deformans) is a disorder of osteoclasts (possibly virally induced) causing increased rate of bone resorption with reactive osteoblastic overproduction of new, weaker, woven bone, producing characteristic "mosaic pattern."

Initially there is a "hot" phase with elevated osteoclastic activity and increased intraosseous vascularity. Osteoblasts lay down a soft, nonlamellar bone. Later a "cool" phase occurs with disappearance of the vascular stroma and osteoblastic activity leaving sclerotic, radiodense, brittle bone[1] ("ivory bone").

### 74.1.2 Malignant degeneration

A misnomer, since the malignant changes actually occur in the reactive osteoblastic cells. About 1% (reported range: 1–14%) degenerate into sarcoma (osteogenic sarcoma, fibrous sarcoma, or chondrosarcoma),[2 (p 2642)] with the possibility of systemic (e.g. pulmonary) metastases. Malignant degeneration is much less common in the spine than in the skull or femur.

### 74.1.3 Epidemiology

Prevalence: ≈ 3% of population > 55 years old in the U.S. and Europe, much lower in Asia.[3] Slight male predominance. Family history of Paget's disease is found in 15–30% of cases (accuracy is poor since most are asymptomatic).

### 74.1.4 Common sites of involvement

Affinity for axial skeleton, long bones and skull. In approximate descending order of frequency: pelvis, thoracic and lumbar spine, skull, femur, tibia, fibula, and clavicles.

### 74.1.5 Neurosurgical involvement

PD may present to the neurosurgeon as a result of:

1. back pain: usually not as a direct result of vertebral bone involvement (see below)
2. spinal cord and/or nerve root symptoms
   a) compression of the spinal cord or cauda equina (relatively rare)
   b) spinal nerve-root compression
   c) vascular steal due to reactive vasodilatation adjacent to involved areas
3. with skull involvement:
   a) compression of cranial nerves as they exit through bony foramina: 8th nerve is most common, producing deafness or ataxia (p.1400)
   b) skull base involvement → basilar invagination
4. to ascertain diagnosis in unclear bone lesions of the spine or skull

### 74.1.6 Presentation

#### General information

Only ≈ 30% of pagetic sites are symptomatic,[4] the rest are discovered incidentally. The overproduction of weak bone may produce bone pain (the most common symptom), predilection for fractures and compressive syndromes: cranial nerve (p.1400), spinal nerve root... Painless bowing of a long bone may be the first manifestation. A number of patients present due to pain from joint dysfunction related to PD.

The overwhelming majority of pagetic lesions are asymptomatic[5 (p 1413)] with lesions detected on radiographs or bone scan obtained for other reasons or as part of a work-up for an elevated alkaline phosphatase. Although the most common complaint in patients with Paget's disease is of back pain, this is attributable to pagetic involvement alone in only ≈ 12%,[6] in the remainder it is secondary to other factors, some of which are described below.

### Symptoms that may be related to the Paget's disease itself

Symptoms from the following are slowly progressive (usually present for >12 months; rarely <6 mos):

1. neural compression
   a) causes of compression
      - due to expansion of woven bone
      - due to osteoid tissue
      - pagetic extension into ligamentum flavum and epidural fat[7]
   b) sites of compression
      - spinal cord (see below)
      - nerve root in neural foramen
2. osteoarthritis of facet joints (Paget's disease may precipitate osteoarthritis[6])

Symptoms from the following tend to progress more rapidly:

1. malignant (sarcomatous) change of involved bone (rare, see above)
2. pathologic fracture (pain usually sudden in onset)
3. neurovascular (compromise of vascular supply to nerves or spinal cord) by
   a) compression of blood vessels (arterial or venous)
   b) pagetic vascular steal (see below)

### Spinal cord symptoms

Myelopathy or cauda equina syndrome may be due to spinal cord compression or from vascular effects (occlusion, or "steal" due to reactive vasodilatation of nearby blood vessels[5 (p 1415)]). Only ≈ 100 cases had been described as of 1981.[8] Characteristically, 3–5 adjacent vertebrae are involved,[9 (p 2307)] whereas monostotic involvement is usually asymptomatic.[10] In case reports in the literature, progressive quadri- or paraparesis was the most common presentation.[11] Sensory changes are usually the first manifestation, progressing to weakness and sphincter disturbance. Pain was the only symptom in a neurologically intact patient in only 5.5%

A rapid course (averaging 6 wks) with a sudden increase in pain is more suggestive of malignant degeneration.

## 74.1.7 Evaluation

1. lab work (serum markers may be normal in monostotic involvement):
   a) serum alkaline phosphatase: usually elevated (this enzyme is involved in bone synthesis and so may not be elevated in purely lytic Paget's disease[5 (p 1416)]); mean 380 ± 318 IU/L (normal range: 9–44).[6] Bone-specific alkaline phosphatase may be more sensitive and may be useful in monostotic involvement[3]
   b) calcium: usually normal (if elevated, one should R/O hyperparathyroidism)
   c) urinary hydroxyproline: hydroxyproline is found almost exclusively in cartilage. Due to the high turnover of bone, urinary hydroxyproline is often increased in PD with a mean of 280 ± 262 mg/24 hrs (normal range 18–38)[6]
2. bone scan: lights up in areas of involvement in most, but not all[6] cases
3. plain x-rays:
   a) localized enlargement of bone: a finding unique to PD (not seen in other osteoclastic diseases, such as prostatic bone mets)
   b) cortical thickening
   c) sclerotic changes
   d) osteolytic areas (in skull → osteoporosis circumscripta; in long bones → "V" shaped lesions)
   e) spinal Paget's disease often involves several contiguous levels. Pedicles and lamina are thickened, vertebral bodies are usually dense and compressed with increased width. Intervening discs are replaced by bone
4. CT: hypertrophic changes at the facet joints with coarse trabeculations

## 74.1.8 Treatment

### Medical treatment for Paget's disease

#### General information

There is no cure for Paget's disease. Medical treatment is indicated for cases that are not rapidly progressive where the diagnosis is certain, for patients who are poor surgical candidates, and pre-op if

excessive bleeding cannot be tolerated. Medical therapy reverses some neurologic deficit in 50% of cases,[12] but generally requires prolonged treatment (≈ 6–8 months) before improvement occurs, and may need to be continued indefinitely due to propensity for relapses. Medications used include the following.

### Calcitonin derivatives

Parenteral salmon calcitonin (Calcimar®)[12]: reduces osteoclastic activity directly, osteoblastic hyperactivity subsides secondarily. Relapse may occur even while on calcitonin. Side effects include nausea, facial flushing, and the development of antibodies to salmon calcitonin (these patients may benefit from a more expensive synthetic human preparation (Cibacalcin®) starting at 0.5 mg SQ q d[13]).

□ 50–100 IU (medical research council units) SQ q d × 1 month, then 3 injections per week for several months.[3] If used pre-op to help decrease bony vascularity, ≈ 6 months of treatment is ideal. Doses as low as ≈ 50 IU units 3 × per week may be used indefinitely post-op or as a sole treatment (alkaline phosphatase and urinary hydroxyproline decline by 30–50% in > half of patients in 3–6 months, but they rarely normalize).

### Bisphosphonates

These drugs are pyrophosphate analogues that bind to hydroxyapatite crystals and inhibit reabsorption. They also alter osteoclastic metabolism, inhibit their activity, and reduce their numbers. They are retained in bone until it is resorbed. Oral absorption of all is poor (especially in the presence of food). Bone formed during treatment is lamellar rather than woven.

Etidronate (Didronel®) (AKA EHDP): reduces normal bone mineralization (especially at doses ≥ 20 mg/kg/d) producing mineralization defects (osteomalacia) which may increase the risk of fracture but which tend to heal between courses.[14] Contraindicated in patients with renal failure, osteomalacia, or severe lytic lesions of a LE. □ 5–10 mg/kg PO daily (average dose: 400 mg/d, or 200–300 mg/d in frail elderly patients) for 6 months, may be repeated after a 3–6 month hiatus if biochemical markers indicate relapse.

Tiludronate (Skelid®): unlike etidronate, does not appear to interfere with bone mineralization at recommended doses. Side effects: abdominal pain, diarrhea, N/V. □ 400 mg PO qd with 6–8 ounces of plain water > 2 hrs before or after eating × 3 months. Available: 200 mg tablets.

Pamidronate (Aredia®): much more potent than etidronate. May cause a transient acute flu-like syndrome. Oral dosing is hindered by GI intolerance, and IV forms may be required. Mineralization defects do not occur in doses < 180 mg/course. □ 90 mg/d IV × 3 days, or as weekly or monthly infusions.

Alendronate (Fosamax®): does not produce mineralization defects (p.1010).

Clodronate (Ostac®, Bonefos®): □ 400–1600 mg/d PO × 3–6 months. 300 mg/d IV × 5 days (may be available outside the U.S.).

Risedronate (Actonel®): does not interfere with bone mineralization in recommended doses.[15] □: 30 mg PO q d with 6–8 oz. of water at least 30 minutes before the first meal of the day.

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## Surgical treatment

### General information

In general, conservative treatment of fractures in PD are associated with a high rate of delayed union.

### Surgical indications for spinal Paget's disease

1. rapid progression: indicating possible malignant change or spinal instability
2. spinal instability: severe kyphosis or compromise of canal by bone fragments from pathologic fracture. Although the collapse is usually gradual, sudden compression may occur
3. uncertain diagnosis: especially to R/O metastatic disease (osteoblastic lesions)
4. failure to improve with medications

### Surgical considerations

1. profuse bleeding is common: if significant bleeding would present an unusual problem, treat for as long as feasible pre-op with a bisphosphonate or calcitonin (see above)
   a) use bone wax to help control bleeding
   b) hemostasis may be difficult

2. to treat resultant spinal stenosis: decompressive laminectomy is the standard procedure in the thoracic region.[11] However, if most of the pathology is anterior, consideration should be given to anterior approach
3. bone is often thickened, and may be fused with obliteration of interspace landmarks. A high-speed drill is usually helpful
4. post-op medical treatment may be necessary to prevent recurrences[12]
5. osteogenic sarcoma
   a) surgery and chemotherapy are used, cure is less likely than in primary osteosarcoma of non-pagetic origin
   b) biopsy proven of the scalp requires en-bloc excision of scalp and tumor

### Surgical outcome

See reference.[11]

In 65 patients treated with decompressive laminectomy, 55 (85%) had definite but variable degrees of improvement. Patients who had only minimal improvement were often ones with malignant changes. One patient was worse after surgery, and the operative mortality was 7 patients (10%). Survival with malignant degeneration is <5.5 mos after admission.

