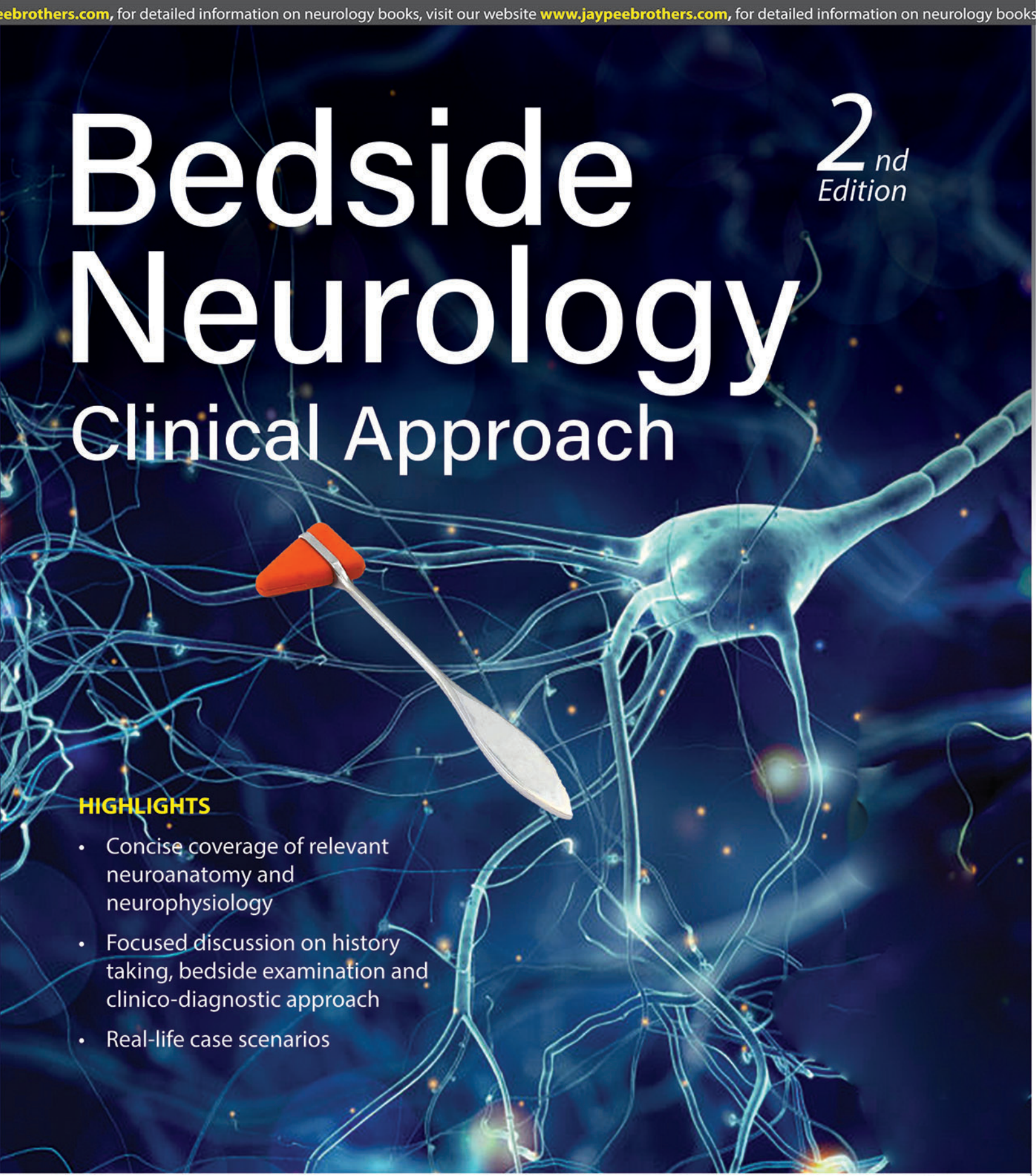
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Bedside Neurology

2nd Edition

Clinical Approach



HIGHLIGHTS

- Concise coverage of relevant neuroanatomy and neurophysiology
- Focused discussion on history taking, bedside examination and clinico-diagnostic approach
- Real-life case scenarios

Biman Kanti Ray

Foreword
Padma Srivastava

JAYPEE

Contents

CHAPTER 1	**Overview of Neurological Examination** *Biman Kanti Ray, Souvik Dubey*	1
CHAPTER 2	**Approach to Motor System Disorders** *Biman Kanti Ray*	2
CHAPTER 3	**Approach to Peripheral Nerve Disease** *Biman Kanti Ray, Adrija Ray*	40
CHAPTER 4	**Approach to Bladder, Bowel, and Sexual Dysfunction** *Biman Kanti Ray*	51
CHAPTER 5	**Approach to Paraparesis** *Biman Kanti Ray*	59
CHAPTER 6	**Approach to Quadriparesis** *Biman Kanti Ray*	73
CHAPTER 7	**Approach to Hemiparesis** *Biman Kanti Ray*	80
CHAPTER 8	**Approach to Monoparesis** *Biman Kanti Ray*	84
CHAPTER 9	**Approach to Cranial Neuropathies** *Biman Kanti Ray*	86
CHAPTER 10	**Approach to Ataxia** *Biman Kanti Ray*	145
CHAPTER 11	**Approach to Parkinsonism** *Adreesh Mukherjee*	158
CHAPTER 12	**Approach to Hyperkinetic Movement Disorders** *Adreesh Mukherjee*	167
CHAPTER 13	**Approach to Gait Disorders** *Biman Kanti Ray*	173
CHAPTER 14	**Approach to Dysphagia** *Biman Kanti Ray*	178

CHAPTER 15 **Approach to Common Neurological Disorders (Headache, Low Back Pain, Seizure, and Unconsciousness)** 180
Subhadeep Gupta, Arpan Dutta, Biman Kanti Ray

CHAPTER 16 **Approach to Speech and Language Dysfunction** 187
Gautam Das, Durjoy Lahiri

CHAPTER 17 **Approach to Dementia** 191
Souvik Dubey, Gautam Das, Subham Chatterjee, Mahua Jana Dubey

CHAPTER 18 **Approach to Neurological Evaluation of a Child** 209
Pradip Paria

Index 217

2 Approach to Motor System Disorders

CHAPTER

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CHAPTER OVERVIEW

- Overview of Neuroanatomy and Physiology of Motor System
- Symptoms of Motor System Disorders
- Bedside Examination of Muscle Tone, Power, and Reflexes
- Muscle Weakness: Differential Diagnosis and Clinical Approach

Case Scenario 1:

A 68-year-old man presented with one and half years history of progressive weakness of right upper limb, slurring of speech, and cramps in his hands and legs. For last 2 months, he complains of crying/laughing spells, cough when eating, especially during drinking. His past medical history was unremarkable. Neurological examination revealed spastic dysarthria, fasciculations of the tongue, arm and thigh, wasting of the tongue and right hand, normal power of lower limbs and proximal part of upper limbs but finger extensors and small muscles of hands were weak more on the right hand than the left. Jaw jerk was present, all deep tendon reflexes were brisk, Hoffman sign was present on right side and bilateral plantar responses were extensor. Sensory and cerebellar examinations were within normal limit.

Case Scenario 2:

A 26-year-old male presented with slowly progressive weakness for last 8 years. His symptoms initially began with difficulty in climbing upstairs, getting up from squatting position which was subsequently followed by difficulty in performing overhead activities. For last 2 years, he noticed thinning of shoulders, arms, and thighs. His elder brother, who is now 40 years old, experienced similar weakness in limbs since 18 years of his age and is now wheelchair bound. Examination showed normal cognition, normal cranial nerves, atrophy of anterior and posterior fibers of deltoids bilaterally and sparing of central fibers, winging of scapula, thinning of thighs, and pseudohypertrophy of both calves. He had proximal weakness of upper and lower extremities with predominant involvement of deltoid and biceps, sparing of triceps, diminished deep tendon reflexes (except at the ankle), bilateral flexor plantar response, normal sensation, and waddling gait.

Questions:

- Where do you localize the lesion?
- What is the likely pathology?
- What is the provisional diagnosis?

OVERVIEW OF NEUROANATOMY AND PHYSIOLOGY OF MOTOR SYSTEM

Components of Motor System at Different Levels

The precision of a movement depends on the motor integration at different levels:

- Suprasegmental level
- Segmental level
- Motor unit level

Suprasegmental Level

This includes brain (pyramidal and extrapyramidal), brainstem, and cerebellum. The key components are discussed here.

Brain

- *Primary motor cortex (i.e., precentral gyrus)*: Muscle groups of contralateral face, arm, trunk, and leg are represented in the primary motor cortex. The pyramidal system arises from the primary motor cortex in the precentral gyrus.

- *Premotor cortex, supplementary motor cortex, and prefrontal cortex*: Involved in initiation and planning of movements.
- *Posterior parietal cortex*: Involved in motor activity initiated by sensory perception.
- *Limbic system*: Subserve motivation and attention.
- *Striato pallidum (basal ganglia) and cerebellum*: The extrapyramidal system originates mainly from the basal ganglia. Both of them project to primary motor cortex and modulate the function of pyramidal system.

Key Point

The primary motor cortex is extensively connected to the other areas and finally executes the voluntary movement.

Brainstem

Various suprasegmental tracts descending through brainstem modulate the activity that occurs at segmental level, i.e., at the level of spinal cord.

- For axial and proximal limb muscles (maintain righting and postural reflexes):
  - *Tectospinal*: Originates from superior colliculus and influences muscles of the neck and upper back.
  - *Reticulospinal*: Originates from the reticular nuclei in pons and medulla. The pontine (medial) reticulospinal tract facilities extensor muscle whereas the medullary (lateral) reticulospinal tract inhibits antigravity muscles and is involved in autonomic function.
  - *Vestibulospinal*: Lateral vestibulospinal tract arises from the lateral vestibular nucleus and facilitates ipsilateral extensor muscle tone of the trunk and extremities.
- For distal limb muscles (manipulative action):
  - *Rubrospinal tract*: Arises from red nucleus in midbrain and facilitates flexor muscle tone mainly in the upper extremities.
  - Corticospinal tract

Segmental Level

Occurs at the level of spinal cord, mediates segmental reflex (e.g., withdrawal reflex), and involves many motor units.

Motor Unit Level

This is the final common pathway for voluntary and involuntary motor activity. A motor unit includes one alpha motor neuron and all the muscle fibers it innervates. Cell bodies of alpha motor neurons lie in the anterior horn cell (AHC) of spinal cord and in the brainstem motor nuclei. Their axons pass through the anterior root, peripheral nerve, and neuromuscular junction (NMJ) to the muscle.

Muscle

A single muscle consists of hundreds to thousands of muscle fibers. The muscle fiber is a complex and multinucleated cell enveloped by plasma membrane (sarcolemma) and the cytoplasm (sarcoplasm) inside contains myofibrils and cellular organelles, e.g., mitochondria and ribosome. The connective tissue surrounding the individual muscle fiber is called endomysium. Multiple muscle fibers are grouped together to form a muscle fascicle lying within a collagen sheath (perimysium). Multiple muscle fascicles are bundled together to form a muscle which is surrounded by epimysium. The ends of muscle fibers are attached to tendon fibers which ultimately anchor them at the skeleton.

Muscle Spindle (Fig. 1A)

- It is a fusiform body, containing about 10 embryonic muscle fibers, called *intrafusal fibers*.
- Spindles lie parallel to skeletal muscle fibers *(extrafusal fibers)* and their ends are attached to endomysium of muscle fibers.
- *Intrafusal fibers* are of two types:
  1. *Nuclear bag fibers*—nuclei are aggregated in the center (center is bulged)
  2. *Nuclear chain fibers*—nuclei are arranged in a single row throughout the length of the fiber

The ends of each intrafusal fiber have contractile elements.

Neural connection of muscle spindles:

- *Afferent (sensory)*:
  - *Primary*: Type la sensory fibers (diameter 17 μm), wind around central parts of both nuclear bag and chain fibers *(annulospiral fibers)*.
  - *Secondary*: Type II fibers (diameter 8 μm), wind around peripheral part of nuclear chain fibers *(flower spray endings)*
- *Efferent (motor)*: *Gamma (ϒ) motor* fibers supply the ends of the intrafusal fibers containing the contractile elements. *Gamma d (dynamic)* fibers supply nuclear bag fibers and *gamma s (static)* fibers supply nuclear chain fibers.

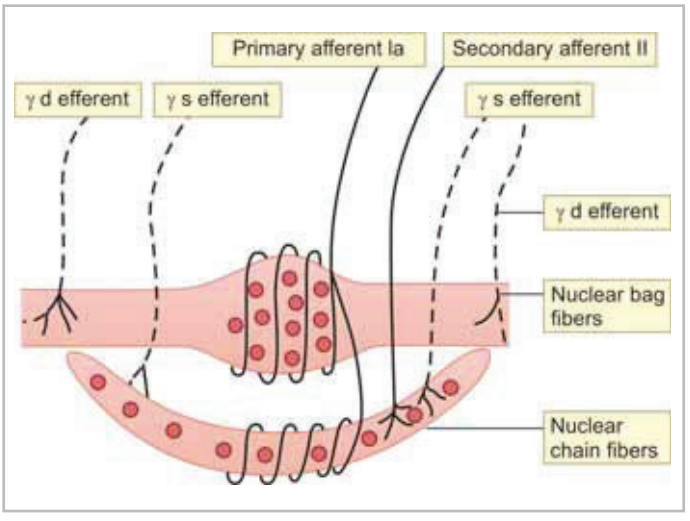


FIG. 1A: Muscle spindle.

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[Two types of motor neurons are present in AHC—alpha and gamma. Alpha (α) motor neurons give rise to large diameter nerve fibers to supply extrafusal muscle fibers. Gamma motor neurons give rise to smaller diameter nerve fibers to supply intrafusal fibers.]

***Dynamic and static response*:** If a muscle fiber is suddenly stretched, the stretch acts immediately on the center of nuclear bag fibers and thus the rate of firing in primary sensory afferents (annulospiral) increases to highest rate immediately after stretch application and then declines.

Muscle spindles lie parallel to extrafusal fibers. When a muscle is suddenly stretched, the muscle spindles in it are also stretched and due to difference in elasticity, the stretch acts immediately on the center of the nuclear bag fiber and thus bag fiber afferents (annulospiral fibers) fire at highest rate during change in length of muscle fibers (phasic). The nuclear bag fibers sense the rate of stretch and thus inform about the change of length of muscle fibers *(dynamic response)*. As soon as the length of muscle fibers becomes constant, the rate of firing decreases even if the stretch is maintained.

Due to uniform elasticity, the afferents of nuclear chain fibers fire uniformly throughout the stretch (tonic). Therefore, nuclear chain fibers sense the amount of stretch and thus inform about length of the muscle fibers (not the change of length of muscle fibers) *(static response)*.

During resting state, sensory nerve fibers have resting firing rate. If the muscle spindle is stretched (either by contraction of intrafusal fibers or stretch of extrafusal fibers), firing rate increases. During ϒ discharge, there is contraction of muscle spindles leading to stimulation of sensory afferents. Stimulation of gamma-d fibers leads to contraction of nuclear bag fibers, whereas stimulation of gamma-s fibers leads to contraction of nuclear chain fibers. In either case, increased firing rate in sensory nerve leads to stimulation of alpha fibers and contraction of extrafusal fibers.

The sequence of events can be summarized as follows:
ϒ motor discharge → contraction of ends of intrafusal fibers → Stretching of midparts of intrafusal fibers → increased firing in sensory afferent → impulse reaches spinal cord and reflex firing of α motor neuron → contraction of extrafusal fibers → increase in muscle tone.

***α-ϒ linkage*:** When a resting muscle is stretched, the muscle spindles are stretched in parallel. However, a subsequent contraction of the muscle will cause slackening of the muscle spindle, thereby rendering it temporarily insensitive to stretch. Activation of gamma motor neurons will contract the muscle spindle, ensuring that the spindle is taut at all times and remains sensitive to changes in length of muscle throughout. Hence, during muscle contraction, central nervous system (CNS) activates both alpha and gamma motor neurons. This coordinated process is referred as *α-ϒ linkage*.

***Jendrassik maneuver*:** The subject is instructed to perform a forceful voluntary activity (e.g., clenching of teeth and interlocking of fingers in a hook-like form). There is increased flow of afferent impulses via the sensory fibers from the spindles. This leads to increased gamma ϒ motor discharge resulting in increased sensitivity of muscle spindles to stretch and facilitation of jerks.

