Chapter 9 – Neck and Back Pain

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General Concepts

Musculoskeletal neck and back pain are common complaints with a prevalence of 60 percent to 90 percent in the general population. Differentiating benign musculoskeletal pathology from a more serious neurologic problem is fairly logical when the differential diagnosis is approached systematically.

Musculoskeletal neck and back pain generally is high and midline, experienced in the upper neck and back muscles. Radicular pain tends to occur medial to the scapula or in the buttock with eventual radiation down the arm or leg. Because sitting increases the intraspinal pressure, radiculopathies are classically much worse with sitting but improved with standing or walking. Also, as a general rule, the pain is maximal proximally, whereas paresthesia and numbness are more prominent distally.

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Cervical spondylitic myelopathy and lumbar spinal stenosis belong in a special category because neither present with arm or leg pain. Both syndromes can be easily overlooked if not specifically considered during the history and physical exam.

Spondylolysis (Figure 9-1) is a weak line that occurs between the inferior and superior portions of the facet joint, creating a break in part of the lamina and allowing slippage of one vertebra forward on the vertebral body below. This slippage is called spondylolisthesis. Commonly a cause of back pain after athletic activities in a young patient, this is one cause of back pain, which has a characteristic radiologic appearance.

Figure 9-1: Schematic drawing of an oblique roentgenogram of the lumbar spine, showing the characteristic “Scotty dog” look of its posterior elements. Note that the defect in the pars interarticularis appears to be a collar around the dog’s neck.
The most commonly used grading system for spondylolisthesis is the one proposed by Meyerding in 1947. The degree of slippage is measured as the percentage of the distance the anterior vertebral body has moved forward relative to the vertebral body below.

- Grade 1: 1–25 percent slippage
- Grade 2: 26–50 percent slippage
- Grade 3: 51–75 percent slippage
- Grade 4: 76–100 percent slippage
- Grade 5: Greater than 100 percent slippage

Cervical myelopathy can be produced by chronic compression of the cervical spinal cord and often presents primarily as a gait disorder, though moderate neck pain and hand atrophy from chronic root involvement are often found on exam.

Lumbar spinal stenosis can produce significant leg and back pain but is often present on exertion and relieved by sitting, and the physical examination is usually entirely normal.

“Look-alike” pathology in the shoulder and hip are actually not at all uncommon and can lead to significant diagnostic confusion. Shoulder joint pathology often causes radiation of pain into the upper arm. Unlike radiculopathy, however, the pain originates from a point anterior to the shoulder joint rather than posteriorly beneath the scapular wing, and movement of the arm at the shoulder usually exacerbates the pain.

Hip pain generally radiates into the groin and the anterior thigh rather than the buttock and calf. Generally hip maneuvers will reproduce the pain. Occasionally diagnostic cortisone injections into the hip are required to differentiate hip pain from radicular pain in patients with generalized arthritis in both the hip and the back.

Musculoskeletal neck pain associated with median and ulnar neuropathy is sometimes confused for cervical radiculopathy, and the occasional patient with left arm radiculopathy can have symptoms which mimic angina.

Spinal cord and nerve root symptoms and signs are logically ordered according to specific neurologic levels. It is usually possible to predict the location of the lesion responsible within the neuraxis by evaluating a few key muscle groups and reflexes.

**Cervical Pathology**

**Neck Pain:** Generally presents in the paraspinal muscles high and bilateral rather than presenting beneath the scapular wing and radiating into the arm which is classic for cervical radiculopathy.

**Cervical Radiculopathy:**

Examination of the upper extremity, with careful attention to the motor and reflex abnormalities, generally allows localization of the cervical nerve root affected. In less completely expressed radiculopathies, occasionally, just the history alone of radiating pain and the description of the location of the paresthesia will be enough to localize the nerve root involved. This is done primarily to ensure that the lesion is a benign radiculopathy that can be expected to resolve spontaneously with conservative management.
Treatment is typically the passage of time and physical therapy coupled with nonsteroidal anti-inflammatory medications. Patients can be taught simple exercises; in addition, use of "over-the-door traction" on a daily basis may be helpful. Some physical therapists use mechanical traction as well, but this modality should be used cautiously. Short-term use of narcotics at night is usually necessary, as a fully expressed cervical radiculopathy can be exquisitely painful. Use of epidural corticosteroid and oral steroids are reserved for patients with intractable symptoms. Prednisone 20 mg taken 4 tabs q day X 3 days, 3 tabs Q day X 3 days, 2 tabs q day X 3 days and 1 tab for 3 days (#30 tabs) is often the last conservative treatment prior to the consideration of surgery.

