Chapter 5
Cervical Radiculopathy and Myelopathy

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Cervical Radiculopathy

Definition and Epidemiology

Cervical radiculopathy represents dysfunction of one or more cervical nerve roots that typically presents with radiating pain in the upper extremity and varying degrees of sensory loss, motor weakness, and reflex changes. Population-based studies have shown an annual incidence of 107/100,000 men and 64/100,000 women, with a peak incidence in the sixth decade of life. About 15% of patients report an antecedent episode of physical exertion or trauma that precedes symptom onset. Identified risk factors for cervical radiculopathy include white race, smoking history, and prior lumbar radiculopathy. The majority of the cases stem from compression of nerve roots in the lower cervical spine, most commonly at C6–7, likely due to greater segmental mobility and smaller neuroforamina in this region.

Clinical Presentation

Cervical radiculopathy is usually the result of neuroforaminal stenosis due to a herniated disc, overgrowth of the uncovertebral joints anteriorly, or facet joint hypertrophy posteriorly. This stenosis can manifest with pain, sensory disturbances,

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diminished reflexes, and muscle weakness that correspond to the affected nerve root. A general understanding of the myotomes and dermatomes of the cervical spine aids in diagnosis (Fig. 5.1). However, radicular symptoms do not always follow a predictable pattern of the affected root, and the type and intensity of symptoms vary widely. Some patients complain of less specific upper trapezial and interscapular pain, or discomfort about the shoulder girdle. There may also be more than a single nerve root involved, or anatomic variations in innervation, such that symptoms seem to cross over dermatomes and/or myotomes. Radiculopathy may also be present in the bilateral upper extremities and can exist concurrently in patients with myelopathy or peripheral nerve compression syndromes.

The physical exam performed in a systematic, root-specific manner, can elucidate sensory disturbances, motor deficits, and diminished reflexes. Pain and sensory changes in the affected root distribution are more commonly seen, while motor weakness and reflex changes are encountered less often. The examiner can sometimes reproduce radicular pain by performing the Spurling test, where the patient extends the neck and bends it toward the affected side. This maneuver narrows the neuroforamina and causes root impingement. As a corollary, patients often endorse relief of radicular symptoms when they sleep with their arm overhead, which enlarges the neuroforamina and decreases root compression. One must examine the shoulder with various maneuvers (refer to shoulder chapter) to rule out intrinsic shoulder pathology which can mimic or coexist with cervical radiculopathy. Shoulder pain that seems to localize anteriorly is generally intrinsic to that joint, but shoulder pain that localizes to the posterior scapular region or radiates past the midarm to the elbow or hand is typically referred from the cervical spine.

For any patient presenting with cervical radiculopathy, care must be taken to screen for concurrent myelopathy. Part of the history should include inquiry about

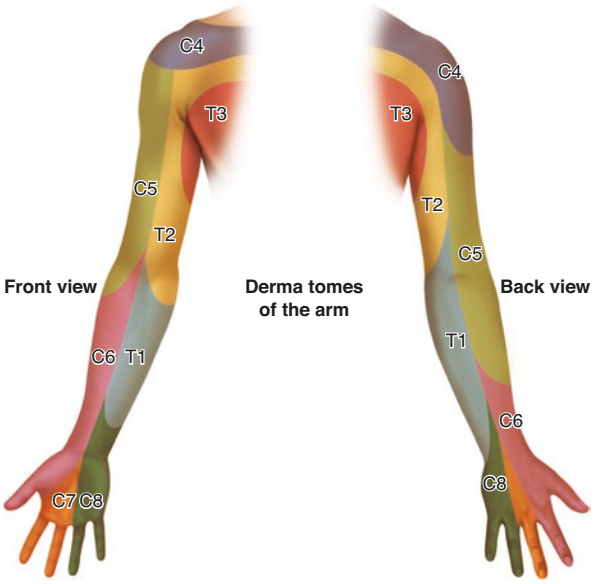

Fig. 5.1 Map of the most common anatomic distribution of cervical dermatomes and myotomes in the arm. The C5 through C8 levels are most frequently affected by radiculopathy

changes in gait, manual dexterity while performing fine motor tasks, and bowel and bladder incontinence. Screening for myelopathy should also include an examination for the presence of long tract signs, including tests for positive Hoffman or Babinski signs, as well as clonus or an inverted brachioradialis reflex. A more detailed discussion of the evaluation for myelopathy will be discussed in a later section.