NEUROPHYSIOLOGY OF MUSCLE TONE, POWER, AND REFLEXES

Muscle Tone

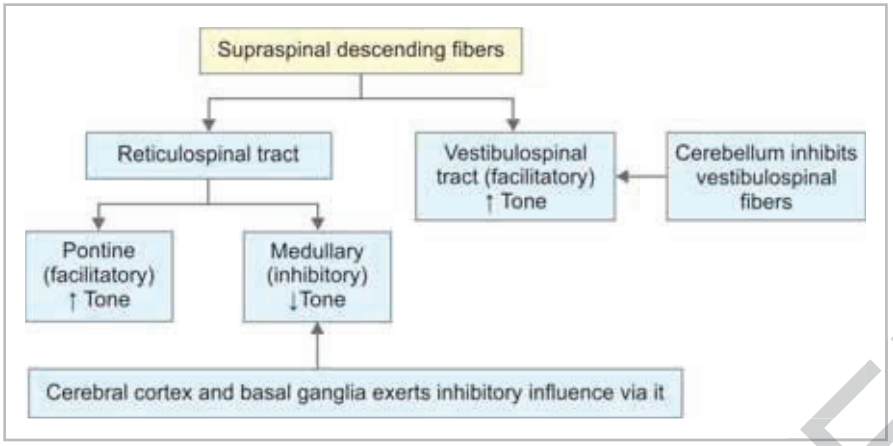
- The state of partial contraction in a muscle at rest
- The resistance felt while moving a limb passively when voluntary contraction is absent

Muscle tone is the force exerted by a muscle to resist being lengthened. At rest, some of the gamma efferent fibers are tonically active and thus keep the muscle spindles taut. Firing of the ϒ motor neurons leads to contraction of the distal ends of the spindle fibers resulting in stretching of the spindle. Then the muscle spindles send afferent stimuli via Ia fibers which in turn causes excitation of the alpha motor neurons leading to contraction of muscle. Thus a steady state of tension can be maintained. Tone can be assessed by observing the reaction that occurs in a muscle when it is stretched. During stretching, the feedback system operates via Ia afferent to counteract the stretch and thus causing muscle contraction to the desired length. This explains how the stretch reflex contributes to muscle tone. In addition, there are several supraspinal descending fibers and muscle tone is ultimately determined by the sum of these ascending and descending influences to the alpha and gamma motor neurons.

Anatomical Substrates

- *Muscle spindles* and their afferent connections via Ia fibers to spinal cord segment. The afferent fibers synapse with alpha motor neurons.
- *The alpha motor neurons*: Supply the extrafusal muscle fibers
- *The gamma neuron*: Supply the muscle spindle (intrafusal fibers)
- *Golgi tendon organs*: Encapsulated receptors which lie in the tendinous and aponeurotic insertions of muscle. Severe muscle stretching activates afferent fibers from Golgi tendon organs that terminate in internuncial cells within gray matter in the spinal cord which projects to alpha motor neurons and thus leads to decrease in muscle tone.
- *Supraspinal descending fibers* which influence the alpha and gamma neurons **(Flowchart 1)**.
  - *Reticulospinal tract*:
    - *Pontine reticulospinal tract* is facilitatory, i.e., increases muscle tone
    - *Medullary reticulospinal tract* is inhibitory, i.e., decreases muscle tone

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FLOWCHART 1: Impact of supraspinal descending fibers on muscle tone.

These effects are mediated by gamma efferents which indirectly activate alpha motor neurons (via Ia spindle afferent activity) *(gamma loop)*.

- *Vestibulospinal tract*: It is facilitatory, directly stimulates the alpha motor neurons of AHCs and the increased tone produced by it is called alpha rigidity.

- *Cerebral cortex and basal ganglia*: Normally, inhibitory influence from cerebral cortex and basal ganglia act through the descending medullary reticulospinal tract.
- *Cerebellum*: Normally, the anterior lobe of cerebellum inhibits vestibular nucleus and thus cerebellum as such inhibits muscle tone by inhibitory influences on the vestibulospinal mechanisms. If the anterior lobe of cerebellum is removed in a decerebrate animal, there is increase of muscle tone ***(decerebellate rigidity)***. Hypotonia in cerebellar disease is related to depression of gamma and alpha motor neuron activity associated with decreased fusimotor efferent and spindle afferent activity.
- *Viscosity and elasticity* of muscle fibers also contribute to muscle tone.

Significance of Muscle Tone

- Helps to maintain normal resting limb positions and attitudes; prevent undue mobility at joints.
- Resting muscle tone is greatest in antigravity muscles because this helps to maintain the steady contraction of antigravity muscles which keeps the body in an erect position.
- Resting muscle tone provides the background activity over which muscle contraction (i.e., increased tone) becomes easier and a muscle with normal tone can perform better than a muscle with abnormal tone.

Muscle Power

Muscle power indicates the capacity of a muscle to exert force. This is determined by the number and size of recruited motor units.

- Weak movement means few, small motor units are involved.
- Powerful movement means many more large sized motor units are called into action.

Paralysis or plegia means complete loss of motor function whereas paresis indicates partial loss of motor function.

Reflex

The examination of reflex is considered one of the most crucial parts of neurological examination. Reflex is defined as an involuntary response to a sensory stimulus.

Components of reflex arc:

- *Afferent part*: Receptor and afferent neuron
- *Centre*: Either in spinal cord or brain
- *Efferent part*: Efferent neuron and effector organ

Bell–Magendie law: In spinal cord, dorsal root is always sensory whereas ventral root is always motor in nature.

If the reflex response is normal, it indicates that the reflex arc is intact.

If the reflex response is abnormal, it suggests that there is lesion either anywhere in the reflex arc (afferent-center-efferent) or in the CNS which influences the activity of reflex arc.

In clinical practice, reflexes can be classified in a number of ways. The most frequently used classifications in clinical practice are:

- *According to the number of synapses in the reflex pathway*:
  - *Monosynaptic*—only one synapse in the reflex pathway and therefore response is very rapid, e.g., knee jerk and ankle jerk.
  - *Polysynaptic*—two or more synapses in the reflex pathway, e.g., plantar reflex and withdrawal reflex.
- *According to the site of stimulus*:
  - *Superficial reflex*—when superficial structures (receptors in skin or mucous membrane) are stimulated, e.g., corneal, conjunctival, abdominal, and plantar reflex.
  - *Deep reflex*—when deeper structures (receptors are located deeper inside, e.g., muscle spindles) are stimulated, e.g., knee jerk and ankle jerk.

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- *According to anatomical position*:
  - *Segmental reflex*—only one segment is involved, i.e., end of afferent limb and beginning of the efferent limb is situated in one segment of spinal cord.
  - *Intersegmental reflex*—more than one segment is involved, i.e., end of afferent limb and beginning of the efferent limb are situated in different segments of spinal cord.
  - *Suprasegmental reflex*—reflexes which involve the integrating center (in brain or brainstem) along with the spinal segments.

Stretch Reflex (Figs. 1B and C)

- Monosynaptic deep tendon reflex

Stimulus—stretch on skeletal muscle
↓
Receptor—muscle spindle stimulated
↓
Afferent arc—type Ia and type II fibers transmit impulse via dorsal root
↓
Center—reaches AHC in spinal cord
↓
Efferent arc—alpha fibers transmit impulse via ventral root
↓
Effector organ—extrafusal fibers
↓
Result—contraction of skeletal muscle

- Two types:
  1. *Phasic*—when response is quick and short lived
  2. *Tonic*—persistent action against a maintained stretch

Stretch reflex opposes sudden increase in the length of the muscle.

- *Dynamic stretch reflex*: Sudden stretching of a muscle leads to its immediate contraction due to direct synapse of sensory afferents with anterior alpha motor neurons (monosynaptic pathway).
- *Static stretch reflex*: When muscle stretch is continued, it continues to contract due to indirect synapse of sensory afferent collaterals with anterior alpha motor neurons through interneurons.
- *Inverse stretch reflex* ***(Fig. 1D)***: When a muscle is stretched, it contracts but if it is stretched forcefully, the muscle relaxes. This is known as inverse stretch reflex *(Golgi tendon reflex)*.

Stretch—contraction. *More stretch*—relaxation (lengthening). Golgi tendon organs are encapsulated receptors, present in the tendinous and aponeurotic insertions of muscles. In relaxed muscle and during passive stretch, Golgi tendon organs are silent. Strong stimulus in required to stimulate these receptors.

Neural Circuitry of Inverse Stretch Reflex

- *Stimulus*—excessive stretch of a muscle
- *Receptor*—Golgi tendon organ

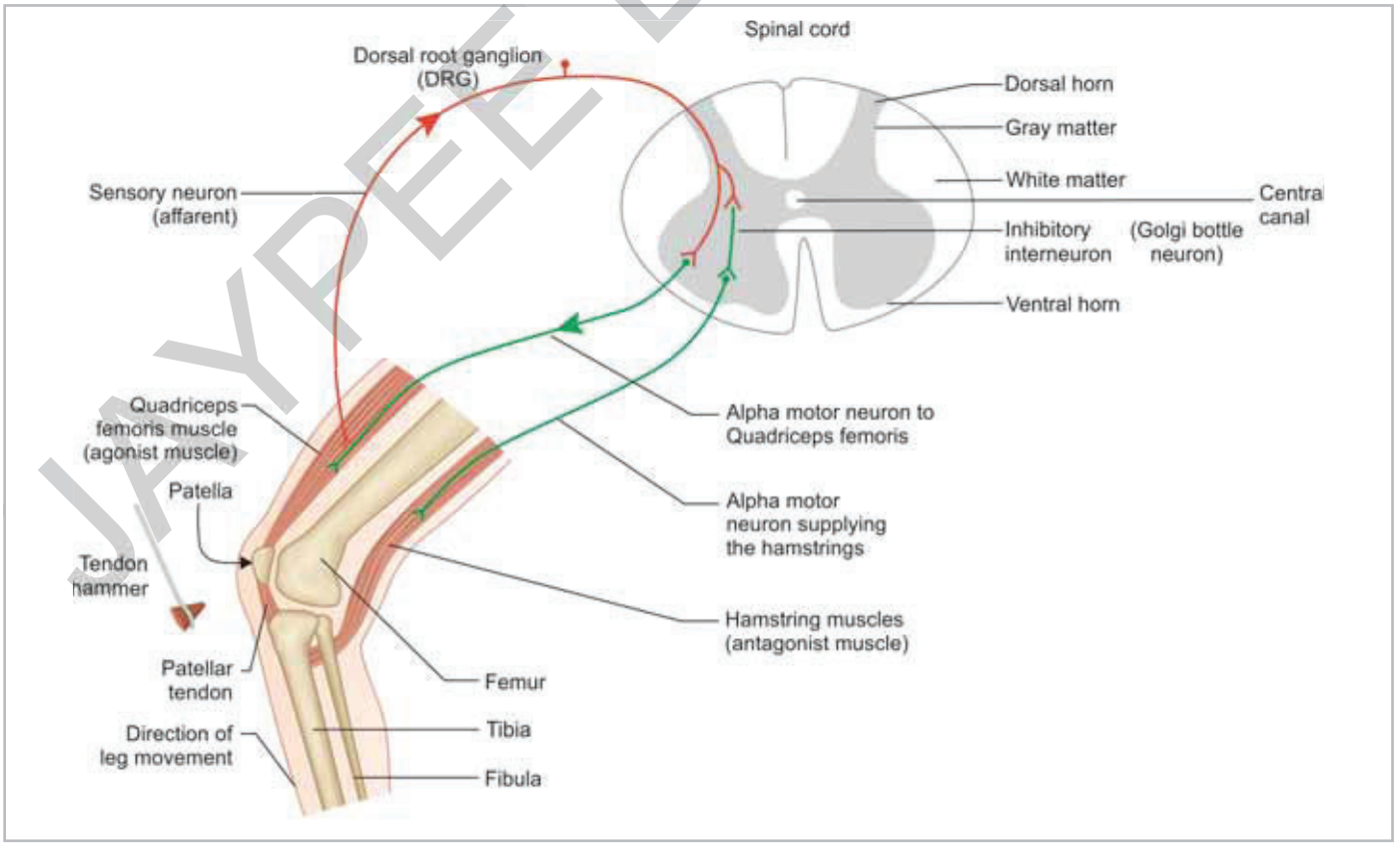


FIG. 1B: Stretch reflex.

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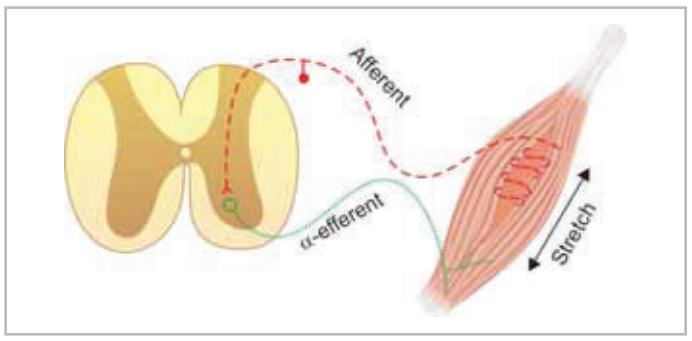


FIG. 1C: Stretch reflex.

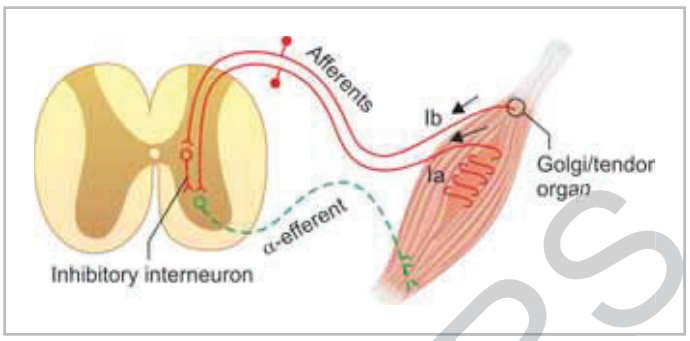


FIG. 1D: Inverse stretch reflex.

- *Afferent*—type Ib (myelinated and rapidly conducting) fibers
- *Center*—spinal cord inhibitory interneurons (internuncial cells) which in turn project to alpha motor neurons (disynaptic reflex)
- *Efferent*—alpha motor neurons
- *Result*—relaxation of same muscle and contraction of antagonist muscle

Some Phenomenon Associated with Reflex Activity

- *Reciprocal innervation*: When a reflex leads to contraction of agonist group of muscles, it is also accompanied by inhibition of the antagonist group (Sherrington's law of reciprocal innervation).
  Example, when we elicit knee jerk, contraction of quadriceps is accompanied by relaxation of its antagonist, i.e., hamstrings. This is brought about by inhibitory interneurons lying in between the afferent neuron and the efferent neuron for the antagonist muscle.
- *Irradiation*: Sometimes, when a stimulus is applied to elicit a reflex, the reflex response may spread beyond the muscle stretched and a wider response is obtained. This is called irradiation.
  Example, a tap on the styloid process of the radius may elicit a contraction not only of the brachioradialis but also of the long flexors of the fingers. This irradiation of reflex is probably due to propagation of vibration wave from bone to muscle resulting in stimulation of excitable muscle spindles in its path.
- *Withdrawal reflex*: This is a polysynaptic reflex (involving internuncial neurons) which withdraws (flexes) a limb from noxious stimuli (also called flexor reflex). This is usually associated with crossed extensor reflex.
- *Crossed extensor reflex*: When a painful noxious stimulus causes contraction of flexors of ipsilateral side (flexor reflex), there is contraction of extensors of contralateral side. This is called crossed extensor reflex. This helps to maintain balance of the body when a limb is withdrawn from the painful stimuli.
- *Mass reflex*: In a complete transverse spinal cord lesion, the period of spinal shock is followed by a stage of heightened reflex activity. At this stage there are flexor spasms, accompanied by contraction of abdominal muscles, automatic evacuation of bladder and bowel, priapism, ejaculation, profuse sweating, and piloerection below the level of lesion. This is called mass reflex which can be precipitated by stimulation of skin of legs or by distended bladder.

Clonus and Clasp Knife Phenomenon

If muscle tone is normal, we get normal stretch reflex. If muscle tone is increased, there is increased stretch reflex and increased resistance during passive movement. If stretch is still continued, this strong stimulus stimulates Golgi tendon organs leading to inverse stretch reflex, i.e., relaxation with increase in the length of the muscle (lengthening reaction/clasp knife phenomenon).

