Neurological Examination of Sensation, Reflexes and Motor Function (Dr. Merchut)

Sensation

1. Definitions and terminology

There are three types of **primary or basic sensation. Exteroceptive sensation** refers to external stimuli, typically light touch, pain, and temperature, detected by various receptors in the skin. **Proprioceptive sensation** refers to stimuli from muscles, tendons, ligaments and joints, in relation to position and movement of the body, limbs and digits, which is important for balance and coordination. **Interoceptive sensation** refers to internal stimuli affecting visceral organs, such as the perception of a distended bladder, or pain from an ulcerated stomach.

Cortical or combined sensation involves the simultaneous perception of several basic stimuli, further integrated and interpreted at the cortical level. For example, a small object could be recognized in the dark solely by its combined tactile characteristics of texture, shape, weight, temperature, and so on.

The special or unique sensations of vision, hearing, taste, and smell involve receptors and pathways that are anatomically localized to specific parts of the body. The clinical evaluation of these special sensations will be discussed elsewhere.

2. Clinical evaluation of patients

At this point, a few introductory comments about the neurological examination are needed. The bedside history and physical is concerned with **symptoms**, the subjective complaints of the patient, and **signs**, the objective abnormalities found on the physical examination. Signs and symptoms are then related to one, or perhaps a few, **lesions** or anatomical sites of abnormality in the nervous system. Initially, the signs and symptoms may appear as a confusing array, which cannot be attributed or related to a single anatomical area. After a closer look, armed with knowledge of neuroanatomy, a unique, single lesion, or "short list" of possible lesions, may become apparent. Try the approach of "Ockham's razor," attributed to Father William of Ockham, a Franciscan friar in the 14th-century, who wrote that "entities must not be multiplied beyond necessity." Some "classical" lesions or syndromes described in this course may be encountered infrequently in clinical practice, yet serve as worthy examples of how unusual groups of signs and symptoms can be related to just one lesion.

This neuroanatomical localization, along with other historical clues (severity and tempo of symptoms, relevant family history, occupational exposure, precipitating events such as trauma) suggests the **etiology**, or cause, of the patient's problem. A **differential diagnosis**, or list of possible diseases which may cause the patient's symptoms, can be narrowed down further by appropriate diagnostic testing. Recognition of a specific disease then allows for its specific treatment. With this practical goal in mind, the "intellectual process" or "mental game" of localization is not just an academic exercise, but a practical approach in clinical neurology. All experienced, skilled neurologists and neurosurgeons know that the history and physical is the most helpful and important part of this process.

3. Clinical evaluation of exteroceptive sensation

Patients may report a lack of sensation over part of the body, or may be unaware of the deficit. With various lesions of the sensory system, but particularly with peripheral nerve disorders, they may complain of **paresthesia or dysesthesia. Paresthesia** is an abnormal, spontaneous sensation, not provoked by stimuli, often described as tingling, or "pins and needles." **Dysesthesia** is an uncomfortable, at times painful, hypersensitivity to non-noxious stimuli and may be elicited when examining the patient.

The sensory examination consists of applying typical test stimuli to the patient's skin, while he or she, with eyes closed, tries to identify the type of stimulus correctly, and whether the stimulus is consistently decreased or absent over certain areas. **Light touch** may be tested with a wisp of cotton or light stroke of the finger. A blunted safety pin or broken cotton-swab stick may be used to test **pain (which is often referred to as "pin" or "pinprick" sensation)**. A cool metallic object (edge of the tuning fork) or tube of warm or cool water may be used to test **temperature** sensation. The examiner applies such test stimuli often at the distal lower limbs, alternating from left to right side, and moving proximally. The process is repeated for the upper limbs, and trunk or face.

Vibration sensation is assessed by applying a vibrating 128 hertz tuning fork to a bony structure such as the ankle or knuckle. The patient, with eyes closed, reports when the vibration is gone. Vibration sensation is abnormally decreased at the ankle or knuckle if the tuning fork is still perceived to vibrate more proximally at the patient's knee or elbow, or at the same bony site in the examiner. The latter comparison of patient to examiner is less valid if one person is much older, since vibration sensation normally declines with age.