Differential Diagnosis and Diagnostic Testing

The diagnosis of cervical radiculopathy is typically made using clinical history and physical exam alone, without the need for imaging or special tests. The differential diagnosis for radicular symptomatology includes peripheral nerve entrapment, brachial plexus injury, and tendonopathies (shoulder and elbow) of the upper extremity. Less commonly, infectious (herpetic zoster) or post-infectious (Parsonage-Turner) etiologies may produce similar symptoms. Some patients will present with a neck-shoulder syndrome where pathology coexists at both anatomic locations. Hence they will have both radicular features as well as intrinsic shoulder pain (rotator cuff pathology) with certain maneuvers and therefore can often be confusing to the clinician. Patients should also be screened for "red flags," such as unexplained weight loss, fever, intravenous drug abuse, and history of previous cancer, which may suggest the possibility of infection or tumor.

Cervical radiculopathy can exist concurrently with peripheral neuropathy, a so-called "double crush syndrome", where there is pathologic compression at more than one location along the course of a peripheral nerve. This may present a diagnostic challenge. For example, a patient with carpal tunnel syndrome may also have a C6 radiculopathy, which may result in an overlapping distribution of numbness and sensory deficits. In patients with carpal tunnel syndrome, it is helpful to inquire if symptoms radiate from the neck and to perform a provocative Spurling test to assess for radiculopathy. Proximal muscles supplied by the C6 nerve, such as the biceps or common wrist extensors, will be spared by carpal tunnel syndrome but may be affected in radiculopathy. By screening patients in this manner, fewer cases of double crush syndrome would go undiagnosed, and patients would benefit from timely treatment of both the cervical radiculopathy and peripheral nerve compression. Interestingly, the diagnosis of double crush syndrome is often made when patients are dissatisfied with the outcomes of a carpal tunnel release, presumably because of coexisting C6 radiculopathy.

Because the diagnosis is reliably made clinically and the natural history is usually self-limiting, it is reasonable to limit the use of diagnostic imaging until patients have been symptomatic for 4–6 weeks. The imaging helps to confirm the diagnosis and to facilitate treatment. Of course, if there is a concern for infection, tumor, or progressive motor deficits, diagnostic imaging should be obtained expeditiously. Plain anterior-posterior and lateral cervical radiographs are of limited diagnostic value, but they do demonstrate overall cervical alignment, and the extent of degeneration as evidenced by intervertebral disc height loss and osteophyte formation.

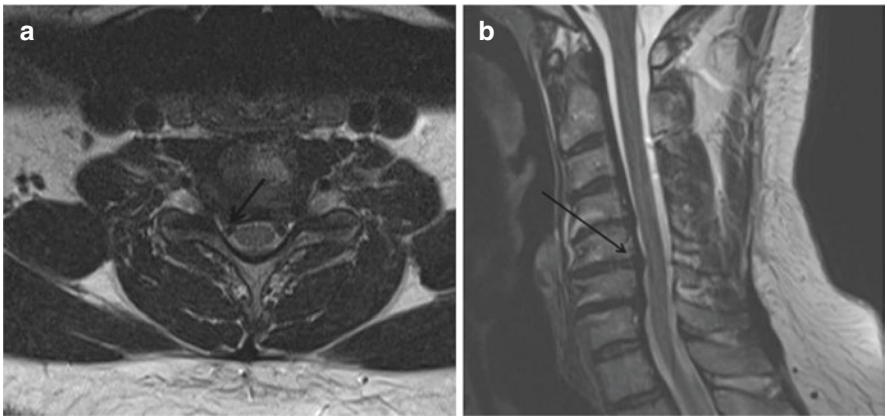

Fig. 5.2 Axial (**a**) and sagittal (**b**) magnetic resonance images (MRI) of a disc process at C5–C6 causing a right-sided radiculopathy (black arrows)

Magnetic resonance imaging (MRI) is the study of choice for cervical radiculopathy. MRI provides detail of the neural elements and surrounding soft tissue structures (Fig. 5.2). When an MRI is contraindicated, a computed tomography (CT)-myelogram can be useful to show focal areas of compression.

