The parietal lobe is the middle lobe of the cerebrum and is found on the lateral surface of the hemisphere and consists of postcentral gyrus, superior and inferior parietal lobules. In this region of the cerebrum one finds major functional differences between the left hemisphere which is dominant for language, and the right hemisphere that is important for body imagery. The pain pathway has been discussed in Chap. 3 – spinal cord, 4 – brain stem, and 6 – diencephalon. In this chapter, we will cover the pathways subserving tactile information for: (1) the extremities, thorax and abdomen, the posterior columns and (2) the trigeminal systems subserving tactile information from the head. The importance of these pathways is apparent when one walks in the dark or drinks or eats.

Postcentral Gyrus: Somatic Sensory Cortex (Primary Sensory S-I)

The postcentral gyrus is not from a histological standpoint homogeneous. Four subtypes may be distinguished.

Organization of the Postcentral Gyrus

Within the postcentral gyrus, four representations of the body and limb surfaces are present in a parallel manner – with each representation relatively modality specific.

The response of the cells in the postcentral:
- **Area 3a** respond primarily to muscle stretch receptors
- **Area 3b** to rapidly and slowly adapting skin receptors (as in movement of a hair or skin indentation)
- **Area 1** to rapidly adapting skin receptors
- **Area 2** to deep pressure and joint position receptors

Within areas 1 and 2, there are additional neurons that do not receive direct thalamic input. These neurons respond instead to more complex properties of the stimulus such as the specific direction of movement and have been labeled complex cells. Studies of cortical potentials evoked by tactile stimulation in the monkey have suggested that there is a secondary somatic sensory projection area (S-II) in addition to the classic postcentral contralateral projection area. This second area has a bilateral representation and is found partially buried in the Sylvian fissure at the lower end of the central sulcus. A similar second area of representation has been reported by Penfield and Jasper (1957), Luders et al. (1985), and Blume et al. (1992) in those seizure patients in whom an abdominal sensation (aura) was followed by a sensation of paresthesias in both sides of the mouth and in both hands. Note also that at times stimulation of the precentral gyrus by the neurosurgeon during surgery has at times produced contralateral tingling or numbness, in addition to the more frequent motor responses.

Area 3a and 3b receive the major projection from ventral posterior medial (VPM) nucleus of the thalamus. Some of the neurons in areas 1 and 2 receive direct input from the ventral posterior thalamic nuclei while other neurons are dependent on collaterals from area 3.

Postcentral Gyrus Stimulation

Stimulation of the postcentral gyrus produces a sensation over the contralateral side of the face, arm, hand, leg, or trunk described by the patient as a tingling or numbness and labeled paresthesias. Less often, a sense of movement is experienced. The patient does not describe the sensation. As painful. These various phenomena, occurring at the onset of a focal seizure, may be described as a somatic sensory aura. There is in the postcentral gyrus a sequence of
sensory representation which, in general, is similar to that noted in the precentral gyrus for motor function (Fig. 14.1). The representation of the face occupies the lower 40%; the representation of the hand, the middle upper 40%; and the representation of the foot, the paracentral lobule. As in the precentral gyrus, certain areas of the body have a disproportionate area of representation, i.e., the thumb, fingers, lips, and tongue. Those areas of the skin surface that are most sensitive to touch have not only the greatest area of cortical representation but also the greatest number and density of receptors projecting to the postcentral gyrus. The peripheral field of each of these receptors is also very small (compared, e.g., to the less sensitive skin areas of the trunk). In addition, at the lower end of the postcentral gyrus, extending into the Sylvian fissure (the parietal operculum), there is found a representation of the alimentary tract, including taste. Gustatory hallucinations may arise from seizure foci in this area. There is also a representation of the genitalia in the paracentral lobule and a rare patient with seizures beginning in this area has reported paroxysmal sexual emotions including orgasm and nymphomania. Microelectrode techniques for recording from single cells in the cerebral cortex of the monkey and cat have allowed a considerable elaboration of the functional localization within the sensory cortex. The studies of indicated a vertical columnar organization from cortical surface to white matter. While each column is modality specific, each neuron in a particular column is activated by that specific sensory modality, e.g., touch, movement of a hair, deep pressure, joint position. Jones (1985), Kasdon and Jacobson (1978), Jones and Powell (1970), and discuss the thalamocortical relationships.