4. Clinical evaluation of proprioception (proprioceptive sensation)

Proprioceptive sensation is also called **position sense** or joint sense. Testing is performed by raising or lowering the patient's finger or toe subtly a few degrees at one joint (perhaps easiest at the metacarpophalangeal or metatarsophalangeal joints). The patient, with eyes closed, identifies the movement as "up" or "down." If the patient detects only large excursions of the finger or toe, but consistently misses smaller movements, position sense is decreased there. If even large joint movements are not perceived, position sense is then absent. In that case, position sense may be preserved more proximally, which can be noted by testing more proximal joints such as the ankle or wrist.

5. Clinical evaluation of cortical or combined sensation

These sensory modalities involve object recognition (**gnosis**) as well as perception of objects or more complex stimuli. A lesion in the parietal sensory cortex or its connecting pathways produces a cortical sensory deficit in the contralateral body, while primary sensations may be relatively intact. **Stereognosis** refers to the tactile recognition of familiar or common objects, such as a penny or paper clip placed in the palm, with the patient's eyes closed. A deficit here may be referred to as astereognosis. **Graphesthesia**

involves the identification of numbers traced on the palm with the patient's eyes closed. A deficit of the latter is termed agraphesthesia or graphanesthesia. **Double simultaneous stimulation** refers to the ability to perceive two tactile stimuli applied simultaneously to the same bilateral parts of the body, such as both hands or both feet, again with the patient's eyes closed. When bilateral tactile stimuli are given, the consistent failure to detect a stimulus on one side is due to a contralateral parietal cortical lesion, and is described as **extinction on double simultaneous stimulation. Two-point discrimination** (also called **fine touch**) is the ability to detect the simultaneous application of two sharp points separated by a minimal distance on the skin. A deficit here consists of perceiving the two points as one point, or failing to feel it at all. The stimulus (special calipers, or an unfolded paper clip) should be perceived as two distinct points when only 3-4 mm apart at the fingertip, 20-30 mm apart at the dorsal hand and at greater distances of separation over the limbs and trunk. Although conveyed by the posterior columns, two-point discrimination is usually considered a cortical sensation.

6. Anatomical localization of sensory deficits

In disorders of peripheral nerves, initial involvement of the larger, more myelinated sensory fibers causes impairment of position sense and vibration, while initial involvement of the smaller, less myelinated or unmyelinated sensory fibers produces early impairment of temperature and pain (or pin) sensation. Eventually, if the peripheral neuropathy becomes extensive and severe, all fibers and all sensory modalities will be impaired.

If lesions exist in specific sensory pathways in the spinal cord or brainstem, certain sensory modalities may be lost while others are preserved. **Lesions of the posterior or dorsal columns** lead to deficits in position sense, vibration, and two-point discrimination. Although generally regarded as a primary or basic exteroceptive sensation, vibration sense is conveyed mainly via the posterior (dorsal) columns, and thus is clinically associated with the proprioceptive sensation of position sense. Isolated deficits of two-point discrimination have been associated with contralateral sensory (parietal) cortex lesions. **Lesions of the spinothalamic tract** create deficits in pain (pin) sensation and temperature. More than one spinal cord pathway conveys the sensation of light touch, so absence of light touch sensation occurs only in extensive lesions of the spinal cord or its dorsal roots (or in severe peripheral neuropathy or thalamic lesions).

It is not only the type of sensory loss that is important for localization of a lesion, but also where the deficit maps out on the patient's body. In the case of a **mononeuropathy,** sensation is decreased or lost in the territory of one peripheral nerve (Fig. 1). If there is a **polyneuropathy (peripheral neuropathy),** sensation is decreased or lost in several peripheral nerves, creating a **"stocking and glove"** distal pattern of deficit. **Dermatomal deficits** (Table 1) are sensory impairments in the territory of one or more dermatomes from one or multiple root lesions (Fig. 1). In the case of a single dermatomal lesion, a sensory deficit may not be clearly detected on examination because of the normal overlap of dermatomal sensory territories. The patient's own reported localization of sensory abnormality, paresthesia or dysesthesia is often more helpful in that situation. In **spinal cord lesions (myelopathies),** dissociation of sensation is characteristic (loss of one modality of sensation with preservation of another), although

