

## ESSENTIALS OF CLINICAL NEUROLOGY

### CHAPTER 6: SENSORY DISORDERS (INCLUDING SELECTED PAIN SYNDROMES)

#### DEFINITIONS

Following lesions of central or peripheral nervous system, sensory disturbances can be characterized by positive features; too much sensation, spontaneous sensations such as pins and needles, or negative sensory deficit such as too little sensation or numbness. Neuralgia refers to tingling, electrical, or shock sensations in specific cranial (trigeminal nerve) or peripheral root (postherpetic neuralgia patterns.) Paresthesias are abnormal sensations of pins and needles or tingling caused by normally nonnoxious events, for example, when socks or bed sheets are applied to the feet of a patient with peripheral neuropathy. Paresthesias may occur spontaneously and may originate along sensory system pathways from sensory cortex to peripheral nerve. These usually result from uncontrolled impulses firing from injured sensory pathways, which have reduced threshold or enhanced excitative in either central or peripheral sensory system. Dysesthesias are uncomfortable painful abnormal sensations that occur with or without any external stimulus being applied. When local anesthetic is given for dental procedures, an area of the mouth is initially “dead or numb” (analgesia), and while sensation in the “blocked” nerve returns, paresthesias are reported. Paresthesias may be transient (following prolonged crossing of patients legs) and not be associated with neurological abnormality; however, if paresthesias are persistent, search carefully for sensory system abnormality.

#### SYMPTOMS

Sensory abnormalities are characterized by loss of sensation, pain, or paresthesias. These subjective symptomatic disturbances can precede or occur without objective neurologic findings. Because sensory symptoms can be very subjective, physicians must attempt to determine these characteristic features, which are listed in Box 6-1.

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#### Box 6-1

1. What is the patient actually experiencing, and what are specific descriptive terms used by patient?
  2. What is the location of the sensations (e.g., localized or diffuse, superficial or deep)?
  3. What is the pattern of onset (e.g., maximal at onset or progressively intensifying, worsen with activity, awaken patient from sleep)?
  4. Are symptoms constant or episodic (intermittent)?
  5. Is there radiation or spread?
  6. What factors exacerbate or relieve sensory disturbances (e.g., position, rest, movement, or sleep)?
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Sensory symptoms can occur in nonneurologic conditions such as vascular insufficiency or arthritis; therefore examination should be directed toward detecting objective neurological findings, for example, areas of abnormal sensation, decreased reflexes, motor weakness, muscle wasting, trophic skin, or joint changes.

Paresthesias can be characterized as “pins and needles,” limbs falling asleep,” or simply as “tingling”; however, if paresthesias are unusually intense they can be perceived as painful (dysesthesias). Paresthesias can occur in absence of involved body parts, such as phantom limb phenomenon after amputation. Paresthesias occur in distribution of isolated sensory nerve (mononeuropathy) or spinal root (radiculopathy), in distal extremities (e.g., peripheral polyneuropathy), over entire body and limbs below level of spinal cord lesion, on one side of the face and on contralateral extremities and trunk (crossed hemianesthesia) in brain stem lesions, or on one side of the body (hemispheric (cortical or subcortical) lesion).

In simple, partial (focal) somatosensory seizures caused by lesions involving the parietal sensory region, paresthesias are episodic and paroxysmal. They usually spread proximally from fingers to arms, shoulders, hips, legs, feet, and then to the face. Paresthesias caused by peripheral neuropathies can be provoked by minimal tactile stimulation (e.g., bed coverings placed on top of bare legs). Neuropathic pain is worse at night. Arterial vascular pain is precipitated by activity; venous vascular pain may intensify with prolonged standing; arthritic pain may be most intense upon awakening and accompanied by morning stiffness. These disorders can be associated with paresthesias. Limb movement or neck flexion can cause electrical paresthesias that extend down the back to thighs and legs (Lhermitte sign) in patients with multiple sclerosis or cervical myelopathy. This is believed caused by irritation of demyelinated posterior columns of the cervical spinal cord. It is clear that paresthesias can be caused by disturbances at any level of neuraxis from peripheral nerve to parietal cortex.

