Chapter 1 – The Neurologic Examination

Section 3

The systems, which work on an unconscious level to modulate motor activity, (basal ganglia and cerebellum), do this by inhibiting or damping the effect of other neurons. Loss of their modulating effect will, thus, cause other systems to be overactive. The substantia nigra has a damping effect on the striatum via the neurotransmitter dopamine. Lack of dopamine, such as occurs in Parkinson syndrome, enables the striatum to discharge excessively. This, in turn, produces a slowing and reduction of motor movements termed bradykinesia. There is also an increase in motor tone to passive movements in all directions (rigidity). Abnormal repetitive discharges lead to the third symptom seen in dopamine deficient states, and that is tremor. This is usually seen at rest, and is a regular alternating tremor with a frequency of 3–4 per second. If the hands and fingers are involved it produces the classic pill rolling tremor. As mentioned, the tremor is seen at rest, and disappears with initiation of motor activity. It should not be confused with essential familial or senile tremor. These tremors are characterized by being initiated with activity and diminishing at rest. There is no associated bradykinesia or rigidity.

Lack of neuronal activity in the basal ganglia diminishes the damping effect on the cerebral cortex and leads to excessive motor activity. This can be seen with degeneration or loss of some basal ganglia neurons or excessive dopaminergic activity, which dampens the caudate nucleus. These states can lead to the production of various movement disorders. The exact mechanism for production of these disorders is not fully understood but some insight into their generation is obtained from knowledge of the specific lesions or biochemical conditions associated with the movement disorder. Some common movement disorders are:

Chorea. This movement consists of brief, random, nonrepetitive movements of fingers, extremities, face and trunk. When present they give the patient the appearance of being fidgety and not being able to sit still. Movements may be exaggerated further by the patients attempt to mask them. (e.g., The patient may attempt to hide an arm elevation movement by following through and scratching his head.) When these actions occur in serial fashion the patient appears to be in constant motion. This may be dismissed by the untrained eye but not the astute observer. If one sees such a patient further evaluation should be performed. The patient should be asked to extend his arms and fingers while extending at the wrist, and at the same time, hold out his tongue. This position enables one to better see the brief choreiform movements, which the patient is unable to prevent. Chorea can be seen with excessive dopamine administration (Sinemet® levodopa/carbidopa), in hereditary diseases (Huntington’s Chorea), and in acquired chorea (Sydenham’s chorea, during pregnancy (chorea gravidarum) and systemic lupus erythematosus.)

Athetosis. This consists of twisting and writhing movements of the extremities, trunk and sometimes face. It is most commonly seen in cerebral palsy where
prenatal or perinatal injury to the motor systems cortex or connections, leaves a fixed motor neurological deficit.

**Dystonia.** This consists of a more sustained abnormal postural movement. It can affect small or larger muscle groups. A common example of dystonia is **torticollis**, whereby the sternocleidomastoid and neck muscles pull the head over to one side.

**Hemiballismus.** This is a dramatic, and fortunately uncommon, movement disorder where an extremity has repetitive flailing movements similar to throwing a ball. These can persist indefinitely and sometimes endanger the person’s health via sheer exhaustion. The movement is usually caused by a lesion in the **subthalamic nucleus**, and commonly is caused by a small infarct.

**Speech** is also affected by these disorders and can be characterized as **hypokinetic** (extrapyramidal), or **hyperkinetic**. Hypokinetic speech is low in volume and pitch and may be affected by tremor. The patient sounds like he is mumbling, while whispering. Hyperkinetic speech on the other hand is irregular, explosive and erratic. Patients with cerebellar disorders have some of these characteristics affecting their speech also.

In a simplistic sense one can think of a smoothly functioning EPS as depending on equal interactions and levels of dopamine (DA) and acetylcholine (ACh). When DA is excessive, or ACh depleted, excessive movement or a hyperkinetic state results. The converse (decreased dopamine or increased ACh) leads to bradykinesia, rigidity and sometimes tremor (Figure 2-32). These observations form the basis for common pharmacologic treatment of these disorders. Hypokinetic states are treated by supplementing dopamine or adding dopamine agonists. Additionally one can block cholinergic receptors with anticholinergic drugs. Hyperkinetic states are treated by blocking dopamine receptors, i.e., with phenothiazines, risperidone, etc., or by depleting dopamine i.e., with reserpine, etc. Adding ACh has been tried but is not as effective.