In the monkey, a secondary somatic sensory projection (SII) has been identified. It has a bilateral representation and is found deep in the lower end of the central sulcus. In several patients with seizures, bilateral sensations have been reported from the mouth and hands (Blume et al. 1992).

**Postcentral Gyrus Lesions**

Immediately following complete destruction of the postcentral gyrus, there will often be found an almost total loss of awareness of all sensory modalities on the contralateral side of the body. Within a short time there is usually a return of some appreciation of painful stimuli. The patient will, however, often continue to note that the quality of the painful stimulus differs from that on the intact side. An awareness of gross pressure, touch, and temperature also returns. Vibratory sensation may return to a certain degree. Certain modalities of sensation, however, never return or return only to a minor degree. (In partial lesions of the postcentral gyrus, however, these various modalities often return to a variable degree. These modalities are often referred to as the cortical modalities of sensation or as discriminative modalities of sensation.

![Figure 14.1: Sensory representation as determined by stimulation studies on the human cerebral cortex at surgery.](image-url)
In contrast, the sensory modalities of pain, gross touch, pressure, temperature, and vibration are referred to as primary modalities of sensation.

Perception of these modalities continues to occur after ablation of the postcentral gyrus although some alteration in quality of sensation is noted. It has been assumed that the anatomical substrate for such awareness must exist at the thalamic level. In terms of more specific localization of specific modalities of cortical sensation, the studies of suggest that in the monkey small lesions restricted to area 3b (hand region) produce deficits in discrimination of texture, size, and shape. Lesions in area 1 interfere with the ability to discriminate texture only; lesions in area 2 – size and shape.

The following types of sensory awareness usually included in this category are summarized in Table 14.1.

The following case histories demonstrate the type of sensory phenomena found in disease involving the postcentral gyrus.

Case 14.1 (Fig. 14.2)

One week prior to evaluation, this 40-year-old right-handed married white male had the onset of repeated 15–21 min duration episodes of focal numbness (tingling or paresthesias) involving the left side of the face, head, ear, and posterior neck and occasionally spreading into the left hand and fingers, and rarely subsequently spreading into the left leg. Biting or eating would trigger the episodes. There was no associated pain or headache or weakness or associated focal motor phenomena.

Initial neurological examination: Totally intact as regards mental status, cranial nerves, motor system, reflexes, and sensory system.

Clinical diagnosis: Focal seizures originating lower third postcentral gyrus of uncertain etiology.

Laboratory data: CT scan. The enhanced study demonstrated a small enhancing lesion just above the right Sylvian fissure. MRI (Fig. 14.2): Demonstrated the more extensive nature of this process involving the operculum – above the right Sylvian fissure including the lower end of the postcentral gyrus, the area of representation of the face. A probable infiltrating tumor was suggested as the most likely diagnosis.

Arteriogram demonstrated a tumor blush at the right Sylvian area, consistent with a glioblastoma, a rapidly growing infiltrating tumor – originating from astrocytic.