this may also occur in brain stem lesions. **Intramedullary spinal cord lesions** occur **within** the spinal cord parenchyma, causing a **suspended** or vestlike sensory loss and **sacral** (dermatome) **sparing** of sensory deficit. **Extramedullary spinal cord lesions** compress the spinal cord from outside, creating an initial sensory loss in sacral segments, progressing up "to a level" because of lamination of the spinothalamic tract. A **hemisensory (hemibody)** deficit of basic sensations on the right or left side of the body including the face is caused by a contralateral **thalamic** lesion, or involvement of sensory pathways to the contralateral parietal lobe. Isolated or predominant deficits involving **cortical or combined sensation** typically occur on one side of the body, and are usually due to a lesion in the contralateral parietal sensory cortex.

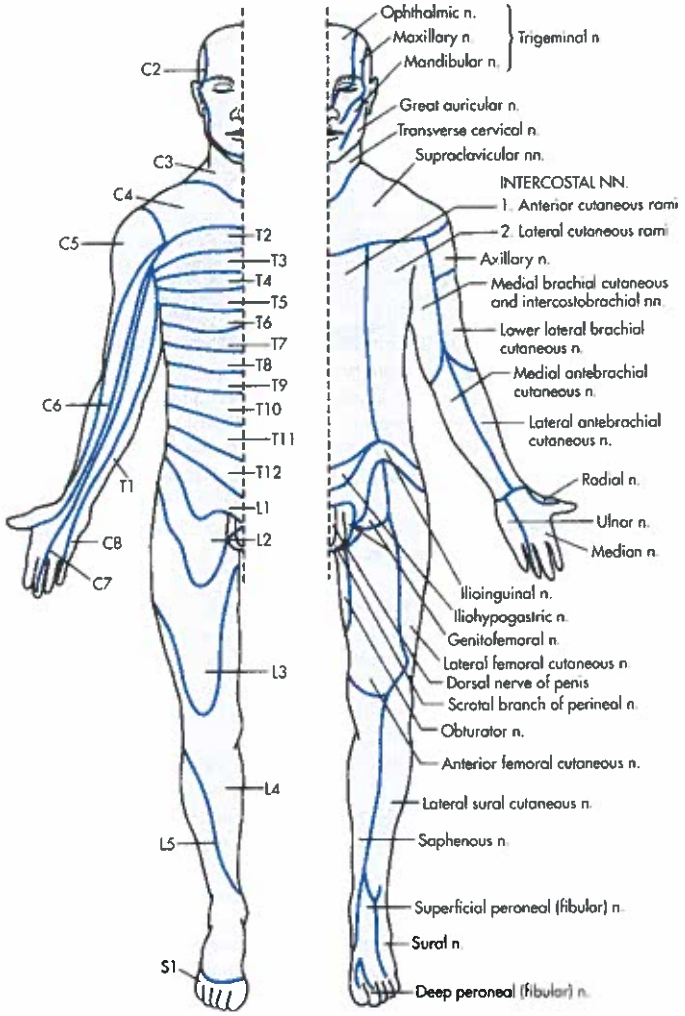

Fig. 1 Sensory territories for dermatomes and peripheral nerves (from Gilman S, Newman SW. Manter and Gatz's Essentials of Clinical Neuroanatomy and Neurophysiology. 9th ed. Philadelphia: FA Davis, 1996)

Table 1 Important Dermatomal Landmarks	
DERMATOME	BODY LANDMARK
C5	Lateral shoulder
C6	Thumb
C7	Index/middle fingers
C8,T1	Ring/little fingers
T4	Nipple
T10	Umbilicus
L3,4	Anterior thigh
L5	Dorsal foot
S1	Lateral foot/sole

Interoceptive sensation typically involves visceral pain, caused by an inflamed internal organ, as in appendicitis, or a sense of fullness or pressure, as from a distended bowel or bladder. It is poorly localized at times, in comparison to other modalities, except for the scenario of **referred pain. Referred pain** is perceived along a dermatome having sensory afferents from the same dorsal root level as the diseased internal organ. A heart attack may be heralded by pain along the inside of the left arm and forearm (C8, T1 dermatomes), while an infection below the right diaphragm may cause pain at the right shoulder (C3,4,5 dermatomes).