**The Extrapyramidal System Examination**

Each portion of the neurological examination should be initiated by observation of the patient including gait, posture, and activities while sitting during the interview. With abnormalities of the EPS many of the previously described abnormalities will be noted on inspection.

The patient with Parkinson syndrome has a characteristic gait that is produced by his abnormal muscle tone (rigidity) and slowness (bradykinesia). The posture is characterized by increased flexor tone and he walks stooped forward. This puts the center of gravity in front of the patient. Slowness in initiating gait may cause the trunk to move forward first, and the patient winds up chasing his center of gravity. Rigidity only permits small steps producing a characteristic **festinating** gait.

![Figure 2-32: Biochemical Basis of Extrapyramidal disorders.](image-url)
At rest, a patient may demonstrate tremor, chorea, or dystonia. The latter two may be seen more readily with the arms outstretched. Dystonia often causes the supinated outstretched arm to pronate.

Rigidity may be appreciated by passive range of motion of the arms or legs of a patient. If the examiner puts his left thumb on the biceps tendon of the patients arm and actively flexes and extends that arm at the elbow with his right arm, he will feel increased resistance in both directions with his right arm (rigidity) and a ratcheting sensation with his left thumb (cogwheeling).

It is thus by inspection, palpation and observation that most extrapyramidal abnormalities are detected. There is usually no muscle weakness and no sensory loss. The abnormalities may be symmetric or asymmetric in their distribution. Parkinson’s disease or a movement disorder may also begin in a single extremity or part of an extremity, but is usually progressive.

Normal development and functioning of the EPS provides the truncal and extremity support for other activities such as individual extremity movement, walking, or even sitting. Abnormalities of this system disrupt the smooth and accurate functioning of this support matrix and leads to the postural and motion abnormalities described above. The voluntary motor system, which initiates individual volitional motor acts, depends on the support matrix of the EPS to carry out its activities.

Summary
- Disorders of the EPS usually present as motor activity which is either hypokinetic or hyperkinetic.
  - Hypokinetic disorders are characterized by rigidity, bradykinesia and tremor. The most common example is Parkinson’s syndrome. There is increased flexor tone, causing a stooped posture with the head, neck, trunk, arms and legs flexed. Tremor and slowness of movement are noted on inspection and cogwheel rigidity may be appreciated by passive range of motion of the extremities. The patient has a festinating gait but no motor weakness is evident.
  - Hyperkinetic disorders are distinguished by excessive motor activity and may take the form of chorea, athetosis, dystonia or hemiballismus. These abnormalities are due to neurotransmitter derangements, degenerative diseases or structural lesions, and are often defined by the clinical setting in which they occur.

The Pyramidal System (Video)

Our marionette is now standing with his trunk and head erect. In the human infant unconscious (indirect) motor systems are fully activated to maintain this posture and to provide the framework upon which other motor activity can occur. The pyramidal system, consisting of a cortical premotor, motor and spinal motor area, is what affects voluntary motor activity.

The infant looks up and is attracted by the objects on a mobile. He reaches for one and his arm extends forward in a nonsmooth, perhaps erratic fashion, in his attempt to grasp. Such early activity gives some insight into what unmodified pyramidal movements are like. This system initiates voluntary motor activity, but without the modifying effects of a mature EPS and cerebellar system, the activity is crude and poorly coordinated. It is, however, the basic foundation upon which
more sophisticated movements are developed. The motor cortex is located in the precentral gyrus of the posterior frontal lobe (face, hand, arm, trunk) and paracentral lobule (hip, leg, foot). Several layers of pyramidal neurons interconnect and give off long axons that travel through the hemispheric white matter (corona radiata) and converge in topographic fashion in the internal capsule (Figure 2-33). Fibers descend in the brain stem where they keep their topographic representation in the pyramidal tract. The cranial nerve fibers are medial and the leg fibers most lateral. Most of this pathway (80%) crosses to the opposite side in the pyramidal decussation, at the cervico-medullary junction region. Descending fibers now travel in the lateral corticospinal tract. Fibers are given off to anterior horn cells at each level. Fibers from the upper body are arranged more medially since they are given off first.