Numbness is the word commonly used by patients to describe sensations of deadness, heaviness, or coldness in affected body part. In certain neuropathies and spinal cord lesions (e.g., syringomyelia), patients are not aware of any sensory impairment and will report no subjective sensory symptoms; however, they may burn, injure, or mutilate themselves because of the profound sensory loss that can be demonstrated on neurological examination.

Sensory disorders arising from tendons joints muscles affect position sense and symptoms include imbalance, gait unsteadiness (most severe in dark environment.) This is basis of Romberg sign as patient gait is most unsteady and wobbly with eyes closed compared with eyes open. With sensory impairment in upper extremities, abnormal (pseudoathetoid) movements occur when patient attempts to hold outstretched hands steady.

## **EXAMINATION FINDINGS**

Sensory examination depends on the patient’s subjective interpretation of the applied stimulus. Primary sensory modalities include pain, temperature, light, touch, vibration and position sensation Cortical sensation includes two-point discrimination, bilateral simultaneous stimulation, graphesthesia (ability to recognize numbers written on patients hand with the eyes closed), and stereognosia (ability to recognize objects such as coins placed in patients hand.)

The sensory exam is not very sensitive because at least 50% of afferent fibers must be dysfunctional before sensory deficit is shown by the clinical examination finding. Thoroughness of examination is determined by the patient's symptomatology and neurologic findings (reflex changes, Babinski sign, motor weakness, muscle wasting, trophic skin, joint changes). These findings suggest the possible level of involvement in central or peripheral nervous system. The patient's response to pinprick, vibration, light touch, and position-sense testing is carefully assessed if patient has sensory symptoms and more of screening exam (compare right to left side, distal and proximal, for one or two sensory modalities) (see Chapter 1). Reduced (hypoesthesia or absent (anesthesia) sensation refer to findings on the clinical examination. Hypalgesia indicates reduced pain threshold; hyperesthesia refers to pain sensation response to non-noxious stimulus e.g. light touch. Allodynia refers to non-noxious stimulus being painful to the patient. In assessing the significance of sensory disturbances, two general rules should be considered: one, sensory symptoms can occur in the absence of objective sensory findings such as paresthesias in radicular pattern caused by herniated disk; and two, objective sensory findings rarely occur in absence of sensory complaints such as anesthetic limb that feels different to the patient than does a normal limb; if anesthetic limb does not feel different, consider functional conversion reaction.

## **CLINICAL SYNDROMES**

### **Polyneuropathy**

Neuropathic disorders include symmetrical polyneuropathies and asymmetric mononeuropathies (single or multiple.) There is symmetric loss of sensory perception that initially involves longest and largest diameter nerve fibers. Sensory impairment is most severe distally, that is feet and hands; there is less deficit proximally, that is, thighs and shoulders, with trunk usually being spared. This causes stocking-and-glove pattern of sensory impairment. The sensory deficit of polyneuropathy does not correspond to any specific peripheral nerve or root and is usually quite symmetric. Sensory symptoms begin on bottom (soles) of feet and later involve the top (dorsum) of the foot. It would be most unusual for polyneuropathy symptoms to begin in hands rather than feet (consider an alternative diagnosis such as carpal tunnel syndrome or cervical radiculopathy), and if initial sensory symptoms are on dorsum of feet or appear asymmetrical, consider diagnosis of radiculopathy. The anesthetic zone gradually merges into zone of less diminished sensation (hypoesthesia); this subsequently blends into region of normal sensory perception in sensory disturbance caused by objective sensory neurological disturbance. Sensory loss usually involves all modalities, but in some cases proprioception and vibration sense seem to be involved earlier and more severely than are pinprick, light touch, and temperature sensation. This indicates that faster-conducting, thickly myelinated fibers (mediating vibration and position sensibility) are usually damaged before slower-conducting, thinly myelinated and unmyelinated fibers (mediating pain, temperature, light touch). Large-fiber nerve dysfunction is characteristic of diabetic neuropathy, whereas early thin fiber nerve involvement is more common with amyloid neuropathy. Other findings in patients with sensorimotor polyneuropathy include one, decreased or absent deep tendon reflexes especially at ankles; two, distal extremity weakness and muscle wasting; and three, trophic skin and joint changes. Loss of deep tendon reflexes in neuropathy is usually due to sensory afferent portion of reflex arc such as large sensory fibers rather than being caused by efferent motor portion of reflex. Polyneuropathies usually include both sensory and motor abnormalities; in rare cases pure sensory neuropathy caused by amyloid or remote effect of carcinoma occurs. These