## 74.2 Ankylosing spondylitis

### 74.2.1 General information

#### Key concepts

- Prototypical spondyloarthritidy, now referred to as radiographic axial spondyloarthritis
- Seronegative (absence of rheumatoid factor), associated with HLA-B27
- Begins in SI joints (sine qua non of involvement) progressing rostrally
- Clinical: morning back stiffness, kyphotic deformity limits chest expansion
- X-ray findings: “bamboo spine”, Andersson lesions, progressive thoracic kyphosis
- Fragile rigid spine highly susceptible to fracture and SCI even after low energy trauma
- Severe deformity, neurologic involvement or unstable fracture warrants surgical intervention



Historically known as Marie-Strümpell disease, Ankylosing spondylitis (AS) is an HLA-B27 associated inflammatory disease and the prototype of a group of diseases known as the spondyloarthritides (SpA). Common features are inflammatory back pain, negative serology for rheumatoid factor and absence of rheumatoid nodules, and asymmetric oligoarthritis predominantly of lower extremities. Currently referred to in the literature as Radiographic axial Spondyloarthritis (RaxSpA), AS is believed to be the late stage of a single disease entity, axial Spondyloarthritis (axSpA) distinguished by definitive evidence of sacroilitis on plain radiographs. Other conditions under this rubric include: psoriatic arthropathy, Reiter’s disease, juvenile spondyloarthropathy… AS has in the past also been known as rheumatoid spondylitis, or rheumatoid arthritis of the spine, but use of these terms is discouraged because of the lack of rheumatoid factor. The spine is the primary skeletal site involved, usually starting in the sacroiliac joints and lumbar spine and progressing rostrally.

Enthesopathy: nongranulomatous inflammatory changes at the entheses (attachment points of ligaments, tendons or capsules on bones; the locus of involvement in AS) stimulates re-placement of ligaments by bone ultimately resulting in osteoporotic VBs, calcified intervertebral discs (sparing the nucleus pulposus), and ossified ligaments, producing square appearing VBs with bridging syndesmophytes, the so-called “bamboo spine” or “poker spine”. Extra-articular manifestations (EAMs) include: anterior uveitis, inflammatory bowel disease (IBD) and psoriasis.

Neurosurgical involvement usually results from the following:

1. cauda equina syndrome (CES-AS) : the etiology of CES in AS is frequently unclear, but is usually not due to stenosis or compressive lesion. Onset is slow and insidious and there is a high incidence of dural ectasia.[16] Any patient with AS and neurologic deficit should be assumed to have CES until proven otherwise. Without treatment most patients’ neurological status continues to deteriorate[16]
2. rotatory subluxation: at occipitoatlantal and atlantoaxial joints. May occur as these are typically the last mobile segments of the spine. Incidence is much less than with rheumatoid arthritis. Lesions that might be stable in otherwise normal spines are often not stable in AS
3. myelopathy secondary to bow-stringing of the cord: laminectomy may aggravate

4. acute spinal cord injury (SCI): risk of SCI or CES due to fracture is increased in AS, and may occur following minimal trauma. Injuries are more common in the lower cervical spine. The akylosed spine of AS when fractured creates long lever arms, limiting the ability to absorb impact and rendering even minor fractures very unstable.[17] Delayed deterioration may be due to spinal epidural hematoma.[18]
5. Andersson lesion: discovertebral lesion that results from inflammation or fracture, mechanical stresses prevent lesion from fusion resulting in pseudarthrosis.[19]
6. spinal deformity
7. spinal stenosis: rare
8. basilar impression

## 74.2.2 Epidemiology

Incidence in the general population is ≈ 0.44-7.3 cases per 100,000.[20] Traditionally reported male: female ratio 3:1, however, likely stems from underdiagnosis in women, and more rapid progression of spinal ankylosis in men.[20] Peak incidence: 17-35 yrs age. >90% of patients with AS are HLA-B27 positive (only 8% of people without AS have this antigen), but only 2% of people with HLA-B27 develop clinical AS. Although AS is not hereditary, first degree relatives are at increased risk.

## 74.2.3 Clinical

□ Symptoms. Typical initial presentation is with nonradiating low back pain, morning back stiffness, hip pain and swelling (due to large joint arthritis), exacerbated by inactivity and improved with exercise.[21]

□ Signs. Patrick's test (p.1048) usually positive. Compressing the pelvis with the patient in the lateral decubitus position produces pain.

Schober test (measure distraction between skin marks on the back before and after forward flexion to detect reduced mobility of the spine due to fusion). Is not specific for inflammatory spondylopathies[22] but may be helpful for monitoring ongoing physical therapy.

## 74.2.4 Diagnosis

Diagnosis by experienced rheumatologist is closest thing to a gold standard.[23] The Assessment of SpondyloArthritis international Society (ASAS) recently presented its recommendations for a modified Berlin Algorithm[23] as a potentially useful tool for rheumatologists in diagnosing AS. SI joint involvement is the sine qua non for definite diagnosis. Diagnosis is very involved, and includes: chronic low back pain, buttock pain, sacroiliitis, family history, psoriasis, an inflammatory bowel disease or an arthritis followed in ≤1 month with urethritis, cervicitis or acute diarrhea, an enthesopathy, and a family history, and positive x-rays. The (obsolete) New York Criteria (□ Table 74.1) are shown here to provide a small insight into early attempts to establish diagnostic criteria, but should no longer be used for definitive diagnosis.



## 74.2.5 Radiographic evaluation

□ Plain x-rays. Vital for diagnosis and follow-up. Sacroiliac (SI) joint involvement (on AP pelvic x-rays or on oblique views through the plane of the SI joints) is one of the earliest findings, and the often symmetric osteoporosis followed by sclerosis is characteristic. "Bamboo spine" (see above) is also classic. X-ray of the entire spine is recommended since multiple, non-contiguous (and often unsuspected) fractures are not unusual. See □ Fig. 74.1.

□ CT. Useful for diagnosis of cervical fracture not apparent on plain radiographs, and in preoperative assessment of bony anatomy.

□ MRI. Can rule out spinal epidural hematoma and the occasional herniated disc. May demonstrate dural ectasia in cases of CES-AS syndrome. Andersson lesions: pathologic changes at ligament insertion sites (MRI signal abnormalities at front and back of the endplates) are characteristic. Erosive changes due to pseudarthrosis at the disc space can mimic discitis (high signal on T1WI & T2WI with enhancement).

□ Bone scan. Ratio of uptake of SI joint to sacrum >1.3:1 is suggestive of AS.

| Table 74.1 Modified New York Criteria for AS[24] – not to be used for diagnosis (obsolete) |
|---|
| Diagnosis (see criteria below) |
| Definite AS: radiologic criterion + ≥ 1 clinical criteria |
| Probable AS: radiological criterion without clinical criteria, or 3 clinical criteria without radiological criterion |
| Clinical criteria |
| low back pain > 3 months, improved by exercise, not relieved by rest |
| limitation of lumbar spine motion in both sagittal and frontal planes |
| limitation of chest expansion relative to normal values for age and sex |
| Radiological criterion |
| sacroiliitis |

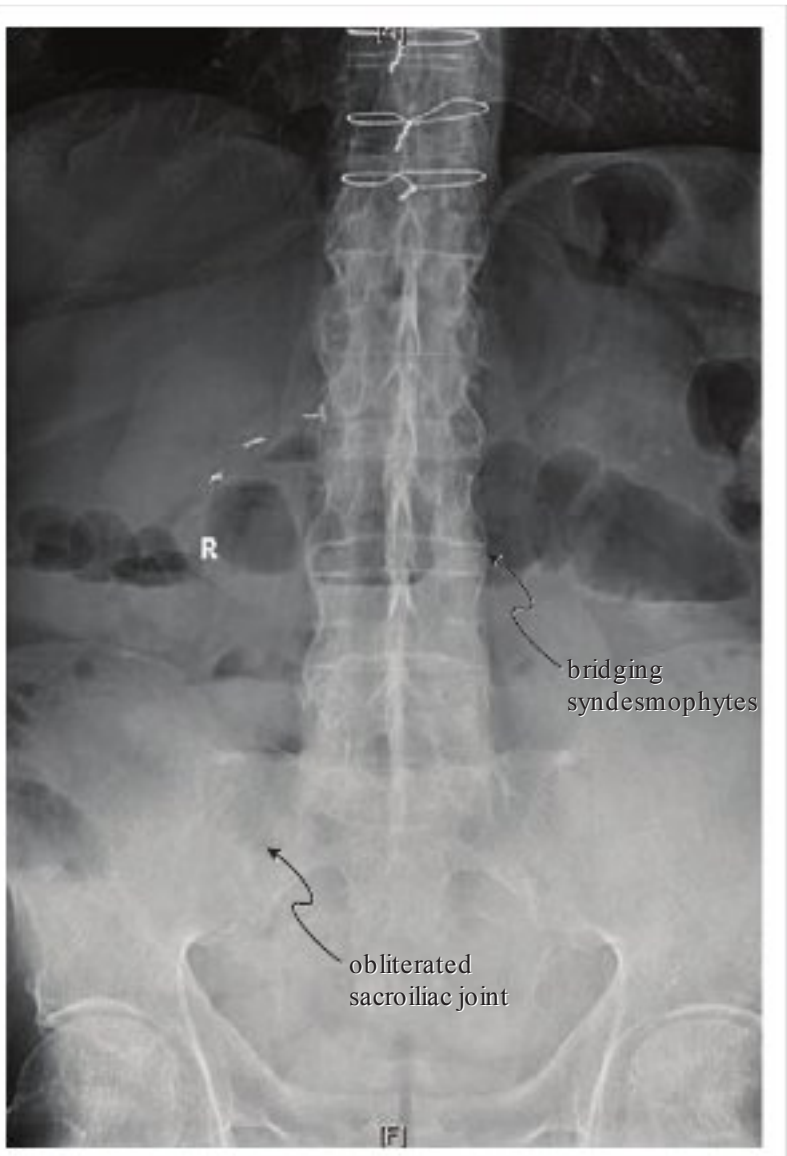


Fig. 74.1 AP lumbar/pelvic x-ray demonstrating "Bamboo spine" and sclerosis of sacroiliac joints



## 74.2.6 Differential diagnosis

1. early on, AS may resemble rheumatoid arthritis. However, in AS nodules do not form in joints, and rheumatoid factor is absent in the serum
2. metastatic prostate Ca in elderly male patients with sacroiliac pain and blastic changes compatible with sacroiliitis
3. Forestier's disease (p.1130) and DISH (p.1129): these overlapping conditions produce exuberant bony overgrowth anterior and lateral to the disc without degeneration and ossification of the disc as in AS. Both spare the facets and SI joints, do not produce flexion deformity, and tend to occur in men > 50 yrs old (older than typical AS)[25]
4. Psoriasis, Reactive Arthritis (Reiter's syndrome), Enteropathic (IBD-related) Arthritis: the spondylitis with these tends to be milder and less uniform, and SI joint involvement is asymmetrical. Cutaneous findings (erythema nodosum, and pyoderma gangrenosum) are absent in AS.[26]

### 74.2.7 Natural history

Progression is slow, and patients usually remain functionally active. Thoracic kyphosis with compensatory increase in cervical and lumbar lordosis is common. The shift in center of gravity together with spine stiffness and fragility predisposes to frequent falls and further spine injuries. Eventually can progress to involvement of costovertebral joints resulting in restrictive lung disease pattern. AS patients are also predisposed to development of fibrotic lung disease in late stages.