Reflexes

1. Definitions and terminology

A **reflex** is a quick, automatic, replicable motor response or muscle contraction provoked by a stimulus. In neurological disease, normal physiologic reflexes may be increased, decreased or lost, and abnormal pathologic reflexes may appear, especially with upper motor neuron lesions. Clinically important reflexes include **muscle stretch reflexes** (also called deep tendon reflexes, or commonly referred to just as "reflexes") and **superficial reflexes**.

2. Muscle stretch reflexes

Muscle stretch reflexes (MSRs) are elicited by the hammer tap of a selected tendon, which causes a brief or single contraction of its muscle. The commonly tested MSRs are listed in Table 2. The tendon tap causes passive stretching of its muscle and

neuromuscular spindles, which activates Ia sensory nerve fibers (**afferent reflex arc**), with subsequent depolarization of the alpha motor neurons (anterior horn cells) at that root level of the spinal cord. This depolarization of motor nerves leads to the contraction of muscle fibers and a visible muscle twitch (**efferent reflex arc**). Sometimes when a muscle stretch reflex cannot be elicited, it may be due to an anxious patient who is subconsciously tensing muscles. In that case, the examiner should perform a reinforcement maneuver to "distract" the patient and obtain the MSR. With the **Jendrassik maneuver,** the patient is asked to hook together his flexed fingers, attempting to pull them apart, while the examiner taps the tendons at the knee or ankle. Other reinforcement maneuvers may be used for obtaining upper limb reflexes, such as clenching the jaw. These maneuvers may actually lessen supraspinal inhibition of the reflex arc, rather than merely serving as a "mental distraction" for a tense, nervous patient.

Table 2 Muscle Stretch Reflexes	
Biceps	C5,6
Brachioradialis (radial)	C5,6
Triceps	C7,8
Finger flexors	C8,T1
Quadriceps (patellar, knee jerk)	L2,L3,L4
Achilles (ankle Jerk)	S1,2

MSRs are clinically graded on a scale of 0 to 4 (Table 3). An absent reflex despite reinforcement maneuvers is graded 0, while a reflex elicited only with reinforcement maneuvers is graded 1. An abnormally brisk reflex grade of 4 occurs when repetitive muscle contractions persist while the tendon is stretched. This is called **clonus,** and is best demonstrated at the Achilles' tendon (Fig. 2).

Table 3 Grading of reflexes (0-4)	
Grade	Response
0	Reflex absent despite reinforcement
1	Reflex present only with reinforcement
2	Average reflex
3	Very brisk reflex without clonus
4	Reflex followed by repetitive jerking movements (clonus)

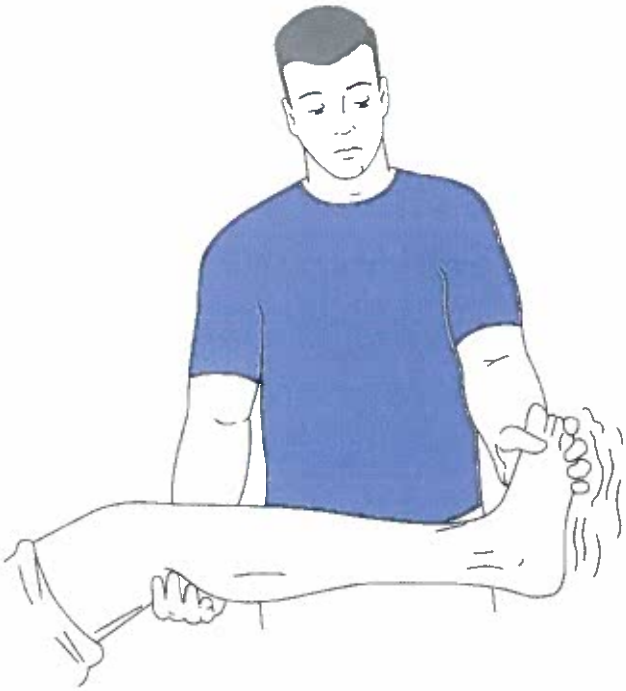

Fig. 2 Ankle clonus (repetitive jerks of plantar flexion as examiner maintains stretch on the Achilles' tendon)