predominantly sensory neuropathies show dorsal column sensory loss such as impaired proprioception and vibration sense with less severely involved pain perception. Sensory ataxia can develop in certain neuropathies. These patients have broad-based unsteady gaits with clumsy, awkward finger manipulations of objects and utensils. There can be involuntary finger movements that simulate athetosis (pseudoathetosis), and these result from severe proprioception impairment. Pseudoathetoid movements are seen in other conditions such as tabes dorsalis, multiple sclerosis, and parietal lobe lesions.

## **Mononeuropathy**

Mononeuropathies involve single nerve trunks. The majority result from trauma; others have vascular or toxic causes. Mononeuropathy of vascular etiology can involve single or multiple (mononeuritis multiplex) nerve(s). Entrapment (compressive) neuropathy is due to pathologic disturbances (demyelination) in isolated nerve segments. Sensory symptoms are frequently the initial complaint in mononeuropathies. Numbness and tingling in the hand (especially first three fingers) are initial symptoms of median nerve compression (carpal tunnel syndrome). Clinical findings such as sensory, motor, and trophic abnormalities are localized to the known distribution of the specific involved nerve. The anesthetic zone can be smaller than the nerve's anatomic cutaneous distribution because of overlap of contiguous sensory nerves. The diagnosis of neuropathy is confirmed by nerve conduction velocity measurement and electromyographic findings.

## **Plexus (Brachial and Lumbosacral) Syndromes**

Sensory symptoms depend on the specific nerve trunks damaged. Brachial plexus dysfunction can be caused by trauma (stab injuries or gunshot wounds), infection (following immunization, viral, or bacterial illness), or neoplasm (lung or breast). If there is involvement of upper portion (C5, C6), sensory impairment occurs in the shoulder, lateral forearm, and arm; there is motor weakness and wasting of intrinsic hand muscles with sensory loss along the medial (ulnar) region of forearm and hand. In most brachial plexus disorders, predominant sensory symptoms is anesthesia; however, in certain conditions (e.g., brachial plexitis, neoplastic infiltration) there can be pain. Certain traumatic injuries can result in causalgia (burning pain).

The lumbosacral plexus is less frequently damaged. Causes include vertebral or hip disorders (pelvic and retroperitoneal neoplasms, osteomyelitis, pelvic surgery or fractures, psoas abscess) and inadvertent injury into the nerve(s) of the plexus from local injections. Patients usually describe pain and paresthesias located in gluteal region or thigh; this can radiate down back of calf and lateral portion of the leg to ankle region. This local pain can be constant, dull, and aching, and the radiating component pain can be intermittent and lancinating. Pain is worsened when sciatic nerve is stretched or palpated. Lumbosacral plexus involvement can be confused with disk disease, but lack of back pain is important differentiating feature in excluding disk disease.

## **Radicular Syndromes**

Subjective symptoms and objective sensory findings of dorsal spinal roots are referred to segmental dermatomal distribution. Because of overlap between several dorsal roots, objective delineation on sensory examination of sensory impairment can be less reliable than patient's description of sensory radiation. Pain and paresthesias of radicular involvement usually are lancinating, radiate along nerve root distribution, intermittent, increased by activities that increase intraspinal pressure (coughing, straining) or stretch nerve root, and respond to treatment (e.g., bed rest and traction). Reflex changes can be present if the dorsal (sensory) root is involved. If ventral root is involved, weakness and muscle wasting can be prominent. Radicular syndromes can be caused by lesions compressing spinal nerve roots (intervertebral disk, extradural or intradural extramedullary tumors). Less common causes include herpes zoster infection and diabetes mellitus. Herpes zoster can invade the dorsal root ganglia. This can cause severe burning trunk (girdle) pain localized to a specific thoracic segmental pattern. Herpes zoster neuralgia is usually accompanied by cutaneous vesicular eruption. Vesicles may resolve, but neuralgia can persist within an anesthetic zone (post-herpetic neuralgia). This pain can be treated with capsaicin (derived from Hungarian red pepper) that is applied topically. This drug blocks the release of the chemical pain mediator substance P. Other drugs used to block substance P include opiates, tricyclic antidepressants, clonidine, and certain anti-epileptic drugs including gabapentin, topiramate, lamotrigine and carbamazepine.

