Paraplegia is impairment of motor and sensory functions of lower extremities often including the lower part of the trunk.

Terminology:
Paraplegia – Total paralysis of lower limbs
Paraparesis - partial weakness

History elicitation

Present history:
Symptoms to be elicited
1. Motor symptoms
2. Sensory symptoms
Plus Symptoms of
3. Sphincter disturbances,
4. Lesions of Higher functions,

Motor symptoms
Primarily distinguish weakness from fatigue or asthenia
Enquire if
- Paralysis or paresis.
- If limb involvement was symmetrical or serial
- Distal or proximal involvement
  Distal- presents as foot drop or frequent tripping
  Proximal as –difficulty in getting up from sitting posture,
  Difficulty in walking, running or climbing stairs.combing hair
- Ask about wobbling while standing – suggestive of hypotonia.
- Ask about symptom of stiffness or crossing of limbs while walking suggestive of hypertonia.
- Elicit history of involuntary movements.
- Record if weakness increases with exercise and improves with rest (myasthenia)
• Ask about associated symptoms
• Ask about use of medication
• Also consider depression as a possible diagnosis

**Sensory symptoms**
Elicit the history of following.
★ Anesthesia or hypoesthesia of the limbs
★ Parasthesia
★ Root pain –aggravates on coughing or straining;
  Radiates along the distribution of spinal nerve.
  Common in extramedullary lesion
★ Girdle pain: Same as root pain but bilateral-common in transverse myelitis
★ Dissociated sensory loss:
  Pain and temperature sense lost but touch, joint and vibration sense retained

**Lhermitte’s sign/Barber chair Sign:**
On hyperextension of his neck, patient develops electric shock like sensation all over the body-occurs in multiple sclerosis

**Sphincter disturbances**
✦ Urgency, precipitancy or hesitancy.
✦ Retention with overflow.
✦ Incontinence (true or false)
✦ Painless bladder distension.
✦ Painful bladder distension.

Bowel disturbances

Symptoms of sexual dysfunctions

**Symptoms** pertaining to higher functions
Alterations in sensorium- occur in cortical paraplegia.
Euphoria can occur in multiple sclerosis /long term cortisone therapy.
Mental deterioration in- Pagets with skull involvement.

**Cranial Nerve dysfunction in paraplegia:**
Elicit symptoms pertaining to all cranial nerves I-XII
Different cranial nerves can be involved in different paraplegias

**Other points to be elicited in present history**
Onset and progress.

**Onset**
Paraplegia of acute onset
1. Trauma of vertebral column.
   Injury to spinal cord may be complete or incomplete.
2. Intervertebral Disc Prolapse
3. Acute transverse myelitis.
5. Hematomyelia.
6. Gullian Barre.
7. Bleed into a spinal cord tumor.

**Sub acute** onset
- Spinal epidural Abscess.
- Sub acute combined degeneration

**Insidious** Onset
- Compressive and non-compressive myelopathy.

Progress:
- Waxing and waning-in demyelinating diseases

**Also elicit**
- Symptoms of raised intracranial tension
- Symptoms pertaining to other systems
- Ask about activities of daily life

**CHART 1. Elicitation of present history**

1. Motor symptoms
2. Sensory symptoms
3. Sphincter disturbances
4. Symptoms of cranial nerves dysfunction
5. Symptoms of Higher functions dysfunction

**Past history (chart2)**

Trauma
- Direct trauma,
- IV disc prolapse due to lifting of heavy weights

Infections
- TB, syphilis, viral, (Rabies, HIV), Rickettsial, Fungal like Actinomycosis, Cryptococcus
- parasitic infections

Neoplastic
- H/O tumors in the past, which can cause secondaries in the spine.

Metabolic
- h/o Liver disease, porphyria.

Immuno-Allergic
- H/o Vaccination-Rabies, tetanus, Polio
H/exanthemata, Chicken pox, measles

Physical
H/O irradiation, Electric Shock

Toxins
Kesari Dal Intake, lathyism, triorthocresyl phosphate exposure

Drugs:

**Occupational history** (Chart2)
Divers, Miners (Caissons Disease).

**Personal history** (Chart2)
DM, HT, IHD, Atherosclerosis (Anterior Spinal Artery thrombosis).
Smoking
Alcoholism: Myopathy.
H/o Living in Endemic area: of flurosis
Strict vegetarian: Sub acute combined degeneration.

*In females:* menstrual and obstetric history
H/o recurrent abortions, suggestive of antiphospholipid syndrome

Chart 2. Elicitation of past, personal and family history
**Family history (Chart2)**
- Hereditary spastic paraplegia
- Friedreich's Ataxia
- Charcot-Marie-tooth neuropathy
- HMSN (Hereditary motor and Sensory Neuropathy)
- Hereditary sensory Neuropathy (rare)
- Potassium related paralysis
- Myopathy
- Muscular dystrophy
- Autoimmune diseases like rheumatoid