### 74.2.8 Treatment

#### General information

Combined ASAS/EULAR recommendations for management of AS are the most comprehensive currently available (copy available at ASAS website). Multidisciplinary, coordinated by a rheumatologist.[27] Goal of treatment is long term quality of life through symptomatic control and prevention of progressive structural damage. NSAIDs are first line pharmacological management.[27] Management of the disease itself may involve TNF inhibitors in patients with persistently high disease activity.[27]

#### Surgical treatment

##### General information

The most common surgical intervention is orthopedic total hip arthroplasty.[27]

##### Cervical fracture

The cervical spine is the most frequent fracture site in AS patients.[28] Patients often cannot distinguish acute fracture pain from chronic inflammatory pain; therefore there should be a low threshold to obtain imaging. It is imperative to determine pre-injury alignment as application of C-collar may cause hyperextension injury.[29] Gentle, low-weight traction with the force vector directed anteriorly and superiorly may be used for initial stabilization.[30,31] Halo vest or surgery for unstable fracture pattern.

Surgical indications:

- irreducible deformity
- deteriorating neurological status in the presence of epidural hematoma or other source of compression[32]
- unstable fracture (e.g. most 3-column fractures which are very unstable as a result of long lever arms of fused segments above and below): halo-vest immobilization is becoming a less frequently employed option for this



Procedures: Decompressive laminectomy if evidence of spinal cord compression.[32] Given poor bone quality and extended lever arms, good fusion bed and a construct extending multiple levels above and below the fracture are critical.[33] Proximally, lateral mass screws up to C3, possible pedicle screw in C2, and pedicle screws in thoracic spine for distal fixation.[33] 360° fusion may provide optimal stabilization in some cases (where feasible).

##### Thoracolumbar fracture

The majority occur at thoracolumbar junction.[33] Can be divided into 3 types[34]:

1. shearing injury: typically acute. Resembles chance fracture, highly unstable 3 column injury[34]
2. wedge compression: typically chronic
3. pseudarthrosis: typically subacute previously missed fracture

Wedge compression or pseudarthrosis; rule out posterior element involvement to determine if fracture is unstable.[33] Stable fracture can be treated with external orthosis. Unstable fractures, consider thicker rods or more rigid rod material to account for increased forces across fracture, and PMMA augmentation to prevent screw pullout.

##### Kyphotic deformity

ASAS/EULAR recommendations include corrective osteotomy for severe disabling deformity.[27] Can be accomplished via open wedge osteotomy, polysegmental wedge oseotomy, or closed wedge osteotomy (lowest complication rate).[35] Cervical deformity is most commonly addressed with wedge

osteotomy at C7 and T1 given absence of vertebral artery in foramen transversarium at these levels. Current trend in literature is to address deformity simultaneously with acute fracture fixation.[31,32]

### Cauda equina syndrome

Although evidence is limited, in the absence of demonstrable neural compression, a lumbo-peritneal (LP) shunt may provide the best chance of improving neurologic dysfunction or halting progression of neurologic deficit.[36]

### Surgical considerations

- Anesthesia team should be aware of Kyphotic deformity and fracture location: nasotracheal or fiberoptic intubation to prevent hyperextension of the neck and exacerbation of neurologic injury.[33]
- Extensive pre-op evaluation: fracture pattern, posterior ligamentous restraint, neurologic compression and function, preexisting bone quality.[33]
- Surgical positioning modified to account for preexisting deformity; support in all regions to prevent hyperextension and exacerbation of neurologic injury.[33]
- ICBG is gold standard; however, often a significant pain source potentially limiting mobilization and increasing likelihood of stasis sequelae (e.g. DVT), consider allograft.[32]
- Extensive knowledge of lateral mass and pedicle anatomy to ensure adequate hardware placement despite distorted bony anatomy and obscuring of typical landmarks.[32]
- Post-op immobilization via halo-vest or TLSO.[33]
- Expedited mobilization out-of-bed as AS patients are predisposed to pulmonary complications.[33]
- Plastic surgery to help manage skin necrosis and wound closure.[33]

## 74.3 Ossification of the posterior longitudinal ligament (OPLL)

### 74.3.1 General information

#### Key concepts

- fibrosis followed by calcification and then ossification of the posterior longitudinal ligament. The process may involve the dura
- more common in Asian population
- most patients have only mild subjective complaints
- 50%of patients have impaired glucose tolerance, respiratory compromise may result from ossification of the costotransverse and costovertebral ligaments
- surgery is best for moderate neuro involvement (Nurick grade 3 &4)



The age of patients with OPLL ranges from 32–81 years (mean=53), with a slight male predominance. The prevalence increases with age. Duration of symptoms averages ≈ 13 months. It is more prevalent in the Japanese population (2–3.5%).[37,38]

### 74.3.2 Pathophysiology

The pathologic basis of OPLL is unknown, but there is an increased incidence of ankylosing hyperostosis which suggests a hereditary basis.

OPLL begins with hypervascular fibrosis in the PLL which is followed by focal areas of calcification, proliferation of periosteal cartilaginous cells and finally ossification.[39] The process frequently extends into the dura. Eventually active bone marrow production may occur. The process progresses at varying rates among patients, with an average annual growth rate of 0.67 mm in the AP direction and 4.1 mm longitudinally.[40]

When hypertrophied or ossified, the posterior longitudinal ligament may cause myelopathy (due to direct spinal cord compression, or ischemia) and/or radiculopathy (by nerve root compression or stretching).

Changes within the spinal cord involve the postero-lateral gray matter more than white matter, suggesting an ischemic basis for the neurologic involvement.

## 74.3.3 Distribution

Average involvement: 2.7–4 levels. Frequency of involvement:

1. cervical: 70–75% of cases of OPLL. Typically begins at C3–4 and proceeds distally, often involving C4–5 and C5–6 but usually sparing C6–7
2. thoracic: 15–20% (usually upper, ≈ T4–6)
3. lumbar: 10–15% (also usually upper, ≈ L1–3)

## 74.3.4 Pathologic classification

See reference.[41]

1. segmental: confined to space behind vertebral bodies, does not cross disc spaces
2. continuous: extends from VB to VB, spanning disc space(s)
3. mixed: combines elements of both of the above with skip areas
4. other variants: includes a rare type of OPLL that is contiguous with the endplates and is confined to the disc space (involves focal hypertrophy of the PLL with punctate calcification)

## 74.3.5 Clinical

Most patients are asymptomatic, or have only mild subjective complaints. This is probably explained by the protective effect of the fusion resulting from OPLL and the very gradual compression.

Natural history: 17% of patients without myelopathy developed myelopathy in one study[42] over 1.6 years mean follow-up. Statistically, the myelopathy-free rate in patients without initially presenting with myelopathy was 71% after 30 years.[42]

## 74.3.6 Evaluation

### Plain x-rays

Often fail to demonstrate OPLL. Flexion/extension views may be helpful in assessing stability.

### MRI

OPLL appears as a hypointense area and is difficult to appreciate until it reaches ≈ 5 mm thickness. On T1WI it blends in with the hypointensity of the ventral subarachnoid space; on T2WI it remains hypointense while the CSF becomes bright. Sagittal images may be very helpful in providing an overview of the extent of involvement, and T2WI may demonstrate intrinsic spinal cord abnormalities which may be associated with a worse outcome.



### Myelography/CT

Myelography with post-myelographic CT (especially with 3D reconstructions) is probably best at demonstrating and accurately diagnosing OPLL.

## 74.3.7 Treatment

### Treatment decisions

Based on clinical grade[41] as follows:

1. Class I: radiographic evidence without clinical signs or symptoms. Most patients with OPLL are asymptomatic.[38] Conservative management unless severe
2. Class II: patients with myelopathy or radiculopathy. Minimal or stable deficit may be followed expectantly. Significant deficit or evidence of progression warrants surgical intervention
3. Class IIIA: moderate to severe myelopathy. Usually requires surgical intervention
4. Class IIIB: severe to complete quadriplegia. Surgery is considered for incomplete quadriplegics showing progressive slow worsening. Rapid deterioration or complete quadriplegia, advanced age or poor medical condition are all associated with worse outcome

In moderate grade patients (Nurick grades 3 & 4,[43] (see □ Table 74.2), surgery provided a statistically significant reduction in deterioration. There was no difference between surgery and conservative treatment in mild grade (Nurick 1 or 2), and surgery was ineffective in severe grade (Nurick 5).[42]

Table 74.2 Nurick grade of disability from cervical spondylosis[43]

| Grade | Description |
|---|---|
| 0 | signs or symptoms of root involvement without myelopathy |
| 1 | myelopathy, but no difficulty in walking |
| 2 | slight difficulty in walking, able to work |
| 3 | difficulty in walking but not needing assistance, unable to work full-time |
| 4 | able to walk only with assistance or walker |
| 5 | chairbound or bedridden |

### Pre-op assessment

Appropriate cardiorespiratory assessment should be made knowing that:

1. respiratory compromise may result from ossification of the costotransverse and costovertebral ligaments
2. 50% of patients have impaired glucose tolerance with the attendant risks associated with diabetes

### Technical considerations for surgery

Severe OPLL increases the risk of spinal cord injury during neck positioning for intubation, and strong consideration should be given to awake nasotracheal intubation.

An anterior approach is generally favored, although laminectomy may be acceptable. SSEP monitoring has been recommended by some.[39] Distraction should be avoided until the spinal cord has been decompressed from the OPLL.

Some authors advocate complete removal of bone from the dura, while others feel it is permissible to leave a thin rim of bone adherent to the dura. Care must be taken in removing bone because it tends to blend imperceptibly with dura and the next thing one may see is bare spinal cord.

Depending on the distance of vertical involvement, vertebral corpectomy with strut grafting may be required. Internal plate fixation is often used as an adjunct. Post-operative immobilization for at least 3 months is employed with rigid collars for single level ACDF or 1–2 level corpectomies, or halo-vest traction for corpectomies >2 levels.

## 74.3.8 Results with surgery

The incidence of pseudarthrosis after vertebral corpectomy and strut graft ranges from 5–10% and increases with the number of levels fused.

In one series there was a 10% incidence of transient worsening of neurologic function following anterior surgery[40] which may have been related to distraction.

The risk of dural tear with CSF leak following an anterior approach depends on the aggressiveness with which bone is removed from the dura, and ranges ≈ 16–25%

Other risks of anterior approaches, e.g. esophageal injury (p.1074), also pertain.

## 74.4 Ossification of the anterior longitudinal ligament (OALL)

OALL of the cervical spine and/or hypertrophic anterior cervical osteophytes may produce dramatic radiographic findings and minimal clinical symptoms. Distinct from Forestier's disease (see below). Cervical involvement may produce dysphagia.[44]

## 74.5 Diffuse idiopathic skeletal hyperostosis (DISH)

### Key concepts

- usually asymptomatic, but may present with globus
- W/U: □ speech therapy consult for dysphagia evaluation (usually includes □ modified barium swallow), □ CT of cervical spine, ± □ digital video esophagoscopy

AKA "DISH", AKA spondylitis ossificans ligamentosa, AKA ankylosing hyperostosis, among others. A condition characterized by flowing osteophytic formation of the spine in the absence of degenerative, traumatic, or post-infectious changes. Affects Caucasians and males more commonly, and usually seen in patients in their mid-60s.