Five to ten percent of the time patients will fail conservative management and will have progressive, unrelenting pain, progressive motor weakness and reflex loss in the distribution of one cervical nerve root. If this is matched by a convincing disc abnormality, with clear neural impingement at the appropriate level, these patients will often need neurosurgical consultation for cervical laminotomy and disectomy.

Radiculopathies, in general, are reliably worsened with laughing, crying, sneezing or any other variant of the Valsalva maneuver, due to an increase in intraspinal pressure. Sitting usually makes the pain worse while standing relieves the discomfort. A patient with an active radiculopathy may actually walk the floor at night to get relief from pain. Cervical 5 (Figures 2 and 3).

![Figure 9-2](image)

Figure 9-2: An easy way to remember that the biceps reflex is innervated by C5 is to associate five fingers with neurologic level C5.
The deltoid and the biceps muscles are the two most important C5 muscles. The biceps reflex is the predominant indicator of C5 involvement; because this muscle also has C6 innervation, however, a comparatively small amount of hyporeflexia may indicate significant nerve root pathology. Sensation testing is unreliable for radicular sensory loss. Rather than test sensation, a careful sensory history will often be most useful, if you encourage the patient to be precise, i.e., “If you had to choose which finger is most involved, which one would you pick?”

Cervical 6

This nerve root is compressed by a disc at C5-6 and is the most common site of disc herniation. Both the bicep and wrist extensor muscles have C6 innervation, but the brachioradialis reflex is the preferred C6 reflex (see Figure 9-4). Sensory history or examination will generally demonstrate involvement of the thumb and first finger (see Figure 9-5).
Figure 9-4: Neurologic level C6.

Figure 9-5: An easy way to remember the sensory distribution of C6.
Cervical 7
A disc at C 6-7 will compress the C7 nerve root. The motor function of the triceps muscle and the wrist flexors are the most accurate for defining a C7 radiculopathy. The triceps reflex is usually hypoactive and there is involvement of the middle finger with paresthesias or numbness (see Figure 9-6).

Figure 9-6: A herniated disc between vertebrae C6 and C7 involves the C7 root.

Cervical 8
A disc at C7-T1 produces a C8 nerve root impingement. Finger flexors are often involved but there is no reflex abnormality and the sensory distortion involves the medial surface of the upper arm. Isolated C8 radiculopathy is quite uncommon in clinical practice, possibly because of the stabilization of the ribcage (see Figure 9-7).

Figure 9-7: A herniated disc between vertebrae C7 and T1 involves the C8 root.
Cervical Spondylitic Myelopathy

Unlike cervical radiculopathies described above, cervical spondylitic myelopathy has a much more subtle clinical presentation. The cervical spinal cord becomes slowly compressed over time by generalized arthritis so that the patient often presents with a slowly progressive gait disorder without neck or arm pain, often obscuring the real nature of the diagnosis.

Degenerative changes in the intervertebral discs are a natural process that occurs with age, often accelerated in patients with a significant history of trauma. As the discs desiccate and the vertebral bodies move more closely together, the posterior longitudinal ligaments lift away from the posterior aspect of the vertebral body allowing “disc-spur complexes” to form. Thickening of the facet joints and growth of the ligaments all combine to produce circumferential narrowing of the spinal canal with compression of the spinal cord as well as selected nerve roots (produced by foraminal stenosis). The most common sites for this spondylitic change are at the most concave portions of the spinal axis at C4-5 and C5-6 (and at L3-4, producing lumbar spinal stenosis See page 258-259. Some patients also are born with a congenitally small spinal canal from foreshortened pedicles. The average depth of the canal in the cervical spine is 17 mm. Cord compression rarely occurs until the diameter is reduced below 10 mm. Patients may present with a gait disorder of months to years duration. Because the posterior columns are the most affected, decreased joint position sense and vibratory loss are common, producing a stiff-legged gait with a tendency to fall backwards or to have exaggeration of gait instability in the dark when visual cues are absent. Bowel and bladder involvement are common, with urinary retention or constipation. On examination, patients have decreased range of motion of the neck with atrophy and weakness in the hands (from chronic nerve root compression) accompanied by spasticity and exaggerated reflexes in the legs, bilateral Babinski signs and a positive Romberg. Wide surgical decompression of the cervical spine by multiple level laminectomies often prevents worsening, though often does not significantly improve symptoms.