Results from MRI should be interpreted cautiously given the high sensitivity for detecting abnormalities. It is well established that asymptomatic patients have a high incidence of positive MRI findings, so areas of nerve root compression must be correlated with clinical findings. From a surgeon's perspective, it is ideal when there is correlation between anatomic abnormalities on neuroradiographic studies, patients' symptoms, and physical exam findings. In cases where imaging studies are equivocal, selective nerve root injections at the suspected level of involvement can be both diagnostic and therapeutic. Furthermore, electromyography studies and nerve conduction tests can be used adjunctively when patient's history and physical exam are inadequate to differentiate cervical radiculopathy from other neurologic causes of pain. For example, the presence of abnormal insertional activity in the paraspinal musculature can differentiate cervical radiculopathy from brachial plexopathy. These studies should be interpreted in the context of the clinical exam and radiographic findings and can effectively rule out other sites of compression. When there is concomitant shoulder pain that coexists and the clinical exam would suggest an intrinsic shoulder problem, an MRI of the shoulder may also be considered to clarify the diagnosis.

Nonoperative Management

Nonsurgical management is the mainstay of treatment for cervical radiculopathy. There is a lack of well-established nonsurgical treatment guidelines based on high-quality scientific evidence, and much of conservative treatment is centered

on level 4 and 5 evidence. In the setting of herniated disc material, chemical inflammatory mediators significantly contribute to radicular pain. These properties make oral anti-inflammatory medications an efficacious first-line treatment. Narcotics should rarely be prescribed for routine analgesia but can be considered on occasion for breakthrough pain or in patients who cannot tolerate NSAIDs. Some patients benefit from a multimodal analgesic regimen, which may include muscle relaxants, antidepressants, and gabapentin in conjunction with oral NSAIDs. For symptoms that are unresponsive to anti-inflammatories, in patients without medical contraindications, an oral tapered steroid regimen may also be prescribed.

Postural education, improved ergonomics, and lifestyle modification help to improve functional capacity. Patients are encouraged to mobilize early and to participate in physical therapy once pain has subsided. There is no proven role for immobilization or bed rest. Nonimpact aerobic exercises such as stationary biking can help relieve symptoms and maintain fitness. Some patients also derive temporary relief from intermittent home traction, which temporarily enlarges the neuroforamina and decompresses the exiting roots. Traction is not advised in patients with myelopathy, since lengthening the spinal column across an area of cord compression can be dangerous.

For persistent symptoms that have not been adequately relieved by oral analgesics, and functional rehabilitation, corticosteroid injections can be considered. Epidural corticosteroid injections offer a powerful, locally concentrated anti-inflammatory effect. Selective nerve root injections target the perineural space surrounding the affected root and avoid the spinal canal. Although relatively safe, epidural injections are invasive and come with risks, which include but are not limited to dural puncture, epidural hematoma, and epidural abscess. Conservative management should be continued for at least 6–8 weeks since the natural history of most cervical radiculopathy is for spontaneous pain resolution within 75–90% of patients. Patients may continue to see symptomatic improvement over more than 6 months.

Indications for Surgery

While conservative management is the predominant treatment for this typically self-limiting condition, there are cases where surgery is warranted and largely beneficial. Ideal surgical candidates have neuroradiographic evidence of root impingement, with corresponding root dysfunction, and persistence of symptoms despite several months of conservative care. Functionally significant motor deficits and debilitating radicular symptoms not responsive to conservative measures are indications for earlier surgical intervention. Subtle motor weakness which can be seen in early acute radiculopathy is often due to inflammation and pain and should spontaneously resolve with conservative management. However, if the weakness persists or progresses and leads to early muscle atrophy, the patient should be referred to a spine specialist for closer surveillance.

Operative Management and Expected Outcomes

Anteriorly based pathologies such as soft and hard disc herniations are the most common causes of cervical radiculopathy. The majority of patients are treated with an anterior cervical discectomy and fusion (ACDF). The anterior approach allows excellent exposure of the cervical spine and involves removal of the offending disc. It is muscle sparing and involves minimal blood loss. Once the discectomy is performed, the posterior longitudinal ligament can be resected, offering directly visualization of the dura and exiting nerve roots. Fashioned iliac crest autograft, allograft, or an interbody device is placed in the decompressed interspace to impart stability and to promote bony fusion across the motion segments. The graft restores intervertebral height and indirectly expands the neuroforaminal space. Advantages of the anterior approach include access to both central and lateral disc herniations, low infection and wound complication rates, and relatively minimal postoperative pain. The major disadvantages of ACDF are the risks for nonunion at the fusion site and persistent speech and swallowing difficulties due to retraction of the esophagus and laryngeal nerves.