Clonus is rhythmic, involuntary muscular contractions when abrupt and sustained stretch stimulus is applied to a muscle.

Pathophysiology

In pyramidal tract lesion, the muscle tone is increased and muscle spindles are hypersensitive due to increased ϒ motor discharge. When a hypertonic muscle is forcefully stretched, it contracts strongly (due to stretching of hypersensitive muscle spindles). The applied stretch also stretches the tendon. These two together stretch the tendon sufficiently to stimulate the Golgi tendon organs leading to inverse stretch reflex. When a hypertonic muscle is suddenly stretched, there is alternate stretch reflex and inverse stretch reflex. The stretch reflex–inverse stretch reflex sequence may cause clonus. However, synchronized motor discharge alone (without Golgi tendon organ discharge) may cause clonus. As the muscles contract, discharge from muscle spindle stops. However, there is sustained stretch and as soon as the muscle relaxes, it is again stretched and the spindles are stimulated.

Sustained clonus means more than five oscillations.

Sites where clonus is elicited:

- Ankle clonus—by brisk, sustained dorsiflexion of the foot
- Patellar clonus—by sharply moving the patella downward
- Wrist clonus—by sudden, forceful dorsiflexion of the wrist

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Prerequisites of clonus:
- Spinal stretch reflex mechanism should be intact
- Sustained hyper excitability of α and ϒ motor neurons
- Contraction—relaxation cycle of muscle spindles should be synchronized

SYMPTOMS OF MOTOR SYSTEM DISORDERS

A comprehensive history is the most important step in the approach to any motor system disorder. A motor system disease may present with some negative or positive symptoms. Typically, weakness is the main negative symptom. Other negative symptoms are fatigue, atrophy, or thinning of muscles. The positive symptoms include muscle pain (myalgia), cramps, stiffness, inability to relax (myotonia), and rippling/mounding.

- *Weakness*: Distribution of weakness-related symptoms vary according to the muscles involved **(Table 1)**.

Remember, the early symptoms of subtle weakness:
  - Decreased range and shape of movement
  - Clumsiness of movement
  - Easy fatigability
  - Tremulousness of limbs or difficulty to perform skilled movements
- *Fatigue*: Increased fatigability means the inability to sustain the performance of an activity that should be normal for age, sex, and size-matched control.
  - Fatigue may be generalized (suggestive of systemic disease, anemia) or may involve specific group of muscles, e.g., eyelids, extraocular muscles, muscle of mastication, etc.
  - Diurnal variation may be present. If present, this is suggestive of NMJ disorder.
- *Cramp*:
  - Painful knotting up of muscles; usually affects calf muscles during sleep or exercise particularly when muscles contract in a shortened position and is immediately relieved on stretching of muscle.

Table 1: Symptoms of muscle weakness and clinico-anatomical correlation.

Symptoms	***Muscles weakened***
Ocular muscle weakness:	
• Drooping of eyelids (ptosis) and bilateral tilting of neck • Binocular diplopia, blurring of vision • Thrusting of head while looking to one side	• Levator palpebrae superioris • Asymmetric weakness of extraocular muscles • Bilateral symmetrical weakness of extraocular muscles
Symptoms of facial and bulbar muscle weakness:	
• Inability to close eyes tight, soap entering eyes while washing face, sleeps with eyes open • Inability to suck liquid through a straw, inability to whistle or to blow up a balloon • Loss of facial expression, incomplete smile • Drooling of saliva from mouth • Difficulty in chewing • Difficulty in swallowing particularly liquids • Nasal regurgitation of food, nasal intonation of speech • Cough and choking while eating	• Orbicularis oculi • Orbicularis oris • Levator anguli oris • Lower facial muscle weakness • Muscles of mastication, e.g., temporalis, masseter • Tongue, palate and pharynx muscles • Palatal muscles—LMN weakness • Pseudobulbar, i.e., UMN weakness
Symptoms of neck musle weakness:	
• Chin falls on the chest • Difficulty in lifting the head off the pillow • Difficulty to hold the neck upright while traveling in car leading to forward or backward drop of head	• Neck extensors • Neck flexors • Neck muscles
Symptoms of diaphragmatic weakness:	
Shortness of breath particularly in lying down posture	• Diaphragm muscle
Symptoms of truncal weakness:	
• Difficulty in moving sideway in supine position or difficulty to get up from supine position • Abnormal posturing of spine while sitting or standing, but correctable on lying down	• Truncal muscles • Paraspinal or pelvic girdle muscles

Continued

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Continued

Symptoms	***Muscles weakened***
Symptoms of limb weakness:	
• Difficulty in combing hair, shampooing, brushing, raising arms above shoulder level, performing overhead activities	• Proximal upper limb muscles
• Difficulty in holding small objects, screwing, buttoning, unbuttoning, opening a jar, writing	• Distal upper limb muscles
• Difficulty in standing from squatting position, climbing upstairs, descending downstairs ○ Difficulty in descending downstairs ○ Difficulty in climbing upstairs	• Proximal lower limb muscles ○ Quadriceps ○ Gluteus maximus
• History of waddling gait	• Hip abductors
• History of slipping of chappals from feet while walking, repeated sprains in the ankle	• Distal lower limb muscles
• Frequent tripping particularly while walking on uneven surfaces	• Ankle dorsiflexor and ankle evertor muscles
• Difficulty in standing on tiptoes—appreciated during sporting activity, dancing	• Posterior calf muscles
• Unsteadiness of gait	• Muscles of both anterior and posterior compartments of legs
(LMN: lower motor neuron; UMN: upper motor neuron)	

  - This may be benign or manifestation of neurogenic condition and occurs due to firing of numerous motor units at high frequencies.
- *Stiffness*: Patient may complain of stiffness of distal extremities (e.g., difficulty to release hand grip after handshake and unscrewing a bottle top). This is found in myotonia and neuromyotonia.
- *Muscle pain*: May originate from muscles, fasciae, bones, joints, or painful neuropathy
- *History of passing dark reddish brown urine*: Suggest myoglobinuria

  Once the symptoms are identified, the next step is to define the temporal pattern of illness and to recognize triggering events (e.g., exercise and meal) if any, past medical history, history of toxin exposure, and family history.
- *Temporal pattern*: Define the age at onset, duration, and evolution of symptoms.
  - Age of onset, e.g., symptoms of Duchenne muscular dystrophy usually begin by 3 years of age, most limb girdle and facioscapulohumeral muscular dystrophies occur in adolescence or later, whereas inclusion body myositis occurs in elderly.
  - *Evolution and duration*: For example:
    - Constant weakness occurs in inflammatory myopathies, muscular dystrophies
    - Episodic weakness occurs in periodic paralysis, glycolytic pathway disorders
    - Acute or subacute progression (over days/weeks/months) is found in inflammatory myopathies
    - Chronic slow progression (over years) in muscular dystrophies
    - Nonprogressive/very slowly progression (over decades) is found in congenital myopathies
- *Exercise-related exacerbation*: If exercise provokes muscle pain, fatigue, or weakness, it implies a mitochondrial dysfunction or disorder of metabolism of carbohydrate or fat. Cues from patient's history may help you to identify the underlying metabolic disorder.
  - *In fatty acid metabolic disorder*:
    - Patients can easily tolerate the initial phase of exercise
    - After 30–60 minute of exercise, patients become incapacitated
    - Patient complains of increased symptoms if they exercise in fasting state, may ingest candy bar or sugar to get relief because that quickly increases the blood sugar level
    - Patients prefer relatively intense and brief exercise
  - *In carbohydrate metabolism disorder*:
    - Patient complains of fatigue and muscle pain in the first few minutes after exercise
  - *In mitochondrial disorder*:
    - Even after limited exercise patient presents with manifestation of hypermetabolism (e.g., resting tachycardia, sweating, etc.)

    Explanation:
    - *At resting stage*: Fatty acids are the main source of energy and conserve glucose.
    - *At initial phase of heavy exercise*: Muscles use glycogen stores as the source of energy.
    - *After 10–15 minutes of exercise*: A mixture of carbohydrate and fat provide the source of energy.
    - *After 30–40 minutes of continued exercise:* Carbohydrates are no longer used due to risk of hypoglycemia following depletion of glycogen stores. At this stage, fatty acid are the chief sources of energy.

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  - In mitochondrial disorders, due to limited availability of ATP, metabolic pathway operates at maximum level even after light exercise.
- *Meal-related exacerbation*:
  - Inactivity after a high carbohydrate meal may precipitate an attack of weakness in hypokalemic periodic paralysis.
  - Ingestion of potassium rich diet provokes hyperkalemic periodic paralysis.
- *Past medical history*: Many medial diseases may be associated with motor system disorders, e.g., inflammatory myopathies and connective tissue disease or myasthenia gravis and other autoimmune disorder. Past medical history should always be addressed during evaluation of weakness.
- *History of toxin exposure*: Toxin can damage the AHC, motor neurons, NMJ, or muscle. Toxin exposure may be related to occupational hazard, home environment medications, or food.
- *Family history*: Take history of both paternal and maternal sides of the family and if possible, try to include information of three generations. You must be very careful because false negative reporting sometimes happens due to wide range of clinical manifestations among different family members and perception of negative social implications. History of skeletal deformities or functional limitations among family members is very informative.

EXAMINATION OF MOTOR SYSTEM DISORDERS

Components of Examination

- Inspection
- Muscle tone
- Muscle power
- Reflexes
- Stance and Gait

Inspection

Expose the patient but ensure that the comfort and dignity of patient is maintained.

- *Look for any abnormal muscle movements*:
  - *Fasciculation:*
    - Brief twitch due to spontaneous discharge of single motor unit, occurs in denervating disease.
    - Benign fasciculation—found in normal individuals, not associated with weakness or wasting of involved muscle.
  - *Myokymia*: Undulating movement of muscle (worm-like) due to repetitive discharge of a motor unit (i.e., grouped fasciculation), occurs in denervating disease as well as in normal individuals.

Key Points

- Differentiate quadriceps muscle wasting from slender thigh. Instruct the patient to tighten the knee firmly. Normally, the medial and lateral bellies of quadriceps stand out prominent in distal part of the thigh above the knee. In quadriceps wasting, normal prominences of these bellies are not visible.
- Groove on lateral aspect of tibia becomes prominent during dorsiflexion of the foot (in anterior tibial muscle wasting)
- Look at extensor digitorum brevis (EDB)
  - Wasted early in neurogenic disorder
  - Hypertrophy in myopathy to compensate for weakness of long toe dorsiflexors.
- Pes cavus (high-arched foot with hammer toes): Indicates intrinsic foot muscle weakness since very early childhood.

  - *Cramp:* Painful and hard on palpation, occurs due to firing of numerous motor units at high frequencies.
  - *Contracture*: Active painful muscle contraction in the absence of electrical activity occurs in metabolic myopathies (e.g., McArdle disease). The three clinical features which differentiate contracture from cramp are longer duration, no relief by muscle stretch, and occurrence only in exercised muscles.
- *Look for atrophy of muscle*: **Figure 2** shows the appearance of different parts of the body in case of atrophy of muscles.
- *Look for hypertrophy of muscle*:
  - Severe hypertrophy suggests muscle disease (look at calf muscles, deltoid, brachioradialis, and glutei)
  - Mild hypertrophy—may be found in spinal muscular atrophy (SMA) and sarcoglycanopathies
  - Well-developed musculature—found in myotonia congenita and hypothyroidism
- *Look for hypertrophy and atrophy*:
  - *Valley sign*: When a patient with Duchenne muscular dystrophy abducts his arms with elbow flexed at 90°, two prominent mounds are seen due to hypertrophy of the infraspinatus and deltoid. A valley is formed between these two muscles due to atrophy of the teres major and minor.
- *Look for myotonia/myoedema*:
  - *Myotonia* is characterized by delayed relaxation after sustained contraction. There are two ways to demonstrate myotonia: (1) If you percuss directly over thenar eminence, there is localized contraction of the muscles persisting for seconds with delayed relaxation of thumb abductors (percussion myotonia) and (2) Ask the patient to sustain a grip for a brief period and then release grip. There is slow relaxation of grip (action myotonia). Myotonia is commonly found in distal muscles. Myotonia usually improves with repetitive activity but paramyotonia worsens

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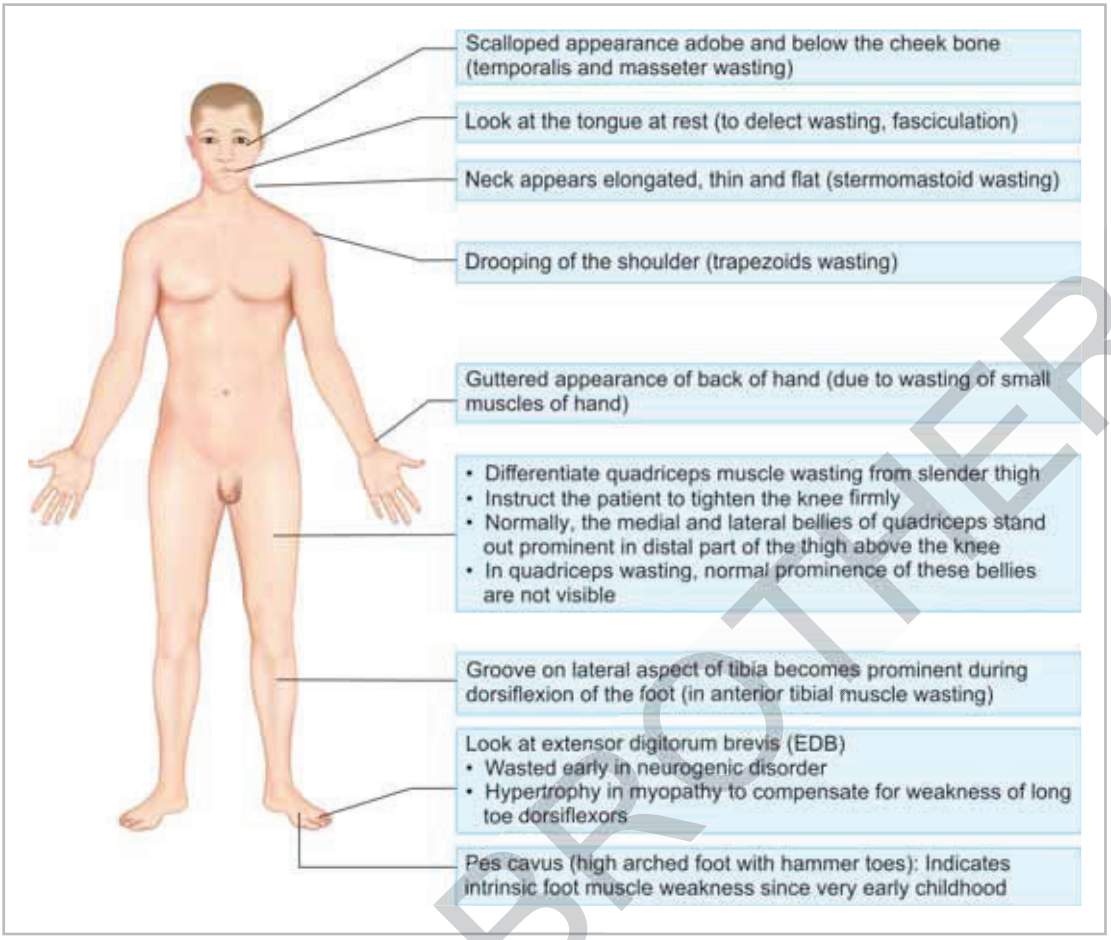


FIG. 2: Findings in atrophy of muscles.

with repetition. Both paramyotonia and myotonia worsen in cold.
  - *Myoedema*—following percussion, a dimple develops in the muscle followed by formation of a small hillock (occurs in thyroid disorder).
- *Look for deformities and contractures*: Look for high arched palate, scoliosis, kyphosis or lordosis, and contracture in joints (occurs in congenital myopathies, muscular dystrophies, polymyositis or dermatomyositis, and SMA).

EXAMINATION OF MUSCLE TONE

Do not order the patient to "relax". Small irrelevant conversation may ensure relaxation or ask the patient to count down from 100. Simple inspection may reveal an abnormal positioning of limb suggestive of change of tone.

What to do?

Move the limb passively through a complete range of motion, first slowly and then at varying speeds. Determine the resistance of relaxed muscle to passive manipulation.