Certain patients with diabetes mellitus complain of severe and constant pain in the upper back, rib, thoracic, or abdominal region. This has either radicular or girdlelike distribution. Clinical findings include decreased sensation in the thoracic region, weakness of abdominal muscles, and weakness of iliopsoas or quadriceps muscles. There is frequently minimal evidence of generalized peripheral neuropathy, although the patient can experience significant weight loss. The presence of pain and weight loss can lead to extensive evaluation for thoracic, abdominal, or pelvic lesions or pathological conditions originating from the spine. The diagnosis of diabetic thoracoabdominal radiculopathy is established by electromyography findings.

## **Spinal Cord Syndromes**

### ***Central Gray Matter Commissural Syndrome***

Sensory fibers subserving pain and temperature travel in lateral spinothalamic tract and decussate centrally in anterior commissure. If these are damaged, sensory loss is usually symmetrical and segmental; less frequently it is asymmetrical. Because certain sensory modalities such as light touch, vibratory sensation, and proprioception do not decussate in this region, they are initially spared. This sensory dissociation (loss of pain sensation with sparing of touch, vibration and position sense) and shawl-like sensory loss that extends for several levels (segments) is characteristic of intramedullary lesions (e.g., syringomyelia). If lesion involves fourth through sixth cervical fibers, sensory loss has shawl-like distribution involving anterior neck, shoulder, and upper arm. Because sensory fibers involving sacral region are located peripherally in spinothalamic tract, buttock (saddle region) is not affected by central spinal cord lesions located above thoracic region. Ventral extension of syringomyelic cavity into anterior horn of cervical region can cause accompanying weakness, fasciculations, muscle wasting, and hypoactive reflexes in upper extremities.

## **Posterior Spinal Cord Syndromes**

If a spinal lesion, for example, cervical spondylosis, neoplasm, or abscess, causes compression and distortion of posterior spinal cord, there is loss of proprioception and vibration sensation below level of the lesion. With proprioceptive impairment in cervical region searching, writhing movements (pseudoanthetoid) of outstretched hands and fingers can occur. With flexion of neck, electrical shocklike sensation is felt in the back and down the legs (Lhermitte sign).

## **Hemisection (Brown-Sequard) Syndrome**

If lesions are restricted to one side of spinal cord, hemiparesis often with impaired position and vibration sense occurs ipsilateral to lesion, and impaired pain and temperature perception are present in extremities and trunk contralateral to the lesion (usually beginning two spinal segments below lesion level). This pattern results from extradural or intradural extramedullary lesions such as meningioma, neurofibroma, and cervical spondylosis.

## **Complete Spinal Cord Transection Syndrome**

Complete transection syndrome is characterized by complete loss of all sensory modalities below level of the lesion with associated paralysis and loss of sphincter function. The deficit develops rapidly in trauma or demyelinating disorders (acute transverse myelitis); it evolves more gradually when caused by spinal cord neoplasm or abscess. Spinothalamic tract fibers have laminar pattern, sacral fibers are lateral, and cervical fibers are medial. If spinal cord is damaged from extrinsic compression (extradural or intradural extramedullary tumor), sensory level gradually can ascend to higher level. Initially, lesion acts to compress externally located sacral fibers; later, internally located (medial) fibers – which represent lumbar, thoracic, and cervical regions- are compressed. It is important to consider evolution of the pattern of sensory involvement caused by spinal cord lesions; this differs depending on the location of spinal cord lesion, for example, intramedullary lesions initially spare sacral region, and extramedullary lesions cause early sacral involvement with prominent autonomic (bowel and bladder) impairment.

## **Anterior Spinal Cord Syndrome**

This is most commonly caused by ischemic spinal artery disease-usually due to atheromatous aneurysm and their surgery. Initial symptoms of spinal stroke caused by infarction within anterior spinal artery territory can be diffuse back pain or radicular dysesthetic pain. Pain is followed by the rapid onset of weakness in arms and legs (occlusion of cervical portion) or legs only (occlusion of thoracic region), usually with incontinence. Initially, limb tone is flaccid; reflexes can be diminished, and bilateral Babinski signs are present. There is absent pain and temperature sensation below the lesion level. Proprioception and vibration sensation are intact because posterior columns are supplied by posterior spinal artery. This pattern of dissociated sensory loss is differentiated from that seen with mass lesions including hematomyelia (hemorrhage into substance of spinal cord), in which there can be complete transection syndrome in which there is involvement of all sensory modalities.