A subset of cervical radiculopathy patients are eligible for cervical disc arthroplasty instead of an ACDF. The approach and manner of decompression are essentially similar to that for a fusion, except an artificial disc is placed in the interspace. The theoretical advantage of cervical disc arthroplasty (CDA) is preservation of motion at the surgical level, potentially mitigating the risk of adjacent segment disease and subsequent need for reoperation. It also eliminates the risk for pseudarthrosis. ACDF and CDA have been shown to have essentially equivalent patient-reported outcomes in medium-term clinical trials (2–10 years); however, debate persists regarding CDA's effectiveness in decreasing adjacent segment disease and need for reoperation. Cervical adjacent segment disease is believed to occur at an annual incidence of about 3%, regardless of the surgery performed, and it is unclear if this is consequence of fusion or due to the natural history of disc degeneration. The long-term mechanical durability and clinical outcomes data for cervical disc arthroplasty have also not yet been realized as long-term prospective trials are only starting to report 10-year data.

A posterior approach involving a laminoforaminotomy can be used to address anterolateral disc herniations or foraminal stenosis. The posterior approach to the spine involves dissection through the muscular raphe in the midline of the neck. Direct access to the compressed nerve root is achieved with removal of bone from the overlying facet and lamina, without destabilizing the motion segment. Proponents of the posterior laminoforaminotomy value the direct visualization of the nerve root, and avoidance of fusion and its attendant complications. Drawbacks of this procedure include inability to restore foraminal height with an interbody graft, as well as risk for recurrence as degenerative changes ensue.

A high rate of clinical success is to be expected for surgical decompression of the cervical nerve roots for cervical radiculopathy, regardless of approach. Patients commonly experience lasting relief of arm pain and improvements in motor and sensory function. Up to 10 years after surgery, patient satisfaction is reported at more than 90%.

Cervical Myelopathy and Myeloradiculopathy

Definition and Epidemiology

Cervical spondylotic myelopathy is the most common cause of spinal cord dysfunction in adults, and its incidence is likely underreported. Cervical spondylotic myelopathy results from age-associated degenerative changes to structures about the spinal cord, including disc degeneration, ligamentous hypertrophy, and osseous changes. These anatomic changes encroach upon the spinal canal and can lead to direct compression of the cord. Congenital spinal stenosis anatomically predisposes the development of cervical myelopathy. Patients with cervical spondylotic myelopathy have a much greater risk for spinal cord injury. Primary care physicians play an important role in the management of cervical myelopathy, as early detection and prompt referral for surgical evaluation can greatly improve patient outcomes.

Clinical Presentation

The pathophysiologic effects of spinal cord compression are thought to be a combination of direct mechanical effects on the neural tissue and related alterations in vascular supply. Presenting symptoms can include gait instability, diminished manual dexterity, motor weakness, sensory loss, incontinence, and permanent functional disability. The spectrum of disease severity and variation in symptomatology are commensurate with the many different manners in which the spinal cord can be functionally compromised by compression. For example, pathology that affects the dorsal column may predominantly manifest with proprioceptive loss in the extremities. The clinical course of cervical spondylotic myelopathy is marked by periods of neurologic stability with stepwise deterioration of neurologic function. Approximately 20–62% of patients will deteriorate neurologically within 3–6 years of diagnosis, and patients with even mild cervical myelopathy may have increasing difficulties with managing activities of daily living as years pass.

A thorough history and physical exam help to illicit subtle cues of spinal cord dysfunction. Patients may endorse subacute changes in their gait, demonstrate instability on exam, and have difficulty with tandem heel-to-toe walking more than a few steps. Patients may also report difficulty performing fine motor tasks, like buttoning a shirt or using chopsticks. The examiner can test hand dexterity with the grip and release test, where patients rapidly open and close their hands while being timed. Patients are normally able to do this about 20 times in 10 seconds. This test of manual dexterity can be used to survey stability of neurologic function over time. Additional evidence of spinal cord dysfunction occurs with extension of the neck causing an electrical shock-like sensation to shoot down the spine, the so-called Lhermitte's sign. This maneuver dynamically decreases the space available for the spinal cord and exacerbates symptoms.