Syringomyelia

With the advent of MRI scanning, the diagnosis of syringomyelia is more common. A syrinx, expansion of the central canal of the spinal cord by fluid under pressure, is a unique structure that can extend many levels up and down the spinal cord, sometimes reaching all the way to the brainstem. Patients describe a severe ripping, burning discomfort which often presents in both shoulders and arms, sometimes extending up the back of the head in a “cape-like” distribution. Reflexes are often diffusely absent in the arms and weakness can be profound, extending beyond the territory of a single cervical nerve. This disorder is treated with a shunt placed within the distended sac, drained to the lumbar space. Even with surgery, this disorder generally produces chronic neurologic problems. Syringomyelia should be considered in any patient who persistently complains of a peculiar bilateral shoulder and arm pain. MRI of the cervical spine will often show the syrinx as a clear CSF filled structure on sagittal T2 weighted images.
Shoulder Pathology

Shoulder joint pathology and cervical radiculopathy are frequently confused, perhaps because patients with shoulder problems often describe the pain in a way that can sound like radicular radiation. Differentiation is relatively easy with careful history and examination. Radicular pain generally radiates beneath the scapular wing before moving on to radiate down the arm, while shoulder pain is usually maximal anteriorly over the point of the shoulder. Movements of the arm at the shoulder exacerbate joint, but not radicular pain. Rolling the bicipital tendon beneath the thumb will often reproduce the pain for patients with bicipital tenosynovitis, while head turning or compression testing (pushing down strongly on the vertex of the head to see if radicular pain is worsened) reproduce radicular pain. Occasionally steroid injections in the shoulder are used to identify which area is most symptomatic in patients with a clinical picture that suggests both cervical osteoarthritis and shoulder pathology.

Median neuropathy

Compression of the median nerve at the wrist, or carpal tunnel syndrome, is extremely common. However, it is not always appreciated that carpal tunnel syndrome can present in an atypical fashion, mimicking the symptoms of a cervical radiculopathy. Carpal tunnel syndrome must be considered whenever a patient complains of hand or wrist pain that is worse at night. This strong nocturnal predilection is sometimes not clear unless patients are asked directly. Most patients have maximal symptoms in their dominant hand, but bilateral involvement is common. Fingers that feel stiff, tight and swollen (like “sausage casings”) in the morning or awaken the person at night is the typical complaint. Shaking the hands, holding them above the head or thrusting them into ice-cold water often brings relief. When only some fingers are involved, it is usually the thumb and first finger, though many patients believe the sensory symptoms involve the whole hand. After some time weakness begins to progress, particularly in the abductor pollicis brevis, or thumb muscle, which in turn produces flattening, or atrophy, of that muscle group with weakness of the pincer grip. Tasks such as using a nail clipper, turning a key in a lock, or opening a jar become difficult. Because the pain in carpal tunnel often radiates up the arm to the shoulder and the sensory symptoms are in the first two fingers, a C6 or C7 radiculopathy might be suspected.

Tinel’s sign (electrical dysesthesia in the hand produced by tapping over the median nerve in the wrist with a reflex hammer) and Phalen’s sign (reproduction of symptoms in the hand with forced compression) are often present, but their absence does not exclude a diagnosis of carpal tunnel. Nerve conduction studies (NCS) are more cost-effective than a cervical MRI and therefore need to be considered in cases with mixed symptomatology. Surgical decompression of the median nerve is usually not necessary unless slowing of conduction velocity in the motor portion of the nerve is evident on NCV (nerve conduction velocity) testing or the patient remains symptomatic after a 4–6 weeks trial of wrist splinting.

