Sensory Disorders

Last updated: September 5, 2017

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!

DIAGNOSIS

- 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
Receptor dysfunction (dermal pathology)	All sensations	Distribution of local cutaneous process
Peripheral mononeuropathy		Distribution of nerve (distal to proximal gradient loss)
Peripheral polyneuropathy		Distal "glove-and-stocking", gradual shading from normal to diminished sensation
Plexopathy		Multiple nerves in single limb
Sensory root		Dermatomes (several roots must be affected to produce loss!)
Conus medullaris or cauda equina		Saddle distribution
Spinal cord (central as in syringomyelia)	Pain & temperature	Dermatomes
Spinal cord (posterior column)	Position, vibration, discriminative touch	Ipsilateral below lesion
Spinal cord (half transection)	Position & vibration	Ipsilateral below lesion
	Pain & temperature	Contralateral below lesion
	Touch relatively preserved!	
Spinal cord (full transection)	All sensations	Below lesion ("sensory level")
Brain stem (medial lemniscus)	Position, vibration, discriminative touch	Contralateral incomplete* hemibody (*due to segmental arrangement of fibers)
Brain stem (spinothalamic tract + nucl. spinalis CN5)	Pain, temperature	Harlequin pattern: ipsilateral face, contralateral hemibody
Thalamus	All sensations ± hyperpathia, spontaneous pain	Contralateral entire hemibody
Subcortical white matter (as in multiple sclerosis)	All sensations	Contralateral multifocal partial peripheral nerve distribution
Sensory cortex	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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