Upper limbs:

- Grasp the patient's hand as if to shake it and hold the forearm. First pronate and supinate the forearm and then roll the hand around the wrist.
- Support the patient's forearm and the elbow and passively flex and extend the elbow through the full range of motion.
- *Shoulder shaking test*: Hold the patient's chest and shake the shoulders briskly back and forth. Observe the reciprocal movements of the arms. In extrapyramidal disease, the range of arm swing will be decreased whereas it will be increased in hypotonia (e.g., cerebellar disease).

Lower limbs:

- *Tone at hip*: Ask the patient to lie down with straight legs. Roll the knee from side to side.
- *Tone at knee*:
  - Place your hand behind the knee and lift it rapidly. Watch the heel. Normally there will be minimal lifting of the heel off the bed. When the knee is lifted quickly, heel does not lift off the bed in hypotonia whereas it easily leaves the bed in hypertonic state.
  - Hold the knee and ankle. Flex and extend the knee passively.

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- *Tone at the ankle*: Hold the ankle and then proceed to passive planter flexion and dorsiflexion of the foot.

What do you find?

- *Normal:* Mild resistance through whole range of motion
- *Hypotonia:* Loss of resistance through movement
- Hypertonia:
  - Sudden increased resistance *(spasticity)*
  - Uniform resistance through whole range of motion *(rigidity)*
  - When the examiner tries to move the limb, there is almost proportional increase of tone in that muscle, as if patient opposes examiner's attempts *(gegenhalten paratonia)*

Pendulousness of the legs:
Test: Ask the patient to sit on the edge of a table with legs hanging freely. Apply a brisk, equal backward push over both legs.

Normal response: Swinging of the legs that progressively diminishes in range and usually disappears over six to seven oscillations.

Extrapyramidal disease: Decrease in swing time.

Spasticity: Forward movements are of greater amplitude and more brisk than backward movements.

Hypotonia: Range of motion is increased and prolonged in time (>6–7 oscillations).

Babinski's trunk-thigh test: Ask the patient to get up from supine position without support. In presence of spasticity in lower limbs, the legs will be elevated from the bed *(leg rising sign)*.

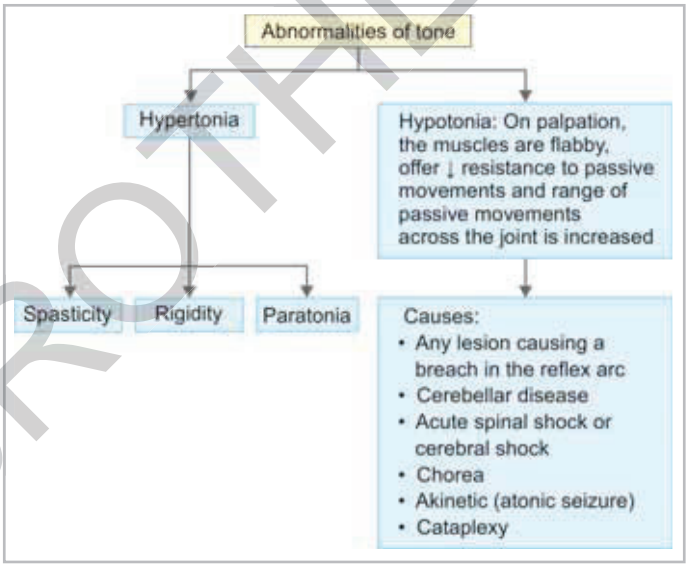
Froment's maneuver: Ask the patient to make alternating closure and opening movement of the hand (not being tested). This will elicit cog-wheel rigidity in the tested hand.

Abnormalities of tone: Refer to **Flowchart 2** and **Table 2**.

Paratonia:

- Occurs due to diffuse frontal lobe disease.
- Inhibitory paratonia (gegenhalten)—already discussed
- Facilitatory paratonia (mitgehen)—patient cooperates too much.

Remember that in addition to pyramidal tract disorder, hypertonia may occur in disease of spinal cord involving interneurons (e.g., stiff-person syndrome) and even in



FLOWCHART 2: Abnormalities of tone.

Table 2: Differentiation between spasticity and rigidity.

Parameter	***Spasticity***	***Rigidity***
Lesions involving	Corticospinal tract (CST) pathways	Extrapyramidal system
Muscles affected	Antigravity muscles (flexors in upper limb and extensors in lower limb)	Both flexors and extensors of limb uniformly
Dependence on velocity	Characterized by velocity dependent increase in the resistance of muscles to a passive stretch stimulus. When muscles are passively stretched slowly, there may be little or no increase of resistance and when stretched fast, resistance increases proportionate to velocity of stretch	Characterized by velocity independent increase in the resistance of muscle to a passive stretch stimulus. During testing of muscle tone, the degree of increased resistance remains same throughout the range of passive movement
Special phenomenon	*Clasp knife phenomenon*: At the initial part of brisk muscle stretching, there is free movement of limb for a short distance (free interval). This is followed by an abrupt catch and resistance is most marked near the middle of the range of motion. With continued movement, the resistance "melts away"	*Types*: • *Lead pipe rigidity*—smooth resistance throughout the movement • *Cogwheel rigidity*—jerky, tremulous variation (due to superimposed tremor)

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muscle disorders with continuous muscle fiber activity syndromes.

Decerebrate rigidity (Fig. 3):
- It occurs due to severe insult to the brainstem at any level between superior colliculus and inferior colliculus.
- There is opisthotonus with all four limbs stiffly extended and the neck is extended.

Explanation:
- The excitatory and inhibitory reticulospinal pathways remain intact. The inhibitory pathway (medullary reticulospinal) is driven by inhibitory influences from the cerebral cortex, basal ganglia, and cerebellum. On the other hand, the excitatory pathway (pontine reticulospinal) gets input from the periphery. Due to the mid-collicular lesion, the inhibitory influence from cortex and basal ganglia are lost. The excitatory input from the reticulospinal pathways gets upper hand and activates gamma motor neurons which indirectly activate alpha motor neuron (called gamma loop)
- The tone of the extensors also increases due to free operation of vestibulospinal reflex.

Decorticate rigidity (Fig. 3):
- Occurs due to removal of cerebral cortex, seen on the hemiplegic side after stroke involving internal capsule.
- There is flexion of upper limbs at the elbow and extensor hyperactivity in the lower limbs.

Explanation: The function of rubrospinal tract is preserved and this enhances flexor tone in the upper limbs. The hyperextension in lower limbs is due to the same changes that occur in decerebrate rigidity.

Flexor spasm: Flexor spasm is characterized by flexion at the hip, knee, and ankle.

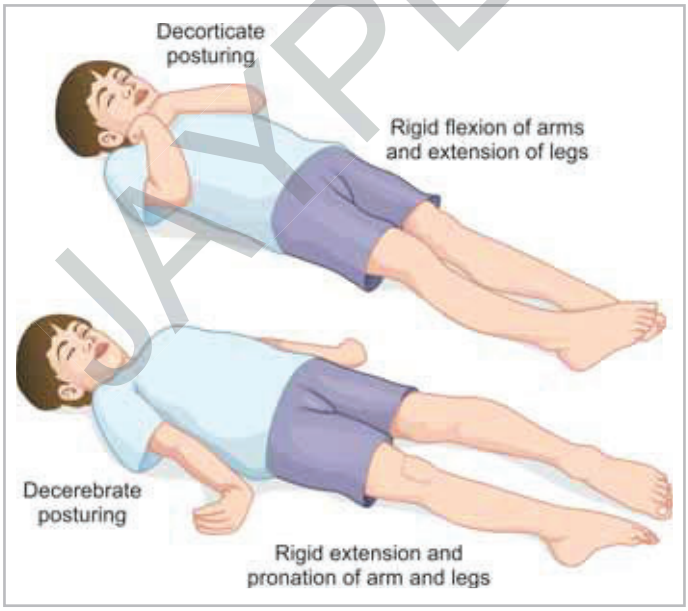


FIG. 3: Decorticate-decerebrate posturing.

Pathophysiology: Normally, two systems of fibers are important.
1. The dorsal reticulospinal tract has an inhibitory effect on stretch reflex.
2. The medial (pontine) reticulospinal tract and lateral vestibulospinal tract facilitate extensor tone. In spinal cord lesion that involves the corticospinal tract, the dorsal reticulospinal tract is involved as well. Dorsal reticulospinal tract particularly inhibits flexor lower limb reflexes. Lesion of pyramidal tract along with dorsal reticulospinal tract leads to disinhibition of affected flexor reflex pathway resulting in flexor spasm. It the dorsal reticulospinal tract is spared, there will be only paresis and extensor plantar response. Damage to only pyramidal system will result in flaccid paralysis.

Extensor spasm: Consists of extension of hip and knee along with flexion and inversion of foot. Flexor spasm is found in complete spinal cord lesion whereas extensor spasm is usually observed with partial cord lesion. The position of the limbs during the early stages of para/quadriplegia also influences the subsequent development of flexor or extensor spasm.

What is the difference between flexor spasm and propriospinal myoclonus?

Flexor spasm:
- Painful, involves hip, knee and ankle joints
- Can be a part of mass reflex

Propriospinal myoclonus (occurs probably due to lesion in propriospinal pathways in spinal cord):
- Painless, involves trunk, hip, and knee (predominantly involves the trunk)
- Increases in supine position
- Not associated with mass reflex
- Stimulus sensitive

Spinal shock: It may develop following acute and extensive spinal cord lesion. This is characterized by loss of motor function, autonomic impairment (e.g., atonic paralysis of bladder and bowel, loss of vasomotor tone, and abnormal sweating) along with muscular flaccidity (hypotonia), and loss of spinal segmental reflex activities below the level of the lesion. This occurs due to sudden interruption of suprasegmental level of control that normally keeps the spinal motor neurons in a continuous state of ready to act.

EXAMINATION OF MUSCLE POWER

There are two ways of testing muscle power:
1. The patient first completes the movement and places the joint in a particular position. Now you try to overcome it against patient's resistance.
2. Apply a fixed resistance and then instruct the patient to move the joint against your resistance.

Both methods are almost equally useful.

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Remember:

- Local pain, swelling, dislocation/ankylosis of joints, and contracture of muscle may lead to the false impression of weakness. Palpation and passive movements of the involved joint help to confirm whether the limitation of the movement is due to weakness or some other reason.
- Failure to comprehend the command of examiner, systemic illness may result in false impression of weakness.
- Beware of "trick" movements, i.e., patient uses another strong muscle with similar function to overcome the disability caused by weakness of the muscle being tested.

Grading of Muscle Power

Five-level Medical Research Council (MRC) scale is most commonly used. This was originally developed in Britain during World War II for evaluation of patients with peripheral nerve injuries.

Grading of power:

0 = No contraction

1 = Flicker of contraction

2 = Active movement with gravity eliminated

3 = Moves against gravity but not resistance

4– = Moves against gravity and slight resistance

4 = Move against gravity and moderate resistance

4+ = Submaximal movement against gravity and strong resistance

5 = Normal power

The grade of muscle power is determined by maximum power attained by the muscle, even if it is only for a brief period.

The main limitation of MRC scale is that the MRC grades are not evenly spaced.

General comments:

- Explain to the patient in simple terms about what to do and demonstrate clearly the movement he is to make.
- Test simple movements across single joints. When testing power, feel the contraction of tested muscle.
- Fix and hold the proximal portion of a limb to isolate the movement you want to test.
  Example: If you want to test the power of forearm pronators, you must fix the arm so that the shoulder does not rotate internally to compensate for pronator weakness.
- Proper positioning is important in some situations. Suppose, a patient has wrist and finger drop due to radial nerve palsy. There may be apparent weakness of grip and finger abduction. But when the same muscles are tested with passive extension of wrist and fingers, these functions are absolutely normal.
- Always consider the lever effect. Suppose, you are examining power of gluteus maximus. It is easier to overpower the muscle if you place your hands on the back of leg with knee extended (i.e., you are using a long lever) compared to placing your hand on back of lower thigh (i.e., using a short lever). So the technique that you practice is of utmost importance.
- When you are testing small muscles of hands, always match them against similar muscle of your hand. Always compare the power of the right side with that of the left side.

Testing Individual Muscles

Muscles of Shoulder Girdle and Scapula

- *Supraspinatus* ***(Fig. 4)***:
  - *Nerve*: Suprascapular nerve
  - *Root*: C5
  - *Action*: Abduction of shoulder (mainly the first 15° of abduction)
  - *Test*: Ask the patient to initiate abduction of the arm against resistance.
- *Deltoid* ***(Fig. 5)***:
  - *Nerve*: Axillary nerve
  - *Root*: C5
  - *Action*: Abduction of shoulder (mainly 15–90°)
    - Flexion and internal rotation of shoulder (anterior fibers)
    - Extension and external rotation (posterior fibers)
  - *Test*: Ask the patient to hold the arm abducted to the horizontal level. Then, ask him to resist the examiner's attempt to push it down.
- *Infraspinatus* ***(Fig. 6)***:
  - *Nerve*: Suprascapular nerve
  - *Root*: C5 and C6
  - *Action*: external rotation of shoulder
  - *Test*: Ask the patient to flex his elbow at an angle of 90° and hold it tightly against his trunk. Now, instruct the patient to turn the forearm outward against resistance.
- *Teres major*:
  - *Nerve*: Subscapular nerve
  - *Root*: C5 and C6



FIG. 4: Supraspinatus.

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FIG. 5: Deltoid.



FIG. 6: Infraspinatus.

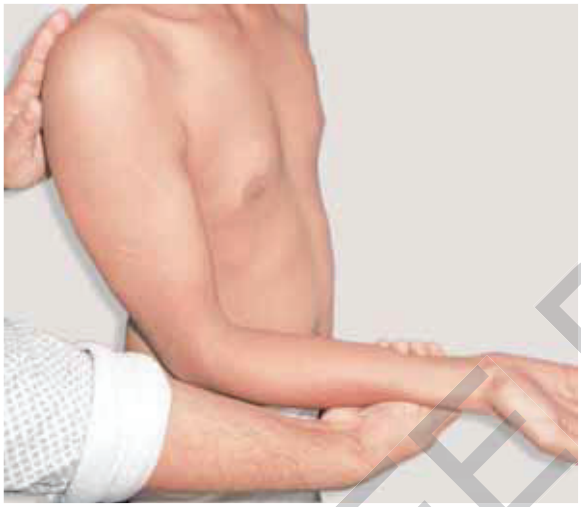


FIG. 7: Teres Major and subscapularis.



FIG. 8: Rhomboids.

- *Subscapularis*:
  - *Nerve*: Subscapular nerve
  - *Root*: C5 and C6
  - *Action*: internal rotation of shoulder
  - *Test* ***(Fig. 7)***: Ask the patient to hold his elbow against his side with the elbow flexed at an angle of 90° and then instruct the patient to turn the forearm internally (medially) against resistance.
- *Rhomboid* ***(Fig. 8)***:
  - *Nerve*: Nerve to rhomboid (dorsal scapular nerve)
  - *Root*: C5
  - *Action*: Acts primarily to retract the scapula

Key Point

The rhomboid is supplied by a twig directly from C5 nerve root. Involvement of rhomboid helps to differentiate between C5 radiculopathy and upper trunk brachial plexopathy.

  - *Test*: Ask the patient to put his hands on hips. Hold his elbow and instruct him to force his elbow backward against resistance. While doing so, you can palpate the muscle with your fingertip under the vertebral border of scapula.
- *Serratus Anterior* ***(Fig. 9)***:
  - *Nerve*: Long thoracic nerve
  - *Root*: C5–C7
  - *Action*: The serratus anterior protracts the scapula, pulling it anteriorly
  - *Test*: Stand behind the patient in front of a wall. Ask the patient to keep his arms straight with hands at shoulder level and then push against the wall. Look at the vertebral border of scapula. If the muscle is weak, the inferior angle is shifted medially and the vertebral border on the entire scapula lifts off the thoracic wall (winging).

Winging of scapula ***(Fig. 10)***:
Normally, when the arms are raised, the medial border of scapula remains close to the thoracic wall. But, if there

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FIG. 9: Serratus anterior.



FIG. 10: Winging of scapula.