The pyramidal axons synapse on anterior horn cells, located in the anterior horn of the spinal cord. This is the final common pathway for initiation of voluntary activity. Impulses from pyramidal motor neurons initiate motor activity by stimulating anterior horn cells whose impulses, in turn, cause peripheral skeletal muscle fibers to contract and initiate joint motion.

It is in the premotor area of the cerebral cortex where connections are developed that act as programs for various motor activities. Repetition and practice help these connections develop and serve as templates for certain motor activities. As the infant continues to reach for the mobile he develops more dexterity, through maturation of this and other developing systems.

Similarly, stimulation of the motor cortex with electrical current induces crude uncoordinated movements, much like that of the infant. Previously we discussed how the cerebellum acts to modify motor activity and makes it smooth and coordinated. We will discuss specific functional areas of the cerebellum in the next section.

In experimental situations, production of a pure pyramidal tract lesion leads to flaccidity and hypotonia of effected muscles. In clinical situations most pyramidal lesions are not “pure” and involve premotor and extrapyramidal structures as well. As a result the clinical picture associated with pyramidal system lesions contains the following signs:

**Weakness.** The weakness associated with pyramidal tract lesions has a characteristic distribution.

**Face:** Only the lower facial muscles are affected since the upper facial nucleus
receives bilateral cortical innervation.

**Upper extremity:** The extensors are weaker than the flexors. Consequently the arm eventually assumes a flexed position. Therefore the biceps will be stronger than the triceps, the wrist and finger flexors stronger than the extensors. For this reason it is not a good idea to monitor for stroke progression by testing grip strength. Test finger extensor strength instead.

**Lower extremity:** Here the converse is true. The extensors remain stronger than the flexors. This has the beneficial effect of ultimately permitting standing and possibly walking. On examination the gluteus maximus is stronger than the iliopsoas, the quadriceps stronger than the hamstrings, and the gastrocnemius stronger than the anterior tibial muscle.

The most severely affected muscles are also the ones with the greatest cortical representation. The hand and fingers have a large area of cortical representation since they are capable of complicated and intricate movements. Besides weakness, lesions of these areas also cause clumsiness and loss of the ability to perform intricate movements. This is especially true of lesions in the premotor areas where patterning of movements is coordinated. As a result, even though the finger flexors may have mild weakness, the ability to manipulate small objects in the hand or to button a button may be seriously impaired.

Unlike lower motor neuron lesions, **muscle atrophy does not occur** with pyramidal tract lesions. The affected muscles may get a little smaller over the years, due to disuse, but true atrophy does not occur.

**Hyperreflexia.** The deep tendon reflexes are increased due to loss of inhibitory motor cortex influences on the anterior horn cell. Sometimes striking or suddenly stretching a muscle tendon will produce repetitive contractions of that muscle. This is called **clonus** and is a sign of pyramidal tract dysfunction. The abdominal reflexes are lost opposite the affected side. They are normally elicited by gently scratching the abdominal muscles in a supine patient, stroking each quadrant upwards or downwards and inwards towards the umbilicus. A normal response is a contraction of the scratched muscle.

In addition to deep tendon reflexes being increased, certain reflexes that are absent in normal individuals now appear. These are called **pathological reflexes.** Some pathological reflexes are:

**Babinski's sign:** The examiner scratches the patient's foot starting laterally at the heel, and moving up and crossing medially at the metatarsal head area. If Babinski's sign is present, the first toe extends and the others fan outward.

**Grasp reflex:** The patient's palm is rubbed with the examiner's fingers. If the motor system of the contralateral frontal lobe is involved, the patient will involuntarily grasp the examiner's fingers. The examiner's fingers may have to forcefully freed.