3. Abnormalities of muscle stretch reflexes

Disorders disrupting the afferent or efferent reflex arcs may cause MSRs to be decreased (**hyporeflexia**) or absent (**areflexia**), which often occurs in polyneuropathy or radiculopathy. However, healthy older adults may have absent ankle reflexes merely due to aging. Abnormally increased MSRs (**hyperreflexia**) occur with upper motor neuron lesions, where the inhibitory effect on the local reflex circuit from descending supraspinal tracts is lessened. Besides the grading of individual reflexes, another significant abnormality is whether or not MSRs are symmetrical between the left and right sides. **Asymmetrical hyperreflexia** on one side of the body strongly suggests an upper motor neuron (corticospinal tract) lesion.

4. Superficial reflexes

Most **superficial reflexes** are cutaneous reflexes, provoked by tactile stimuli to a localized area of skin or mucous membrane. An exception is the pupillary light reflex, where stimulation involves shining light into the pupil of the eye. The **cranial nerve--mediated superficial reflexes** are characteristically **consensual**, with a bilateral response to a unilateral stimulus: touching one cornea produces a bilateral blink for the corneal reflex. The afferent and efferent reflex arcs consist of different cranial nerves (Table 4).

Table 4 Cranial Nerve Superficial Reflexes		
REFLEX	UNILATERAL STIMULUS (AFFERENT NERVE)	BILATERAL REFLEX (EFFERENT NERVE)
Pupillary	Shine light (CN II)	Pupils constrict (CN III)
Corneal	Touch cornea (CN V)	Eyes blink (CN VII)
Palpebral	Touch eyelid/lash (CN V)	Eyes blink (CN VII)
Gag (pharyngeal)	Touch pharynx (CN IX)	Gag (CN X)

Other cutaneous reflexes do not involve cranial nerves and may be diminished or absent from an interrupted reflex arc at the lower motor neuron level or from an upper motor neuron (corticospinal tract) lesion. The **abdominal reflex** consists of stroking the skin over each abdominal quadrant, whereby local muscle contraction causes retraction or deviation of the umbilicus toward the stimulus (reflex arcs at T7 through T12 and upper lumbar spinal cord segments). The abdominal reflex may be difficult to assess in obese patients or those with scars from abdominal surgery. The **cremasteric reflex** consists of stroking up the inner thigh, eliciting ipsilateral elevation of the testicle (reflex arcs at L1,2 spinal cord segments).

5. Pathological (abnormal) reflexes

The most important and reliable pathological reflex is the **Babinski sign,** which **indicates an upper motor neuron (corticospinal tract) lesion in the adult**. In infants, however, supraspinal tracts may take 1-2 years to normally myelinate, so the presence of a Babinski sign is not abnormal. The stimulus consists of stroking the lateral sole, from the heel to the ball of the foot (Fig. 3), causing slow dorsiflexion of the great toe (and less importantly fanning of the little toes). The Babinski sign notably is not a monosynaptic muscle stretch reflex. Other stimuli may also elicit "an upgoing toe," and these signs are named after those who first reported them. The Chaddock sign is elicited by stroking around the lateral ankle, then down the dorsolateral foot, and is a useful alternative to the Babinski sign in patients with ticklish feet.

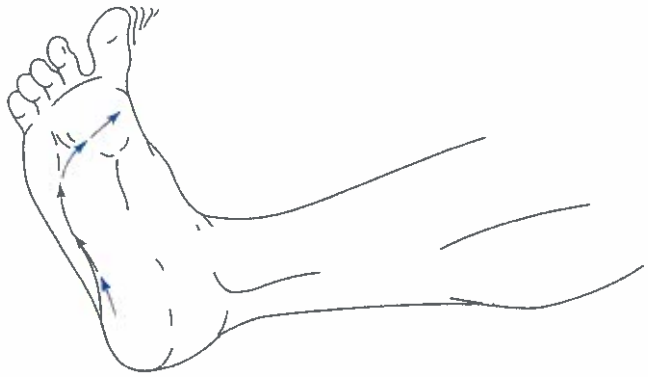

Fig. 3 The Babinski sign (stimulus applied in direction of the arrows)