Subsequent course: Treatment with an anticonvulsant reduced the frequency of the episodes. Neurosurgical consultation suggested an additional observation period and periodic CT scans. Three months after onset of symptoms, the patient developed a difficulty in coordination of left arm. Exam now demonstrated left central facial weakness, mild weakness of

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**Table 14.1** Modalities of sensation in postcentral gyrus

<table>
<thead>
<tr>
<th>Cortical discriminative sensory modalities</th>
<th>Type of stimulus</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Position sense</td>
<td>Ability to perceive movement and the direction of movement when the finger or toe is moved passively at the interphalangeal joint, the hand at the wrist or the foot at the ankle</td>
</tr>
<tr>
<td>2. Tactile localization</td>
<td>Ability to accurately localize the specific point on the body or extremity which has been stimulated</td>
</tr>
<tr>
<td>3. Two-point discrimination</td>
<td>Ability to perceive that a double stimulus with a small separation in space has touched a given area on the body or limb or hand</td>
</tr>
<tr>
<td>4. Stereognosis</td>
<td>Ability to distinguish the shape of an object and thus to recognize objects based on their three-dimensional tactile form</td>
</tr>
<tr>
<td>5. Graphesthesia</td>
<td>Ability to recognize numbers or letters which have been drawn on the fingers, hand, hand face, or leg</td>
</tr>
<tr>
<td>6. Weight discrimination</td>
<td>Ability to recognize differences in weight placed on the hand or foot</td>
</tr>
<tr>
<td>7. Perception of simultaneous stimuli</td>
<td>Ability to perceive that both sides of the body have been simultaneously stimulated. When bilateral stimuli are presented but only a unilateral stimulus is perceived, “extinction” is said to have occurred</td>
</tr>
<tr>
<td>8. Perception of texture</td>
<td>Ability to perceive the pattern of texture surface stimuli encountered by the moving tactile receptors</td>
</tr>
</tbody>
</table>

In contrast, the sensory modalities of pain, gross touch, pressure, temperature, and vibration are referred to as primary modalities of sensation.)
the left hand, and in the left hand a loss of stereognosis and graphesthesia with a relative alteration in pinprick perception. The CT scan showed significant enlargement of the previous lesion. Doctor Bernard Stone performed a subtotal resection of a glioblastoma – in the right temporal parietal area. Radiation therapy was administered following surgery, 4,000 rad total whole brain and 5,210 rad total to the tumor area. Reevaluation 3 months after surgery indicated only rare focal seizures involving the face. The neurological examination was normal. Repeat CT scans, however, continued to demonstrate a large area of enhancing tumor in the right temporal-frontal-parietal area. Despite the use of dexamethasone and arterial chemotherapy with cis-Platinum, he continued to progress with the development of a left field defect, left hemiparesis, and a loss of all modalities of sensation on the left side. Subsequently, he developed significant changes in memory and cognition. CT scan demonstrated progression with extensive involvement of the right side of the brain and spread to the left hemisphere. Death occurred 33 months after onset of symptoms. At post-mortem examination of the brain, almost the entire right hemisphere was replaced by necrotic infiltrating tumor. The tumor now had spread into the corpus callosum and temporal and occipital lobes. The internal capsule, thalamus, hypothalamus, and basal ganglia were destroyed.

The following case presents a patient with episodic pain from a tumor in the left postcentral gyrus.

Case 14.2

This 62-year-old white, right-handed housewife, 6 years prior to admission had undergone a left radical mastectomy for carcinoma of the breast. Five months prior to admission, the patient developed a persistent cough, left pleuritic pain, and a collection of fluid in the left pleural space (pleural effusion). She now had the onset of daily headaches in the right orbit, occasionally awakening her from sleep. Four months prior to admission, over a 3–4 week period, the patient developed progressive “weakness” and difficulty in control of the right lower extremity. Several weeks later, the patient now experienced, aching pain in the right index finger in addition to a progressive deficit in the use of the right hand. This was more a “stiffness and in coordination” than any actual weakness. One month prior to admission the patient noted episodes of pain in the toes of the right foot and numbness of right foot occurred, lasting 2–3 days at a time. She subsequently developed, difficulty in memory and some minor language deficit, suggesting a nominal aphasia, prompting hospital admission.

Neurological examination:

Mental status: Slowness in naming objects was present although she missed only one item of 6. Delayed recall was slightly reduced to three out of five in 5 min.

Cranial nerves: Early papilledema was noted: absence of venous pulsations, indistinctness of disk margins, and minimal elevation of vessels as they passed over the disk margin. A right central facial weakness was present.