**General examination (chart3)**

**Vital signs:**
- Respiratory distress can occur in;
- Motor neuron Disease, Gullaine-Barre, Duchene muscular dystrophy
- BP: Orthostatic hypotension – in autonomic system involvement
- Others
  - Neurofibromata,
  - Cafeau-lais spots,
  - Leafy macules
  - Adenoma sebacium
  - Subcutaneous nodules (secondaries)
  - Spider nevi
  - Icthyosis-SACD, Pellagra, leprosy
  - Trophic ulcer- Syrinx, Tabes, ulcers of leprosy/thickened peripheral nerves
  - bed sores
  - Fasciculation.
  - Anemia (B12 deficiency);
  - Clubbing-syrigomyelgia
  - Cyanosis- respiratory paralysis in-
    - Guillain- Barre syndrome
    - Cervical cord compression
  - Jaundice – in hepatic encephalopathy
  - Lymph adenopathy
    - i) Associated with TB spine’TB endarteritis, tuberculoma spine
    - ii) Leukemia, reticulosis with deposits in spine
    - iii) Malignancy
  - Cardio vascular; evidence of aneurysm of aorta, cardiac failure
  - Examination of tongue, mouth and lymph glands
  - Kyphosis, scoliosis, Pescavus (spino cerebellar degeneration) spina bifida/tuft of hair
  - Muscle tenderness – polymyositis
Chart 3. General examination and Examination of higher functions and cranial nerves.

**Examination of higher functions (Chart 3)**

Altered sensorium – in cortical paraplegia  
Euphoria–in multiple sclerosis  
Mental disturbances –  
   in pagets with skull involvement  
   Tabes with General paralysis of insane(not common now)  
   Tumor of falx cerebri, unpaired antr cerebral artery thrombosis  
   Mental retardation in Muscular dystrophy

**Examination of cranial nerves (Chart 3)**

I cranial nerve may be involved in Taboparasis.

**II cranial nerve in:**
Devis syndrome(Neuromyelitis optica)  
Multiple sclerosis.(optic neuritis)  
Tabes (optic atrophy)  
Spino cerebellar degeneration (retinitis pigmentosa)  
SMON – Sub acute myelo optic neuropathy  
Papilloedema/optic neuritis in Gullian Barre syndrome.  
Subacute combined degeneration

**III, IV, VI**  
External ophthalmoplegia can occur in Gullian Barre.
Argyl Robertson pupil-in syphilis
Reversed Argyll Robertson pupil; in encephalitis lethargica

V cranial nerve (descending tract of Trigeminal)
in high cervical cord lesion.

VI cranial nerve
– in cortical paraplegia like tumor of Falx Cerebri. (False localizing sign)

VII cranial nerve LMN type
– in Gullian Barre.

VIII cranial Nerve
– in Madras Motor neurone Disease
(Sensory neuronal deafness)

IX, X, XII cranial nerves
– in MND – bulbar or pseudo bulbar type.
Pharyngeal palsy with dysphagia in Gullian Barre
Cranial polyneuritis in Gullian Barre
CV junction anomaly.

Multiple cranial Nerve involvement in Paget’s
(1st, 2nd, 6th, 8th and lower cranial nerves)

Paraplegia with cranial nerve involvement occurs in
Motor neuron disease
Multiple sclerosis.
Poliomyelitis
Acute idiopathic poly neuritis
Diphtheria

Examination of spinal motor system (Chart4)

Routine examination

Components of the examination of the spinal motor system:

1. Nutrition: (Bulk of the muscles)
   - Wasting of the muscles is a late phenomenon in UMN lesions/
     occurs early in LMN lesion
   - Minimal in UMN lesion, marked in LMN lesion
   - Atrophy is sign of LMN lesion; but in LMN of short duration-no atrophy.
   - Disuse atrophy–sign of long term UMN lesion
   - Wasting can be examined by:
Observation or by Measuring the bulk with a tape
- Pseudohypertrophy- of calf muscles occurs in Duchene,Beckers
  Look for Gower’s sign.when pseudo hypertrophy is found
- In lesions between T4-T10- No wasting can be made out
- Non neurological causes of atrophy: Joint injury, joint disease.
- If myositis suspected-*palpate* for tenderness of the muscles

2. **Power:**
Often groups of muscles are involved-(in UMN Lesion); Individual muscles in LMN lesions
Test muscle power in groups initially.
(Muscles around major joints (shoulder,elbow wrist,hip,knee ankle)
Knowledge of root values of movement around these joints is essential for diagnosis.
In case of individual muscle wasting, test the power in appropriate muscle.

*Grading of power*

- 0. absent movements
- 1.-flicker of contraction
- 2. Movements with gravity eliminated
- 3. Movements against gravity, not against resistance
- 4. Movement against gravity and resistance but less than normal
- 5. Normal power

**Note:**
- Inter observer variations occur in grading
- Effectiveness of grading is diminished in the presence of joint swelling and pain
- Above grading is usable for proximal muscle group
- Distal weakness - graded as normal, weak or absent

3. **Tone:**
Tone is a state of partial contraction of resting muscles which helps in maintenance of posture.
(While testing tone, muscle should be totally relaxed (distract the patient’s attention)
(Testing power may leave the patient tense,hence tone may be tested before power.),

i) **Spasticity**: is Velocity dependant increase of muscle tone in response to passive muscle stretch.

ii) **Clonus**: sustained clonus is a sign of upper motor lesion.
*Clonus* is a series of rhythmical contraction occurring in response to maintenance of tension in muscle tendon (in ‘pseudo clonus’ contractions are not sustained)
Clonus is due to **gamma** neuron discharge.