97%of cases occur in the thoracic spine, also in the lumbar spine in 90%, cervical spine in 78%, and all three segments in 70% Sacroiliac joints are spared, unlike ankylosing spondylitis (AS) (p.1123). As with AS, unfused levels may be very unstable.

Risk factors for DISH include: elevated body mass index,[45] elevated serum uric acid,[45] diabetes mellitus,[45] elevated growth hormone or insulin levels.[46]

Usually does not produce clinical symptoms. Patients may have early morning stiffness and mild limitations of activities. Cervical involvement may present with dysphagia or globus pharyngis (the subjective sensation of a lump in the throat, not to be confused with globus hystericus which refers to the sensation of a lump in the throat where there is no identifiable pathology) due to compression of the esophagus between the osteophytes and the rigid laryngeal structures[47] (part of Forestier's disease[48]).

Plain x-rays and CT scan demonstrate the pathology. In cases of dysphagia, evaluation should include speech therapy consult for dysphagia evaluation, barium swallow to help localize the site of obstruction, and DVE (digital video esophagoscopy) to rule-out intrinsic esophageal disease. Plain x-rays and CT scan helps demonstrate the pathology. Cases that do not respond satisfactorily to dietary modifications in patients who are losing weight or are having recurrent episodes of choking or pneumonia should be considered for surgery. An anterior cervical approach, and utilization of a high-speed drill with careful protection of soft-tissue structures (esophagus, carotid sheath) without need for discectomy nor spine stabilization has been recommended.[47] Patients need to be made aware that post-op they are likely to be worse initially (from manipulation of esophagus and possibly disruption of some of the autonomic innervation of the esophagus) and will probably need a gastrostomy feeding tube. By 1 year post-op there may be some improvement.

## 74.6 Scheuermann's kyphosis

### 74.6.1 General information

AKA Scheuermann juvenile kyphosis AKA Scheuermann disease AKA juvenile osteochondrosis of the spine.

Definition: anterior wedging of at least 5° of ≥3 adjacent thoracic vertebral bodies.

Other findings include: Schmorl nodes (p.1060) and endplate narrowing.



### 74.6.2 Presentation

Adolescents: often present as a result of the cosmetic deformity associated with progressive kyphosis which may be mistaken for "slouching."

Adults: often present with pain.

### 74.6.3 Evaluation

Patients require standing scoliosis x-rays and a careful neurologic exam.

The role of MRI is controversial, and not all practitioners obtain this.

### 74.6.4 Radiographic findings

Anterior wedge deformities at multiple levels. End plate irregularities and Schmorl's nodes.

### 74.6.5 Treatment

Bracing may be used in adolescents, unless the patient meets surgical criteria listed below.

Adults presenting with pain often respond to nonsurgical treatment including: physical therapy and NSAIDs.

Surgery is generally indicated for:
1. Curves exceeding 70–75°
2. Unacceptable cosmetic appearance
3. Refractory pain
4. Progressive kyphosis
5. Neurologic deficit

## 74.7 Spinal epidural hematoma

### 74.7.1 General information

Rare. Over 200 cases of varying etiology have been reported,[49] although one–third of recent cases have been associated with anticoagulation therapy.[50] NSAIDs may also be a risk factor.[51] Etiologies include:
1. traumatic: including following LP or epidural anesthesia,[49,52,53,54] fracture (see below), spinal surgery[55] or chiropractic manipulation.[56] Occurs predominantly in patient who is: anticoagulated,[57] thrombocytopenic, or has bleeding diathesis or a vascular lesion
2. spontaneous[58]: rare. Etiologies: hemorrhage from spinal cord AVM (p.1140), from vertebral hemangioma (p.794) or tumor

May occur at any level of the spine, however, thoracic is most common. Most often located posterior to spinal cord (except for hematomas following anterior cervical procedures), facilitating removal via laminectomy.[50]

### 74.7.2 Traumatic spinal epidural hematoma (TSEH) associated with spine fracture

In one series,[59] among 74 trauma patients who underwent emergent spinal MRI, ≈ half of the patients with spine fractures also had TSEH. Treatment was based solely on the fracture, and the outcome in patients with neurologic deficits was no worse in the group with TSEH than in the group without.

### 74.7.3 Presentation

The clinical picture of spontaneous spinal epidural hematoma is fairly consistent but nonspecific. Usually starts with severe back pain with radicular component. It may occasionally follow minor straining, and is less commonly preceded by major straining or back trauma. Spinal neurologic deficits follow, usually progressing over hours, occasionally over days. Motor weakness may go unnoticed when patients are bedridden with pain.



### 74.7.4 Treatment

Recovery of neurologic deficit without surgery is rare (only a handful of case reports in the literature[51]), therefore optimal treatment is immediate decompressive laminectomy in those patients who can tolerate surgery.[50] In one series, most patients who recovered underwent decompression within 72 hrs of onset of symptoms.[60] In another, decompression within 6 hours was associated with better outcome.[55]

High-risk patients: for medically high-risk patients (e.g. acute MI) on anticoagulation, surgical mortality and morbidity is extremely high, and this must be considered when making the decision of whether or not to operate. In patients not operated, anticoagulants should be stopped, and reversed if possible; see Correction of coagulopathies or reversal of anticoagulants (p.166). Consider use of high dose methylprednisolone to minimize cord injury; see Methylprednisolone under spinal cord injury (p.951). Percutaneous needle aspiration may be a consideration in high-risk patients.

## 74.8 Spinal subdural hematoma

Rare. May be posttraumatic (including iatrogenic causes) or may occur spontaneously. Spinal subdural hematomas (SSH) that occur spontaneously or following lumbar puncture usually occur in patients with coagulopathies (primary or iatrogenic).[61]

Conservative treatment is possible in nontraumatic SSHs with minimal neurologic impairment.[61]

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# 75 Other Non-Spine Conditions with Spine Implications

## 75.1 Rheumatoid arthritis

### 75.1.1 General information

More than 85%of patients with moderate or severe rheumatoid arthritis (RA) have radiographic evidence of C-spine involvement.[1]

The grading system of Ranawat et al.[1] for myelopathy is shown in □ Table 75.1 is used in RA as well as some other etiologies of myelopathy.

### 75.1.2 Cervical spine involvement in RA

Common involvement

1. upper cervical spine: involved in 44–88%of RA cases[2] (often found together):
   a) anterior atlantoaxial subluxation: the most common manifestation of RA in the cervical spine, found in up to 25%of patients with RA (see below)
   b) basilar impression (BI): upward translocation of the odontoid process, found in ≈ 8%of patients with RA (p.1138)
   c) pannus of granulation tissue: forms around the odontoid
2. subaxial C-spine (i.e. below C2) subluxation (p.1138)

Less common involvement of the cervical spine in RA

1. posterior subluxation of the atlantoaxial joint: must have either associated fracture of or near total arthritic erosion of odontoid
2. vertebral artery insufficiency secondary to changes at the cranio-cervical junction[3]

### 75.1.3 Atlantoaxial subluxation (AAS) in RA

#### General information

Inflammatory involvement of the atlantoaxial synovial joints causes erosive changes in the odontoid process (anteriorly at the synovial joint with the C1 arch, and posteriorly at the synovial joint with the transverse ligament) and decalcification and loosening of the insertion of the transverse ligament on the atlas. These changes lead to instability allowing a scissoring effect with anterior subluxation of C1 on C2. AAS occurs in ≈ 25% of patients with RA.[3] Mean time between onset of RA symptoms to the diagnosis of AAS in 15 patients: 14 years.[4]



#### Clinical

Signs and symptoms of AAS are shown in □ Table 75.2.

AAS is usually slowly progressive. Mean age at onset of AAS symptoms: 57 years.

Pain is experienced locally (upper cervical and suboccipital regions, often from compression of C2 nerve root) or is referred (to mastoid, occipital, temporal, or frontal regions).

VBI may occur from VA involvement (p.1305).

Table 75.1 Ranawat classification of myelopathy

| Class | Description |
|---|---|
| I | no neural deficit |
| II | subjective weakness + hyperreflexia + dysesthesia |
| III | objective weakness + long tract signs<br>III A = ambulatory<br>III B = quadriparetic & non ambulatory |

Table 75.2 Signs and symptoms of AAS[a] (15 patients with AAS[4])

| Finding | % |
|---|---|
| pain | |
| • local | 67% |
| • referred | 27% |
| hyperreflexia | 67% |
| spasticity | 27% |
| paresis | 27% |
| sensory disturbance | 20% |

[a]other possible findings not reported in this series: clumsiness, neurogenic bladder, Babinski sign

## Radiographic evaluation

### General information

The magnitude of AAS is usually increased with neck flexion.

### Lateral C-spine x-ray

Both the ADI and PADI below are surrogate markers for instability and for spinal cord compression. With the availability of MRI, the ability to directly assess spinal cord compression has diminished the usefulness of these measures, especially for that aspect.

#### Anterior atlantodental interval (ADI)

The ADI (p.213) only gives information about the stability of the C1–2 joint. The normal ADI in adults is <3–4 mm.[5,6] Widening of the ADI suggests possible incompetence of the transverse ligament. However, the ADI does not correlate with the risk of neurologic injury[7,8] and is not predictive of progression from asymptomatic AAS to symptomatic AAS.

#### Posterior atlantodental interval (PADI)

The amount of room available for the spinal cord can vary for any given ADI depending on the AP diameter of the spinal canal and the thickness of any pannus. The PADI (p.213) and the AP diameter of the subaxial canal measured on a lateral C-spine x-ray correlates with the presence and severity of paralysis.[7]

The PADI also predicts neurologic recovery following surgery. Patients with paralysis from AAS showed no recovery if the pre-op PADI was <10 mm.[7]

PADI ≤14 mm has been proposed as an indication for surgical stabilization.



### MRI

The optimal test to evaluate the source and magnitude of upper cord or medulla compression. Demonstrates location of odontoid process, extent of pannus, and effects of subluxation (may need to be performed with head flexed to evaluate this).