**Increased muscle tone:** In the acute phase of a pyramidal tract lesion muscle tone may be diminished and muscles are **hypotonic** to passive range of motion. Over days to weeks, however, muscle tone increases, albeit in a specific manner. The muscle that are stronger, as previously described (flexors in the arm and extensors in the leg), also have increased tone. This leads to two observable clinical findings.

**Posture.** The difference in tone causes the patient to keep his upper extremity
flexed, and his lower extremity extended. Gait now assumes a characteristic pattern. On the normal side the leg moves smoothly and the arm swings normally as the patient walks. On the affected side the leg has to swing outwards (circumduction) to clear the toes because increased extensor tone points the foot down and in. The affected arm stays flexed and does not swing with each step. This is the classic hemiparetic gait. As a result patients may fall more frequently due to catching the toes of the affected foot on uneven surfaces such as carpeting.

**Spasticity.** This is the clinical term for the alteration in muscle tone seen with pyramidal tract lesions. When the examiner flexes and extends the patients relaxed arm at a joint he will feel a resistance when he tries to overcome flexion (i.e., when he extends the arm). The resistance is at the beginning of the movement and then it diminishes. The sensation is like that of opening the blade of pocketknife where there is initial resistance to extending the blade. As a result this finding has been termed clasped-knife spasticity. It is best appreciated at the elbow and knee joints of affected extremities.

**Summary**

- Pyramidal tract lesions affect the lower face, arm extensors and leg flexors.
- Spasticity causes increased tone in arm flexors and leg extensors, with clasped-knife spasticity noted on passive range of motion.
- The patient walks with the affected arm flexed and leg extended (hemiparetic gait).
- There is hyperreflexia on the affected side.
- Pathological reflexes, such as Babinski's sign and the grasp reflex, may be noted on the affected side.
- Muscle atrophy does not occur in affected muscles.
- There is loss of fine coordinated activity in the affected limb.

This concludes the section on the pyramidal tract or upper motor neuron system. This is intended to be a broad overview and the student should use this as an adjunct along with individual instruction, video materials and clinical experience. These principals will be alluded to again in sections dealing with diseases specifically affecting this system.

**The Cerebellum and Coordination**

At this time our marionette is able to stand erect, and initiate motor activities, albeit crude ones. Our infant is able to stand erect and reach for the mobile also in an uncoordinated fashion. We discussed how the cerebellum, by comparing the intended activity to what is actually being achieved, as communicated by sensory receptors, was able to smooth out motor movements and make them more coordinated. In this fashion, and through learning by multiple repetitions, structural connections are developed between these interactive systems, which facilitate the performance of often-repeated acts, such as writing or tying shoelaces.

We mentioned that motor incoordination, or ataxia, can be sensory, motor or cerebellar in origin. If motor and sensory functions are intact, then involvement
of the cerebellar pathways is suspected. Having determined this, our next goal is to localize the lesion to a particular portion of the cerebellum. Cerebellar anatomy is quite complex; but in terms of clinical utilization, the cerebellum can be broken down to a few useful concepts (Figure 2-34).

We will divide cerebellar lesions into those that involve the midline structures, the anterior lobe, and the lateral hemispheres.

**Midline Structure Lesions**

A useful way to remember cerebellar functional anatomy is to think in terms of phylogeny. The first creatures to require a coordination system basically consisted of a trunk and head (fish and eels). This is, developmentally, the oldest portion of the cerebellum and is termed the archicerebellum. The portion of the cerebellum that controls head and trunk movements lies in the midline and consists of the vermis and flocculo-nodular lobe region. Of interest is that its shape is similar to the elongated trunk and head of the organism (Figure 2-35). Extremities, except for fins, have not evolved yet so the vermis is primarily concerned with truncal coordination. Lesions that affect the vermis produce truncal ataxia. An individual who has truncal ataxia is unable to sit up, stand or walk. The reason for this is that the trunk cannot be stabilized to maintain the upright position. Even when the patient is sitting up his trunk will sway and his head may seem to bobble on the shoulders (titubation). When lying down, however, the trunk is supported and there is no problem performing coordinated acts with the upper or lower extremities.