Ulnar Neuropathy

Compression of the ulnar nerve at the elbow can be confused with a C7 or T1 radiculopathy based on sensory symptoms in the fingers. Ulnar neuropathy is much less prevalent than median neuropathy and has a less stereotyped
presentation. Sensory complaints are present in the fourth and fifth fingers and are not more common during nighttime hours. When weakness is present, the fourth and fifth fingers sometimes develop a characteristic arched appearance so that the hand cannot easily lie flat on a surface. Treatment is simply avoidance of overuse of the arm at the elbow and elbow pads. Surgery requires transposition of the ulnar nerve at the elbow rather than simple decompression and is, therefore, performed less often. In some cases a fibrous band in the cubital tunnel may compress the ulnar nerve. Lack of improvement by conservative treatment may warrant further investigation with MRI since surgical lysis of the band is beneficial.

**Thoracic Pathology**

Because of the stabilization provided by the ribcage, herniation of thoracic discs, while possible, is not common. Thoracic radiculopathy is, however, seen in diabetic patients who suffer from peripheral neuropathy. The pain is quite intense, described as burning, shooting or tingling and generally in the distribution of one thoracic nerve root involving the back and radiating to one half of the chest wall. Unless this diagnosis is entertained in a diabetic patient, the pain can often be confused with pain from an abdominal or cardiac source. Herpes sine zoster can also produce severe neuropathic pain which can be quite puzzling in the few days prior to the outbreak of the characteristic vesicles.

**Malignant Cord Compression**

Hematogenous spread of cancer to vertebral bodies occurs more commonly in the thoracic spine because the amount of bone marrow is greatest due to the size and number of vertebral bodies. While benign musculoskeletal pain is common in the neck and back, it is relatively uncommon in the thoracic area. Therefore, any worsening pain localized to the thoracic area must be assumed to be cancer-related until proven otherwise. Typically, pain precedes the development of neurologic signs and symptoms by weeks to months. The pain is often encircling and can be misunderstood as visceral pain. Often, unlike benign disc pathology, the pain worsens at night. Most often the malignant process grows from the marrow bearing space in the vertebral body itself, breaking out backwards to compress the spinal cord. Breast, lung, renal cell and prostate carcinoma are the most common sources for malignant cord compression.

The spinocerebellar tracts and the posterior columns are often involved first, creating an ataxic gait from poor position sense. Antigravity muscles are then selectively affected leading to weakness in the iliopsoas muscles and dorsiflexors of the feet. Bowel and bladder function are affected relatively late in the course leading to complaints of constipation and urinary retention. If pain is the only presenting complaint, an elective MRI of the spinal column can be ordered the following day.

It is important to remember:

- To specify the spinal level of concern
- The lesion is often 2–3 vertebral bodies above the sensory or motor level seen on examination
Emergency study is indicated for any patient with rapidly advancing motor/sensory or bowel and bladder complaints.

Once the spinal cord compression creates infarction of the anterior spinal artery, the patient becomes permanently paralyzed without return of neurologic function. Dexamethasone 4–24 mg qid given urgently can provide almost complete relief of pain and allow testing to proceed while edema is treated, thereby reducing the risk of abrupt neurologic decline.

**Lumbar Pathology—Low Back Pain**

Musculoskeletal low back pain is one of the most common causes of missed work and disability. Most such pain is benign, self-limited and self-correcting. Generally best treated with activity and exercise, there is now little justification for prolonged bed rest. Lumbar x-rays are not routinely necessary in the first four weeks of symptoms and should only be performed in the presence of “red flags” or warning signs that the pain is due to something other than benign pathology. Fever, history of intravenous drug use, immunosuppression or prolonged steroid use, cancer or unexplained weight loss, significant trauma, or bowel and bladder complaints are warning signs that should prompt earlier additional testing.

Nonsteroidal anti-inflammatory drugs and physical therapy are the best first-line treatments for low back pain. Muscle relaxants may be used at night to decrease spasm and promote sleep but should not be used for more than four weeks because of risks of habituation. Chiropractic manipulation may be somewhat beneficial acutely, though there is nothing to support its use after the first four weeks of symptoms. Lumbar bracing and lumbar traction have no demonstrated benefit and should not be used.

**Lumbar Radiculopathy**

Radicular pain generally radiates from the buttock near the sciatic notch down the back or side of the leg to the foot. This pain, created by neural impingement from a disc herniation or osteophyte, is generally made worse by sitting and relieved by standing or lying. Generally the pain is worse proximally, in the back and buttock, while the sensory symptoms are more intense distally, in the tips of the toes or bottom of foot. Paraspinal muscular spasm commonly accompanies lumbar disc herniation with limitation of range of motion on forward bending.