**Sites** where elicitable:
- Ankle clonus: elicited by forcibly dorsiflexing the foot after flexing the hip and knee
- Patellar clonus: by sharply moving the patella downwards.
- Wrist clonus: by suddenly and forcibly dorsiflexing the wrist.
Finger clonus: by suddenly extending the fingers
Pseudo clonus: is ill sustained; and is seen in tense individuals normally.

iv) Flaccidity:
Found in Spinal shock, flaccid \textit{paraplegia} (and associated cerebellar lesion)

4. Coordination:
Test for: Romberg’s

- Ataxia occurs in Friedrich’s ataxia
- Ataxia and nystagmus can occur in complex form of hereditary spastic paraplegia
- Poor coordination: may also result from: peripheral pathology-impaired proprioception in nerves, dorsal root ganglion and roots; with positive. Romberg’s sign

\textbf{Note:} lower limb coordination cannot be tested in paraplegia/can be tested only in paraparesis.

5. Involuntary movements:
Accompany complex form of hereditary spastic paraplegia
\textit{Athetosis} is seen in cerebral palsy
\textit{Fasciculations}: Spontaneous contraction of a group of muscle fibers innervated by a single motor neuron
Best muscles to look for: deltoid, biceps, thigh muscles
\textit{occur in} – LMN lesions - motor cells/nerve root, peripheral nerve

- Motor neuron disease,
- Syringomyelia
- Poliomyelitis
- Cervical spondylosis
- Primary muscular atrophy
- Peroneal muscular atrophy
- Thyrotoxic myopathy
- Carcinomatous myopathy
- Organophosphorus poisoning
- Drugs-edrophonium/neostigmine
- Benign

Also look for Fibrillations in the tongue-a sign of MND

\textbf{Examination of sensory systems (Chart4)}

\textbf{Routine}
- Test various modalities of sensation - superficial and deep
  - Test cortical sensation - Two point discrimination, graphesthesia, stereognosis
- Test if there is a definite upper horizontal level for the sensory loss.
  - possibilities are: Transverse myelitis - acute, Cord compression - insidious
    - rapidly growing spinal tumor may be acute onset (rare)

\textbf{Note:} Small fiber sensory loss involves pain and temperature
Large fiber sensory loss involves proprioception, vibration
- Test the pattern of sensory loss in the limbs
  Pattern in Peripheral sensory neuropathy
  Pattern in Spinal cord lesion
  Pattern of cerebral cortical lesions

**Examination of Spine**

**Look for:**
- Gibbus
- Kyphosis, scoliosis,
- Tenderness
- Para vertebral swelling (infection or malignant disease)
- Tuft of hair/sacral dimple – Spina bifida occulta/Dermoid.
- Straight Leg Raising Test: if positive – indicates root lesion.
- Restricted spinal mobility – indicates bone disorder/disc disease/root disease.

**Examination of Autonomic nervous system**

1. Monitor vital signs; pulse, BP, Respiration
2. Temperature (raised also in intercurrent infection like pneumonia)
3. Skin: look for pallor, flushing, dryness, sweating
4. GI tract: nausea, vomiting, constipation, diarrhea in diabetics
   Check for: diminished bowel sounds,
5. Bladder, bowel incontinence
6. Bladder distension
7. Orthostatic hypotension

**Bladder involvement**

**Early:** In intramedullary lesion
Simultaneously: In transverse myelitis
Late: Extradural compression
   - Pyramidal lesion
**Bowel disturbances:** Constipation/incontinence

*Autonomic disturbances occur in:*
- Guillain-Barre
- Spinal cord disease

**Applied anatomy:**

a. Sympathetic neurons are located in - Thoracic and lumbar spinal cord segments
   Lesions of thoraco lumbar sympathetic neurons at or above T2 cause – Ipsilateral Horners syndrome
b. Parasympathetic neurons are located in Sacral spinal cord segments
   Lesions of sacral parasympathetic neurons between S2-4 cause - bladder, bowel dysfunction
Routine examination of Motor system:
  - Nutrition,
  - Power,
  - Tone,
  - Coordination,
  - Involuntary movements.

Routine Examination of Sensory system:
  - Test for touch, pain, temperature,
  - vibration, position sense.
  - If primary sensations intact,
  - Test cortical sensation:
    - two point discrimination, graphesthesia, Sterognosis. Look for a definite upper horizontal level

Chart4. Routine examination of motor and sensory system

Anatomic and clinical diagnosis of paraplegia

Motor examination specially tailored to diagnose a case of paraplegia

Components to be examined

For anatomical diagnosis (refer chart5)
1. Pattern of motor loss in the limbs
2. Site of neurological deficit in the neuroaxis
3. Segment pointer muscles testing

For clinical diagnosis (refer chart5)
4. Type of muscle weakness spastic or flaccid
5. Distribution of muscle weakness- proximal/distal/focal
6. Temporal profile of weakness-acute/sub acute/chronic
   Episodic or disseminated in space and time or variable weakness with fatiguability
7. If pure motor paraplegia or mixed with sensory signs

Table1. Patterns of muscle weakness

<table>
<thead>
<tr>
<th>Features</th>
<th>Upper motor neuron</th>
<th>Lower motor neuron</th>
<th>Myopathy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Distribution</td>
<td>Extensors in upper limb, flexors in</td>
<td>Follows root or nerve innervations</td>
<td>Proximal symmetric</td>
</tr>
</tbody>
</table>
lower limb pattern

<table>
<thead>
<tr>
<th>Atrophy</th>
<th>Absent</th>
<th>Present</th>
<th>Present</th>
</tr>
</thead>
<tbody>
<tr>
<td>DTR</td>
<td>increased</td>
<td>decreased</td>
<td>decreased</td>
</tr>
<tr>
<td>Muscle tone</td>
<td>increased</td>
<td>decreased</td>
<td>Not affected</td>
</tr>
</tbody>
</table>