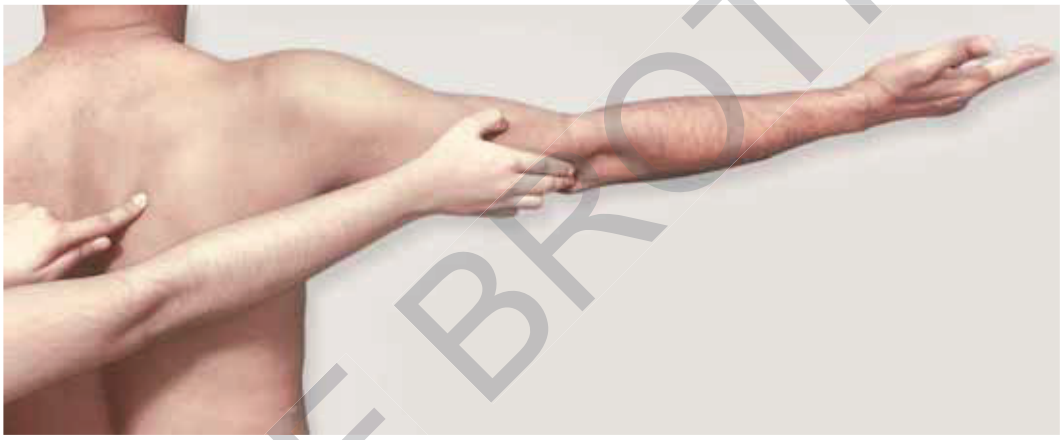


FIG. 11: Trapezius.

is weakness of the serratus anterior or the trapezius, the vertebral border of scapula or the entire scapula lifts off the thoracic wall. This is called "winging of scapula".

Key Points

- In serratus anterior weakness, winging is most prominent when the patient tries to elevate the arm in front in sagittal plane of the body.
- In trapezius weakness, winging is more obvious when the patient tries to abduct the arm in the coronal plane of the body.

- *Trapezius* ***(Fig. 11)***:
  - *Nerve*: Spinal part of the accessory nerve and ventral rami of C3 and C4
  - *Root*: C3 and C4
  - *Action*:
    - Upper fibers: Elevation of scapula
    - Middle fibers: Retraction of scapula
    - Lower fibers: Depression of medial part of spine of scapula
    - Abduction at shoulder joint beyond 90°
  - *Test*: Traditionally, trapezius is tested by asking the patient to shrug the shoulders against resistance. However, it may be erroneous as shoulder shrugging is predominantly done by levator scapulae. Hence, the better method of testing the upper fibers of trapezius is by asking the patient to try to approximate the occiput to the acromion against resistance. For examining the middle and lower fibers of trapezius, instruct the patient to place the abducted arm horizontally with palm upward. Now, the examiner should attempt to push the elbow forward against patient's resistance and palpate the muscle.

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- *Pectoralis major* ***(Figs. 12A and B)***:
  - *Nerve*: lateral and medial pectoral nerves
  - *Root*: C5-T1
  - *Action*: mainly adduction and internal rotation of the shoulder
  - *Test*:
    - Sternal head: Ask the patient to put the hand on the hip and try to push the arm inward against resistance.
    - Clavicular head: Ask the patient to stretch the arms forward and raise them above 90° at the level of face. Now instruct the patient to try to adduct the arms against resistance.
- *Latissimus dorsi* **(Fig. 13)**:
  - *Nerve*: Thoracodorsal nerve
  - *Root*: C6–C8
  - *Action*: Adducts raised arm against resistance (e.g., during climbing). The posterior axillary fold is formed by latissimus dorsi and teres major. When the arm is adducted against resistance, the muscle belly becomes prominent and can be palpated.
  - *Test*:
    - Ask the patient to keep the arm abducted above 90°. Then resist the patient's attempt to adduct the horizontally raised upper arm.
    - Ask the patient to cough and palpate the muscle belly in posterior axillary fold.



FIG. 12A: Pectoralis major sternal head.

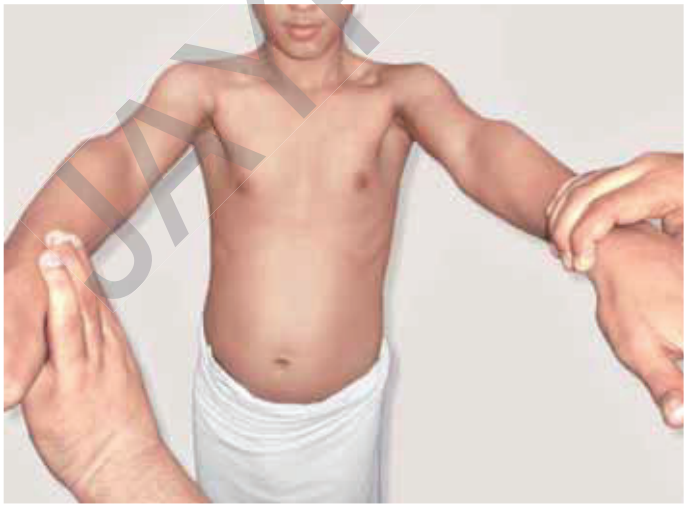


FIG. 12B: Pectoralis major clavicular head.

Muscles Acting on the Elbow Joint

- *Biceps* **(Fig. 14)**:
  - *Nerve*: Musculocutaneous nerve
  - *Root*: C5 and C6
  - *Action*: Flexion of elbow and supination of forearm (when elbow is flexed at 90°)
  - *Test*: Ensure that the forearm is supinated and then ask the patient to flex his elbow against resistance.

  (*Beware of trick movements that involve midpronation of the arm to use brachioradialis*)
- *Brachioradialis* ***(Fig. 15)***:
  - *Nerve*: Radial nerve
  - *Root*: C5 and C6
  - *Action*: Flexion of elbow (The muscle is most active when the forearm is in some pronation)
  - *Test*: Hold the patient's forearm and wrist with the forearm in mid-prone position (i.e., midway between pronation and supination, thumb up). Ask the patient to pull his thumb toward his nose against resistance.
- *Triceps* ***(Fig. 16)***:
  - *Nerve*: Radial nerve
  - *Root*: C6–C8
  - *Action*: Extensor of elbow
  - *Test*: Hold the patient's elbow and wrist and place the elbow midway between flexion and extension position. Ask the patient to extend elbow against resistance.



FIG. 13: Latissimus dorsi.

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FIG. 14: Biceps.



FIG. 15: Brachioradialis.



FIG. 16: Triceps.



FIG. 17: Extensor carpi radialis.

Key Points

- Biceps, brachioradialis, and brachialis are flexors of elbow
- Biceps flexes elbow—best in supinated position of forearm
- Brachioradialis flexes elbow—best in midprone position of forearm
- Brachialis flexes the elbow—regardless of forearm position

Muscles Acting on the Wrist Joint and Fingers

- *Extensor carpi radialis longus* ***(Fig. 17)***:
  - *Nerve*: Radial nerve
  - *Root*: C6 and C7
  - *Action*: Extension and radial deviation of wrist
  - *Test*: Forearm is fully pronated. Then ask the patient to dorsiflex the wrist toward the radial side against resistance.
- *Extensor carpi ulnaris* ***(Fig. 18)***:
  - *Nerve*: Radial nerve
  - *Root*: C7
  - *Action*: Extension and ulnar deviation of wrist
  - *Test*: Same as before, but dorsiflexes the wrist toward the ulnar side.
- *Extensor digitorum*:
  - *Nerve*: Posterior interosseous nerve and branch of radial nerve
  - *Root*: C7
  - *Action*: Extension of the metacarpophalangeal (MP) joints, digits 2–5
  - *Test*: The forearm is pronated, wrist is stabilized and patient keeps his finger extended. Then instruct the patient to resist your attempt to flex the fingers at the MP joints.
- *Flexor carpi radialis* ***(Fig. 19)***:
  - *Nerve*: Median nerve
  - Root: C6 and C7
  - *Action*: Flexion and radial deviation of wrist
  - *Test*: Forearm is supinated. Then ask the patient to flex the wrist toward the radial side against resistance directed toward the thumb.

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FIG. 18: Extensor carpi ulnaris.



FIG. 19: Flexor carpi radialis.



FIG. 20: Flexor carpi ulnaris.



FIG. 21: Flexor digitorum superficialis.

- *Flexor carpi ulnaris* ***(Fig. 20)***:
  - *Nerve*: Ulnar nerve
  - *Root*: C8
  - *Action*: Flexion and ulnar deviation of wrist
  - *Test*: Same as before, flexes the wrist toward the ulnar side against resistance directed toward the hypothenar region.
- *Flexor digitorum superficialis* ***(Fig. 21)***:
  - *Nerve*: Median nerve
  - *Root*: C7–T1
  - *Action*: Flexes middle phalanges of 2nd, 3rd, 4th, and 5th finger
  - *Test*: Ask the patient to flex the finger at the interphalangeal joint against resistance with the proximal phalanx fixed.
- *Flexor digitorum profundus I and II* ***(Fig. 22)***:
  - *Nerve*: Anterior interosseous nerve, branch of median nerve (C7–C8)
  - *Action*: Flexes terminal phalanges of the 2nd and 3rd finger
- *Flexor digitorum profundus III and IV*:
  - *Nerve*: Ulnar nerve (C7–C8)
  - *Action*: Flexes terminal phalanges of the 4th and 5th finger
  - *Test*: Ask the patient to flex the distal phalanx against resistance with the middle phalanx fixed

Muscles of the Thumb

- Movements in thumb include abduction, adduction, flexion, extension, and opposition **(Figs. 23 to 27)**.
- Abduction and adduction movements occur at carpometacarpal joint (CMJ). Abduction is upward movement of thumb at right angle to the plane of palm. Adduction is movement of thumb which brings first and second metacarpals together.

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FIG. 22: Flexor digitorum profundus.

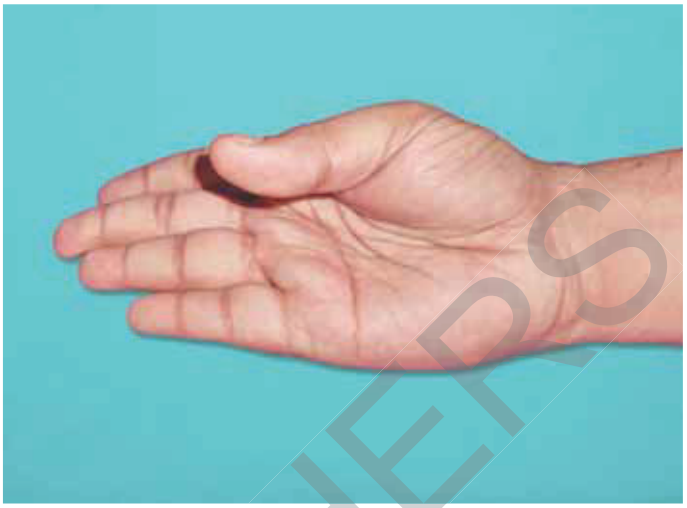


FIG. 23: Thumb flexion.



FIG. 24: Thumb extension.



FIG. 25: Thumb abduction.

- Flexion and extension movements occur at the level of interphalangeal and MP joints. Extension is movement of thumb away in the plane of palm.
- Opposition is circumduction of the thumb so that palmar surface of the tip of the thumb touches the palmar surface of the tip of the small finger. It occurs at the first CMJ.

Forearm Muscles which Control Thumb (Extrinsic Muscles)

- Abductor pollicis longus (APL)
- Extensor pollicis longus (EPL)
- Extensor pollicis brevis (EPB)
- Flexor pollicis longus (FPL)

Thenar Eminence Muscles which Control Thumb (Intrinsic Muscles)

- Abductor pollicis brevis (APB)
- Opponens pollicis (OP)
- Flexor pollicis brevis (FPB)



FIG. 26: Thumb adduction.

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FIG. 27: Thumb opposition.



FIG. 28: Abductor pollicis longus.



FIG. 29: Extensor pollicis longus.



FIG. 30: Extensor pollicis brevis.

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Abductor Pollicis Longus (Fig. 28)

- *Nerve*: Posterior interosseous branch of radial nerve
- *Root*: C7 and C8
- *Action*: Abduction of the metacarpal of the thumb at carpometacarpal joint (CMC).
- *Test*: Ask the patient to maintain his thumb in abduction against resistance.
- Resistance is applied on the distal end of the first metacarpal in the direction of adduction.

Extensor Pollicis Longus (Fig. 29)

- *Nerve*: Posterior interosseous branch of radial nerve
- *Root*: C7 and C8
- *Action:* Extension of thumb at all joints:
  - At distal phalanx (alone)
  - At MP joint and CMC (in concert with EPB and APL)
- *Test*: Ask the patient to extend the thumb at the interphalangeal joint against resistance while the proximal phalanx is immobilized.

Extensor Pollicis Brevis (Fig. 30)

- *Nerve:* Posterior interosseous branch of radial nerve
- *Root:* C7 and C8
- *Action:* Extension of MP joint of thumb
- *Test:* Ask the patient to extend the thumb at the metacarpophalangeal joint against resistance while the metacarpal bone is immobilized.

Flexor Pollicis Longus (Fig. 31)

- *Nerve:* Anterior interosseous branch of the median nerve
- *Root:* C8 and T1
- *Action:* Flexes the terminal phalanx of the thumb
- *Test:* Ask the patient to flex the distal phalanx of the thumb against resistance while the proximal phalanx is immobilized.

Flexor Pollicis Brevis

- *Nerve*:
  - Superficial head—median nerve
  - Deep head—ulnar nerve



FIG. 31: Flexor pollicis longus.



FIG. 32: Abductor pollicis brevis.

- *Root*: C8 and T1
- *Action*: Flexion of the MP and CMC joints of the thumb
- *Test:* Ask the patient to flex the proximal phalanx of the thumb against resistance at MP joint.

Abductor Pollicis Brevis (Fig. 32)

- *Nerve*: Median nerve
- *Root*: C8 and T1
- *Action*: Abduction of the metacarpal of thumb
- *Test*: During testing of abduction, both the patient and the physician can easily confuse between abduction and extension. Therefore, to avoid such confusion, place a pencil between the thumb and palm perpendicular to the palm. Now, ask the patient to raise the thumb vertically against resistance, with the thumb parallel to the pencil. In fully abducted position, the thumbnail lies perpendicular to the palm. If there is no abduction, the thumb is adducted with the thumbnail parallel to the fingernails.

Opponens Pollicis (Fig. 33)

- *Nerve*: Median nerve
- *Root*: C8 and T1
- *Action*: The opposition movement is a combination of abduction, flexion, and medial rotation of the thumb
- *Test*: Ask the patient to touch the little finger with the thumb against resistance (the plane of thumbnail should be parallel to that of the palm). Apply resistance at the head of the first metacarpal.

Adductor Pollicis (Fig. 34)

- *Nerve*: Ulnar nerve
- *Root*: C8 and T1
- *Test*: The patient resists withdrawal of a piece of paper held between the thumb and the palm as the examiner tries to extract it. *When the adductor* pollicis is weak, patient may flex the interphalangeal joint with the help of FPL and thus tries to secure the paper with the tip of the thumb (Froment's sign). This is found in ulnar neuropathy.



FIG. 33: Opponens pollicis.

Intrinsic Muscles of the Hand and Fingers

Lumbricals (Fig. 35)

- *Nerve*:
  - Lumbrical I and II—median nerve
  - Lumbrical III and IV—ulnar nerve
- *Root*: C8 and T1
- *Action*:
  - Flexion of MP joint
  - Extensor of interphalangeal joints
- *Test*: Ask the patient to resist extension of the MP joints while the interphalangeal joints remain extended. To avoid substitution of lumbricals by long finger flexors, make sure that interphalangeal joints are fully extended.
- *Another test*: Patient resists flexion of the terminal phalanx.

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FIG. 34: Adductor pollicis.



FIG. 35: Lumbricals.



FIG. 36: Palmar interossei.



FIG. 37: Dorsal interossei.

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Interossei

- *Nerve*: Ulnar nerve
- *Root*: C8 and T1
- *Actions*:
  - Palmar interossei—adduction of fingers
  - Dorsal interossei—abduction of fingers
    (Remember the mnemonic—DAB and PAD; dorsal—abduction/palmar—adduction)
- Test:
  - *Palmar interossei* ***(Fig. 36)***:
    - Ask the patient to bring the fingers together with the fingers remaining straight. Patient resists attempts to abduct the fingers.
    - *Card test*: The patient holds a card between two adjacent fingers and resists attempts to pull it out.
  - *Dorsal interossei* ***(Fig. 37)***:
    - Demonstrate to the patient how to spread out his fully extended fingers. Once the patient has done so, instruct him to resist the examiner's attempt to bring the fingers together (i.e., adduction).
  - *First dorsal interossei*:
    - *Test:* Place the patient's hand flat on a table. Next, instruct the patient to abduct the index finger against resistance.