Lumbar root pathology can be localized to one level with the careful application of sensory, motor and reflex testing. In general, surgery for lumbar root compression from simple disc herniation should not be entertained unless:

- The patient has failed 6 to 8 weeks of conservative therapy
- Has worsening intractable pain
- Has signs on exam confined to the distribution of one nerve root (which must match the sight of neural impingement on MRI or myelogram/post myelogram CT testing)

Although L3 can be involved, the most commonly involved nerve roots are L4, L5 and S1. The pain in all cases will radiate down the leg posteriorly from the buttock; on occasion, L4 pathology produces pain that radiates into the anterior aspect of the thigh before radiating into the foot. Sensory testing is the least useful or reliable part of the examination; though a sensory history
of involvement of the sole of the foot (versus the dorsum) can be a reliable indicator of S1 impingement (see Figure 9-8).

**Figure 9-8**: The sensory dermatomes (A) and (B) a practical method of testing sensation across the dorsum of the foot.

**L4 Root Level (Figure 9-9)**
Figure 9-9: A herniated disc between vertebrae L3 and L4 involves the L4 nerve root.

Having the patient attempt to walk on the heels can also test for early foot drop. Remember that the knee reflex can also be absent if the patient has had knee surgery and, therefore, in the presence of this history, the importance of an absent knee jerk is diminished as a localizing sign.

L5 Root Level (Figure 9-10)

Figure 9-10: A herniated disc between vertebrae L4 and L5 involves the L5 nerve root. This is the second most common level of disc herniation in the lumbar spine.

Lumbar 5-root pathology can be difficult to distinguish from L4 unless you remember that there is no reflex abnormality caused by compression of the L5 nerve root. Even in its most fully expressed form, the patient will have weakness of the extensor of the great toe and some tingling or numbness on the top of the foot.

S1 Root Level (Figure 9-11)
Lumbar Spinal Stenosis

This disorder is quite common and yet often overlooked as a source for back and leg pain because the physical examination is often completely normal. The diagnosis must be suspected on the basis of the history alone. Spinal stenosis is created from circumferential narrowing of the diameter of the spinal canal caused by facet hypertrophy and thickening of the posterior longitudinal ligament, processes which occur naturally with aging. Some people are also born with a congenitally narrow spinal canal from foreshortened pedicles, which predisposes them to the development of this disorder.

Patients with lumbar spinal stenosis sometimes do not complain of back pain at all, but instead describe intense aching or cramping in the thighs and calves. This pain is reliably induced by walking and relieved by sitting. Typically patients will tell you, when asked, that the symptoms come on more quickly when attempting to walk uphill than when walking on level ground. Often they will find that bending the torso forward will decrease discomfort. This allows them to go grocery shopping bent over a cart, but unable to walk the same distance around the store upright, without the cart. Likewise, they may be unable to walk for 20 minutes, but can easily sit on an exercise bike and pedal for the same amount of time without pain. This discomfort is best termed “pseudoclaudication” as it is identical to the complaint of patients with significant arterial compromise to the legs. Commonly these patients are sent first for vascular studies or to a vascular surgeon before referral to a neurologist or neurosurgeon. MRI of the lumbar spine is the best way to look for spinal stenosis. Surgical treatment is necessary with a generous posterior decompressive laminectomy to remove the bony compression of the cauda equina. In patients who are sedentary and quite comfortable to spend much of their time sitting, surgery may not be necessary. Short-term relief of symptoms can often by achieved with lumbar epidural corticosteroid injections. Because the compression is bony, physical therapy is not helpful. Hyperextension exercises may also exacerbate the pain.

Cauda Equina Syndrome

Compression of all of the lumbar nerve roots after they leave the spinal cord (which ends at vertebral level L1) produces a cauda equina (“horse’s tail”) syndrome. This produces a characteristic clinical syndrome of weakness, numbness, tingling and lack of deep tendon reflexes symmetrically in the legs. This syndrome can be seen with far advanced lumbar spinal stenosis (see above) but, because spinal stenosis is painful, the stenosis is often discovered and treated before a true cauda equina syndrome develops. Tumors are a more common cause of this syndrome, allowing compression of nerve roots to occur from a neurofibroma, menigioma or ependymoma. Metastatic disease to the vertebral bodies can also produce this syndrome, though pain preceding the onset of neurologic symptoms would be expected. Recognition of a patient’s
bilateral leg weakness as reflective of compression of nerve roots with essentially all of the findings listed for individual nerve roots above (especially L4, L5 and S1) allows targeting of diagnostic studies to the vertebral level L1 and below and prevents a fruitless search for spinal cord pathology.