**II. Site of neurological deficit in the neuraxis**

Summary: distribution of weakness and diagnostic clues
- Segmental weakness (nerve root)
- Territory of one peripheral nerve (mononeuropathy)
- Glove and stocking region (polyneuropathy)
- Proximal (Myopathy)
- Distal (Neuropathy)
- One sided (upper motor neuron)
- Spinal level /Paraplegia (spinal cord)
- Generalised (Wasting disorder)

**IIIB. Segment pointer muscles**

**Learning point:**
*Anterior root dysfunction* causes typical motor disturbance
This is distinct from peripheral nerve or plexus lesion
The pattern of distribution helps to differentiate these three
Monoradiculopathy produces
–partial muscle wasting since each muscle is innervated by several nerve roots;
but one single muscle suffers more than others in mono radiculopathy
These are called segment pointer muscles
Weakness of these muscles indicates the segment involved

**Table 2. Segment pointer muscles**

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Root and function</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diaphragm</td>
<td>C3+c4 –respiration</td>
</tr>
<tr>
<td>Deltoid</td>
<td>C5 arm abduction</td>
</tr>
<tr>
<td>Biceps</td>
<td>C6 Forearm flexion</td>
</tr>
<tr>
<td>Triceps</td>
<td>C7 Forearm extension</td>
</tr>
<tr>
<td>Intrinsic hand muscles</td>
<td>C8T1 finger adduction/abduction</td>
</tr>
<tr>
<td>Iliopsoas</td>
<td>L1 hipflexion</td>
</tr>
<tr>
<td>Quadriceps femoris</td>
<td>L3L4,knee extension</td>
</tr>
<tr>
<td>Tibialis anterior</td>
<td>L4 foot dorsiflexion</td>
</tr>
<tr>
<td>Extensor hallucis longus</td>
<td>L5 great toe dorsiflexion</td>
</tr>
<tr>
<td>Gastrocnemius</td>
<td>S1 plantar flexion</td>
</tr>
<tr>
<td>Rectal Sphincter</td>
<td>S3S4</td>
</tr>
</tbody>
</table>

**A special sign**

*Beevors sign:* When patient raises the head against resistance umbilicus moves upwards-
due to weakness of lower abdominal muscles.
Beevor’s sign is positive in Lesion at T10 level
T10 is a common site of spinal metastasis

Points which help in’ Clinical’ diagnosis

IV. Type of muscle weakness—Spastic or flaccid

Spastic paraplegia-causes
1. Paraplegia of cortical lesions
2. Compressive myelopathy
3. Non compressive myelopathy
   Congenital-familial spastic paraplegia, Friedreich’s ataxia
   Demyelinating-transverse myelitis, multiple sclerosis
   Degenerative-Motor neuron disease
   Nutritional- sub acute combined degeneration of spinal cord
   Toxic agents- lathyrisim
   Physical agents-Radiation, electricity
   Vascular-anterior spinal artery thrombosis
   Paraneoplastic
   Tropical spastic paraplegia

Flaccid paraplegia-causes
1. Spinal shock
2. Anterior horn cell lesion
   Anterior poliomyelitis
   Toxic anterior horn cell disease
   Inherited anterior horn cell disease
      Werdnig-Hoffman disease
      KugelbergWelander disease
3. Radiculopathy
   Guillain-Barre syndrome
   Arachnoiditis TB, Fungal
4. Peripheral neuropathy
   Of various etiologies
5. Muscle disease
   Acute onset
Poliomyelitis
Dermatomyositis
Alcoholic myopathy
Boutilism
Organophosphorous poisoning
Rarely myasthenia gravis
Gradual onset
Progressive muscular dystrophy
Chronic poliomyelitis
Thyrotoxic myopathy
Episodic
Periodic paralysis
6. Apart from Spinal shock,
   Muscles supplied at the level of
   the lesion-show hypotonia
   Muscles supplied below the level
   of lesion show hypertonia
   Hypotonia is also found in cerebellar
   lesion

V. Distribution of muscle weakness

- Proximal or Distal-wasting
- Symmetric or Asymmetric

Proximal muscle weakness

Involves shoulder girdle or pelvic girdle muscles or both
Sign: difficulty in standing from sitting position and raising the arm above the head.

Conditions causing: Proximal weakness

Anterior horn cells involvement
1. Poliomyelitis –
2. Genetically determined disease of motor neuron
3. Spinal muscular atrophy

Root involvement
- Guillain –Barre Syndrome (GBS)-
Peripheral nerve involvement

Neuromuscular junction
- Myasthenia/Eaton-Lambert syndrome (Variable weakness)

Muscle disease
- Myopathy

Dystrophic myopathy
- Duchene
- Beckers
- Fascioscapulohumeral dystrophy
- Limb girdle muscular dystrophy
- Primary inflammatory myopathy

Inflammation primarily against muscles
- polymyositis

Channelopathy
( Ion channels in skeletal muscle cell membrane)
- Periodic paralysis
- Myotonia

Metabolic
- Diabetic Mononeuritis multiplex
  (Sciatic and femoral nerve)
Diabetic myopathy involving proximal shoulder, pelvic girdle muscles—unilaterally or bilaterally.
Cushings syndrome
Osteomalacia