Motor system: Mild weakness of the right upper limb was present. A more marked weakness was present in the lower limb (most marked distally). Spasticity was present at the right elbow and knee. In walking, the right leg was circumducted; the right arm was held in a flexed posture.

Reflexes: Deep tendon reflexes were increased on the right. A right Babinski response was present.

Sensation: An ill-defined alteration in pain perception was present in the right arm and leg – more of a relative difference in quality of the pain than any actual deficit. Repeated stimulation of the right lower extremity (pain or touch) produced dysesthesias (painful sensation). Position sense was markedly defective in the right fingers with errors in perception of fine and medium amplitude movement in the right toes. Simultaneous stimulation resulted in extinction in the right lower extremity. Graphesthesia (identification of numbers, e.g., 8, 5, 4 drawn on cutaneous surface) was absent in the right hand and fingers and poor in the right leg. Tactile localization and two-point discrimination were decreased on the right side.

Clinical diagnosis: Metastatic tumor to the left postcentral and precentral gyri with a cortical pain syndrome (pseudothalamic pain syndrome).

Laboratory data: Chest X-ray: Several metastatic nodules were present in the left lung.

Imaging studies were consistent with a focal metastatic lesion in the parasagittal upper left parietal area.

Subsequent course: Because there was evidence in this case that the disease had spread to multiple organs, radiation and hormonal therapy were, therefore, administered rather than any attempt at surgical removal of the left parietal metastatic lesion. Temporary improvement occurred in motor function in the arm but the patient eventually expired 5 months after admission. Autopsy performed by Dr. Humphrey Lloyd of the Beverly Hospital disclosed extensive metastatic disease in the lungs, liver, and lymph nodes and a single necrotic metastatic brain lesion in the upper left parietal area. 1.0 cm below the pial surface and measuring 1.2 x 1.0 x 0.6 cm. The occurrence of episodic pain in the involved arms and legs along with the production of an experienced painful sensation on repetitive tactile stimulation (dysesthesias) in patients with sensory pathway lesions is sometimes referred to as a “thalamic” or “pseudothalamic” syndrome.

Comments: In this case, the anatomical locus for the pseudothalamic syndrome is apparent. The effects of deficits in cortical sensation on the total sensory motor function of a limb are apparent in this case. The actual disability and disuse of the
right arm and leg were far out of proportion to any actual weakness. Such an extremity is often referred to as a “useless limb.” The actual weakness that was present undoubtedly reflected pressure effects on the precentral gyrus and the descending motor fibers in adjacent white matter. Destructive lesions of the postcentral gyrus during infancy or early childhood often produce a retardation of skeletal growth on the contralateral side of the body. Such a patient examined as an adolescent or adult will be found to have not only cortical sensory deficits in the contralateral arm and leg but also a relative smallness of these extremities (shorter arm or leg, smaller hand and glove size, smaller shoe size).

**Superior and Inferior Parietal Lobules**

The superior parietal lobule is composed of Brodmann’s cytoarchitectural areas 5 and 7, the inferior parietal lobule of areas 40 and 39 (the supramarginal and angular gyri). All of these areas may be classified as varieties of homotypical cerebral cortex. The major afferent input of the superior parietal lobule area 5 in the monkey is from the primary sensory areas of the postcentral gyrus. Area 7 of the monkey cortex receives indirect cortico-cortical connections. The superior parietal lobule receives projections from the posterior lateral nuclei of the thalamus and the inferior parietal lobule from the pulvinar (see Chap. 6). These secondary sensory areas project to an adjacent tertiary sensory area and then to a multimodal sensory association area at the temporal parietal junctional area. This latter area in the posterior parietal cortex then projects to another multimodal sensory association area in the frontal – premotor and frontal eye fields (discussed earlier in relationship to premotor and prefrontal motor function). Area 7 has connections to the limbic cortex: cingulate gyrus. The implications of these connections for the integration of complex movements are clear. It is not surprising then that single cell studies as reviewed by Darian-Smith et al. (1979) demonstrate responses of neurons in area 5 to manipulation of joints: hand manipulation as in grasping and manipulation objects, or in projecting the hand to obtain a specific object associate with reward. Neurons in area 7 discharged in relationship to complex eye and limb movements Lynch (1998). When damage to parietal cortex occurs, the patient manifests a variety of spatial deficits. Neglect occurs in relation to multiple sensory modalities but is most prominent with regard to contralateral visual space as we will discuss later. There are corresponding deficits in the generation of spatially directed actions.