**Conus Syndrome**

Bowel and bladder fibers travel in the spinal cord. The conus is the tapered end of the spinal cord just before the cauda equina (rostral to caudal). When patients have what appears to be a cauda equina syndrome but with prominent or early involvement of bowel and bladder function, a lesion in the tip of the conus needs to be considered. Because this structure is often at spinal level T11 or T12, it can be missed on routine lumbar MRI studies and certainly on lumbar CT studies which often don’t even start the imaging until below L2. Always remember the localization of the problem and make sure the imaging studies show you the involved area as well as one or two vertebral bodies higher in order to ensure that pathology has not been missed.

**Hip Pathology**

Pain from the hip generally radiates into the groin but can also be present into the anterior and lateral surface of the thigh, mimicking an L3 radiculopathy. Hip maneuvers should exacerbate the pain and should be part of the examination of any patient with back and leg pain. Sometimes accurate diagnosis will require hip radiographs and even diagnostic cortisone injections. Patients with generalized arthritis in the back, hips and legs, or those with knee replacements (which render the localizing value of an absent knee jerk useless) are the most likely candidates for this group of problems. Hip pain is also more likely to be present with weight bearing while radicular pain tends to be better with standing and worse with sitting.

It is important to remember that the lumbosacral plexus travels through the pelvis after exiting the lumbar spine.

On occasion hemorrhage into the psoas muscle in patients taking anticoagulants, or neoplastic spread from pelvic tumors (prostate cancer for example), can compress lumbar nerve roots in the pelvis. A pelvic CT scan needs to be ordered. A pelvic CT scan needs to be ordered for patients with clear involvement of high lumbar nerve roots and an unremarkable lumbar MRI scan.

**Peripheral Neuropathy**

Peripheral neuropathy is fiber-length dependant. Axonal loss occurs in the spinal cord and the longest fibers “die-back” first. This produces a symmetrical complaint of numbness in the tips of the toes that spreads slowly up the leg like a knee sock (not a complaint of sole numbness, which would be characteristic of an S1 nerve root compression). Neuropathies can be pure sensory, pure motor or mixed sensorimotor. Bilateral foot drop generally does not occur until the sensory involvement is fairly pronounced. A severe mixed sensorimotor axonal polyneuropathy, therefore, could easily mimic lumbar spinal stenosis with compression of nerve roots as both could produce absent ankle jerks and sensory loss below the knees. Searching for a history of exertional claudication in the legs should serve to separate the two entities, as peripheral neuropathy
should not produce back and leg pain. In addition, because of the fiber length
dependant features of neuropathy, when a patient is affected in the legs so that
sensory loss extends to the knees, careful search for numbness in the tips of the
fingers and hands should allow diagnosis of a more widespread problem. Axonal
polyneuropathy is generally produced by a systemic illness such as diabetes.
Demyelinating neuropathy, where the major site of tissue destruction is the
myelin sheath, is often immune mediated and potentially treatable. The most
common example of a demyelinating neuropathy is Guillain-Barré syndrome.
Vitamin B12 testing, RPR, serum protein electrophoresis (myeloma), and thyroid
function studies should be part of the routine evaluation of patients with
neuropathy.

Separating neuropathies into axonal versus demyelinating becomes important
as one type is treatable and the other is largely untreatable. Symptom onset
is fairly slow for the axonal variety and more fulminant for the demyelinating
type. The best method to distinguish them from each other, however, is
electromyography and nerve conduction studies. Demyelination, which
occurs randomly through the plexus and nerve root, produces areas of
complete conduction block and very slow conduction times. Axonal pathology
produces more modest changes in conduction velocities, allowing the
electrophysiologist to tell the clinician which variety of neuropathy is likely.
Nerve biopsies and extensive evaluation for the cause of severe neuropathies
should be left to tertiary care subspecialty centers.