## **Tabes Dorsalis**

Tabes dorsalis is usually due to neurosyphilis but can be caused by diabetes. There is damage to proprioceptive fibers of dorsal roots. This damage initially involves lumbosacral roots but can extend to the thoracic and cervical regions. Clinical symptoms include lightning or lancinating pains. Findings include hypotonia, areflexia, and loss of proprioception and vibration sensations.

## **Brain Stem Syndromes**

With lesions in medulla and those extending to midpontine level, there is crossed anesthesia; loss of pain and temperature sensation of the face is on the same side as the lesion (because fibers traveling in trigeminothalamic tract initially descend in the brain stem on same side before their synapses) and on trunk and extremities contralateral to lesion (caused by the crossing of the ascending lateral spinothalamic tract within the spinal cord). If lesion is located above nucleus of spinal tract or the trigeminal nerve, all sensory loss is located contralateral to lesion. There is impairment of sensation of sensory modalities supplied by lateral spinothalamic tract (pain and temperature) and medial lemniscus (position sense and vibration) because at this level of neuraxis these sensory tracts are parallel and contiguous.

## **Thalamic Sensory Syndromes**

Patients with thalamic sensory syndromes invariably demonstrate contralateral hemianesthesia for superficial and deep sensations as well as having astereognosia (inability to recognize objects placed in hand). There can sometimes be accompanying hemiparesis, hemiataxia, or hemichorea. Sensory abnormalities include all modalities involving the face, arm trunk, and leg contralateral to the lesion. This is usually caused by infarction that is due to occlusion of thalamogeniculate branches of posterior cerebral artery or hemorrhage involving thalamus, it can also be due to parietal white matter subcortical lesions. Several days to weeks after onset of vascular episode, painful sensations develop in region of sensory impairment (Dejerine-Roussy syndrome). These sensations have a burning and unpleasant causalgic quality. Tactile stimulation of the involved region or emotional disturbances can evoke dysesthesias in anesthetic regions (anesthesia dolorosa). Pain can be spontaneous or evoked by touching the limb. This "thalamic pain" is usually unresponsive to any analgesic medication. It may respond to tricyclic antidepressants or anticonvulsants, agents effective in treating neuropathic pain.

## **Cortical Sensory Syndromes**

Sensory information is modulated and interpreted in neocortex (parietal lobe). In parietal lobe lesions there can be inattention or neglect of sensory stimulation on contralateral limbs and trunk. These are most common with nondominant lesions but can also occur with dominant cerebral hemispheric lesions. These cortical sensory parameters are mentioned in Box 6-2.

In simple partial seizures originating in parietal (somatosensory) cortex, the patient may describe paresthesias, dysesthesias, or the sensation and the motionless limb feels as if it is moving; these sensations occur contralateral to the side of the parietal lesion.

## Functional Sensory Loss

Patients with conversion symptoms and hysteria frequently have sensory disturbances. The possibility of functional sensory dysfunction is considered when there is discrepancy between the patient's symptoms and examination findings with neuroanatomical and neurophysiologic realities. For example, the patient may report stocking-and-glove anesthesia; however, this pattern is characterized by abrupt or precise change from abnormal to normal without intervening area of partial anesthesia as is consistent with polyneuropathy.