Patients may also exhibit long tract signs, which are indicative of damage to the corticospinal tracts. The Hoffman's reflex, for example, should raise concern for cervical myelopathy when positive. To test this the examiner flicks the distal phalanx of the index or middle finger, and a positive finding is seen with flexion of the distal phalanx of the thumb. Other clinical findings of upper motor neuron dysfunction include an extensor plantar response known as the Babinski sign, where firmly stroking the lateral border of the foot results in extension of the great toe, or the inverted radial reflex, where a strike by a reflex hammer to the brachioradialis tendon elicits not only wrist extension but also finger flexion.

It is important to note that the absence of upper motor neuron signs (i.e., hyperreflexia, Hoffman sign, inverted brachioradialis reflex, clonus, and Babinski sign) does not preclude the diagnosis of myelopathy. The presence of long tract signs is not highly sensitive, and patients with unequivocal cervical myelopathy may in fact manifest no such signs. Up to one-fifth of patients who otherwise are myelopathic on the basis of history, correlative advanced imaging, and subjective improvement after decompression do not have long tract signs on presentation. Certain coexisting conditions can diminish the reliability of long tract signs in detecting spinal cord dysfunction. For example, in patients with myeloradiculopathy, concurrent radiculopathy can diminish the transmission of long tract signs. Diabetes, through its effect on peripheral nerves, is also thought to have a dampening effect on the transmission of neurologic reflexes. A higher index of suspicion for myelopathy should be had for patients with diabetic peripheral neuropathy. Even in the absence of long tract signs, concerning clinical symptoms combined with correlative imaging studies should guide treatment decisions.

Primary care physicians should remain vigilant for cervical myelopathy even in patients presenting with lumbar spine symptoms, such as neurogenic claudication and radiculopathy. A red flag symptom such as gait instability should immediately stoke concern for concomitant cervical myelopathy. Interestingly, it is not an uncommon presenting clinical scenario for patients with primarily low back symptomatology to have an underlying cervical disease. In fact, the coexistence of lumbar and cervical spinal stenosis has been reported in up to 15% of patients. A focused lower extremity exam may not illicit positive long tract signs, since concomitant lumbar spinal stenosis may dampen CNS signal transmission. It is therefore appropriate to screen patients presenting with lumbar spinal stenosis for concomitant cervical myelopathy by thoroughly examining both the upper and lower extremities.

Differential Diagnosis and Diagnostic Testing

The differential diagnosis for cervical myelopathy includes other central nervous system disorders as well as neuropathy and the long-term effects of alcohol abuse or certain vitamin deficiencies. When cervical myelopathy is suspected, upright plain radiographs are used to assess for alignment, segmental stability, and degree of degeneration. The condition of the spinal cord and influence of surrounding

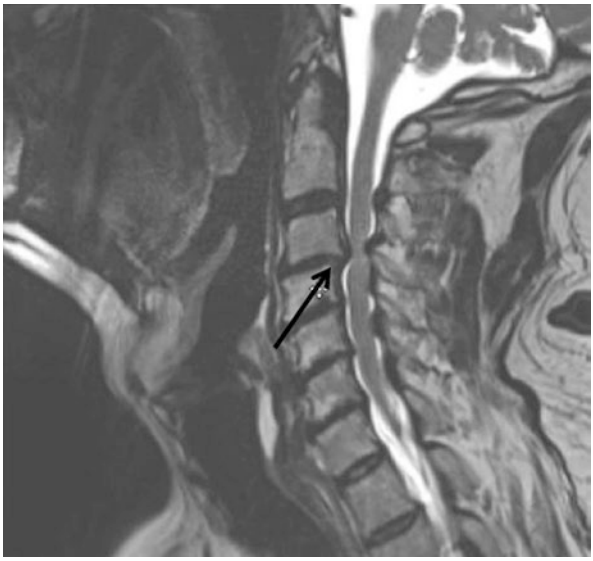

Fig. 5.3 Sagittal MR image demonstrating severe spinal stenosis and myelomalacia (black arrow) at the level of C3–4 in a patient with cervical spondylotic myelopathy

structures is evaluated with an MRI, or CT-myelogram in cases where MRI is contraindicated (Fig. 5.3). The patient should be referred to a spine surgeon to discuss treatment options and establish care for routine surveillance.