There is no equivalent of the Babinski sign in the upper limbs. Digit flexor responses are the next best sign suggestive of an upper motor neuron lesion there. The examiner lifts or supports the patient's proximal middle finger, and then flicks its distal phalanx downwards (**Hoffman's sign**) or upwards (**Tromner's sign**), eliciting flexion of the fingers of that hand. This response, however, appears to involve the muscle stretch reflex of the finger flexors, which one would expect to be increased anyway in the presence of an upper motor neuron lesion.

6. Miscellaneous clinical signs

Meningeal signs are noted when the meninges are inflamed and irritated from infection or subarachnoid hemorrhage. Nuchal rigidity is the stiffness felt by the examiner when the patient's head is passively flexed in the anterior direction. **Kernig's sign** is present when the examiner feels resistance while attempting to fully extend the patient's knee with the hip in 90 degrees of flexion. **Brudzinski's sign** is present when the patient's hips and knees flex after the examiner passively flexes the neck.

Signs suggestive of nerve **root irritation or compression** include **Lasegue's sign,** the replication of radicular pain when the patient's hip is passively flexed with the knee in extension (straight leg raise maneuver).

Motor Function

1. Clinical evaluation of strength

Observation of the patient's gross body movements "in action" may reveal weakness, such as the dragging of a lower limb when walking across the room or standing up from a chair. The presence of a **pronator drift** suggests a subtle proximal upper limb weakness from a corticospinal tract lesion. The examiner would see slow pronation (palm turning towards the floor) and downward drift of an outstretched, supinated arm. Subtle weakness of the hand may be manifest more as impaired finger dexterity rather than gross weakness of certain muscles. The strength of individual

muscles can be graded on a scale of 0 to 5 (Table 5). As an example, a patient with a weak deltoid muscle may be able to abduct the arm only when lying supine in bed, since the arm is not abducted against gravity, but only moves in the horizontal plane of the bed: this would be graded as 2 out of 5. If the arm could be barely abducted against gravity, as when seated, but not offer much resistance to the examiner's effort, it would be graded as 3 out of 5.

Table 5 Grading of strength

GRADE	MUSCLE MOVEMENT
0	No movement
1	Flicker of movement
2	Movement only if gravity eliminated
3	Movement only against gravity
4	Movement against partial resistance
5	Movement against full resistance

2. Patterns of weakness

Focal weakness may occur in the territory of one peripheral nerve, or segmentally, such as the thigh or hand, where the weak muscles are innervated by more than one nerve or spinal root. The weakness of one limb may be partial **(monoparesis)** or complete **(monoplegia). Myelopathic** weakness, from a bilateral spinal cord lesion, may involve both lower limbs, from a lesion at the thoracic level. This lower limb weakness may be partial **(paraparesis)** or complete **(paraplegia).** All four limbs may be weak from a cervical spinal cord lesion, whether partial **(quadriparesis)** or complete **(quadriplegia).** Bilateral upper motor neuron lesions in the brain or brain stem could also cause paraparesis (or paraplegia) or quadriparesis (or quadriplegia). A **hemiparetic** (partial) or **hemiplegic** (complete) weakness of the upper and lower limbs on one side is typically due to an upper motor neuron lesion in the ipsilateral spinal cord or contralateral brain or brain stem. In a patient with muscle disease, the typical **myopathic** pattern of weakness involves the **proximal** limbs, namely at the shoulders and hips. In a patient with a peripheral neuropathy (polyneuropathy), the **neuropathic** pattern of weakness is **distal**, involving the feet, and later the hands.