Distal weakness pertaining to paraplegia/quadriplegia

Conditions causing

2. Wasting of small muscles of hand
A. Vertebral lesion
   - Vertebral metastasis
B. Spinal cord lesion
   i. Syringomyelia
   ii. Compressive myelopathy with tumor
C. Anterior horn cell lesion
   i. Motor neuron disease
   ii. Poliomyelitis
   iii. Spinal muscular atrophy
D. Root Lesion
   i. Cervical spondylitis
   ii. Cervical cord tumor
   iii. Cervical pachymeningitis
   iv. Cervical disc prolapse
   v. Peroneal muscular atrophy
E. Peripheral nerve lesion
   i. Leprosy
   ii. Carpal tunnel syndrome
   iii. Lead poisoning
   iv. Bilateral median and ulnar nerve lesion
F. Muscle disease
   already mentioned above

2. Certain Myopathies with distal weakness:
   - Myotonic dystrophy
   - Inclusion body myositis
   - Genetic distal myopathy

Either Proximal or distal weakness
   - Alcohol, statin toxicity

Asymmetric muscle weakness
Multifocal motor Neuropathy
   (More common in upper limbs)
Root lesions

VI. Temporal profile of weakness
Acute
Sub acute
Chronic

**Acute paraplegia**
- Superior sagittal sinus thrombosis (cortical)
- Thrombosis of unpaired anterior cerebral artery (cortical)
- Multiple sclerosis (cortex/spinal cord)
- Trauma, hematomyelia, epidural abscess & anterior spinal artery thrombosis (Spinal cord)
- Guillain-Barre syndrome (multiple roots)
- Ticks paralysis, Boutilism (neuro muscular junction)
- Hypokalemia (muscles)

**Sub acute paraplegia**
- Sub acute combined degeneration (cord)
- Multiple sclerosis
- Amotrophic sclerosis (Antr horn cells)
- Disc lesion
- Compression, tumor, abscess (cord)
- Myesthenia (neuro muscular junction)
- Eaton–Lambert Syndrome (neuro muscular junction)
- Dermatomyositis (muscle)

- **Acute versus chronic radiculopathy**
  - In acute radiculopathy — (as in acute disc prolapsed) weakness precedes wasting
  - In chronic radiculopathy — (as in cervical spondylosis) wasting precedes weakness

**Episodic paraplegia**
- Occur in
  - Periodic paralysis
  - Myesthenia gravis
  - Hyperthyroidism

**Fluctuating muscle weakness**
- Myesthenia gravis
- Metabolic myopathies like McArdles

**Pure motor paraplegia**
- Lesions of para sagittal lesion
- Familial spastic paraplegia
- Motor neuron disease
- Lathyism
- Anterior poliomyelitis
- Lead palsy
- Acute idiopathic poly neuritis
- Diabetic amyotrophy
- Primary muscle disease
- Erb’s spastic paraplegia

**Extra muscular involvement**
- Examine also for extra muscular involvement
Arthralgia, myalgia, abdominal pain, dysphagia, rash around eyelids, exercise provoked weakness, features of cushings.

**Learning point**
Myotome is a group of muscle innervated by a single spinal cord segment
Dermatome – area of skin supplied by a single posterior nerve root

Examination of sensory system tailored to diagnose Paraplegia

*Modalities of sensation to be tested:*
1. Superficial sensations: pain, temperature touch.
2. Deep sensations: proprioception, Vibration
3. Cortical Sensations: Two point discrimination, graphesthesia, stereognosis

*Note:* Primary sensation must be intact to test cortical sensations
5. Compare both sides; compare upper and lower limbs

Anatomical diagnosis (Chart 6)

**Table 3. Sensory modalities and their anatomic correlation.**

<table>
<thead>
<tr>
<th>Modality</th>
<th>Fiber type (Periphery)</th>
<th>Tract (Central)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Light touch</td>
<td>small fiber</td>
<td>Spinothalamic</td>
</tr>
<tr>
<td>Temperature</td>
<td>small fiber</td>
<td>Spinothalamic</td>
</tr>
<tr>
<td>Pinprick</td>
<td>small fiber</td>
<td>Spinothalamic</td>
</tr>
<tr>
<td>Two-point discrimination</td>
<td>small fiber</td>
<td>Spinothalamic (parietal)</td>
</tr>
<tr>
<td>Proprioception</td>
<td>large fiber</td>
<td>Dorsal columns</td>
</tr>
<tr>
<td>Vibration</td>
<td>both small and large fiber</td>
<td></td>
</tr>
</tbody>
</table>

**Table 4. Body landmark and sensory dermatomes**

<table>
<thead>
<tr>
<th>Body landmark</th>
<th>Dermatome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Back of head</td>
<td>C2</td>
</tr>
<tr>
<td>Shoulder</td>
<td>C4</td>
</tr>
<tr>
<td>Thumb</td>
<td>C6</td>
</tr>
<tr>
<td><strong>Middle finger</strong></td>
<td></td>
</tr>
<tr>
<td>Thumb</td>
<td>C6</td>
</tr>
<tr>
<td>Small finger</td>
<td>C8</td>
</tr>
<tr>
<td><strong>Nipple</strong></td>
<td></td>
</tr>
<tr>
<td>Nipple</td>
<td>T4</td>
</tr>
<tr>
<td><strong>Umbilicus</strong></td>
<td></td>
</tr>
<tr>
<td>Umbilicus</td>
<td>T10</td>
</tr>
<tr>
<td>Inguinal region</td>
<td>L1</td>
</tr>
<tr>
<td>Big toe</td>
<td>L4L5</td>
</tr>
<tr>
<td>Small toe</td>
<td>S1</td>
</tr>
<tr>
<td>Genetalia, perianal region</td>
<td>S4S5</td>
</tr>
<tr>
<td><em>(sparing helps to differentiate intrinsic and extrinsic cord lesion)</em></td>
<td></td>
</tr>
</tbody>
</table>
**Pattern of sensory loss and anatomic diagnosis**

Very useful information from sensory examination is from distribution of deficit.