Cerebellar midline lesions are usually neoplastic and are most often seen in childhood. An example of such a lesion is the medulloblastoma, a primitive tumor that may develop near the roof of the fourth ventricle. In its early stages it exerts pressure on the flocculus and vermis. The mild degree of truncal ataxia...
that it induces may cause instability while running, consequently the history of the previously normal child who now has some falls while running. If the neurological exam is normal it is easy to dismiss this complaint or ascribe it to something like a “growth spurt.” It is only when the tumor has reached sufficient size to occlude the fourth ventricle or aqueduct of Sylvius, causing acute hydrocephalus, severe headache and projectile vomiting, that the seriousness of the condition is fully appreciated. At this time the child will need an emergency ventriculo-peritoneal shunt and then surgery to remove the tumor.

If one encounters a child with a similar history, early investigation with an MRI scan may demonstrate the lesion. MRI is the test of choice, since CT scans do not demonstrate the posterior fossa as well.

**Clinical Testing**

If the patient has truncal ataxia he will not be able to sit or stand unsupported. The patient is asked to sit on the edge of a bed or chair with the back unsupported and the arms folded across the chest. If truncal ataxia is present he will tend to fall over in any direction. The head may titubate upon the shoulders. If he is unable to sit without falling over, standing will be impossible as well. Truncal ataxia while standing must meet similar criteria and be unassociated with joint position sense loss, (no difference in ataxia with eyes open or closed), or motor weakness.

**Anterior Lobe Lesions**

The next major phylogenetic evolutionary step was that certain creatures, which previously only had trunks, became land dwellers. This necessitated the development of extremities ([Figure 2-36](#)). The first land dwellers had four extremities and a central nervous system structure (paleocerebellum) that was necessary to coordinate the synergistic movement of these extremities for the action of walking and running. This structure is the anterior lobe of the cerebellum and is located superior to the vermis. Lesions or other dysfunctions of the anterior lobe produce **gait ataxia**.

Of all forms of cerebellar ataxia, gait ataxia is the most common. The anterior lobe Purkinje cells, or main cerebellar neurons, are very sensitive to certain chemicals especially ethanol. Weekend alcohol consumption increases the incidence of cerebellar gait ataxia and enables police to perform mini-neurological exams on suspected offenders, i.e., by asking the person to walk

---

![Figure 2-36](#)  
**Figure 2-36:** Paleocerebellum: Controls the synergies of walking.
along a straight line and to tandem walk. Fortunately, this form of ataxia is reversible but may become permanent in chronic alcoholics.

Other toxins that affect the anterior lobe are drugs such as phenytoin, and other anticonvulsants. Toxic levels may induce gait ataxia and nystagmus.

Certain neoplasms may produce cerebellar ataxia in a poorly understood and perhaps autoimmune fashion. Some malignancies known to do this are small cell lung cancer, ovarian cancer and lymphoma. Certain anti-Purkinje cell antibodies can be elevated with this type of remote effect of cancer. Anti-Hu antibodies are seen with small cell lung cancer and anti-Yo antibodies with ovarian cancer. Although rare as causes of cerebellar ataxia, they should be thought of in any type of acquired cerebellar ataxia where no structural lesion exists, especially if the onset is acute or sub-acute.

**Clinical Testing**

The patient is able to sit or stand unsupported. If he lies on his back he can make normal pedaling motions with his legs and do heel-to-shin testing without any problems. This excludes the presence of truncal and individual extremity ataxia. If he attempts to walk however, he will stagger about and have to hold onto objects to prevent falls.

**Lateral Hemisphere Lesions**

A later stage of evolution sees the development of primates who can ambulate with their lower extremities and have opposable thumbs to grasp and manipulate objects. These intricate and complex movements require large areas of integrating neurons and are seen in the expanded cerebellar hemispheres *(neocerebellum)*. These have evolved with topographical representation of the extremities as seen in Figure 2-37. Included also are the related areas for eye movement and speech.