3. Muscle tone

Muscle tone clinically is the resistance felt by the examiner when passively moving a patient's limb. Tone may be increased, **hypertonicity,** or decreased, **hypotonicity.** There are two types of hypertonicity: spasticity and rigidity. The increased tone in **spasticity** is unequal between agonist and antagonist muscles. This increased tone is especially increased in antigravity muscles (upper limb flexors, lower

limb extensors), which may allow patients the ability to stand and walk again. The perceived resistance varies as the limb is moved passively (**clasp-knife spasticity**). (Analogous to opening a clasp-knife or pocket-knife, there is more resistance when first pulling the blade out of the handle, and then the motion gets easier.). **Spasticity indicates an upper motor neuron lesion involving the pyramidal or corticospinal tract.** Loss or impairment of some descending motor pathways to the spinal cord presumably reduces some of the inhibition at the spinal cord level, leading to a relative overactivity of the gamma motor neurons. The gamma motor neurons tend to activate muscle spindles by shortening their intrafusal muscle fibers, making extrafusal muscle fibers very sensitive to stretch, thus creating the clinical signs of spasticity and hyperreflexia. However, clinical upper motor neuron lesions in patients vary from the precise upper motor neuron lesions seen in animal models.

Rigidity is the type of hypertonicity where the increased tone feels equal between agonist and antagonist muscles, so resistance feels constant as the limb is moved passively (**lead pipe rigidity**). (Analogous to bending a lead pipe, the resistance is constant throughout the task.). Rigidity is from a lesion in the **extrapyramidal** system. If tremor is present, passive limb movements produce a ratchety feeling (**cogwheel rigidity,** analogous to the jerky turning of a cogwheel inside a clock).

Hypotonicity is decreased muscle tone, usually from an afferent sensory or lower motor neuron lesion that interrupts the reflex arc of the muscle stretch reflex relevant to the tested limb. It has also been described in cerebellar disease, but may be difficult to observe clinically.

4. Muscle atrophy

Decreased bulk or wasting of a muscle indicates **muscle atrophy**, which is most prominent or severe in lower motor neuron lesions or myopathies (muscle disease). Milder degrees of atrophy occur in upper motor neuron lesions, or from disuse (actually "non use") of a limb, as in a patient confined to bed for other medical reasons.

Other signs of muscle denervation from a lower motor neuron lesion include fasciculations and fibrillations. A **fasciculation** is the grossly observable, spontaneous twitch of a group of muscle fibers innervated by a single lower motor neuron. More precisely, this is a spontaneous discharge of a **motor unit,** which consists of the lower motor neuron, its axon, and all the muscle fibers it controls. A **fibrillation** is the spontaneous twitch of an individual muscle fiber, which is visible only in the "naked" muscles of the tongue, not in limb muscles. Fibrillations always indicate denervation, while fasciculations occur with denervation as well as in benign conditions such as muscle fatigue. Generally, fasciculations are considered a serious sign of underlying neurological disease if other abnormal signs are present as well.

5. Upper versus lower motor neuron lesions

Upper or lower motor neuron lesions are the clinical syndromes which consist of distinct findings or signs pertaining to patterns and severity of weakness, changes in muscle tone or muscle stretch reflexes, and the presence or absence of fasciculations or pathological reflexes (Table 6). A precise, surgical lesion of the upper motor neurons or

corticospinal tract in an animal model may produce different deficits. Nonetheless, the presence of these findings in patients correlates very well with localization to an upper or lower motor neuron lesion, and is an **important concept for clinical neurology**. One scenario is an important exception to this clinical rule: **spinal or neurogenic shock**. In a sudden, severe spinal cord injury, typically from trauma, the expected upper motor neuron clinical signs are initially absent, only to gradually emerge days or weeks later. Thus, in spinal shock, paralysis is initially accompanied by diffuse hypotonia and areflexia. The latter findings may occasionally be seen also in acutely severe or extensive lesions of the brain or brainstem, as in a massive stroke.

Table 6 Clinical Signs of Motor Neuron Lesions

SIGN	UPPER MOTOR NEURON LESION	LOWER MOTOR NEURON LESION
Weakness	More diffuse	More focal
Atrophy	Mild, general	Severe, focal
Atrophy versus weakness	Severe weakness with relatively mild atrophy	Severe atrophy with milder weakness
Fasciculations	Never seen	May be present
Muscle tone	Increased (spasticity)*	Decreased
Muscle stretch reflexes	Increased*	Decreased to absent
Clonus	May be present*	Never present
Pathological reflexes (Babinski sign)	May be present*	Absent

*(except in spinal or neurogenic shock)