**Stimulation:** The threshold of the superior and inferior parietal lobules for discharge is relatively high. Although Foerster reported the occurrence of some contralateral paresthesias on stimulation in man, Penfield did not confirm these results. Stimulation in the inferior parietal areas of the dominant hemisphere did produce arrest of speech, but this is a nonspecific effect, occurring on stimulation of any of the speech areas of the dominant hemisphere. It must, of course, be noted that space-occupying lesions in the parietal lobules may produce sensory or motor seizures by virtue of their pressure effects on the lower threshold post and precentral gyri.

**Lesions:** Darian-Smith et al. (1979) have summarized the effects of selective lesions of area 5 and/or area 7 in the monkey. The most selective studies were those of using reversible cooling lesions. Cooling of area 5 resulted in a clumsiness of the contralateral arm and hand so that the animal was unable to search for a small object. Cooling of area 7 produced a much more complex clumsiness which was apparent only when the arm was moved into the contralateral visual field. Movements of the hand to the mouth were intact (refer to discussion of Wise 1985, Wise et al. 1997, for a more recent discussion of the topic). Earlier clinical studies in humans had suggested possible sensory deficits in relation to ablation of the inferior or superior parietal areas. The detailed studies of on patients subjected to limited cortical ablations of the inferior or superior parietal areas (in the treatment of focal epilepsy) clearly indicated that no significant sensory deficits occurred. The postcentral gyrus rather than the parietal lobules is critical for somatic sensory discrimination. The more recent studies of confirmed that anterior parietal (postcentral lesions) resulted in somatosensory disturbances including surface sensibility, two-point discrimination, position sense, as well as more complex tactile recognition. In contrast, in posterior parietal lesions, there was a preferential impairment of complex somatosensory and motor functions involving exploration and manipulation by the fingers – not explained by any sensory deficit. There was a deficit in the conception and execution in the spatial and temporal patterns of movement. The end result is an impairment of purposive movement as discussed earlier. The response to visual stimuli in terms of attending to and reaching for objects is impaired. Lesions involving the white matter deep to the inferior parietal lobule may damage the superior portion of the optic (geniculocalcarine) radiation, producing a deficit in the inferior half of the contralateral visual field, an inferior quadrantanopsia.

**Dominant Hemisphere in the Parietal Lobules**

**Gerstmann’s syndrome.** Destructive lesions of the parietal lobules produce additional effects on more complex cortical functions. Those lesions involving particularly the supramarginal and angular gyri of the dominant (usually the left
hemisphere) inferior parietal lobule may produce one or more of a complex of symptoms and signs known as Gerstmann’s syndrome.

Gerstmann’s syndrome:
(a) Dysgraphia (a deficit in writing in the presence of intact motor and sensory function in the upper extremities)
(b) Dyscalculia (deficits in the performance of calculations)
(c) Left–right confusion
(d) Errors in finger recognition, for example, middle finger, index finger, ring finger, in the presence of intact sensation (finger agnosia)
(e) In addition, disturbances in the capacity for reading may be present. Some patients may also manifest problems in performing skilled movements on command (an apraxia) at a time when strength, sensation, and coordination are intact. Usually only partial forms of the syndrome are present. The problem of the dominant parietal lobe in language function will be considered in greater detail and illustrated in the section on language and aphasia. The reader should refer to Chap. 15 for an illustrative