Afferent and efferent pathways cross on entering and leaving the cerebellum, therefore representation is ipsilateral. The right upper extremity is controlled by the right upper lobe, the right lower extremity by the right lower lobe, and so forth. Consequently a right upper lobe lesion would produce ataxia of the right upper extremity alone.

Cerebellar lesions produce loss of ability to be the servomechanism that coordinates movement. The abnormal movements thus generated, may be defective in rate, range, direction and force. The loss of coordination leads to a movement abnormality termed *dyssynergia*. Defects in range are termed *dysmetria*. The gross movements may have a coarse undulating quality during execution of the movement. This has been called *intention tremor*. Overall what we see is *limb ataxia*. The affected extremity usually has decreased muscle tone and diminished ability to correct and change direction rapidly. Thus there is defective
performance of rapid alternating movements of the hands, feet and fingers. Finger-to-nose testing will be performed less well with the affected upper extremity.

The lower extremities can be tested individually with rapid alternating movements such as foot tapping or with the heel-to-shin test. This test is performed with the patient supine. On the side being tested, the patient first puts his heel to the ipsilateral knee, and then rapidly slides it up and down the shin. The action should be smooth, and rapid with the heel staying on the shin. If a lower extremity develops ataxia there is some difficulty with ambulation, but is not as severe as gait ataxia secondary to an anterior lobe cerebellar lesion.

Lesions that produce lateral hemisphere dysfunction are usually primary tumors, metastases, infarcts, multiple sclerosis plaques or hemorrhages. Infarcts and hemorrhages are acute in onset and metastases and tumors have a more chronic temporal profile.

Central cerebellar lesions or toxic and degenerative disorders may affect speech and eye movements. Ataxic speech is usually explosive with erratic volume, rate and rhythm.

Cerebellar nystagmus is usually horizontal and most pronounced looking towards the lesion. There may be a null point (no nystagmus) somewhere past the midline in the opposite direction.

Emergency Situation
A patient presents to the emergency department with a history of sudden onset of headache and collapse. On examination he is drowsy but arousable and conversant. He has nausea and vomiting, hypertension and horizontal nystagmus. At this point, determine if the patient can sit or stand unsupported and whether he has any extremity ataxia. If he does, the presumptive diagnosis is cerebellar hemorrhage which may be life threatening. You should proceed to obtain an emergency CT scan and neurology/neurosurgical consultation.

Generalized, slowly developing ataxia may be seen in hereditary spino-cerebellar degenerations or with cumulative lesions such as occur with stroke or multiple sclerosis.

Summary
- Midline cerebellar lesions produce truncal ataxia, and can be seen in children with posterior fossa tumors.
- Anterior lobe cerebellar lesions produce gait ataxia and can be seen with neoplasms, infarcts, toxins, MS (multiple sclerosis) plaques and as a remote effect of cancer.
- Lateral hemisphere lesions produce individual extremity ataxia and are seen with infarcts, neoplasms, hemorrhage, and MS plaques.
- Generalized ataxia is seen with cerebellar degenerative disorders and remote effect of cancer.
- Trauma may produce any combination of the above depending on which portion of the cerebellum is involved.
The Peripheral Nervous System

The peripheral nervous system (PNS) contains the motor, sensory and autonomic nerve fibers that are traveling to, or exiting from, the spinal cord. Many peripheral nerves contain a mixture of the above different nerve types while some branches may be purely motor or sensory in composition.

Peripheral nerve fibers may contain an insulating coating of myelin, which is invested around the nerve cell by Schwann cells (Figure 2-38). This coating aids in axonal metabolism and enables more rapid conduction called saltatory conduction. The depolarization potential travels more rapidly by jumping from node to node. Large rapidly conducting fibers have thick myelin coats. These are the proprioceptive and motor fibers of peripheral nerves. Nerve fibers that subserve pain and temperature sensibility are unmyelinated or poorly myelinated. They conduct impulses more slowly.

Figure 2-38: Peripheral nerve axon and Schwann cell with myelin sheath.