## Treatment

### General information

Requires knowledge of the following information:

1. natural history: AAS in most patients progresses, with a small percentage either stabilizing or fusing spontaneously. In one series[9] with 4.5 years mean follow-up, 45%of patients with 3.5–5 mm subluxation progressed to 5–8 mm, and 10%of these progressed to >8 mm
2. once myelopathy occurs, it may be irreversible
3. the worse the myelopathy, the higher the risk for sudden death
4. the chances of finding myelopathy are significantly increased once the subluxation reaches ≥9 mm[10]

5. associated cranial settling further decreases the tolerance for AAS
6. the life expectancy of patients with RA is 10 years less than the general population[9]
7. the morbidity and mortality of surgical treatment (see below)
8. pannus may regress some after medical treatment

### When to treat?

1. symptomatic patients with AAS: almost all require surgical treatment (C1–2 fusion in most cases). For management, see below. Some surgeons do not operate if the maximal dens-C1 distance is <6 mm
2. asymptomatic patients: controversial
   a) some authors feel surgical fusion is not necessary in asymptomatic patient if the dens-C1 distance is below a certain cutoff. Recommendations for this cutoff have ranged from 6 to 10 mm,[11] with 8 mm commonly cited (an unvalidated delineation)
   b) these patients are often placed in a rigid cervical collar, e.g. while outside the home, even though it is generally acknowledged that a collar probably does not provide significant support or protection
   c) NB: some cases of sudden death in previously asymptomatic RA patients may be due to AAS and may then be erroneously attributed to cardiac arrhythmias, etc.[12]

### Surgical management

It is necessary to either reduce the subluxation or to decompress the upper cord before doing a C1-C2 or occipital-C1-C2 fusion.

Menezes assesses all subluxed patients for reducibility using MRI compatible Halo cervical traction as follows: start with 5 lbs, and gradually increase over a period of a week. Most cases reduce within 2–3 days. If not reduced after 7 days then it is probably not reducible. Only ≈ 20% of cases are not reducible (most of these have odontoid >15 mm above foramen magnum).

Most require stabilization via posterior wiring and fusion, either of C1 to C2, or of occiput to C2. The latter is used when fusion is combined with decompression (posterior laminectomy of C1 with posterior enlargement of the foramen magnum). See Atlantoaxial fusion (C1–2 arthrodesis) (p.1479).

Posterior fusion alone does not provide adequate relief if the subluxation is irreducible, or if pannus causes significant compression (however, there may be some reduction of pannus after fusion). In these cases, transoral odontoidectomy may be indicated. Performing the posterior stabilization and decompression first allows some patients to avoid a second operation, and permits the remainder to undergo the anterior approach without becoming destabilized. Still, some surgeons do the odontoidectomy first[11] (requires the patient to remain in traction until the fusion).

Reminder: the patient must be able to open the mouth greater than ≈ 25 mm in order to perform transoral odontoidectomy without splitting the mandible.



### Posterior fusion

See Atlantoaxial fusion (C1–2 arthrodesis) (p.1479) for technique. In RA, erosion and osteoporosis weakens the C1 arch, and extra care is needed to avoid fracturing it.

## 75.1.4 Surgical morbidity and mortality

Because of the frequency of simultaneous involvement of other systems in RA including pulmonary, cardiac, and endocrine, operative mortality ranges from 5–15%[11]

The non-fusion rate for C1–2 wiring and fusion has been reported as high as 50%,[13] typical rates are lower (with 18% of patients in one series developing a fibrous union[11]). The most common site of failure of osseous fusion is the interface between the bone graft and the posterior arch of C1.[14]

## 75.1.5 Post-operative care

The patient is usually mobilized almost immediately post-op in halo vest traction (some use an optional period of maintained traction before mobilization). Impaired healing in RA dictates that the Halo be worn until fusion is well established, as seen on x-ray (usually 8–12 weeks). Sonntag evaluates the patient with flexion-extension lateral C-spine x-rays by disconnecting the halo ring from the vest.

## 75.1.6 Basilar impression in rheumatoid arthritis

### General information

AKA atlantoaxial impaction. Erosive changes in the lateral masses of C1 → telescoping of the atlas onto the body of C2 causing ventral migration of C1 with resultant ↓ in AP diameter of the spinal canal. There is concomitant upward displacement of the dens. The posterior arch of C1 often protrudes superiorly through the foramen magnum. All of these factors lead to compression of the pons and medulla. Rheumatoid granulation tissue behind the odontoid also contributes to the brainstem compression. Vertebral artery and/or anterior spinal artery compression may also cause neurologic dysfunction.

The degree of erosion of C1 correlates with the extent of odontoid invagination.

### Clinical

See □ Table 75.3 for signs & symptoms.

Pain may occur as a result of compression of the C1 and/or C2 nerve roots. Compression of the medulla can cause cranial nerve dysfunction.

Motor exam usually difficult because of severe polyarticular degeneration and associated pain. Sensory findings (all non-localizing): diminished vibratory, position, and light touch.

### Radiographic evaluation

See Basilar invagination & basilar impression (BI) (p.217) for radiographic criteria of BI. Erosion of the tip of the odontoid, commonly seen in RA, obviates use of any measurement that is based on the location of the tip of the odontoid.[15] For this reason, other measures have been developed, including the Clark station,[14] Redlund-Johnell criteria,[16] and Ranawat criteria.[1] Since even these methods will miss up to 6% of cases of BI in RA,[15] it is recommended that suspicious cases be investigated further (e.g. with CT and/or MRI).

MRI: optimal for demonstrating brain stem impingement, poor for showing bone. Cervicomedullary angle: the angle between a line drawn through the long axis of the medulla on a sagittal MRI

Table 75.3 Symptoms and signs of BI (45 patients with RA[2])

| Finding | % |
|---|---|
| headache | 100% |
| progressive difficulty ambulating | 80% |
| hyperreflexia + Babinski | 80% |
| limb paresthesias | 71% |
| neurogenic bladder | 31% |
| cranial nerve dysfunction | 22% |
| • trigeminal nerve anesthesia | 20% |
| • glossopharyngeal | |
| • vagus | |
| • hypoglossal | |
| miscellaneous findings | |
| • internuclear ophthalmoplegia | |
| • vertigo | |
| • diplopia | |
| • downbeat nystagmus | |
| • sleep apnea | |
| • spastic quadriparesis | |

and a line drawn through the cervical spinal cord. The normal CMA is 135–170°. A CMA < 135° correlates with signs of cervicomedullary compression, myelopathy or C2 radiculopathy.[17]

CT: primarily done to assess bony anatomy (erosion, fractures…).

CTA should be performed when surgery is contemplated, to show detail of VA anatomy.

Myelography (water soluble) with CT: also good for delineating bony pathology.

#### Treatment

See also Craniocervical junction and upper cervical spine abnormalities (p.1151).

##### Cervical traction

May attempt with Gardner-Wells tongs. Begin with ≈ 7 lbs, and slowly increase up to 15 lbs. Some may require several weeks of traction to reduce.

##### Surgery

Reducible cases: posterior occipitocervical fusion ± C1 decompressive laminectomy.

Irreducible cases: requires transoral resection of odontoid. May perform before posterior fusion (but then must be kept in traction while waiting for posterior fusion).

### 75.1.7 Subaxial subluxation in rheumatoid arthritis

The direct effects of RA on the subaxial spine involves the facet joints posteriorly. Degenerative disc disease, which is generally a late manifestation in RA, is not the result of synovitis.[18] Involvement is most common at C2–3 and C3–4.

## 75.2 Down syndrome

### 75.2.1 General information

Down syndrome is associated with ligamentous laxity of the spine. This has implications whenever a fusion is contemplated, as adjacent segment failure with kyphosis is very common. Ligamentous laxity may also result in atlantoaxial subluxation (AAS).

### 75.2.2 Atlantoaxial subluxation (AAS) in Down syndrome

#### General information

Not all cases of AAS are unstable (an unstable spine, by definition, needs treatment).

Incidence of AAS in Down syndrome (DS) is 20%[19] but only 1–2% of DS patients have symptomatic AAS.[20] AAS in DS appears to be due to laxity of the transverse atlantal ligament (TAL). This laxity may decrease with age as the TAL stiffens.



#### Management

Controversial. There have been position statements[21] and rebuttals.[20,22]

Recommendations (modified[23]); ADI = atlantodental interval (p.213), PADI = posterior atlantodental interval (p.213):

1. children who have been screened and do not have AAS: no further screening after age 10 years (since AAS does not develop later; the cutoff age is controversial)
2. os odontoideum: surgical fusion
3. symptomatic AAS
   a) symptoms may include: gait difficulties, neck pain, limited neck motion, torticollis, clumsiness, sensory deficits, and other symptoms of myelopathy
   b) for ADI > 4.5 mm or PADI < 14 mm or spinal cord damage on cervical MRI: surgical fusion
4. asymptomatic AAS seen on lateral C-spine x-ray:
   a) for ADI ≤ 4.5 mm and PADI ≥ 14 mm: no need for further testing
   b) for ADI > 4.5 mm or PADI < 14 mm: cervical MRI
      - if the MRI shows spinal cord damage: surgical fusion
      - if MRI shows no spinal cord damage: surgical fusion is optional. If fusion is not done, prohibit high-risk activities and restudy in 1 year

## 75.3 Morbid obesity

Morbid obesity, defined as a body mass index (BMI)>40. Approximately doubles risks (13.6% vs. 6.9%) of complications of all types (cardiac, renal, pulmonary, wound complications...)[24] with spine surgery. Mortality is tripled (but is still low, 0.41 in morbid obese vs. 0.13).[24] Hospital costs and length of stay is also longer.

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# 76 Special Conditions Affecting the Spinal Cord

## 76.1 Spinal vascular malformations

### 76.1.1 General information

Often also referred to by the term spinal AVMs which technically refers to a subset of spinal vascular malformations (SVMs). Incidence of SVM is about 4% of primary intraspinal masses. 80% occur between age 20 and 60 years.[1 (p 1850–3)]

### 76.1.2 Classification

For a review of the history of classification systems, see the excellent review by Black[2]).

There are three current era classification systems.