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### Box 6-2

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1. Stereognosis, or the ability to recognize objects placed in hand (impairment is astereognosia)
  2. Graphesthesiam, or the ability to recognize numbers traced on skin of hand (impairment is agraphesthesia)
  3. Recognition of shape, weight, or texture of objects placed in hand
  4. Two-point discrimination, or the ability to recognize two points as separate when applied simultaneously 3 mm apart on fingertips
  5. Double simultaneous stimulation, or the ability to recognize two independently applied or presented sensory stimuli (e.g., tactile and visual) on symmetrical body regions
  6. Position and vibration sense; impairment involving sensory modalities is valid only if primary sensory modalities (e.g., pain, temperature, and light touch) are intact
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In other cases functional sensory pattern does not conform to that seen in peripheral nerve or spinal root lesions. In patients with functional hemianesthesia other special systems can be involved (hearing, vision, and olfaction), and functional loss has sharply defined borders between areas of absent and normal sensibility without an intervening zone of mildly impaired sensation. In functional sensory disorders, impairment of position sense is equally impaired in distal and proximal joints (equal numbers of errors when toes and knees are tested), whereas there is most severe impairment in distal joints in true proprioception abnormalities. Despite a profound loss of position sensation, the patient does not fall and does not appear unsteady. In functional hemianesthesia there is an abrupt change in patient's ability to perceive vibration in central region of face and sternum. Because this sensation is transmitted through one bone, the patient should perceive vibration equally on both sides of this bone except if sensory loss is functional. Also, in functional sensory anesthesia, the patient does not report that the limb feels different than the normal limb.

## SELECTED PAIN SYNDROMES

**Neuropathic Pain** – This is defined as pain that occurs after injury to the nervous system. These may be classified as peripheral or central type.

## **Pain Related to Peripheral Nerve Lesions**

Neuralgia literally means “nerve pain” and is used to describe paroxysms of electrical or shocklike sensation caused by spontaneous discharges within demyelinated areas of the nerve. Common peripheral neuropathic pain syndromes include – polyneuropathy, traumatic mononeuropathy, neuroma, stump pain. Post-herpetic neuralgia may be combination of central and peripheral type. Neuralgia can result from compression or stretching of nerves. Following nerve trauma, this neuropathic pain is due to abnormal discharges involving thinly myelinated or unmyelinated fibers. The pain is usually intermittent as is the characteristic of short-lived, but frequent paroxysms of trigeminal neuralgia. With partial nerve injuries, the painful limb usually shows other characteristics (Box 6-3).

In patients with partial nerve injuries there is reduced function of large, thickly myelinated fibers serving vibration sensibility, proprioception, and two point discrimination. These large-fibers are believed to initiate a mechanism within the dorsal horn of the spinal cord (substantia gelatinosa) that should normally inhibit conduction of painful impulses to CNS. When this mechanism is impaired, CNS is flooded with painful impulses, and the patient reports limb pain. By using transcutaneous electrical stimulation to activate afferent sensory fibers or by applying a sympathetic nerve block used to terminate abnormal electrical impulses, neuropathic pain can be reduced.

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### **Box 6-3**

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1. Painful discomfort to testing with normally nonnoxious stimulus such as pin, cotton, or temperature (cold or warm)
  2. Delayed appreciation of sensory stimulus.
  3. Continued pain after stimulation with the pin has stopped
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## **Phantom-Limb Pain**

Amputation of a limb can be followed by awareness of deafferented (amputated) body part. This experience can range from a vague diffuse tingling in the region of the amputated limb to reporting the exact experience that previously emanated from the amputated limb. Less commonly, the patient experiences painful sensations in the region of the missing limb or at the remaining amputation stump. This pain can have these characteristics: stabbing, throbbing, deep ache. The pain is exacerbated by emotional stress, autonomic (sympathetic) reflex activity, or touching the stump; this pain can be relieved by rest or massage of the stump. Pain usually disappears with one year of amputation. The mechanism of phantom-limb pain is not established but may relate to persistence of sensory reflex signals transmitted to spinal cord and brain following amputation of the painful limb.

## **Peripheral Neuropathy**

Certain patients with neuropathies report severe uncomfortable paresthesias and limb pain; this can be deep aching in limbs or superficial burning pain. It would seem logical that painful

neuropathies would have selective large fiber loss; however, there are other painful neuropathies (amyloid) in which there is selective small-fiber type loss. This indicates that the mechanism for pain in neuropathy is not established.