Myelinated fibers are more prone to pressure injury and may be affected by pressure on bony prominences, e.g., the ulnar nerve at the elbow and the peroneal nerve at the knee.

Clinical symptoms of peripheral nerve disease include:

- Loss of feeling (numbness)
- Abnormal tingling sensations (paresthesias)
- Pain

Clinical signs include:

- Sensory loss
- Weakness
- Muscle atrophy
- Decreased muscle tone
- Diminished or absent deep tendon reflexes
- Distal paresthesias on tapping the lesion site (Tinel’s sign)

The characteristics of the above symptoms and signs depend on the extent and pattern of peripheral nerve involvement. Some clinical examples of peripheral nerve involvement include:
• **Mononeuropathy**: Only one peripheral nerve is affected.
• **Mononeuritis multiplex**: Multiple peripheral nerves are randomly affected.
• **Polyneuropathy** or **Peripheral neuropathy**: Symmetrical, distal greater than proximal, sensory, motor, autonomic, or combined nerve involvement.
• **Plexopathy**: Involvement of the nerve complexes in the retroperitoneal or brachial regions.
• **Radiculopathy**: Involvement of the nerve roots prior to exiting or entering the spinal cord. Roots contain all the nerve elements.

**Mononeuropathy**

Median neuropathy at the wrist (Carpal tunnel syndrome)
• Sensory loss as in Figure 2-25.
• Weakness of the abductor and opponens pollicis muscles.
• Thenar atrophy
• Pain in the forearm and arm.
• Tinel’s sign at the volar wrist.

Ulnar neuropathy at the elbow (leaning elbow excessively on a hard surface)
• Sensory loss as in Figure 2-26.
• Weakness of the interossei, and hypothenar muscles.
• Hypothenar and interosseous atrophy.
• Elbow, forearm and arm pain
• Tinel’s sign at the elbow.

Radial neuropathy at the upper arm, i.e., radial groove (usually caused by pressure from leaning or lying on this area for prolonged periods after excessive alcohol or sedative usage)
• Sensory loss on the dorsum of the hand.
• Weakness of wrist and finger extensors. (wrist drop).
• Wasting of dorsal forearm muscles.
• Arm pain and radial groove tenderness.
• Tinel’s sign at the radial groove.
• Diminished brachioradialis reflex.

**Ermoral neuropathy** (may be seen with diabetes or surgical trauma during pelvic surgery)
• Sensory loss on the anterior thigh.
• Weakness of the iliopsoas and quadriceps muscle (thigh buckles with weight bearing).
• Quadriceps atrophy
• Thigh pain
• Diminished knee reflex

**Sciatic Neuropathy** (usually due to pressure or trauma at the sciatic notch)
• Sensory loss below the knee
• Severe distal leg pain
• Weakness of thigh flexors and all muscles below the knee.
• Absent Achilles (ankle) reflex

Peroneal Neuropathy, i.e., deep peroneal nerve, (usually due to pressure from leg crossing or trauma at the fibular head)
• Minimal sensory loss (web space between 1st and 2nd toe).
• Weakness of the anterior tibial, peronei and toe extensor muscles. The posterior tibial muscle (inversion of the foot) is spared.
• Usually not painful.
• Tinel’s sign at the fibular head.

Mononeuritis Multiplex
This is due to multiple peripheral nerve lesions. They can occur sequentially and in a random fashion. Any combination of the above nerves or other peripheral nerves can be encountered. The lesions are usually secondary to nerve infarcts due to disease of the vasa vasorum which can be seen in diabetes or vasculitis.

Peripheral Neuropathy
Symmetric, distal greater than proximal, involvement of sensory, motor or autonomic nerves. Numbness or weakness usually starts in the toes and moves proximally, eventually involving the upper extremities. Peripheral neuropathy (PN) is usually due to systemic disease or toxins that affect the Schwann cell or nerve cell body. Metabolic demands on the cell body and axon, or larger number of Schwann cells affected on longer nerves, causes the longest nerves to be more severely affected, thus symptoms begin distally in the toes. The symptoms and signs of peripheral neuropathy therefore involve: symmetric distal weakness; symmetric distal sensory loss and symmetric diminished deep tendon reflexes.