Abductor Digiti Minimi (Fig. 38)

- *Nerve*: Ulnar
- *Root:* T1
- *Action:* Abduction of the fifth digit away from ring finger
- *Test:* The patient places the back of his hand on the table. Then ask the patient to abduct little finger against resistance.

Opponens Digiti Minimi

- *Nerve*: Ulnar nerve
- *Root*: C8 and T1
- *Test*: Ask the patient to touch the tip of the thumb with the tip of the little finger against resistance. Apply resistance on the palmar surface of the fifth metacarpal.



FIG. 38: Abductor digiti minimi.

Movements and Muscle Groups of the Upper Extremities

- *Movements of scapula*:
  - Elevation—upper fibers of trapezius and levator scapulae
  - Depression—lower fibers of trapezius, pectoralis minor, and subclavius
  - Retraction (brace the shoulder backward, i.e., movement of scapula away from the chest wall)—rhomboid and middle fibers of trapezius
  - Protraction (pull it anteriorly, i.e., movement of scapula toward the chest wall)—serratus anterior and pectoralis minor

The principal movements at shoulder joint include abduction, adduction, flexion, extension, internal rotation, and external rotation. These movements occur in the plane of body of scapula rather than the plane of entire body.

- *Shoulder abduction* (anterolateral movement of arm away from trunk in the plane of body of scapula):
  - First 15°—supraspinatus
  - Up to horizontal (90°)—deltoid
  - Above horizontal (>90°)—upper fibers of trapezius
- *Shoulder adduction* (posteromedial movement of arm toward trunk in the plane of body of scapula):
  - Pectoralis major
  - Latissimus dorsi
- *Shoulder flexion* (forward and medial movement of arm in the plane of glenoid cavity, i.e., midway between coronal and sagittal plane):
  - Deltoid (anterior fibers), pectoralis major, biceps, and coracobrachialis
- *Shoulder extension* (backward and lateral movement of arm in the plane of glenoid cavity)
  - Deltoid (posterior fibers), latissimus dorsi, and triceps
- *External rotation of shoulder* (lateral movement of hand with midflexed elbow)
  - Infraspinatus
  - Teres minor
- *Internal rotation of shoulder* (medial movement of hand with midflexed elbow)
  - Subscapularis
  - Teres major

Movements at Elbow

Elbow Joint

- *Flexion*: Biceps, brachioradialis, and brachialis
- *Extension*: Triceps

Radioulnar Joint

In semiflexed and midprone position of the elbow, the palm is turned upward in supination and downward in pronation.

- *Supination*:
  - Supinator and biceps (with forearm in flexion)
  - Brachioradialis (with forearm in extension)
  - *Test*: Ask the patient to supinate against examiner's resistance
- *Pronation*: Pronator quadratus and pronator teres
  - *Test*: Ask the patient to pronate against the examiner's resistance

Movements at Wrist

- *Flexion*:
  - Flexor carpi radialis
  - Flexor carpi ulnaris
- *Extension*: Extensor carpi radialis longus and brevis and extensor carpi ulnaris

Movement of Fingers

- *Flexion of fingers*:
  - *At metacarpophalangeal joint (MCP)*:
    - Lumbricals
    - Interossei
  - *At proximal interphalangeal (PIP) joint*: Flexor digitorum superficialis
  - *At distal interphalangeal (DIP) joint*: Flexor digitorum profundus
- *Extension of fingers*:
  - At MCP:
    - Extensor digitorum communis (EDC)
    - Extensor indicis proprius (EIP) for index finger
    - Extensor digiti minimi (EDM) for little finger
  - At PIP and DIP:
    - Lumbricals
    - Interossei
    - EDC (through the extensor expansion complexes)
- *Abduction of fingers*: Dorsal interossei (abduction of little finger—by abductor digiti minimi)
- *Adduction of fingers*: Palmar interossei [Weakness of intrinsic muscles of hand leads to loss of MCP joint flexion, loss of PIP and DIP joint extension along with loss of abduction and adduction of fingers. Due to relative preservation of long flexors and EDC, the MCP joint in held in extension and the PIP and DIP joints are flexed (claw hand)]

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Muscles of Neck

Neck Flexors

Sternocleidomastoid (SCM), platysma, suprahyoid, infrahyoid, scalene, and the prevertebral group of muscles (longus colli and capitis rectus capitis).

Test

Position of patient—sitting or supine. Apply extension force to the forehead. Ask the patient to place the chin on the chest against resistance.

Neck Extensors

Trapezius, paravertebral muscles (that lie in between the transverse and spinous process of vertebrae, e.g., splenii, erector spinae, transversospinalis, interspinal, and intertransverse)

Test

Position of patient—sitting or prone. Ask the patient to extend the neck against resistance applied to the occiput.

Sternocleidomastoid (SCM)

Function

- *One SCM*:
  - Turns the head to the contralateral side
  - Tilts the head to the ipsilateral side
- Both SCM acting together:
  - Flex the neck

Test

- *One SCM*:
  - Ask the patient to turn the head fully to one side and keep it there. Now you try to bring the head back to midline. You can see and feel the contraction of the SCM.
- *Both SCM*:
  - Same as test of neck flexor power. If one SCM is weak, the head will deviate toward the weak side.

Trapezius

Already discussed earlier.

Muscles of Thorax

- *Major muscles*:
  - Internal and external intercostals
  - Diaphragm
- *Nerve supply*:
  - *Internal and external intercostals*: Intercostal nerve (anterior division of 12 thoracic spinal nerves)
  - *Diaphragm*: Phrenic nerve (C3–C5)

Action

- *During quiet respiration:*
  - Intercostals—increase the anteroposterior and transverse diameters of thorax
  - Diaphragm—increases the vertical diameter of thorax

Test

Observe the movements of the ribs during inspiration and expiration. In intercostals weakness, there may be retraction of intercostal spaces during inspiration with alternate bulging and retraction of the epigastrium (abdominal breathing) due to increased diaphragmatic contraction.

How to test diaphragmatic function?

Place your one hand lightly over the patient's abdomen in the epigastric region, just below the xiphoid process. Ask the patient to inhale with maximum effort and hold it against your resistance given in a downward direction.

- **In diaphragmatic paralysis (bilateral)**:
  - Epigastrium does not bulge during inspiration
  - Inability to cough and sniff

Muscles of the Abdomen

- Rectum abdominis, pyramidalis, transverses abdominis and obliques
- Nerve supply—anterior division of T5 to L1

Test

The patient lies supine and tries to raise his head against resistance. In presence of weakness of neck or hip flexion, first you should stabilize the neck and pelvis respectively before proceeding for the test. Normally, a person can complete range of motion till the inferior angles of scapulae are off the table. In paralysis of lower abdominal muscles (e.g., in T10 myelopathy), the umbilicus is pulled upwards (Beevor's sign).

Extensors of the Spine

- Erector spinae, paravertebral, or paraspinal muscles
- Nerve—posterior rami of the spinal nerves

(Paraspinal muscle involvement indicates nerve root disease and thus helps to differentiate nerve root disease from plexus or peripheral nerve disease.)

Test

Patient lies prone, tries to raise the head, shoulders, and chest off the table (clears umbilicus). *Weakness of spine extensors leads to lordotic posture.*

Explanation: When a person bends forward, spine extensors contract to prevent tripping over and thus help to balance the spine above the pelvis. Patients with weak spine extensors lean backward as a compensatory mechanism and thus avoid falling forward.

Muscles of the Hip Girdle

Iliopsoas (Fig. 39)

- *Nerve*:
  - Lumbosacral plexus (L1–L4) for the psoas portion
  - Femoral nerve (L2–L4) for the iliacus portion
- *Action*:
  - Flexion of hip
  - Flexion of trunk (when the legs are fixed)

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- *Test*: May be done in sitting or supine position.
  - *Sitting position*: Patient sits with thighs fully supported on table, legs hanging over the edge, and should not lean backward. You give resistance over distal thigh in a downward direction toward the floor and then ask the patient to lift the thigh of the table.
  - *Supine position*: The patient lies on his back, the knee is flexed, and the leg rests on the examiner's arm. Now ask the patient to flex the thigh against resistance.

Gluteus Maximus (Fig. 40)

- *Nerve*: Inferior gluteal nerve
- *Root*: L5–S2
- *Action*: Hip extension and hip internal rotation
- *Test*: The patient lies prone with the knee in flexed position. Please one hand over the posterior thigh just above knee and give resistance in a downward direction. Ask the patient to lift off the thigh against resistance.

Flexing the knee helps to isolate the function of gluteus maximum and thus minimizes any contribution to hip extension by hamstring.

Gluteus Medius (Fig. 41)

- *Nerve*: Superior gluteal nerve
- *Root*: L4–S1
- *Action*: Hip abduction and hip internal rotation
- *Test*:
  - The patient lies prone with the knee flexed. Then he pushes the leg outward against resistance.
  - The patient lies supine with the knee extended. Ask the patient to move the extended leg outward against resistance (contraction of the gluteus medius can be palpated)

Adductors of Hip (Adductor Longus, Brevis, and Magnus) (Fig. 42)

- *Nerve*: Obturator nerve (L2–L4)
- *Test*: Can be tested in sitting or supine position. The patient tries to bring the legs together as the examiner tries to keep them apart.

Internal Rotation of Hip

- *Main muscles*: Hip abductors (i.e., gluteus medius, gluteus minimus, and tensor fascia lata)
- *Test*: Patient lies supine with the leg extended. Ask the patient to rotate the foot medially as if to touch the big toe to bed against resistance.

External Rotation of Hip

- *Main muscle*: Gluteus maximus
- *Test*: Similar to those for testing internal rotation except that the patient rotates the foot laterally.

Muscles of the Thigh and Knee

Hamstrings (Biceps, Semitendinosus, and Semimembranosus) (Fig. 43)

- *Nerve*: Sciatic nerve
- *Root*: L4, L5, S1, and S2

(There are two heads of biceps femoris—long and short. The long head is supplied by the tibial division and the short head is supplied by the peroneal division of sciatic nerve. If electromyography detects involvement of short head of biceps femoris it indicates lesion involving the peroneal division of the sciatic nerve rather than lesion involving peroneal nerve at the knee.)

- *Action*:
  - Flexion of knee
  - Hip extension
- *Test*: The patient, lying prone, tries to flex the knee against resistance; you can palpate the muscle contractions.

Quadriceps Femoris

It is composed of four muscles, i.e., rectus femoris, vastus lateralis, vastus medialis, and vastus intermedius (**Fig. 44**).



FIG. 39: Illio psoas.



FIG. 40: Gluteus maximus.

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FIG. 41: Gluteus medius.



FIG. 42: Hip adductors.



FIG. 43: Hamstrings.



FIG. 44: Quadriceps.

- *Nerve*: Femoral nerve
- *Root*: L2–L4
- *Action*: Extension of knee
- *Test*:
  - The patient, lying supine with the knee semiflexed, tries to extend the knee against resistance.
  - Patient sits on a table. Ask the patient to straighten his knee and hold it against resistance.

Muscles of the Lower Leg and Ankle

Tibialis Anterior (Fig. 45)

- *Nerve*: Deep peroneal nerve
- *Root*: L4–L5
- *Action*:
  - Dorsiflexion of foot (at talocrural joint)
  - Inversion of foot (at subtalar and midtarsal joints)
- *Test*:
  - Patient lying on his back, with the legs extended tries to dorsiflex the foot against resistance of the examiner's hand placed across the dorsum of the foot.
  - Ask the patient to stand on the heels, raising the toes as high as possible.

[Weakness of dorsiflexion leads to foot drop. In order to clear the toes during stride phase, patient raises the affected leg higher off the ground by increased flexion at hip and knee (steppage gait). During normal walking, heel strikes the ground first. In presence of foot drop, the toes touch the floor first followed by the heel leading to an audible double slap.]

Tibialis Posterior (Fig. 46)

- *Nerve*: Tibial nerve (L5–S1) (medial popliteal nerve)
- *Action*:
  - Foot inversion (elevation of the inner border of foot which turns the sole medially)
  - Ankle plantar flexion (accessory)

During inversion, the sole turns medially, with elevation of the inner border of the foot.

- *Test*: Ask the patient to plantar flex the foot slightly and then try to invert it against resistance.

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FIG. 45: Tibialis anterior.



FIG. 46: Tibialis posterior.

Peronei (Peronei Longus and Brevis)

- *Nerve*: Superficial peroneal nerve
- *Root*: L5 and S1
- *Action*:
  - Foot eversion (elevation of the outer border of foot which turns the sole laterally)
  - Ankle plantar flexion (accessory)
- *Test*: Ask the patient to evert the foot against resistance applied to the lateral border of the foot.

Gastrocnemius (Fig. 47)

- *Nerve*: Tibial nerve (medial popliteal nerve)
- *Root*: S1
- *Action*: Plantar flexion of foot
- *Test*:
  - Ask the patient to plantar flex the foot while you offer resistance by applying pressure against the sole of the foot.
  - Ask the patient to stand on tiptoe. (Normally, a person can support the entire body weight on one tiptoe). You can easily compare the power of plantar flexors between two sides.



FIG. 47: Gastrocnemius.

Muscles of the Foot and Great Toe

Extensors of Toe

- Extensor digitorum longus (EDL)
- Extensor digitorum brevis (EDB)
- Extensor hallucis longus (EHL)
- Extensor hallucis brevis (EHB)
- *Nerve*: Deep peroneal nerve
- *Root*: EHL—L5, EDL and EDB—L5–S1
- *Action*:
  - EHL—extensor of the large toe and dorsiflexor of foot
  - EDL—extensor of the four lateral toes and dorsiflexor of the foot
  - EDB—extensor of the great toe and three medial toes
- *Test*:
  - *Extensor hallucis longus* ***(Fig. 48)***: Ask the patient to dorsiflex the great toe against resistance. Try to push down the distal phalanx of his great toe.
  - *Extensor digitorum longus*: Ask the patient to dorsiflex the toes against resistance applied over the dorsal surface of the proximal phalanges of the toes. You can palpate the tendons of EDL over dorsum of metatarsals.
  - *Extensor digitorum brevis* ***(Fig. 49)***: Same method as in EDL. You can observe or feel the prominent bulk of EDB on the dorsolateral aspect of foot just in front of the lateral malleolus.



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FIG. 48: Extensor hallucis longus.



FIG. 49: Extensor digitorum brevis.

Flexor Digitorum Longus

- *Nerve*: Tibial nerve
- *Root*: L5, S1, and S2
- *Test*: Ask the patient to flex the terminal phalanges against resistance

Movements and Muscle Groups of Lower Extremities

Hip Joint

- *Flexors*: Iliopsoas, rectus femoris, sartorius, and tensor fascia lata
- *Extensors*: Gluteus maximus, gluteus medius and minimus, and hamstrings
- *Abductors*: Gluteus medius, gluteus minimums, and tensor fasciae lata
- *Adductors*: Adductor longus, brevis, and magnus
- *Internal rotation*: Hip abduction, i.e., gluteus medius, gluteus minimus, and tensor fasciae lata
- *External rotation*: Gluteus maximus

Knee Joint

- *Flexors*: Hamstrings, popliteus, gracilis, and sartorius
- *Extensors*: Quadriceps femoris

Ankle Joint

- *Planter flexion*: Gastrocnemius and soleus
- *Dorsiflexion*: Tibialis anterior, extensor digitorum longus, and extensor hallucis longus
- *Inversion*: Tibialis posterior (best in plantar flexed position), tibialis anterior (in dorsiflexed position)
- *Eversion*: Peroneus longus, brevis and tertius and the extensor digitorum longus

EXAMINATION OF REFLEXES

Deep Tendon or Muscle Stretch Reflexes

What to Do?

- Use a high quality, heavy, and soft rubber percussion hammer.
- The patient should be relaxed; the target muscle is to be placed in optimum position which is usually about midway in the range of motion of the muscle. The position of the extremities on the two sides should be symmetrical.
- Use your wrists flexibly to ensure that hammer strike is quick, sharp, and forceful.
- Ask the patient to keep the head straight. If the patient looks to one side, that may affect reflex tone, particularly in the arms (tonic neck reflex).
- You can observe or feel the contraction of the target muscle.