#### The "American/English/French Connection" classification

References for classification: include[2,3,4,5,6,7,8,9,10]

1. Type I: dural AVM AKA AV-fistula (AVF). The most common type (80%) of SVM in the adult.[11] Fed by radicular artery which forms an AV shunt (fistula) at the dural root sleeve (located in the intervertebral foramen),[8] drains into an engorged spinal vein on posterior cord. Usually in lumbar or lower thoracic spine. Slow flow. High pressure in draining vein may cause venous congestion of the cord. Cord involvement may be distant to the fistula. Symptoms: LBP and progressive myeloradiculopathy or cauda equina syndrome (due to venous congestion) with urinary retention usually in middle-aged patients, 90%males. Up to 35%have pain. 15–20%are associated with other AVMs (cutaneous or other). Rarely bleed
   a) Type IA: single arterial feeder
   b) Type IB: two or more arterial feeders
2. intradural AVMs (high flow): 75%present with acute onset of symptoms, usually from hemorrhage (SAH or intramedullary)
   a) Type II: AKA spinal glomus AVM. Intramedullary. True AVM of the spinal cord. 15–20%of all SVMs. Compact nidus fed by medullary arteries with the AV shunt contained at least partially within the spinal cord or pia. May be associated with feeding artery aneurysms. Worse prognosis than dural AVM.[8] Fed by 1, or at most 2–3 feeders 80%of the time
   b) Type III: AKA juvenile spinal AVM. Essentially an enlarged glomus AVM which occupies the entire cross-section of the cord and invades the vertebral body which may cause scoliosis
   c) Type IV[7]: intradural perimedullary AVM (also called arteriovenous fistulae (AVF)). Direct fistula between artery supplying spinal cord (usually anterior spinal artery, often artery of Adamkiewicz) and draining veins. Typically occur in younger patients than Type I, and may present catastrophically with hemorrhage into the subarachnoid space.[12] □ Table 76.1 shows the 3 subtypes.[9]
3. miscellaneous spinal vascular lesions:
   a) spinal cord cavernomas
   b) spinal cord venous angiomas: extremely rare. Difficult to visualize angiographically
   c) vertebral body hemangiomas (p.794)

#### Hôpital Bicêtre classification

See reference.[13]

Table 76.1 Merland's subclassification of Type IV (perimedullary) AV fistulas[a]

| Subtype | Arterial supply | AVF | Venous drainage |
|---|---|---|---|
| I | single (thin ASA) | single, small | slowly ascending perimedullary venous system |
| II | multiple (dilated ASA & PSA) | multiple, medium | |
| III | | single, giant | giant venous ectasia, rapid metameric venous drainage |

[a]AVF=arteriovenous fistula; ASA=anterior spinal artery; PSA=posterior spinal artery

1. AVMs
2. fistulae: micro- or macrofistulae
3. genetic classification of spinal cord

AV shunts
a) genetic hereditary lesions: macrofistulae and hereditary hemorrhagic telangiectasias
b) genetic nonhereditary lesions: multiple lesions with metameric or myelomeric associations
c) single lesions: incomplete associations of categories a or b

### Spetzler et al. classification

See reference.[14]

This system reincorporated vascular spinal neoplasms.

1. Neoplastic vascular lesions
   a) hemangioblastoma
   b) cavernous malformation
2. spinal aneurysms (rare)
3. arteriovenous lesions
   a) AVFs
      - extradural
      - intradural: dorsal or ventral
   b) AVMs
      - extradural-intradural
      - intradural
      - intramedullary
      - intramedullary-extramedullary
      - conus medullaris

## 76.1.3 Presentation

85%present as progressive neuro deficit (back pain associated with progressive sensory loss and LE weakness over months to years). Yet, SVMs account for<5% of lesions presenting as spinal cord "tumors." 10–20%of SVMs present as sudden onset of myelopathy usually in patients<30 yrs age,[15,16] secondary to hemorrhage (causing SAH, hematomyelia, epidural hematoma, or watershed infarction). Coup de poignard of Michon=sudden excruciating back pain with SAH (clinical evidence of SVM).

Foix-Alajouanine syndrome (subacute necrotic myelopathy): acute or subacute neurologic deterioration in a patient with a SVM without evidence of hemorrhage. Presents as spastic → flaccid paraplegia, with ascending sensory level and loss of sphincter control. Initially thought to be due to spontaneous thrombosis of the AVM causing subacute necrotizing myelopathy[17] which would be irreversible. However, more recent evidence suggests that the myelopathy may be due to venous hypertension with secondary ischemia, and there may be improvement with treatment.[18]

 Clinical. Auscultation over spine reveals a bruit in 2–3%of cases. Cutaneous angioma over back is present in 3–25%; valsalva maneuver may enhance the redness of the angioma.[16]



## 76.1.4 Evaluation

Spinal angiography: necessary for treatment planning. Best performed at centers that do this study regularly. For Type I dural AVMs, angiography must encompass all dural feeders of the neuraxis, which includes:

1. ICAs: because of the artery of Bernasconi & Cassinari (p.79)
2. every radicular artery including the artery of Adamkiewicz (p.87)
3. internal iliac arteries: for sacral feeders

MRI: detects some SVMs with greater sensitivity and safety than angiography,[19] but is inadequate for treatment planning. 82%show extramedullary flow voids. Variable degree of cord enhancement (from venous congestion or venous infarction). Negative MRI does not rule out diagnosis.

Myelography: classically shows serpiginous intradural filling defects. Generally superseded by MRI. If done, patient should be imaged prone and supine (to avoid missing a dorsal AVM)  Risk of bleeding from puncture of a dilated artery/vein with myelography needle.

### 76.1.5 Treatment

Type I (dural AVMs): usually require treatment. Usually amenable to endovascular techniques using glue, in which case the proximal vein must be taken as well. If you don't completely eliminate a dural fistula (spinal or intracranial) it will come back!

Type II (spinal glomus AVMs): may be amenable to interventional neuroradiologic procedures including embolization,[20] especially type IIA (single feeder). However recurrence may be higher with endovascular treatment than surgery, and surgery is often preferred for Type IIB (≥2 feeders). Surgical strategy: similar to intracranial AVMs except that the parenchyma cannot be retracted, bleeding is rarely life threatening, and arteries of passage must be preserved to avoid devastating deficits. Intraoperative ICG angio is often helpful. The nidus is compact, and the hemosiderin ring around the nidus on MRI often represents a plane that can be exploited.

Type III (juvenile spinal AVMS): the natural history is probably better than the prognosis with any type of treatment.

Type IV (perimedullary fistulae): suggested management[10] is shown in □ Table 76.2.

## 76.2 Spinal meningeal cysts

### 76.2.1 General information

Spinal meningeal cysts (SMC): diverticula of the meningeal sac, nerve root sheath or arachnoid. May have familial tendency.

Terminology in literature is confusing. One classification system is shown in □ Table 76.3. Previously AKA Tarlov's perineural cysts, spinal arachnoid cysts, and extradural diverticula, pouches or cysts. Only congenital lesions are considered here.

1. Type I SMCs above the sacrum usually have a pedicle adjacent to entrance of dorsal nerve root
2. Type II SMCs: formerly called Tarlov's cysts and were differentiated from nerve root diverticula because the former were defined as communicating with subarachnoid space, and the latter not. However, intrathecal contrast CT (ICCT) shows both communicate. Often multiple, occur on dorsal roots anywhere, but are most prominent and symptomatic in sacrum
3. Type III SMCs: may also be multiple and asymptomatic. More common along posterior subarachnoid space. Attributed to proliferation of arachnoid trabeculae

Table 76.2 Suggested management for Type IV arteriovenous fistulae[10]

| Subtype | Diagnosis | Embolization | Surgery |
|---|---|---|---|
| Subtype I | difficult; ? reliability of MRI[a]; tomomyelography; angiotomomyelography | difficult | easy on filum terminale; difficult on conus medullaris |
| Subtype II | easy: MRI or myelography | incomplete occlusion | on posterolateral AVFs |
| Subtype III | | effective | difficult, dangerous |

[a]due to inaccuracy, do not delay angiogram to get MRA, etc.

Table 76.3 Types of spinal meningeal cysts[21]

| Type | Description |
|---|---|
| Type I | extradural meningeal cysts without spinal nerve root fibers |
| IA | "extradural meningeal/arachnoid cyst" |
| IB | (occult) "sacral meningocele" |
| Type II | extradural meningeal cysts with spinal nerve root fibers ("Tarlov's perineural cyst", "spinal nerve root diverticulum") |
| Type III | spinal intradural meningeal cysts ("intradural arachnoid cyst") |

### 76.2.2 Presentation

May be asymptomatic (i.e. incidental finding). May cause radiculopathy by pressure on adjacent nerve root (may or may not cause symptoms of nerve root from which it actually arises). Symptom complex depends on size of SMC, and proximity to spinal cord and nerve roots.

1. Type I SMCs: in thoracic and cervical region, may present with acute myelopathy (spasticity and sensory level); lumbar region →LBP and radiculopathy; sacral region →sphincter disturbance
2. Type II SMCs: often asymptomatic, but sacral lesions may →sciatica and/or sphincter disturbance
3. Type III SMCs: may also be multiple and asymptomatic; more common along posterior subarachnoid space

### 76.2.3 Evaluation

MRI to identify the mass, then water-soluble ICCT scan to evaluate communication of cyst with subarachnoid space.

1. Type II SMCs: all 18 cases had bony erosion (demonstrated by canal widening, pedicle erosion, foraminal enlargement, or vertebral body scalloping)
2. Type III SMCs: may also cause bony erosion; appear on myelogram as intradural defect, may not appear on ICCT if they communicate with subarachnoid space which causes them to blend with adjacent subarachnoid space

### 76.2.4 Treatment

1. Type I SMCs: close ostium between cyst and subarachnoid space. Above sacrum, can usually be dissected from dura; occasionally fibrous adhesions prevent this
2. Type II SMCs: no pedicle, thus either partially resect and oversew cyst wall, or excise cyst and involved nerve root. Simple aspiration is not recommended
3. Type III SMCs: excise completely unless dense fibrous adhesions prevent this, in which case marsupialize cyst. Tend to recur if incompletely excised

## 76.3 Juxtafacet cysts of the lumbar spine

### 76.3.1 General information

The term juxtafacet cyst (JFC) was originated by Kao et al.[22] in 1974 and includes both synovial cysts (those having a synovial lining membrane) and ganglion cysts (those lacking synovial lining) adjacent to a spinal facet joint or arising from the ligamentum flavum. Distinction between these two types of cysts may be difficult without histology (see below) and is clinically unimportant.[23]

JFC occur primarily in the lumbar spine (although cysts in the cervical[24,25,26] and thoracic[27] spine have been described). They were first reported in 1880 by von Gruker during an autopsy,[28] and were first diagnosed clinically in 1968.[29] The etiology is unknown (possibilities include: synovial fluid extrusion from the joint capsule, latent growth of a developmental rest, myxoid degeneration and cyst formation in collagenous connective tissue…), increased motion seems to have a role in many cysts, and the role of trauma in the pathogenesis is debated[25,30] but probably plays a role in a small number (≈ 14%).[31] JFC are relatively rare, only 3 cases were identified in a series of 1,500 spinal CT exams,[32] but the frequency of diagnosis may be on the rise due to the widespread use of MRI and an increasing awareness of the condition.