Symmetric/asymmetric and quality

Table 5. Characteristics of lesion at each level

<table>
<thead>
<tr>
<th>Peripheral Nerve</th>
<th>All modalities of sensation affected along the distribution of affected nerve</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Borders—sharply demarcated</td>
</tr>
<tr>
<td></td>
<td>Hyperesthesia, discomfort, pain may be present</td>
</tr>
<tr>
<td></td>
<td>Glove and stocking type of anesthesia present in polyneuropathy</td>
</tr>
<tr>
<td></td>
<td><em>Often symmetrical distribution</em></td>
</tr>
<tr>
<td></td>
<td>Longest nerves are first affected (lower limb first and then upper limb.)</td>
</tr>
<tr>
<td></td>
<td>Sensory fibres are first affected, then only motor</td>
</tr>
<tr>
<td></td>
<td>Vibration affected 1st, earlier than pain, temperature and touch. Since vibration fibers are largest and heavily myelinated and most metabolically demanding.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Root</th>
<th>All modalities of sensation affected</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Distribution along the distribution of that root</td>
</tr>
<tr>
<td></td>
<td>Borders vague;</td>
</tr>
<tr>
<td></td>
<td>Pain present—radiates along distribution of root.</td>
</tr>
<tr>
<td></td>
<td>Pain felt in muscles than in dermatomes.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Spinal cord</th>
<th>Segmental signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>(Composite picture of tract and segment signs)</td>
<td>Tract signs depend on tracts involved. Lesion can be complete trans section or hemisection or of intra medullary portion of the cord or of</td>
</tr>
</tbody>
</table>
posterior column alone or antero lateral column alone; hence findings vary. Dissociated sensory loss can be present.

| Medulla | • Dissociated sensory loss present  
• Pain and temperature lost over ipsilateral face- and over contra lateral body.  
• Deep sensation lost on contra lateral side of body. |
| Upper brain stem | • Sensory dissociation not found  
• All sensory modalities are now crossed and are on same side  
• Unilateral lesion causes contra lateral loss of sensory modalities  
• Brain stem lesion associates with cranial nerve involvement |
| Thalamus | • Sensory dissociation no longer present  
• Ipsilateral lesion causes contra lateral loss of all modalities of sensation  
• Presence of thalamic pain |
| Cerebral cortex (Parietal lobe) | • Sensory dissociation absent  
• Ipsilateral lesion causes contra lateral loss of all modalities of sensation  
• Discriminative sensory functions are lost |

**Summary:**

Loss of all sensation symmetrical and distal, glove and stocking type—Peripheral neuritis
Loss of all sensation in distribution of one peripheral nerve—mononeuropathy
Loss of all sensation in a dermatome—root lesion
Loss of pain and temperature and preservation of other senses—central cord—syringomyelia; In syringomyelia sensory loss is suspended.——called suspended sensory loss.
Loss of position and vibration (dorsal column or its afferent nerves)
Ipsilateral loss of position and vibration sense with contra lateral loss of pain and temperature below the level of lesion—hemisection of the cord—Brown-Sequard syndrome.
Bilateral loss of all sensations below a level—Transection of spinal cord, and at top of area of sensory loss a band of paresthesia or hyperesthesia occurs.
Loss of all sensation on opposite side of lesion—(hemianesthesia)—Thalamic lesion
Loss of cortical sensation on opposite side with some impairment of other sensations—Sensory cortex lesion

**Points to remember about root lesion:**
• Most frequent in cervical and lumbosacral lesion
• Associated with root pain
• Commonly caused by intervertebral disc herniation and spondylosis
• Sensory loss is not sharply demarcated—because of overlap of sensation of roots above and below
  o But root ‘pain’ is sharp and well localized; intensifies on coughing, sneezing, etc
  o Referred pain is less localized and is felt in muscles or skeletal structures supplied by the same root.

**Points to remember about tract lesions**
• Spinothalamic tracts cross almost immediately after entering the spinal cord
• But tracts of Posterior column cross only at medulla
• These two tracts are separated up to rostral pons, after which they climb in close proximity until reaching sensory cortex.
• Hence lesions in lesions in certain level of spinal cord and brain stem can affect specific sensations and not other sensations—causes dissociated sensory loss.