Reflexes can be graded as follows:

- 0 = Absent
- 1+ = Present but diminished
- 2+ = Normal
- 3+ = Increased but not a pathological degree
- 4+ = Pathologic, often with clonus

Reinforcement

If a deep tendon reflex appears diminished or absent, reinforcement techniques are used.

- *For upper limbs*: Ask the patient to clench the teeth tightly or clench one fist while the other arm is being tested or take a deep breath.
- *For lower limbs*: In addition to above measures, Jendrassik maneuver is useful. The patient is asked to

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hook the flexed fingers of two hands against one another. Then he is asked to pull the hands apart as strongly as possible at the moment of application of the tap on the tendon.

Biceps Jerk (Fig. 50)

- *Nerve*: Musculocutaneous nerve
- *Root*: C5 and C6
- *Test*: The elbow is semiflexed with the forearm semiprone, may lie on patient's abdomen or on the left hand of the examiner. Place the palmar surface of your extended thumb or finger on biceps tendon and then strike the finger sharply with the hammer.
  - *Normal response*: Visible contraction of biceps and flexion of elbow joint. Biceps is also a supinator. So some supination may occur.
  - *Abnormal response*:
    - *Reflex spread (irradiation)*: Accompanying wrist and finger flexion, adduction of the thumb.
    - *Inverted reflex:* Biceps reflex is absent but there is triceps response. Localizes lesion in cervical cord at C5 level [lower motor neuron (LMN) lesion at C5 level with upper motor neuron (UMN) signs below this level]

Supinator Jerk (Fig. 51)

- *Nerve*: Radial nerve
- *Root*: C6 and C5
- *Test*: The elbow is flexed at right angle with forearm semi pronated. A sharp tap is applied over the lower end of the radius about two inches above the wrist.
  - *Normal response*: Contraction of brachioradialis and flexion of elbow. Slight flexion of finger may occur.
  - *Abnormal response*:
    - *Exaggerated supinator jerk*: Supinator jerk present plus accompanying flexion of wrist and fingers.
    - *Inverted supinator jerk*: Supinator jerk absent but finger flexion is present. Usually associated with absent biceps jerk but exaggerated triceps and finger jerk. Inverted supinator jerk localizes the lesion in cervical cord at C5–C6 level.
- *Common mistake*: At midforearm level, the brachioradialis muscle become tendinous. Instead of tendon, if you strike the muscle belly of brachioradialis, you may elicit a local contraction (idiomuscular contraction). This is not a deep tendon reflex. Remember while we examine deep tendon reflex our basic objective is to lengthen the muscle by stretching its tendon.

Triceps Jerk (Fig. 52)

- *Nerve*: Radial nerve
- *Root*: C7
- *Test*: The elbow is placed at right angle with the forearm resting across the chest. A sharp tap is applied over the triceps tendon about 2″ (5 cm) above the elbow.
  - *Normal response*: Extension of the elbow and visible contraction of the triceps.



FIG. 51: Supinator jerk.



FIG. 50: Biceps jerk.



FIG. 52: Triceps jerk.

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- *Abnormal response:* Paradoxical or inverted triceps jerk
  - Flexion of elbow by tapping the triceps tendon.
  - Localizes lesion in cervical cord at C7 and C8 segments

Finger Flexion Jerk

- *Nerve*: Median nerve and ulnar nerve
- *Root*: C8–T1
- *Test*: Place patient's hand on a table with palm upward and fingers slightly flexed. Gently interlock your fingers against the patient's fingers. Strike the back of your fingers with a hammer.
  - *Normal response*: Slight flexion of all four fingers and of the interphalangeal joint of the thumb.

Pectoral Reflex

- *Nerve*: *Lateral and medial pectoral nerve*
- *Root*: *C5–T1*
- *Test:* Place the patient's arm in midposition between abduction and adduction. Place the tip of your finger on the pectoralis muscle on the anterior margin of axilla and then tap on the finger.
  - *Normal response*: Adduction and slight internal rotation of the arm at the shoulder. You can feel the contraction of the muscle.
  - *Abnormal response*: Exaggerated pectoral reflex indicates spinal cord lesion above the level of C5.

Knee Jerk (Fig. 53)

- Nerve: Femoral nerve
- *Root*: L3–L4
- *Test*: The patient lies supine. Partially flex the knee by placing one hand beneath it and then apply a sharp tap over patellar tendon. If you place your hand under both knees in order to flex them together, you can compare knee jerk between two sides.
  - *Normal response*: The knee is extended and there is visible contraction of the quadriceps muscles.
    Alternative methods:
    - Patient sits on the examination table with the legs dangling. Strike patellar tendon just distal to patella.
    - Legs are extended, place your finger just above the patella and strike it in a peripheral direction. This method is helpful when the lower leg is amputated or the patella is removed.
  - *Abnormal response*:
    - *Irradiation of reflex (reflex spread)*: Extension of knee + adduction of hip or bilateral extension of knee
    - *Inversion of knee jerk*: Knee jerk is absent but there is flexion of knee due to flexion of hamstrings. *Cause*: Lesion in nerve or nerve roots innervating quadriceps.

Ankle Jerk (Fig. 54)

- *Nerve*: Tibial nerve
- *Root*: S1 and S2
- *Test*: The patient lies supine with leg externally rotated and slightly flexed at the knee. Dorsiflex the foot slightly by giving passive tension to the tendoachilles and then strike the tendon directly.
  - *Normal response*: Contraction of gastrocnemius and planter flexion of foot.
    Alternative methods:
    - Ask the patient to kneel on a chair so that the feet are hanging loose at right angles. Strike the Achilles tendon directly.
    - Patient lies supine with legs straight. Place your hand against the ball of the foot with the ankles



FIG. 53: Knee jerk.



FIG. 54: Ankle jerk.

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at the 90 degrees. Strike your hand and watch the response.

- *Common mistake*: If ankle jerk appears reduced or absent, always consider whether proper stretching and efficient striking was applied during the procedure. Avoid too little or too much dorsiflexion of the ankle.

Other Reflexes

Hoffmann Reflex (Fig. 55)

Hold the patient's hand with the wrist dorsiflexed and fingers partially flexed. With a sharp, forcible flick downward between your thumb and index finger, the terminal phalanx of middle finger is suddenly flexed followed by sudden release. The rebound of the distal phalanx leads to stretching of finger flexors.

Positive Response

- Thumb flexes and adducts, flexion of index finger, and sometimes other fingers
- Positive response may be found in any state of hyperreflexia. This reflex has little clinical significance except when positive response is found unilaterally (early sign of unilateral corticospinal tract involvement)

Wartenberg's Sign

Ask the patient to supinate his hand with slight flexion of the fingers. Pronate your hand, interlock your similarly flexed fingers with the patient's fingers and then both of you flex your fingers further against each other's resistance.

- *Normal response*: Extension of the thumb
- In *pyramidal tract lesion*: Adduction and flexion of the thumb
- Unilateral findings are more significant

Rossolimo's Sign

Strike the ball of the foot while patient is in a supine position with leg extended and the foot partially dorsiflexed.



FIG. 55: Hoffmann's reflex.

- *Positive response*: Plantar flexion of all the toes.
- *Significance*: Manifestation of hyperactive plantar muscle reflex, similar to finger flexion reflex in the upper limb.

Interpretation of Deep Tendon Reflexes: Some Comments

- Diminished or absent deep tendon reflex suggest LMN lesion, i.e., a breach in any part of the reflex arc. But always take help of reinforcement technique because reflexes that are normal on reinforcement are considered normal.
- In neurogenic illness, loss of deep tendon reflex is out of proportion to wasting and weakness whereas in myopathic illness, loss of reflexes is proportional to wasting and weakness.
- Deep tendon reflexes may be absent in deep coma, heavy sedation, and deep sleep.
- Deep tendon reflexes may be diminished or absent in severe rigidity, spasticity, or muscle contracture due to loss of movements of joints.
- Apart from corticospinal tract lesions, jerks are exaggerated in anxiety states, thyrotoxicosis, and hypocalcemia.

Pendular Knee Jerk

Strike the patellar tendon while the legs are hanging free. In cerebellar disease, there is a series of to-and-fro pendular oscillation of lower limb (>3 oscillation) (1–2 oscillation of leg is normal).

Explanation: Hypotonia and lack of restraining influences in limb muscles.

"Hung" reflex—may be observed in chorea (due to prolonged contraction of quadriceps, the knee is held in extension for a longer period)

Superficial Reflexes

The common superficial reflexes studied in clinical practice are:

- Plantar reflex
- Abdominal reflex
- Cremasteric reflex
- Anal reflex

These reflexes are polysynaptic.

Significance:

- Absent in pyramidal tract lesion
- Help to localize the level of lesion in spinal cord

Abdominal Reflex (Fig. 56)

Test

The patient lies relaxed in supine position with the abdomen uncovered. Lightly scratch the abdominal wall with a blunt object from outside inward at three levels (at, above, and below the umbilicus) preferably at the end of expiration. Normally, the abdominal muscles on the same side contract and draw the umbilicus to the same side.

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FIG. 56: Abdominal reflex.

Segmental Innervation
- Upper quadrants (supraumbilical)—by the intercostal nerves (T7–T9)
- Lower quadrants (infraumbilical)—by intercostal, iliohypogastric, and ilioinguinal nerves (T10–L1)

Abdominal Reflex Absent
Causes: Pyramidal tract lesion above the T7 level, LMN lesion affecting T7–T12 thoracic roots causing a breach of the reflex arc, obesity, multiple pregnancy, previous abdominal surgery, distended bladder, etc.

Abdominal reflex exaggerated in anxiety states.

Abdominal Reflex Inverted
In unilateral abdominal paralysis, if the paralyzed side is stimulated the reflex is absent on that side but produce abdominal muscle contraction on the normal side leading to deviation of the umbilicus to the opposite side (i.e., the normal side).

Fallacy
- Strong, too painful stimuli may produce deep abdominal reflex. Actually this is muscle stretch reflex which is often hyperactive while the superficial abdominal reflex is absent.
- Loss of abdominal reflex does not always correlate with severity of pyramidal tract lesion, e.g., it is lost very early in multiple sclerosis but preserved till late in spastic diplegia and motor neuron disease.

Cremasteric Reflex
Lightly scratch the upper inner part of the thigh in a downward and inward direction. Normally, there is a sharp contraction of the cremasteric muscle, drawing the corresponding testicle upward.

(Do not confuse cremasteric reflex with scrotal reflex which produces a slow and writhing contraction of scrotal skin.)

Segmental Innervation
L1–L2 (Ilioinguinal and Genitofemoral Nerves)

Cremasteric Reflex Absent
Causes:
- Any breach in the corresponding reflex arc or pyramidal tract lesion above that level
- Elderly male, presence of hydrocele, and orchitis.

Anal Reflex
Lightly stroke or scratch the skin in perianal region. Normally, there is contraction of external anal sphincter.

Segmental Innervation
S2–S5 (inferior hemorrhoidal nerve)

Anal Reflex Absent
In cauda equina or conus medullaris lesion due to loss of integrity in the corresponding reflex arc.

Bulbocavernosus Reflex
Squeeze the patient's glans penis. Normally there is contraction of bulbocavernosus muscle which can be palpated with a gloved finger in the rectum.

Segmental Innervation
S2–S4

Bulbocavernosus Reflex Absent
In cauda equina, conus medullaris lesion.

Plantar Reflex
How do you elicit it? (Fig. 57)

Explain to the patient about the procedure and potential discomfort. The patient lies supine with hip and knee extended, heels resting on the bed. In early stage, extensor plantar may disappear when the knee is flexed. If the patient is in sitting position, ask him to extend his knee and hold his foot either by your hand or on your knee.

Now lightly hold the leg just above the ankle and apply a gentle and steady scratch over the outer border of the sole from the heel to the base of the fifth toe and then inward along the heads of the metatarsal (except the first one)

What do you find?
- *Normal response*: The great toe will flex at the metatarsophalangeal joint along with plantar flexion of other toes, sometimes associated with ankle dorsiflexion and contraction of the tensor fasciae lata. The first movement of the great toe on the first stimulation is most important and what happens to the other toes is not important.
- *Extensor plantar response (Babinski response)*: Extension of the great toe of metatarsophalangeal joint, fanning, and extension of other toes. There may be dorsiflexion of the ankle and contraction of the hamstring. There may be isolated extension of the great toe only or extension of the great toe with flexion of other toes. If you repeatedly

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FIG. 57: Plantar reflex.

stimulate the sole, extensor plantar response may disappear. So, observation of the first toe movement on first stimulation is very crucial.

Extensor plantar response occurs in UMN lesion anywhere in spinal cord above L5 or in brain. Extensor plantar response does not necessarily mean a structural lesion of pyramidal tract; rather it indicates dysfunction of the pyramidal system. Due to incomplete myelination of pyramidal tract, the plantar response is extensor below 1 year of age. Extensor plantar response may also be found in deep coma due to any cause, postictally or during sleep. Paralysis of the toe flexors may cause a false positive extensor plantar response.

- *Equivocal plantar response*: Sometimes it is difficult to differentiate between an upgoing toe due to extensor plantar response from an upgoing toe due to voluntary withdrawal. These are called equivocal plantar responses. Contrary to extensor plantar response, voluntary withdrawal usually does not cause dorsiflexion of the ankle, usually associated with plantar flexion of the toes and usually occurs when the stimulus is very uncomfortable to the patient.
- *Mute or silent plantar response*: No elicitable plantar response. This can be found in thick soles, joint deformity, especially hallux valgus, severe weakness of extensor hallucis longus (power less than grade 2), incomplete pyramidal tract lesion, and spinal shock. Even in pyramidal tract lesion, a patient may not have conventional extensor plantar response, if he has concomitant LMN paralysis of toe extension.
- *Asymmetry of plantar response*: One great toe does not flex as crisply as the other great toe.

Segmental Innervation

- *Afferent*: In the S1 root/sural nerve sensory distribution. This explains why a more medial plantar stimulation should be avoided. Actually, this may cause a plantar grasp response leading to toe flexion.
- *Efferent*: Through the L5, S1 roots to the sciatic nerve to its bifurcation. Toe flexors are innervated by the tibial nerve and toe extensors are innervated by the deep peroneal nerve.

What is the physiologic perspective of Babinski sign?

The extension of the great toe is a component of larger synergistic primitive flexor response that helps to withdraw from a noxious stimulus. This is characterized by a triple flexion response—hip, knee, and ankle flexion (dorsiflexion). Maturation of pyramidal system suppresses this flexion response and thus facilitates normal ambulation. In pyramidal tract lesion, this primitive flexion response reappears and Babinski sign is found clinically.

Pseudo-Babinski Sign

Striatal toe in extrapyramidal disorder mimics extensor plantar response. However, contrary to true Babinski sign, there is lack of contraction of hamstring, tibialis anterior, and tensor fasciae lata and can be inhibited by pressure over the base of the great toe.

Alternative Methods to Elicit Plantar Response

- Chaddock's reflex—stimulus on lateral aspect of foot below lateral malleolus
- Oppenheim's reflex—thumb and index finger to drag down the medial side of tibia
- Gordon reflex—squeeze the calf muscles

Primitive Reflexes

These reflexes are normally found in neonates and infants but disappear when the cerebrum sufficiently matures in adult life. In adult life, these reflexes may reappear due to acquired disorders of one or both frontal lobes.

- *Rooting reflex*: When you touch the cheek of the patient, he will turn his lips, mouth, and even head toward the tactile stimulus.
- *Sucking reflex*: Stimulation of the perioral region leads to sucking movement of the lip, tongue, and jaw.
- *Snout reflex*: If you press firmly backward on the philtrum of the upper lip, there will be reflex puckering and protrusion of the lips.
- *Grasp reflex*: Place your index and middle fingers into the patient's palm in between his thumb and forefinger. The patient should be told not to grab the examiner's fingers. Normally, there will be no response. In presence of grasp reflex, there will be flexion and grasping of the fingers. Unilateral presence of grasp reflex localizes the lesion to contralateral frontal lobe whereas bilateral grasp reflex occurs in diffuse disorders of brain, e.g., advanced Alzheimer's disease.
- *Palmomental reflex*: Scratch a blunt point over the thenar eminence of the patient (either from wrist toward thumb or vice versa) and look at the face of the patient. If palmomental reflex is present, there will be contraction of the mentalis and orbicularis oris. Presence of this reflex becomes significant, if there is marked exaggeration of

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the response or there is significant asymmetry between the two sides.
- *Glabellar tap*: The examiner stands behind the patient and repeatedly taps the glabellar prominence (between the eye brows) with the tip of his index finger. In a normal subject, the blink response stops after three to four taps.