### 76.3.2 Pathology

Cyst walls are composed of fibrous connective tissue of varying thickness and cellularity. There is usually no signs of infection or inflammation. There may be a synovial lining[33] (synovial cyst) or it may be absent[34] (ganglion cyst). The distinction between the two may be difficult,[23] possibly owing in part to the fact that fibroblasts in ganglion cysts may form an incomplete synovial-like lining.[35] Proliferation of small venules is seen in the connective tissue. Hemosiderin staining may be present, and may or may not be associated with a history of trauma.[31]

### 76.3.3 Clinical

The average age was 63 years in one series[31] and 58 years in a review of 54 cases in the literature[33] (range: 33–87) with a slight female preponderance in both series. Most occur in patients with severe spondylosis and facet joint degeneration,[34] 25% had degenerative spondylolisthesis.[31] L4–5 is the

most common level.[31,36] They may be bilateral. Pain is the most common symptom, and is usually radicular. Some JFC may contribute to canal stenosis and can produce neurogenic claudication (p.1100) [37] or on occasion a cauda equina syndrome. Symptoms may be more intermittent in nature than with firm compressive lesions, such as HLD. A sudden exacerbation in pain may be due to hemorrhage within the cyst. Some JFC may be asymptomatic.[38]

### 76.3.4 Differential diagnosis

Also see Differential diagnosis, Sciatica (p.1410). Differentiating JFC from other masses relies largely on the appearance and location. Other distinguishing features include:

1. neurofibroma: unlikely to be calcified
2. free fragment of HLD: not cystic in appearance
3. epidural or nerve root metastases: not cystic
4. dural subarachnoid root sleeve dilatation (p.1142)
5. arachnoid cyst (from arachnoid herniation through a dural defect): not associated with facet joint, margins thinner than JFC[39]
6. perineurial cysts (Tarlov's cyst): arise in space between perineurium and endoneurium, usually on sacral roots,[40] occasionally show delayed filling on myelography. Usually associated with remodelling of adjacent bone

### 76.3.5 Evaluation

Identifying a JFC pre-op helps the surgeon, as the approach differs slightly from that for HLD, and the cyst might otherwise be missed or unknowingly deflated and unnecessary time wasted afterwards trying to find a compressive lesion. Or, the unwitting surgeon may misinterpret the cyst as a "transdural disc extrusion" and needlessly open the dura. Pre-op diagnoses were incorrect in 30%of operated cases of JFC.[31]

Myelography: posterolateral filling defect (whereas most discs are situated anteriorly, an occasional fragment may migrate posterolaterally, whereas a JFC will always be posterolateral), often with a round extradural appearance.

CT scan: shows a low density epidural cystic lesion typically with a posterolateral juxtaarticular location. Some have calcified rim,[38] and some may have gas within.[41] Erosion of bony lamina is occasionally seen.[36,42]

MRI: variable findings (may be due to differing composition of cyst fluid: serous vs. proteinaceous[43]). Unenhanced signal characteristics of non-hemorrhagic JFC are very similar to CSF. Hemorrhagic JFC are hyperintense. MRI usually misses bony erosion.

### 76.3.6 Treatment

Optimal treatment is not known. There is one case report of a cyst that resolved spontaneously.[32] If symptoms persist with conservative treatment, some promote cyst aspiration or facet injection with steroids,[44] while most advocate surgical excision of the cyst.

Surgical treatment considerations: The cyst may be adherent to the dura. The cyst may also collapse during the surgical approach and may be missed. A JFC may serve as a marker for possible instability and should prompt an evaluation for the same. Some argue for performing a fusion since JFC may arise from instability, however, it appears that fusion is not required for a good result in many cases.[44] Therefore it is suggested that consideration for fusion be made on the basis of any instability and not merely on the basis of the presence of a JFC.

Minimally invasive spine surgery (MISS) has also been used for removal,[45] long-term follow-up is lacking. A 15 mm entry incision is made 1.5 cm lateral to midline.

Following surgical treatment, symptomatic JFCs may recur or may develop on the contralateral side.[31]



## 76.4 Syringomyelia

### 76.4.1 General information

**Key concepts**

- AKA syrinx. Cystic cavitation of the spinal cord
- 70%are associated with Chiari I malformation, 10%with basilar invagination. May also be posttraumatic or associated with tumor, infection…

- symptoms: progressive neurologic deterioration over months to years, usually affecting UE first
- diameter > 5 mm + associated edema predict a more rapid deterioration
- preferred treatment is directed at correcting the causative pathophysiology

AKA syrinx. Cystic cavitation of the spinal cord. Other terms not precisely defined include: hydrosyringomyelia, communicating or noncommunicating syringomyelia.

Syringobulbia: Rostral extension into brainstem (usually medulla). May present with (bilateral) peri-oral tingling and numbness, due to compression of the spinal trigeminal tracts as the fibers decussate.

## 76.4.2 Etiologies

### Primary syringomyelia

This term is used differently by different authors.[46] Herein, refers to syrinx in the absence of identifiable cause

### Secondary syringomyelia

Most cases are thought to be secondary to partial obstruction of the spinal subarachnoid space.[46] Unanswered question: Why then do patients with varying degrees of degenerative cervical spinal stenosis generally not get syringomyelia? Etiologies include:

1. Chiari I malformation: the most common cause ofsyrinx (p.277)
2. postinflammatory
   a) postinfectious
      - granulomatous meningitides (TB and fungal)
      - postoperative meningitis, especially after intradural procedure
   b) chemical or other sterile inflammations
      - rarely after SAH
      - after myelography: especially with older oil-based agents no longer in use
3. posttraumatic: also see below
   a) with severe posttraumatic kyphotic deformity: e.g. with retropulsed bone, scarring...
   b) arachnoid scarring without recognized trauma
   c) severe injury to spinal cord and/or its coverings. Blood may be a contributing factor

 the older concept of syrinx developing as a coalescence of foci of traumatic hematomyelia has not been borne out

1. postsurgical: has been identified many years after uncomplicated intradural neoplasm removal (e.g. neurofibromas)
2. basilar arachnoiditis:
   a) idiopathic
   b) postinfectious: see above
3. basilar impression, with constriction of the foramen magnum (p.217)
4. associated with spinal tumors: this is distinct from a tumor cyst
5. associated with disc protrusion:
6. cerebellar ectopia
7. Dandy Walker syndrome

## 76.4.3 Epidemiology

See reference.[47]

Prevalence of non-posttraumatic syringomyelia: 8.4 cases/100,000 population. Usually presents between ages 20–50.

Associated clinical syndromes are shown in  Table 76.4.

## 76.4.4 Pathophysiology

Major theories of formation of the cyst:

1. hydrodynamic ("water-hammer") theory of Gardner: systolic pulsations are transmitted with each heartbeat from the intracranial cavity to the central canal. Has been essentially disproven using MRI[48]

| Table 76.4 Conditions associated with syringomyelia | |
|---|---|
| Condition | %[a] |
| Chiari type 1 malformation | 70 |
| basilar invagination | 10 |
| intramedullary spinal cord tumors | 4 |
| [a]percent of cases of syringomyelia | |

2. Williams' ("craniospinal dissociation") theory: maneuvers that raise CSF pressure (valsalva, coughing...) cause "hydrodissection" through the spinal cord tissue. May be more common in noncommunicating syringomyelia
3. Heiss-Oldfield theory: occlusion at the foramen magnum causes CSF pulsations during cardiac systole to be transmitted through the Virchow-Robin spaces which increases the extracellular fluid which coalesce to form a syrinx[47] (i.e. through the cord parenchyma)

## 76.4.5 Clinical

Presentation: highly variable. Usually progresses over months to years, with a more rapid deterioration early that gradually slows.[47] Initially, pain, weakness, atrophy and loss of pain & temperature sensation in the upper extremities (with cervical syrinx) is common. Myelopathy that progresses slowly over years ensues.

## 76.4.6 Characteristic syndrome

(nonspecific for intramedullary spinal cord pathology):

1. sensory loss (similar to central cord syndrome) with a suspended ("cape") dissociated sensory loss (loss of pain and temperature sensation with preserved touch and joint position sense → painless ulcerations from unperceived injuries and/or burns)
2. pain: commonly cervical and occipital. Dysesthetic pain often occurs in the distribution of the sensory loss[47]
3. weakness: lower motor neuron weakness of the hand and arm
4. painless (neurogenic) arthropathies (Charcot's joints) especially in the shoulder & neck due to loss of pain & temperature sensation: seen in <5%

## 76.4.7 Evaluation

Prior to the CT/MRI era, diagnosis relied on myelography or on autopsy.

MRI: defines anatomy in sagittal as well as axial plane. Test of choice. Cervical & thoracic spine and brain MRI (without & with contrast, to include craniocervical junction) should be obtained. Syringomyelic cavities may be complex, with noncommunicating channels (more common with posttraumatic syrinx).

CT: low attenuation area within cord seen on either plain CT or myelogram/CT (with water soluble contrast).

Myelogram: rarely used alone (usually performed in conjunction with CT). When used alone: often normal (false negative), some →complete block at level of syrinx; iodine contrast studies may show fusiform widening of spinal cord, whereas air contrast studies may show collapse of the cord.[49] Dye may slowly leach into the cyst.

EMG: no characteristic findings, but may be useful to R/O other conditions that may be responsible for symptoms (e.g. peripheral neuropathy causing paresthesias).

## 76.4.8 Distinguishing from similar entities

1. tumor cyst:
   a) especially with intramedullary spinal cord gliomas. Tumors may secrete fluid, or may cause microcysts that eventually coalesce. Most (but not all) intramedullary tumors will enhance with IV contrast on MRI
   b) tumor cyst fluid is usually highly proteinaceous, syrinx fluid usually has the same MRI characteristics as CSF (NB: true syrinx can occur with tumor)

2. central spinal canal:
   a) residual central spinal canal: the central canal is present within the spinal cord at birth and normally gradually involutes with age.[50] Persistence of the canal is a normal variant. Characteristic imaging features:
      - linear or fusiform on sagittal MRI
      - ≤2–4 mm in maximal width
      - may be singular, or there may be several discontinuous regions in the rostral-caudal direction
      - perfectly round in cross-section and centrally located on axial MRI
      - if IV contrast is given, there should be no enhancement
   b) simple dilatation of the central canal with ependymal cell lining has sometimes been called hydromyelia, but this usage is ambiguous

## 76.4.9 Management

### General information

For an incidentally discovered syrinx (i.e. asymptomatic and no neurologic deficit) with no identified etiology, if the size remains stable over 2–3 years of observation, F/U studies at 2–3 year intervals can be done if there are no changes in symptoms.

### Surgical treatment

Intervention is considered for symptomatic lesions (not all are symptomatic). If an underlying cause cannot be determined, it may be very difficult to treat a very small syrinx directly (however, these are unlikely to be causing reversible symptoms).