### **Pain Related to Central Nervous System Lesions**

These include post-stroke and spinal cord injury, trigeminal neuralgia, and post herpetic neuralgia. There is damage to synapses within spinal cord dorsal horn and is related to changes within CNS sensory neurons. Trauma and cerebrovascular disease are common cause of central neuropathic pain. The pain may be brief, intense and shock-like, lasting 15 to 30 seconds, while superimposed on constant baseline pain located within the region of neurological impairment. Movement of the involved limb exacerbates the pain and this may interfere with neurorehabilitation efforts. Injury to spinothalamic tract and its thalamocortical pathways are major pathophysiological mechanisms. Treatment with adjuvant analgesics (medications whose primary indication is not for pain management) include tricyclic antidepressants, antiepileptics, cardiac and local topical agents such as capsaicin and lidocaine. Treatment of central and peripheral neuropathic pain is identical.

### **Reflex Sympathetic Dystrophy (RSD) (CRPS)**

The term “complex regional pain syndrome” is the newly accepted diagnostic label. CRPS type I is utilized when there is no associated demonstrable nerve injury and type II is associated with demonstrable nerve injury. CRPS may follow injury to a peripheral nerve or may follow a traumatic limb injury, which may be severe or trivial. The cardinal features are the following: one, burning pain (causalgia); two, sympathetic dysfunction (edema, increased sweating pattern, cold limb temperature, thin shiny skin, cracked brittle nails, reduced extremity hair pattern, osteoporosis); three, pain evoked by usually painless stimulus (allodynia) and hyperesthesia. Allodynia is invariable present, however, physical signs suggesting autonomic dysfunction may be absent. The pain is usually located distally (hand, foot) and is exacerbated by limb movement; therefore the involved limb is held motionless, usually in a guarded position. If untreated, the pain may progress more proximally or to homologous regions of the opposite limb. This disuse of the limb can lead to joint fibrosis, muscle atrophy, contractures, and osteoporosis. The diagnosis of CRPS can be confirmed by thermography (reduced limb temperature caused by reduced blood flow), limb radiogram (demineralization, osteoporosis), and bone scan abnormalities (impaired blood flow, abnormal soft tissue and bone uptake of radionuclide isotope: however, symptom relief by nerve block is essential to establish the diagnosis. Nerve block can abolish symptoms for several weeks; however, symptoms can recur. Some patients’ symptoms respond to physical therapy and transcutaneous nerve stimulation; however, in persistent cases where there has been response to nerve block but the effect wears off after several weeks, sympathectomy may be necessary. It is important to establish the diagnosis of CRPS in the early stages when the condition responds to nerve block and sympathectomy. In later stages of CRPS, the disorder may no longer respond to these treatments and patients may require neurosurgical pain-relieving procedures including dorsal column stimulation, morphine pump, or oral narcotics to control pain. CRPS is an important consideration in any patient who reports unexplained limb pain. Early mobilization of the limb is critical to avoiding the debilitating late effects on the limb and the behavioral effects of anxiety and depression (pain

behavior) which occur when the patient is shunted between multiple physicians who fail to recognize CRPS and its appropriate treatment.

## **Herpes Zoster**

Varicella-zoster causes chicken pox in children. Reactivation of latent virus in dorsal root ganglia (usually in thoracic or abdominal region) results in cutaneous vesicular eruption in adults (shingles). Advanced age and immunosuppression increases risk of shingles. This begins with radicular abnormal itching, tingling or painful sensation. The skin lesion and pain resolve over two to four weeks. Postherpetic neuralgia is defined as pain that persists more than 30 days after rash onset and may last for several months. Therapy for herpes zoster includes antiviral therapy (acyclovir, valacyclovir, famciclovir) and treatment of postherpetic neuralgia includes opioids, tricyclics, and gabapentin.

## **SUMMARY**

Sensory disturbances can be reported as symptoms by the patient even when there are no objective neurological findings. These symptoms can include numbness, paresthesias, or pain. Based upon the history, it is crucial to ascertain if those symptoms are of neurological or nonneurological (vascular, rheumatological) origin. If these symptoms are of possible neurological origin, ascertain the sensory symptom characteristics (pattern, duration) and the exact distribution of the sensory abnormalities. This permits localization of the lesion causing sensory disturbance within the peripheral or central nervous system. It is important to recognize that sensory symptoms can be caused by neurological processes, for example, numbness or paresthesias in the arm or hand caused by cervical nerve root compression or compression of the median nerve at the wrist, even if the neurological examination is normal. Careful history and examination are crucial to determine the exact nature of the reported sensory symptoms, which can be caused by neurological, medical-systemic, or psychiatric disturbances.

## **Suggested Readings**

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