Nonoperative Management

Although surgical decompression of cervical myelopathy is the only manner in which the natural history of the disease can be altered, not all patients desire to undergo surgery. Many patients function well with mild forms of myelopathy and remain neurologically stable for years. However, there is always a risk for functional decline, which patients should reasonably be made aware of. A treatment plan is formulated between the care team and the patient after discussing the risks and benefits of surgery versus expectant management. Medical comorbidities such as diabetes, significant cardiac or renal disease, and advanced age may sway the balance of surgical risks and benefits toward nonoperative care.

There is no role for injections in patients with cervical spondylotic myelopathy. Physical therapy may improve the functional capacity of the patient, but will not alter the natural history of the disease. Anti-inflammatory medications and neuromodulators may help to alleviate radicular symptoms when simultaneously present. Rigid cervical orthoses have not been shown to be beneficial. Nonoperative interventions such as cervical traction and manipulation are not supported by high-quality evidence and associated with case reports of catastrophic complications. In general, when patients present with myelopathy, it is advised that the patient be referred to a spine specialist for consideration of surgery.

Indications for Surgery

The goal of surgery is to decompress the spinal cord and arrest further neurological decline. The thought process surrounding decompression is that the patient is far less likely to worsen in the absence of ongoing cord compression, and this is overwhelmingly the case. Indeed, some patients experience improvement of neurologic symptoms postoperatively. Others may experience further deterioration, even after a successful decompression, but these patients are in the minority. The most common etiology for neurological decline after an adequate decompression is the development of a new, adjacent focus of cord compression.

Patients may elect to defer surgery when there is mild evidence of spinal cord dysfunction, though this is not without some risk. It is difficult to predict which patients will have stable disease without decompression and which are at risk for further progression. These patients can be screened at regular intervals for evidence of neurological decline. Such deterioration may be subtle, and it is advantageous for patients to be followed by the same physician over time. Evidence of decline should be indicative of the capacity for progression and once again prompt a discussion regarding surgery. Patients with more pronounced or progressive clinical findings, and/or evidence of severe cord compression, should consider surgical intervention as soon as is reasonably possible. These patients are likely to be at greater risk for further functional decline, with the possibility of a devastating spinal cord injury in the event of a traumatic event that stresses a spinal cord that is already compromised.

Operative Management and Expected Outcomes

Anterior, posterior, and combined surgical approaches may be utilized, depending on a variety of factors, including anatomic location of the compression, alignment of the spine, and consideration of distinct complications associated with each approach. Decompression is the chief goal of surgery, and selection of the approach is performed with this priority in mind. In the lordotic cervical spine, in the setting of ventral compression, a posterior laminectomy can effectively allow the cord to freely float away dorsally. The most commonly used posterior surgical technique is a laminectomy and instrumented fusion. This involves removal of the posterior lamina and segmental instrumented fusion. Advantages of this include the potential for wide decompression, stabilization to prevent subsequent post-laminectomy kyphosis, and fusion to improve pain related to spondylosis. Laminoplasty, an alternative technique, expands the diameter of the spinal canal by expanding the lamina only on one side. Laminoplasty directly decompresses posterior impinging structures and indirectly decompresses the ventral cord. Advantages of this procedure include maintained segmental distribution of axial and rotational forces and preservation of motion. This is a reasonable option in patients with poor biologic potential for bony fusion.

An anterior approach can directly address anterior pathology, such as a central disc herniation. This approach involves discectomy or corpectomy (Fig. 5.4),