Options include:

1. current philosophy is to treat the underlying pathophysiology (and to use syrinx draining procedures as second choice when this is not feasible)
   a) posterior decompression: procedure of choice when posterior anomalies (e.g. Chiari malformation) are present
   b) decompression if a different site of compression is identified
2. shunts:
   a) disadvantages:
      - complication rate: 16%
      - clinical stabilization rate: 54%at 10 yrs
      - may produce traction on spinal cord with potential for further injury
      - prone to obstruction: 50%at 4 years
      - does not correct underlying pathophysiology and so syrinx may recur
   b) indications: cases of diffuse arachnoiditis (e.g. following tuberculous or chemical meningitis) where the obstruction extends over many levels, and syrinx diameter >3–4 mm
   c) K or T tube drainage. Choice of distal sites includes:
      - peritoneum[51] (difficult in cervical region): e.g. Edwards-Barbaro syringoperitoneal shunt (distributed by Integra)
      - pleural cavity
      - subarachnoid space (e.g. Heyer-Schulte-Pudenz system): requires normal CSF flow in subarachnoid space, therefore cannot use in arachnoiditis
3. percutaneous aspiration of the cyst[52] (may be used repeatedly)
4. □ no longer recommended:
   a) plugging the obex with muscle, teflon or other material
   b) opening the subarachnoid space & removing inferior tonsils
   c) syringostomy: usually fails to remain patent, therefore using a stent or a shunt (syringosubarachnoid or syringoperitoneal) is recommended

Technical considerations:

1. intraoperative ultrasound is often helpful for:
   a) localizing the cyst
   b) assessing for septations (to avoid shunting only part of cyst)
2. if Chiari malformation is not present, consider syringosubarachnoid shunt as the initial procedure. If this fails, then syringoperitoneal shunt may be inserted
3. Rhoton suggests performing the myelotomy in the dorsal root entry zone (DREZ), between the lateral and posterior columns (instead of the midline as with a tumor) because this is consistently

the thinnest part and there is usually already an upper extremity proprioceptive deficit from the syrinx.[53 (p 1317)] There is ≈ 10%incidence of posterior column dysfunction with shunting

4. with syringosubarachnoid shunts, be sure the distal shunt tip is subarachnoid (and not just subdural) or else it will not function
5. with syringopleural shunt, the pleural opening can be made posteriorly, adjacent to one of the ribs as described for ventriculopleural shunt (p.1515)
6. for peritoneal terminus of shunt: the proximal shunt may be placed in the syrinx with the patient in the prone position, and the peritoneal end of the catheter may be tunneled to an intermediate position in the flank in the midaxillary line where it can be coiled into a small subcutaneous pouch which can be temporarily closed with staples and dressed with Tegaderm. After the spinal incision is closed, the patient can be undraped and rotated to the supine position, the Tegaderm is removed, and the abdomen and the flank incision are prepped, the staples are removed and the catheter is retrieved from the sub-Q pocket and tunneled to the peritoneal opening that is made at this time. A small bump (rolled sheet…) to elevate the flank may be employed in both parts of the operation to facilitate access to the pouch

### 76.4.10 Outcome

Assessing treatment results is difficult due to rarity of the condition, variability of natural history (which may arrest spontaneously), and too short follow-up.[54] Enthusiasm for direct treatment (shunts, fenestration…) is low among neurosurgeons because of the perceived poor response and risk of iatrogenic neurologic worsening. However, this may remain as the only option for a deteriorating patient and positive outcomes do occur.[55]

## 76.5 Posttraumatic syringomyelia

### 76.5.1 General information

Posttraumatic syringomyelia (PTSx) may follow significant spinal trauma (with or without clinical spinal cord injury). Includes penetrating injury or non-penetrating “violent” trauma to the spinal cord (injuries such as post-spinal anesthesia or following thoracic disc herniation are not included).

### 76.5.2 Epidemiology

Often a late presentation following spinal cord injury, therefore incidence is higher in series with longer follow-up. Incidence increasing with increasing survival following spinal cord injury and with increasing use of MRI. Range: ≈ 0.3–3%of cord injured patients (see □ Table 76.5).

In a large number of patients followed via multicenter cooperative data bank, there were fewer cases of syrinx following cervical injuries than following thoracic injuries[57] (may be artifactual since patients with lower lesions may be more aware of ascending levels).

Latency following spinal cord injury:

1. latency to symptoms: 3 mos to 34 yrs (mean 9 yrs) (earlier in complete cord lesions than incomplete: mean 7.5 vs. 9.9 yrs)
2. latency to diagnosis: up to 12 yrs (mean 2.8 yrs) after onset of new symptoms



Table 76.5 Incidence of posttraumatic syringomyelia

| Type of injury | No./risk[a] | Incidence |
|---|---|---|
| all spinal cord injury patients | 30/951 | 3.2% |
| complete quadriplegics | 14/177 | 7.9% |
| incomplete quadriplegics | 4/181 | 4.5% |
| complete paraplegics | 4/282 | 1.7% |
| incomplete paraplegics | 4/181 | 2.2% |

[a]number occurring over number at risk in 951 patients followed for 11 years[56]

Table 76.6 Presentation (in 30 SCI patients with syrinx[56])

| Symptom | Initial | At time of diagnosis |
|---|---|---|
| pain[a] | 57% | 70% |
| numbness | 27% | 40% |
| increased motor deficit | 23% | 40% |
| increased spasticity | 10% | 23% |
| increased sweating (hyperhidrosis) | 3% | 13% |
| autonomic dysreflexia | 3% | 3% |
| no symptoms | 7% | 7% |
| Signs | Frequency | |
| ascending sensory level | 93% | |
| depressed tendon reflexes | 77% | |
| increased motor deficits | 40% | |

[a]pain is often quite severe, and unrelieved with analgesics[59]

## 76.5.3 Clinical

The presentation of patients with PTSx is shown in Table 76.6. The late appearance of upper extremity symptoms in a paraplegic patient should raise a high index of suspicion of posttraumatic syringomyelia.[58]

Hyperhidrosis may be the only feature of descending syringomyelia in patients with complete cord lesions.[60] For the differential diagnosis, see Delayed deterioration following spinal cord injuries (p.1019).

## 76.5.4 Evaluation

One end of the cavity is often found at a site of spinal column fracture or abnormal angulation.

## 76.5.5 Management

### General information

Many authors advocate early surgical drainage of cyst as a means of reducing increased delayed deficit.[61] Some authors feel that aside from disturbing sensory symptoms, that motor loss was infrequent and therefore conservative management is indicated in most cases.[62]



### Medical

Managed non-surgically: 31%remained stable, 68%progressed over yrs (longer F/U in latter).

### Surgical

There is probably no benefit in operating on a patient with a small syrinx.[56]

Surgical options

Same as in Communicating syringomyelia, with the following differences:

1. cord transection (cordectomy)[63]: an option in complete injuries only
2. plugging the obex is probably not indicated (controversial in congenital syrinx)

### Outcome

In 9 PTSx patients treated with syringosubarachnoid shunt[56]: pain relieved in all 9 (1 only slightly), motor recovery in 5/8, improved tendon reflex in 1/10. Some post-op complications in 9 patients included: 1 incomplete lesion became complete, 1 sensorimotor deterioration, transient pain in 3.

Most results are good for radicular symptoms, with dubious efficacy for autonomic symptoms or spasticity.

## 76.6 Spinal cord herniation (idiopathic)

### 76.6.1 General information

Rare. Spinal cord herniates though a defect in the dura usually located anteriorly or anterolaterally between T2–8.[64] Bone erosion anterior to the dural defect may occasionally be seen. Frequently associated with a calcified disc fragment which theoretically may have gradually eroded through the dura.

### 76.6.2 Differential diagnosis

Main DDx is with dorsal arachnoid cyst (p.1407). Both result in increased subarachnoid space posterior to the cord, and a ventral kinking of the spinal cord. Contiguous CSF pulsation artifact on MRI can be seen with cord herniation, whereas an arachnoid cyst tends to interrupt this.

### 76.6.3 Presentation

Commonly presents as an incomplete Brown-Séquard syndrome (with relative sparing of posterior columns). Symptoms may be due to distortion of the spinal cord, but vascular injury may also play a role.

### 76.6.4 Surgery

Requires a lateral or anterolateral approach to minimize spinal cord manipulation (p.1061). The dural defect is widened which usually results in reduction of the spinal cord herniation. A sling of dural substitute can then be slid anterior to the cord to prevent reherniation.

## 76.7 Spinal epidural lipomatosis (SEL)

### 76.7.1 General information

Hypertrophy of epidural fat. Most commonly seen with prolonged exogenous steroid therapy (in 75% of cases[65]) usually moderate to high dosage for years,[66] but may also be associated with: Cushing's disease, Cushing's syndrome, obesity,[67] hypothyroidism or may be idiopathic.[68] Male: female = 3:1.[66]

Back pain usually precedes all other symptoms. Progressive LE weakness and sensory changes are common. Sphincter disturbance occurs but is rare. SEL is most common in the thoracic spine (≈ 60% of cases), the rest are in the lumbar spine (no cases reported in cervical spine).

It is often very difficult to differentiate the patient with increased epidural fat (even to the point that CSF may be obliterated at the levels of involvement) that is not causing symptoms, from those cases where the exuberant fat is responsible for the findings.



### 76.7.2 Evaluation

CT: density of adipose tissue is extremely low (-80 to -120 Hounsfield units)[69] which distinguishes SEL from most other lesions (except lipoma).

MRI: signal follows fat (high signal on T1WI, intermediate on T2WI). Suggested diagnostic criteria: epidural adipose should be > 7 mm thick to be considered SEL.[67,70]

### 76.7.3 Treatment

In those patients who can be weaned off steroids and lose weight, surgery may be avoided in some cases.[71] If SEL is related to obesity, weight loss alone can be successful.

Surgery is indicated for symptomatic patients in whom the above interventions are unsuccessful or not feasible. An effort to normalize cortisol levels in those with endogenous hypercortisolism (Cushing's disease...) should be made before laminectomy is performed. Due to potential complications and slow growth of the tissue, the decision to operate should be made with caution.

Surgery usually consists of laminectomy with removal of adipose tissue. Occasionally repeat surgery is needed for reaccumulation of adipose tissue.

### 76.7.4 Outcome

Surgery usually results in significant improvement.[70] Idiopathic cases may fare better than those due to steroid excess. Cauda equina compression responds better than thoracic myelopathy.

Complications rates may be higher than expected in part due to medical comorbidities. Fessler et al.[72] reported 22%1-year mortality.

## 76.8 Craniocervical junction and upper cervical spine abnormalities

### 76.8.1 Associated conditions

Also see Axis (C2) vertebra lesions (p.1391).

Abnormalities in this region are seen in a number of conditions including:

1. rheumatoid arthritis (p.1134)
2. traumatic & post-traumatic: including fractures of odontoid, occipital condyles…
3. ankylosing spondylitis (p.1123): may result in fusion of the entire spine which spares the occipitoatlantal and/or atlantoaxial joints which can lead to instability there
4. congenital conditions:
   a) Chiari malformations (p.277)
   b) Klippel-Feil syndrome (p.271)
   c) Down syndrome
   d) atlantoaxial dislocation (AAD)
   e) occipitalization of the atlas: seen in 40%of congenital AAD[73]
   f) Morquio syndrome (a mucopolysaccharidosis): atlantoaxial subluxation occurs due to hypoplasia of the odontoid process and joint laxity
5. neoplasms: metastatic (p.815) or primary
6. infection
7. following surgical procedures of the skull base or cervical spine: e.g. transoral resection of the odontoid

### 76.8.2 Types of abnormalities

Abnormalities include:

1. basilar impression/invagination: as with Paget's disease
2. atlanto-occipital dislocation
3. atlantoaxial dislocation
4. occipitalization of the atlas, or thin or deficient posterior arch of atlas[74]

### 76.8.3 Treatment



Fractures of the occipital condyles, atlas or axis are usually adequately treated with external immobilization; also see Occipital condyle fractures (p.966). Because traumatic occipitocervical dislocations are usually fatal, optimal treatment is not well defined. Occipitalization of the atlas may be treated by creating an "artificial atlas" from the base of the occiput and wiring to that.[74]

Indications and techniques are outlined in Atlantoaxial fusion (C1–2 arthrodesis) (p.1479).

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