Meralgia Paresthetica

Meralgia paresthetica, or compression of the lateral cutaneous nerve of the thigh
can produce symptoms that overlap with symptoms caused by compression of
nerve roots in the back. Patients have a very characteristic complaint of tingling
dysesthesias in the lateral aspect of the thigh. The discomfort is in a rounded
area in the upper lateral thigh and does not involve the leg below the knee. The
discomfort is often maximal at night so that the patient awakens rubbing or
slapping the thigh to relieve the pain.

The lateral femoral cutaneous nerve enters the leg after diving beneath the
inguinal ligament. This nerve is often compressed by excessive weight such as
in pregnancy or with morbid obesity. The nerve can also be compressed
from tight clothing or activities that involve hyperextension of the leg at the
hip such as cross-country skiing or walking with an excessively long stride. The
typical patient to be affected with meralgia paresthetica, therefore, would be an
overweight truck driver who sits for long hours in a pair of tight jeans wearing a
thick billfold in the back pocket on his affected side.

The pain can be quite intense but generally is not affected by positioning. On
examination, these patients generally have no abnormalities with the exception
of a very small area of numbness within the dyesthetic area. Careful evaluation
of iliopsoas strength and presence of knee jerks should serve to differentiate
this condition from a high lumbar disc. Treatment is often education to remove
precipitating factors though, occasionally, corticosteroid block of the nerve in
the groin is necessary for the patient to obtain relief.
References


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Self-Assessment Questions

1. Which of the following presenting symptoms is most likely to represent a cervical radiculopathy?
   A. anterior shoulder pain made worse with arm movement
   B. high, midline bilateral cervical pain
   C. elbow and forearm discomfort made worse with sitting and straining
   D. hand tingling which is maximal at night

2. Which of the following presenting symptoms is most likely to represent a lumbar radiculopathy?
   A. groin pain made worse by walking
   B. buttock pain made worse by sitting
   C. bilateral calf and thigh pain made worse by walking
   D. tingling paresthesias in the lateral thigh

3. What signs on physical examination are most often seen in patients with lumbar spinal stenosis?
   A. absent deep tendon reflexes at the knees
   B. weakness of the dorsiflexors of one foot
   C. difficulty walking on the toes
   D. none

4. What signs on physical examination are most often seen in patients with cervical spondylitic myelopathy?
   A. spasticity and hyperreflexia in the legs
   B. Horner’s syndrome
   C. diminished deep tendon reflexes at the ankles
   D. down going toes (or a plantar response which is flexor)
Provide the most likely radicular level or other diagnoses for the following patients:

5. A hairdresser who awakens reliably at night with right hand pain and tingling in the thumb and first two fingers. Examination shows some blunting of sensation in the involved fingers without weakness or reflex asymmetry.
   A. C6
   B. Compression of the median nerve at the wrist
   C. C7
   D. C8

6. An elderly man with left buttock pain made worse by sitting. On exam he is unable to rise up on the toes of the left foot and has an absent deep tendon reflex at the left ankle.
   A. L5
   B. L4
   C. S1
   D. lumbar spinal stenosis

7. An elderly woman complains of progressively severe thoracic discomfort that worsens at night. Examination reveals proximal leg weakness manifested by difficulty arising from a low stool.
   A. malignant cord compression
   B. right L5-S1 disc herniation
   C. syringomyelia
   D. lumbar spinal stenosis

8. A young woman presents with urinary retention and constipation with tingling sensations in the soles of both feet. On exam she has only back pain and absent ankle jerks.
   A. polyneuropathy
   B. spondylolisthesis of L4 on L5
   C. cauda equina syndrome
   D. conus syndrome

9. A woman complains of numbness and tingling bilaterally from the knees down. The most reliable way to distinguish polyneuropathy from lumbar spinal stenosis by examination is:
   A. absent ankle jerks
   B. symmetric sensory loss to pinprick testing
   C. absent knee jerks
   D. sensory involvement in the hands
10. When carpal tunnel syndrome is the working diagnosis, the most important muscle to test for weakness is:

A. abductor digiti minimi
B. abductor pollicis brevis
C. finger flexors
D. finger extensors

ANSWERS
1. C
2. B
3. D
4. A
5. B
6. C
7. A
8. D
9. D
10. B