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### Chart 6. Sensory examination specially tailored to diagnose a case of paraplegia

#### Clinical diagnosis

**Dissociated sensory loss**

i. Bilateral: in Intramedullary lesion—Syringo myelia and in Anterior spinal artery thrombosis antr ½ of cord involved

ii. Unilateral: in Intramedullary lesion or Lateral medullary syndrome (PICA syndrome)

**Loss of ‘all modalities’ of sensation and diagnosis**

i. Symmetrical and distal: peripheral neuropathy

ii. Segmental sensory loss: root lesion

iii. Complete trans section type: transeverse myelitis

iv. Saddle shaped anesthesia: cauda equine lesion

v. Hemisensory loss: brain stem, thalamus, and capsular lesion

vi. Crossed hemisensory loss; (like ipsilateral face contra lateral body): brain stem lesion

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### Points to remember about tract lesions

- Spinothalamic tracts cross almost immediately after entering the spinal cord.
- But tracts of Posterior column cross only at medulla.
- These two tracts are separated up to rostral pons, after which they climb in close proximity until reaching sensory cortex.
- Hence lesions in certain level of spinal cord and brain stem can affect specific sensations and not other sensations—causes dissociated sensory loss.
‘Spinothalamic’ sensory loss –and diagnosis
Syringo myelia: loss marked in one or both upper limbs
Thrombosis of anterior spinal artery: similar to transaction of cord
Loss below a particular level of cord
Lateral medullary syndrome: loss on opposite side of body and same side of face

Diagnosis of sensory loss ‘with’ motor paralysis

I. Sensory loss with UMN palsy
a. With bilateral UMN palsy:
   Transverse myelitis
   Anterior spinal artery thrombosis
   Syringomelia (pyramidal lesion late and minimal)

b. I. Sensory loss with unilateral UMN lesion
a. With posterior column lesion
b. Hemisection of cord

II. Sensory loss with LMN palsy
a. Bilateral - Peripheral neuritis, Cauda equine lesions
b. Unilateral: Root lesion

Etiological diagnosis of peripheral neuritis
Neuropathic sensory loss and systemic diseases
Table 6. Key clinical signs which help in etiology of peripheral neuropathy

<table>
<thead>
<tr>
<th>Clinical Sign</th>
<th>May suggest:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rash</td>
<td>Lupus</td>
</tr>
<tr>
<td>Funduscopic examination</td>
<td>Diabetes, Vasculitis</td>
</tr>
<tr>
<td>Adenopathy</td>
<td>Infection, Cancer</td>
</tr>
<tr>
<td>Weight loss</td>
<td>Diabetes, Cancer, Endocrinopathy</td>
</tr>
<tr>
<td>Bony or Cutaneous abnormalities</td>
<td>Inherited neuropathy, Endocrinopathy</td>
</tr>
<tr>
<td>Organomegaly</td>
<td>“POEMS” syndrome¹</td>
</tr>
</tbody>
</table>

Examination of reflexes
Table 7. List of normal deep and superficial reflexes and their root values

<table>
<thead>
<tr>
<th>reflex</th>
<th>To elicit</th>
<th>result</th>
<th>muscle(s)</th>
<th>peripheral nerve</th>
<th>Root value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Biceps reflex</td>
<td>Tap bicep tendon with elbow flexed and forearm supinated</td>
<td>Elbow flexion</td>
<td>Biceps</td>
<td>Musculocutaneous nerve</td>
<td>C5 - C6</td>
</tr>
<tr>
<td>Brachioradial reflex</td>
<td>Tap distal end of radius with elbow</td>
<td>Elbow flexion</td>
<td>Brachioradialis</td>
<td>Radial (musculocutaneous)</td>
<td>C5 - C6</td>
</tr>
</tbody>
</table>
Triceps reflex
- Tap elbow with elbow flexed
- Extension of elbow
- Triceps
- Radial nerve
- C7 - C8

Epigastric reflex
- Quick stroke w/ a pin from nipple downward
- Dimpling of the epigastrium
- Upper fibers of the transversus abdominis
- Intercostal nerves
- T5 - T6

Abdominal reflex
- Quick stroke of skin toward the midline
- Shift of abdominal skin and umbilicus toward stimulated side
- Abdominal muscles
- Intercostal nerves, hypogastric, ilioinguinal nerves
- T6 - T12

Cremasteric reflex
- Stroke of the skin at the inner, upper aspect of thigh
- Elevation of the testicle
- Cremasteric muscle
- Genital branch of genitofemoral nerve
- L1 - L2

Knee jerk
- Tap quads tendon below patella while knee is flexed
- Extension of the knee
- Quadriceps femoris
- Femoral nerve
- L3 - L4 (L2)

Ankle jerk
- Tap the Achilles tendon
- Plantar flexion of the foot
- Triceps surae
- Tibial nerve
- S1 - S2

Bulbocavernous reflex
- Pinch glans penis
- Contraction of bulbocavernous
- Bulbocavernous
- Pudendal nerve
- S3 - S4

Anal reflex
- Pinprick perianal skin
- Visible anal contraction
- External sphincter ani
- Pudendal nerve
- S3 - S5

Grading of Reflexes:
0 No response
1 hypoactive
2 normal
3 hyperactive; no clonus
4 hyperactive with clonus

Hypoactive deep tendon reflexes in paraplegia
Special conditions associated with hypoactive reflexes
- Spinal shock
- Guillain –Barre Syndrome
- Boutilism
- Upper level of transverse cord lesions

Asymmetric or absent reflexes suggest- radiculopathy or neuropathy
Isolated loss of a reflex denotes radiculopathy of that segment

Hyper active deep tendon reflexes
Cerebral palsy
Motor neuron Disease
Multiple sclerosis
Suacute combined degeneration-
In 50%-absent ankle jerk with-hyper reflexia at knee with extensor plantar.
Transverse myelitis
Compressive myelopathy
Lathyrism

**Inverted deep tendon reflexes**
Inverted radial reflex is a sign of lesion at C5 level
(On tapping the supinator finger flexion>elbow flexion)
Inverted triceps reflex is assign of lesion at C7 level
(No contraction of triceps but exaggerated contraction of biceps)