EXAMINATION OF GAIT, STANCE, AND POSTURE

Examination sequence:
- Ask the patient to stand with feet close together and eyes open.
- Ask him to close both eyes with feet close together but you must be prepared to support him if there is a tendency to fall.
- Ask the patient to walk.
- Always examine the posture of the patient while sitting, standing, and walking.
- Gait has been discussed in details under the chapter, "Approach to Gait Disorders".

What happens in shoulder girdle weakness?
Due to weakness of interscapular muscles (e.g., rhomboids and trapezius), scapula slides forward and outward. This leads to rotation of the whole upper limb with dorsum of hand facing forward at rest.

What happens in weakness of hip and back extensors?
Normally continuous activity of hip and back extensors helps us to maintain the erect posture by placing the center of gravity slightly forward of the hip joint. When these muscles are weak, the patient tries to ensure that the weight of the body falls behind the hip joint by throwing the shoulder back. This leads to exaggerated lordosis and protuberant abdomen.

What happens in hip abductor weakness?
Patient waddles when hip abductors (gluteus medius) are weak on both sides. Normally, when one lower limb is lifted in the swing phase, the other takes the entire body weight. During the stance phase, as the heel hits the ground, the hip abductors counteract the shock by stabilizing the pelvis. The pelvis tilts downward on the weight-bearing extremity and hikes up on the non-weight-bearing extremity. In unilateral hip abductor weakness, when the heel of the affected side hits the ground, the weak hip abductors on that side cannot stabilize the pelvis leading to downward tilting of pelvis instead of upward on the non-weight-bearing extremity. In an attempt to lessen this effect, the person compensates by lateral tilt of the trunk away from the affected hip. Bilateral hip abductor weakness produces alternating lateral trunk movements leading to waddling gait.

What happens in hip girdle (hip extensor and quadriceps) weakness?
Following sequence of events occur when a patient tries to stand from a supine or seated position:



FIG. 58: Genu recurvatum.

- First puts the hands on the floor and pushes on the ground with extended forearms.
- Next, straighten the knees to lift the hips high in the air (called "butt first" maneuver) and an angle is formed with buttocks at the apex and both hands and feet on the floor forming the base.
- Then, places one hand on a knee, thus support formed by both feet and one hand (tripod sign). In more severe weakness, the other hand and knee are also used and pushes on the things with both hands to lift up trunk. The whole maneuver is called "Gower's sign".

What happens in quadriceps weakness?
In quadriceps weakness, the knee extensors fail to lock the knee joint that leads to falls. To compensate this, the patient hyperextends the knee (genu recurvatum), because in this position, the knee gets locked and thus ensures maximum stability of the knee joint **(Fig. 58)**.

MUSCLE WEAKNESS: DIFFERENTIAL DIAGNOSIS AND CLINICAL APPROACH

Weakness means loss of muscle power. First, try to clarify what the patient actually means by the word, "weakness". Is it due to loss of muscle strength or due to sensory loss, joint contracture, joint pain, or malingering? Once you confirm true weakness, then proceed to step I.

Step I: History

- *Identify which muscle groups are affected:* Symptoms of weakness depend on the muscles that are involved. Manifestations of weakness in different muscle groups have already been discussed in the previous section on "symptoms of neuromuscular disorders".
- *Define the temporal pattern of weakness*:
  - Onset—age at onset, acute, subacute, or chronic
  - Course—progressive, static, or relapsing

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- *Recognize other negative and positive symptoms*: These include history of fatigue, atrophy, muscle pain, cramp, and stiffness. The other important components are history of past medical illness, toxin exposure, and family history.

Step II: Examination

- Look for any abnormal movements, atrophy/hypertrophy of muscle, and examine tone, power, reflexes, stance, and gait.
- Differentiate whether it is LMN or UMN weakness. **Table 3** shows how to differentiate between UMN and LMN lesion. There is predilection for involvement of certain muscle groups in UMN lesion. Even in the severest form of hemiplegia, the weakness never involves all muscles in the hemiplegic side.

Step III: Where is the Lesion?

Localize the Anatomical Substrates of Lesion

A UMN lesion may be seen at any point along the course of corticospinal tract.

- Cerebrum (including cortex, subcortical white matter, and internal capsule)
- Brainstem
- Spinal cord

Manifestations of UMN lesions at different levels will be discussed in the Chapter on hemiplegia and paraplegia.

Lower motor neuron lesion may be at any point from the AHC to muscle.

- Anterior (ventral) root
- Plexus
- Peripheral nerve or purely motor branch of a peripheral nerve
- NMJ
- Muscle

First look for coexistent sensory changes. If sensory changes are present, the possible sites of lesion are:

- Both anterior and posterior nerve roots
- Plexus
- Mixed motor sensory nerve

If sensory nerve changes are absent, the possible sites of lesion are:

- AHC in the spinal cord
- Anterior nerve root
- A purely motor branch of a peripheral nerve or in motor axons alone
- Muscle

At this point, try to differentiate between two groups of pure motor localizations: Neurogenic (AHC, anterior root, and motor nerve) versus myopathic (muscle).

Table 4 shows the clinical clues that help to differentiate these two groups of motor localizations.

Localization at Different Lower Motor Neuron Levels

In addition to the findings common to all LMN lesions, there are some specific clinical clues for localization at different anatomical substrates.

Anterior Horn Cell Lesion

- Wasting appears early
- Wasting is more prominent compared to weakness
- Asymmetric in distribution, may involve both proximal and distal parts of limbs
- Presence of fasciculation

Nerve Root Lesion

- Multiple muscles innervated by different peripheral nerves and different plexus components may be involved, but all the involved muscles are innervated by the same root.
- Lesion may involve a single root or multiple roots (polyradiculopathy). Radiculopathy may be predominantly motor, predominantly sensory or mixed. Compression of a sensory root causes sharp, lancinating pain along the distribution of that particular root which increases on coughing or sneezing and lesion of a

Table 3: Differentiation between upper motor neuron (UMN) and lower motor neuron (LMN) lesion.

UMN lesion	*LMN lesion*
Involvement of a group of muscles, never individual muscles	Individual muscles may be involved
Mild atrophy (disuse atrophy)	Pronounced atrophy
• Deep tendon reflexes—exaggerated • Plantar reflexes—extensor • Tone—increased (spastic)	• Deep tendon reflexes diminished/lost • Plantar reflex—flexor • Tone—hypotonic (flaccid type)
Fasciculation—absent	Fasciculation—may be present

Table 4: Differentiation between neurogenic and myopathic disorders.

Neurogenic	*Myopathic*
Wasting is more prominent than weakness	Weakness is more pronounced than wasting
Wasting appears early	Wasting appears late
Asymmetric, may involve both proximal and distal parts of limbs	Symmetric, predominant, and proximal involvement
Early loss of deep tendon reflexes	May be well preserved except in advanced stage
Fasciculation—may be seen	Fasciculation—absent
Featureless in distribution	Depending on disease myopathies follow a definite pattern

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motor root causes weakness of muscles innervated by that particular root. Polyradiculopathy is recognized by predominant weakness in proximal limbs and truncal weakness. Truncal power is maintained by paraspinal muscles and these muscles are supplied by roots.

- Sometimes, it is difficult to differentiate weakness due to AHC/anterior (motor) root from weakness due to disease of a motor nerve. Topographical pattern of weakness is different in these two groups. Suppose a patient has presented with foot drop on left side. It may be due to left L5 root lesion or left deep peroneal neuropathy. In L5 root lesion, there is weakness of dorsiflexion of left foot (supplied by L5 via deep peroneal nerve) along with weakness of inversion of left foot (supplied by L5 but via the tibial nerve). In deep peroneal nerve palsy, these are only weakness of dorsiflexion of foot.

Plexus Lesion

Plexus is network of numerous axons that come together, exchange, separate, and advance peripherally. The anatomical components of plexus include root, trunk, division, cord, and nerve.

- Complete plexopathy causes weakness of the whole limb while partial plexopathy causes weakness of only some of the muscles supplied by the concerned plexus components.
- Presence of sensory impairment.
- The distribution of sensory loss and motor deficit does not follow the territory of a single nerve, rather follows multiple contiguous root distributions.

Nerve Lesion

- There is weakness of muscles supplied by the particular nerve.
- Impairment or loss of sensation occurs in the distribution of the nerve.
- Vasomotor, sudomotor, and trophic changes occur in skin, subcutaneous tissues, and nails in the territory of the affected nerve.
- In polyneuropathy, there is sensory disturbance in glove and stocking distribution.

Neuromuscular Junction Disorder

- Predominant proximal weakness
- History of diurnal variation, fluctuating weakness, and fatigability
- Usually, accompanied by evidence of ocular and bulbar involvement
- Sensory examination and reflexes—normal

Muscle Disorder

- Weakness is more pronounced compared to wasting.
- Wasting appears late.
- There is bilateral, symmetrical, and predominantly proximal involvement of limbs.
- Deep tendon reflexes are well preserved till the advanced stage.
- Fasciculations are usually absent.
- Myopathies may follow a definite pattern of involvement. In muscular dystrophies there may be selective muscle involvement, e.g., differential involvement of biceps with relative sparing of triceps (suggest intermuscular specificity) or there may be involvement of only anterior and posterior fibers of deltoid, sparing the central fibers, suggesting intramuscular specificity.

Step IV: Differential Diagnosis

Concentrate on two features:

1. Site of lesion/distribution of weakness
2. What is the likely pathology?

Examples:

- *Site of lesion*:
  - AHC—motor neuron disease, spinal muscular atrophy, and poliomyelitis
  - Polyradiculopathy/polyneuropathy—(discussed in detail in Chapter 6)
  - Neuromuscular junction disorder—myasthenia gravis, Eaton–Lambert syndrome, and botulism
  - Muscle disorder—congenital myopathy, muscular dystrophy, polymyositis, dermatomyositis, toxic myopathy, and metabolic myopathy
- *Distribution of weakness:*
  - *Prominent ocular weakness:*
    - Muscle
      - Oculopharyngeal muscular dystrophy
      - Kearns–Sayre syndrome
      - Congenital myopathy (centronuclear myopathy)
    - Nerve root
      - Miller Fisher syndrome [variant of Guillain–Barré (GB) syndrome]
    - Neuromuscular junction
      - Botulism
      - Myasthenia gravis
  - *Prominent facial or bulbar weakness:*
    - Muscle
      - Facioscapulohumeral muscular dystrophy
      - Myotonic dystrophy—Type 1
    - AHC
      - Bulbar onset amyotrophic lateral sclerosis (ALS)
      - Kennedy disease
  - *Prominent neck muscle weakness:*
    - Muscle
      - Dermatomyositis/polymyositis
      - Myotonic dystrophy
      - Facioscapulohumeral muscular dystrophy
    - Neuromuscular junction
      - Myasthenia gravis
      - Snake bite
      - Botulism
    - Root
      - GB syndrome

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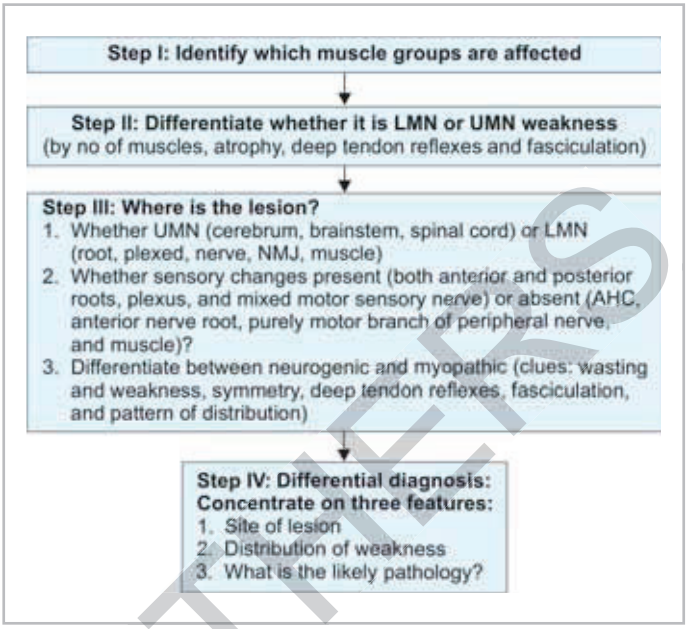
- AHC
  - ALS
- Cervical cord
  - High cervical cord lesion

○ *Prominent proximal limb weakness:*
- Muscle
  - DMD, sarcoglycanopathies, inclusion body myositis, polymyositis toxic myopathies
- AHC
  - Spinal muscular atrophy (SMA)

○ *Proximal arm/distal leg weakness:*
- Affects the periscapular muscles of the proximal arm and the distal muscles of lower limb, e.g. scapuloperoneal dystrophy and facioscapulohumeral dystrophy.

○ *Distal arm/proximal leg weakness:*
- Affects the distal forearm muscles (wrist and finger flexors) and proximal lower limb muscles (knee extensors). This pattern is seen in inclusion body myositis.

○ *Distal weakness:*
- Weakness involving the distal muscles of upper and lower limbs is more commonly a feature of peripheral neuropathies (where sensory system is usually affected). However, distal myopathies may present with only weakness atrophy in distal extremities.



FLOWCHART 3: Algorithm for differential diagnosis of muscle weakness.

(AHC: anterior horn cell; LMN: lower motor neuron; NMJ: neuromuscular junction; UMN: upper motor neuron)

What is the likely pathology?

Hereditary: Present from birth or early age, very slow progression, and history of similar illness in family.

Degenerative: Long duration, insidious onset, and slowly progressive course.

Inflammatory: Subacute onset, slowly progressive, relatively short duration, fever, and systemic symptoms may be present, e.g., infective (TB, HIV, etc.), noninfectious (sarcoidosis, Sjögren syndrome, etc.)

Demyelination: Acute/subacute onset, progressive/fluctuating course, e.g., acute inflammatory demyelinating polyneuropathy (AIDP) and chronic inflammatory demyelinating polyneuropathy (CIDP)

Finally you get answers of two vital questions:

1. Where is the problem?
2. What is the likely pathology?

Now you can reasonably guess about the provisional diagnosis and three to four differential diagnoses **(Flowchart 3)**.

DISCUSSION

Case Scenario 1:

- *Localization*:
  - Patient has motor system disease (sensory and cerebellar examination—normal).
  - Presence of weakness, wasting, and fasciculations suggests LMN involvement in the distribution of bulbar, cervical, and lumbosacral segments.
  - Presence of pseudo bulbar affect, jaw jerks, exaggerated deep tendon reflexes, Hoffman's sign, and extensor plantar response suggests UMN involvement in the distribution of bulbar, cervical, and lumbosacral segments (corticobulbar and corticospinal tracts are affected).
- *Pathology*: Most likely degenerative.
  - *Clues*: Long history, insidious onset, slowly progressive course, and involvement of multiple discrete sites of neuroaxis (diffuse AHCs, corticospinal, and corticobulbar tract)
- *Etiology*:
  - *Provisional diagnosis*: Amyotrophic lateral sclerosis

Continued

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Continued

Case Scenario 2:

- *Localization*:
  - LMN is involved.
    - *Clues*: Presence of weakness with wasting, diminished deep tendon reflexes with normal plantar response bilaterally.
  - Motor system disease (since sensory system is normal). The possible sites are AHC, motor root, muscle, and NMJ.
  - Early, symmetric, and disproportionate weakness with late appearance of atrophy, no fasciculations, no diurnal variation and presence of pseudohypertrophy of both calves suggest localization in muscle.
- *Pathology*: Most likely muscular dystrophy.
  - *Clues*: Long history, slow progression, presence of family history, absence of muscle pain, selective muscle involvement (intramuscular and intermuscular specificity: For example,
    - *Only anterior and posterior fibers of deltoid are involved, sparing the central fibers*—suggests intramuscular specificity
    - *Differential involvement of biceps with relative sparing of triceps*—suggests intermuscular specificity
- *Etiology*:
  - *Provisional diagnosis*: Limb girdle muscular dystrophy (LGMD)
  - *Differential diagnosis*: Becker muscular dystrophy
  - *Clues*: Age of onset, family history, pattern of involvement (limb girdle distribution), calf hypertrophy, and lack of significant joint contracture.

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