Plexopathy

Brachial plexopathy

<table>
<thead>
<tr>
<th>Upper Extremity:</th>
<th>C-5 and C-6.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>• Weakness of the deltoid, infraspinatus, biceps and brachioradialis.</td>
</tr>
<tr>
<td></td>
<td>• Diminished biceps and brachioradialis reflexes.</td>
</tr>
<tr>
<td></td>
<td>• Sensory symptoms or loss in deltoid area or thumb.</td>
</tr>
<tr>
<td>C-7</td>
<td>• Weakness of the triceps, pronator teres, wrist and finger extensor muscles.</td>
</tr>
<tr>
<td></td>
<td>• Diminished triceps reflex.</td>
</tr>
<tr>
<td></td>
<td>• Sensory symptoms or loss in the middle finger.</td>
</tr>
<tr>
<td>C-8</td>
<td>• Weakness of the wrist flexors and intrinsic hand muscles (median and ulnar).</td>
</tr>
<tr>
<td></td>
<td>• Diminished triceps and finger flexor reflex.</td>
</tr>
<tr>
<td></td>
<td>• Sensory symptoms or loss in the hand (fifth finger).</td>
</tr>
</tbody>
</table>
### Lower Extremity:

<table>
<thead>
<tr>
<th>Level</th>
<th>Symptoms/Reflexes</th>
</tr>
</thead>
</table>
| L-3   | Weakness of the iliopsoas, quadriceps and adductor muscles.  
|       | Sensory symptoms or loss on the anterior thigh.  
|       | Diminished or absent knee reflex.  
| L-5   | Weakness of the anterior tibial, peronei, posterior tibial, and toe extensor muscles.  
|       | Sensory symptoms or loss on the dorsum of the foot and great toe.  
|       | Diminished or absent internal hamstring reflex.  
|       | Pain on straight leg raising.  
| S-1   | Weakness of the gastrocnemius (can’t walk on toes on affected side) and toe flexor muscles.  
|       | Sensory symptoms or loss on sole of foot.  
|       | Diminished or absent Achilles (ankle) reflex.  
|       | Pain on straight leg raising.  

### Reflex Testing

Brisk or diminished deep tendon reflexes often give clues to the presence of disease of the upper or lower motor neuron or sensory portion of the reflex arc. In isolation, however, increased or decreased reflexes may not be pathological. Normal individuals can normally have exaggerated or diminished reflexes. It is the associated clinical findings, which support the presence of a pathological state. Brisk reflexes, associated with spasticity on passive range of motion and upper motor neuron pattern weakness, are abnormal.

Reflexes are tested by stretching the tendon with a brisk tap of a reflex hammer and then observing a contraction of the associated muscle. Technique will be demonstrated on the video portion of the syllabus.

### Deep Tendon Reflexes

- **Biceps Tendon:** C-5, C-6.  
  ![Figure 2-39](image-url): Biceps reflex.

- **Triceps Tendon:** C-7  
  ![Figure 2-40](image-url): Triceps reflex.
Brachioradialis Tendon: C-6. **Figure 2-41**

Finger Flexor Tendons: C-8, T-1. **Figure 2-42**

![Figure 2-41: Brachioradialis reflex.](image1)

![Figure 2-42: Finger Flexor Tendons.](image2)

Quadriceps (Knee) Reflex: L-3, L-4. **Figure 2-43**

Internal Hamstring Reflex: L-5, S-1. **Figure 2-44**

Gastrocnemius (Ankle) Reflex: S-1, S-2. **Figure 2-45**

![Figure 2-43: Quadriceps (Knee) reflex.](image3)

![Figure 2-44: Internal Hamstring reflex.](image4)

![Figure 2-45: Gastrocnemius (Ankle) reflex.](image5)

In testing reflexes multiple samples of the reflex should be made to establish its reactivity. Look for asymmetry by testing the reflex on one side and then the same reflex on the other and continue comparing both sides in succession. The patient should be completely relaxed during testing or the results may not be valid.