

# Sensory Disorders

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**NEGATIVE PHENOMENA** - *failure* along sensory channels (conduction block, fiber loss, etc):

**HYPESTHESIA** - **diminution** of any sensory modality (most frequently in discussion of tactile stimulation).

N.B. at least half afferent fibers must be lost in order for sensory deficit to be demonstrated!

**POSITIVE PHENOMENA** – ectopic neural *hyperactivity* anywhere along sensory pathway (as such, it may be not localizable); physical examination may be normal!

1. **HYPERESTHESIA** - **exaggeration** of any sensory modality response.

- **HYPERPILAPHESIE** - augmentation of tactile faculties in response to other sensory deprivation (e.g. touch in blind).

2. **PARESTHESIA** - **SPONTANEOUS** abnormal sensation (i.e. sensations of purely subjective nature).

- often described as *pins-and-needles sensation*.
- often coexist with pain syndromes.
- abnormality anywhere along sensory pathway (from peripheral nerves to sensory cortex).
- **CNS disorders** may cause particular kinds of paresthesias:
  - *FOCAL SENSORY SEIZURES* with **cortical lesions**;
  - *SPONTANEOUS PAIN* in **thalamic syndrome**;
  - *BURSTS OF PARESTHESIAS* down back or into arms on neck flexing (*Lhermitte symptoms*) in **disorders of cervical spinal cord** (e.g. multiple sclerosis).
  - **level lesions of spinal cord** → "*BAND SENSATION*" / "*GIRDLE SENSATION*" (altered sensation encircling abdomen) or *SENSORY LEVEL* (i.e. altered sensation below level of spinal cord lesion).
- most intense and annoying paresthesia in **PNS disorders** is due to multiple symmetric peripheral neuropathy (**polyneuropathy**).

1) if paresthesias **do not persist**, they are *not likely to imply neurologic lesion*.

2) **persistent** paresthesias *imply abnormality of sensory pathways!*

- if paresthesias persist and fail to correspond abnormality to explain symptom, patient should be re-examined.

3. **DYSESTHESIA** - disagreeably abnormal sensations WHEN AREA IS TOUCHED (sometimes even pressure of bedclothes cannot be tolerated by patient).

**Sensory symptoms** may be either positive or negative, but **sensory signs** on examination are always negative phenomena!

- abnormalities of **touch** sensation are *readily recognized* by patient.
- impairment of **vibration** is generally *not noted* by patients (because this sensory inflow is not part of daily conscious experience).
- **proprioceptive** loss is recognized by patient as *lack of coordination* in limbs\* or *impairment in gait*; (early proprioceptive loss may be unmasked by asking if patient has difficulty walking or reaching for objects in dark).

\*limb may show PSEUDOATHETOSIS if patient closes eyes

**Distribution & modality** of sensory loss are chief features for lesion localization:

LESION LOCATION	SENSORY LOSS MODALITY	SENSORY LOSS DISTRIBUTION
<b>Receptor dysfunction</b> (dermal pathology)	All sensations	Distribution of local cutaneous process
<b>Peripheral mononeuropathy</b>		Distribution of nerve (distal to proximal gradient loss)
<b>Peripheral polyneuropathy</b>		Distal “glove-and-socking”, gradual shading from normal to diminished sensation
<b>Plexopathy</b>		Multiple nerves in single limb
<b>Sensory root</b>		Dermatomes (several roots must be affected to produce loss!)
<b>Conus medullaris or cauda equina</b>		Saddle distribution
<b>Spinal cord (central as in syringomyelia)</b>	Pain & temperature	Dermatomes
<b>Spinal cord (posterior column)</b>	Position, vibration, discriminative touch	Ipsilateral below lesion
<b>Spinal cord (half transection)</b>	Position & vibration	Ipsilateral below lesion
	Pain & temperature	Contralateral below lesion
Touch relatively preserved!		
<b>Spinal cord (full transection)</b>	All sensations	Below lesion (“sensory level”)
<b>Brain stem (medial lemniscus)</b>	Position, vibration, discriminative touch	Contralateral incomplete* hemibody (*due to segmental arrangement of fibers)
<b>Brain stem (spinothalamic tract + nucl. spinalis CN5)</b>	Pain, temperature	Harlequin pattern: ipsilateral face, contralateral hemibody
<b>Thalamus</b>	All sensations ± hyperpathia, spontaneous pain	Contralateral entire hemibody
<b>Subcortical white matter</b> (as in multiple sclerosis)	All sensations	Contralateral multifocal partial peripheral nerve distribution
<b>Sensory cortex</b>	Permanent loss of discriminative touch & proprioception. Mild transient loss of pain, temperature, vibration.	Contralateral entire hemibody (or different body parts); limbs > trunk & face; variability in distribution and severity from moment to moment; sensory phenomena (inability to recognize own limbs, alien hand syndrome).

PNS lesions - *intense* sensory loss, with *fixed, clearly defined* zones.

CNS lesions - deficit is more *mild* with *vague boundaries*; there may be considerable *variation* in both distribution and intensity of sensory deficit.

**ELECTROPHYSIOLOGIC TESTING**

Lesions distal to dorsal root ganglion → **sensory nerve conduction studies**.

Lesions proximal to dorsal root ganglion → **somatosensory evoked potentials** (most frequent site of stimulation: in upper extremity - **median nerve**; in lower extremity - **tibial nerve**).

**GENERAL MANAGEMENT**

1. Rehabilitative *sensory re-education*.
2. Increase *patient's awareness* of potential injuries:
  - 1) do not to use insensate hand when cooking or smoking;
  - 2) do not to place insensate extremity in hot water.

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