Sensory Disorders

Last updated: May 8, 2019

DIAGNOSIS

Electrophysiologic testing

GENERAL MANAGEMENT

-ALGESIA, -DYNIA – disorders of pain perception – see p. S20 >>

PSYCHOGENIC SENSORY DISORDERS – see p. D1 >>

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

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

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

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**GENERAL MANAGEMENT**

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

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**BIBLIOGRAPHY**

“Harrison's Principles of Internal Medicine”, 1998 (ch. 23)