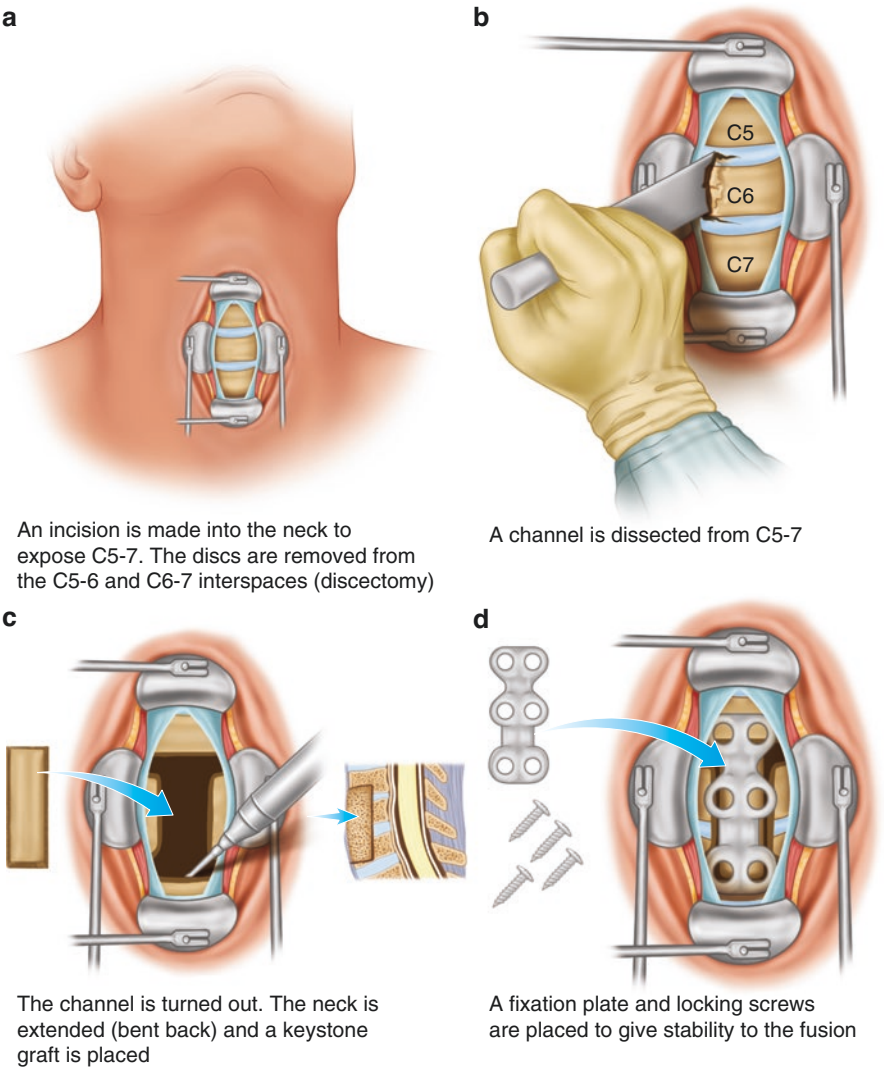

Fig. 5.4 Depiction of an anterior cervical approach with corpectomy and reconstruction using a strut graft, anterior plate, and instrumentation

depending in part on the location and extent of anterior pathology. It is particularly useful when ventral compression exists in the setting of neutral or kyphotic cervical spine alignment, precluding the possibility of indirect decompression with a posterior procedure. Both anterior and posterior approaches are effective in improving patient's quality of life and have comparable outcomes. Posterior approaches have a higher rate of complications, particularly infection or wound breakdown. Ultimately, the success of the surgery is most closely linked to the adequacy of the

Synopsis of presentation, diagnostic testing, and treatment options for patients with cervical radiculopathy or myelopathy

Clinical entity	Presentation	Diagnostic testing	Conservative management	Indications for surgery	Operative management
Cervical radiculopathy	Radiating pain, with possible sensory deficits, and motor weakness in the distribution of the affected nerve root	MRI or CT-myelogram	Physical therapy Anti-inflammatory medications	Radicular symptoms refractory to conservative management Significant motor weakness	Anterior cervical discectomy and fusion Cervical disc arthroplasty Posterior laminoforaminotomy
Cervical myelopathy	Gait instability, diminished fine motor dexterity, sensory deficits and motor weakness, hyperreflexia, bowel and/or bladder incontinence	MRI or CT-myelogram	Generally not advocated Counseling about risks of disease progression Routine surveillance of neurologic function	Myelopathy in the setting of static or dynamic spinal cord compression	Anterior cervical discectomy/ corpectomy and fusion Posterior cervical decompression and instrumented fusion Posterior cervical laminoplasty

MRI magnetic resonance imaging, *CT* computed tomography

spinal cord decompression. Surgical intervention has a better prognosis if patients with myelopathy are treated at an earlier clinical stage before severe spasticity or loss of ambulatory function occurs. Once the spinal cord undergoes irreversible chronic changes, the surgical goal is to prevent further neurologic deterioration since full recovery is often a challenge. Classically, patients were told to expect surgical decompression would arrest the progressive decline in their neurologic function. Recent studies have shown that patients, especially those with a shorter duration of neurologic deficit, can expect to regain a portion of their lost function after surgery.

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