**Inverted superficial reflex**
Inverted plantar if short flexors of the toe is paralysed-
On eliciting plantar reflex extensor response occurs in absence of UMN lesion

**Diagnostic value of abnormal reflexes:**
Reflex abnormality may not show up in acute stage

**Superficial reflexes in paraplegia**
Loss of abdominal,cremasteric reflex and extensor plantar –sign of UMN lesion
Loss of cremastric reflex alone is a sign of LMN lesion at L1
Loss of upper abdominal reflexes and presence of normal lower abdominal reflexes –
is a sign of T10 lesion

**Abdominal reflex and cremastric reflex** loss-is a sign of UMN lesion.
But loss is not dependent on the severity of pyramidal lesion.
In congenital diplegia and motor neuron disease –not lost till late.
But lost very early in multiple sclerosis

**Plantar reflex:**
Eliciting plantar reflex:
- With the patient lying supine, ‘flex the knee, fix the foot’ with ankle externally rotated
  and using blunt object stroke on lateral aspect of foot
  Normal response: Plantar flexion of great toe and plantar flexion of other toes
- Root value: L5, S1

Abnormal extensor plantar reflex is ‘positive babinski sign’

**Uncommon babinski signs** (chart 7)
- **Minimal Babinski:**
  No movements of the toes
  Contraction of tensor facia lata and of adductor muscles of thigh - only felt.
- **Equivocal** Babinski sign: Initial flexor movement of the toe followed by extension
- Babinski mimickers
  - **Pseudo** Babinski sign occurs-
    a. If the short flexors of the toe are paralyzed (Inverted plantar)
b. In athetosis or chorea when the involuntary movement is super imposed at the time of eliciting the reflex, the great toe may extend as a part of athetoid movement
c. In case of plantar hyperesthesia, application of strong or painful stimulus to the sole of the foot produces ‘withdrawal response’

- **Positive bilateral Babinski** in absence of pyramidal lesion:
  a. Infants before age one  
  b. deep sleep  
  c. deep anesthesia  
  d. post ictal state  
  e. coma.

- **Absent Babinski** response in the presence of pyramidal lesion:
  In S1 lesion with paralysis of extensor hallucis longus.

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**BABINSKI RESPONSE**

- True Babinski
- Pseudo Babinski
- minimal Babinski
- absent Babinski
- Bilateral Babinski

*Other methods* of eliciting plantar reflex:
(These methods test positive in extensive pyramidal lesions)
- Oppenheim’s method: firm stroke applied down on either side of anterior border of tibia with finger and thumb.
- 2. Gorden reflex: the calf muscles are squeezed.
- 3. Chaddock method: A curved stroke applied below the lateral malleolus.

**Gait Disorders and their diagnostic value**

**Normal gait**: characteristics
- erect posture, moderate sized steps and the medial malleoli of tibia trace a straight line

**Various types of gait that may be found in paraplegia**

**Scissors gait**
- Found typically in spastic cerebral diplegia
There is hypertonia of lower limb with increased adductor tone:
So thighs and knees cross in scissor like movement; patient walks on tip toe

**Ataxic gait**

1. **Cerebellar ataxia:**
   Feet are separated widely when standing or walking (wide based gait)
   Trunk sways forward, steps are jerky varying in size,
   In mild cases; tandem gait; heel- toe walking is impaired; patient tends to falls to the side of lesion (Lateral reeling)

2. **Sensory ataxia**
   Caused by impaired proprioception
   Impairment of posterior column sensation in spinal cord, /impairment of afferents in peripheral nerves
   On examination-
   Positive Romberg test and impaired joint position test
   Nature of Gait;
   Steps of variable length, foot lifted higher than necessary, audiable stamp can be heard
   –feet being brought down forcibly (feet ‘stamp’ the ground)
   Gait becomes more irregular in dark, or when eyes closed

Conditions with ‘stomping’ gait
Frederic’s ataxia
Pernicious anemia,
Tabes dorsalis
Peripheral neuropathy
Multiple sclerosis

**High steppage /equine gait**
In cases of peroneal muscle and pretibial muscle paralysis (LMN) with foot drop
Because of foot drop patient lifts the affected foot high for the toes to clear the ground
Clinical conditions;
Poliomyelitis
CharcotMarie tooth disease

**Waddling gait: myopathic gait**
Seas in proximal muscle weakness
Trunk and pelvic muscle weakness results in sway-back, pot bellied appearance with difficulty in pelvic fixation when walking

**Hysterical gait**
Takes various forms
Lacks neurological signs
Neurological disorders of bladder and paraplegia.

I. Uninhibited bladder:
In lesions involving cortical center of bladder e.g. para sagittal meningioma
Bladder is as that of infants
Urine is voided anywhere any time without control

II. Bladder in spinal shock:
Retention with overflow

III. Automatic bladder:
Occurs after recovery from spinal shock
Empties suddenly and reflexly.
Reflex occurs even when bladder volume is low (300 ml)
Usually no residual urine.

IV. Autonomous bladder:
Bladder is autocratic—behaves as it wants.
Motor and sensory fibers of reflex arc are damaged e.g. Cauda equine lesion
Bladder sensation is lost
Bladder is over distended with continuous dribbling.
If motor component of bladder reflex is lost,
Over distension with dribbling.
Sensory bladder:
On account of damage to sensory fibres of bladder.
As in above case, over distension with dribbling incontinence
Pain of bladder distension not perceived
Condition causing:
Diabetic autonomic neuropathy
Multiplesclerosis
Tabes.