An inability to interpret drawings. The patient has difficulty with a map or in picking out objects from a complex figure. The patient is confused as to figure background relationship and is disoriented in attempting to locate objects in a room. When asked to locate cities on an outline map of the United States, the patient manifests disorientation as the west and east coasts and as to the relationship of one city to the next. Chicago and New Orleans may be placed on the Pacific Ocean, Boston on the Florida peninsula, and New York City somewhere west of the Great Lakes. The disturbance in capacity for the construction of drawings has been termed a constructional apraxia or dyspraxia. An apraxia may be defined as an inability to perform a previously well-performed act at a time when voluntary movement, sensation, coordination, and understand are otherwise all intact. The following deficits may be present: the patient may be unable to draw a house or the face of a clock, or to copy a complex figure such as a three-dimensional cube, a locomotive, and so forth; in severe disturbances the patient may be unable to copy even a simple square, circle, or triangle. The following case demonstrates many of these features.

Case 14.3
This 70-year-old, single, white female, right-handed, underwent a left radical mastectomy for carcinoma of the breast, 3 years prior to admission. Four months prior to evaluation, the patient became unsteady with a sensation of rocking as though on a boat. She no longer attended to her housekeeping and to dressing. Over a 3-week period, prior to evaluation, a relatively rapid progression occurred with deterioration of recent memory. A perseveration occurred in motor activities and speech. The patient was incontinent but was no longer concerned with urinary and fecal incontinence. For 2 weeks, right temporal headaches had been present. During this time, her sister noted the patient to be neglecting the left side of her body. She would fail to put on the left shoe when dressing. In undressing, the stocking on the left would be only half removed.

Family history: The patient’s mother died of metastatic carcinoma of the breast.

Neurological examination: Mental status: Intact except for the following features: The patient often wandered in her conversation. She often asked irrelevant questions and was often impersistent in motor activities. There was marked disorganization in the drawing of a house or of a clock. A similar marked disorganization was noted in attempts at copying the picture of a railroad engine (Fig. 14.3). There was a marked neglect of the left side of space and of the left side of the body. The patient failed to read the left half of a page. When she put her glasses on, she did not put the left bow over the ear. When getting into bed, she did not move the left leg into bed. She had slipped off her dress on the right side,
Superior and Inferior Parietal Lobules

but was lying in bed with the dress still covering the left side. The patient had been reluctant to come for neurological consultation. Although she complained of headache and nausea, she denied any other deficits. Her relatives provided information concerning these problems. Much additional persuasion over a 2-week period was required before the patient would agree to be hospitalized.

Cranial nerves: A dense left homonymous hemianopsia was present. When reading, the patient left off the left side of a page. She bisected a line markedly off center. Disk margins were blurred and venous pulsations were absent, indicating papilledema. The right pupil was slightly larger than the left. A minimal left central facial weakness was present.

Motor system: Although strength was intact, there was little spontaneous movement of the left arm and leg. The patient was ataxic on a narrow base with eyes open with a tendency to fall to the left, and was unable to stand with eyes closed even on a broad base.

Sensation: Although pain, touch and vibration were intact, there was at times a decreased awareness of stimuli on the left side. Errors were made in position sense at toes and fingers on the left. Tactile localization was poor over the left arm and leg. With double simultaneous stimulation, the patient neglected stimuli on the left face, arm, and leg.

Clinical diagnosis: Metastatic breast tumor to right nondominant parietal cortex.

Laboratory data: Chest X-ray indicated a possible metastatic lesion at the right hilum. EEG was abnormal because of frequent focal 3–4 cps slow waves in the right temporal and parietal areas, suggesting focal damage in these areas.

Subsequent course: Treatment with steroids (dexamethasone and estrogens) resulted in temporary improvement. The patient refused surgery. Her condition soon deteriorated with increasing obtundation of consciousness. She expired 2 months following her initial neurological consultation.

A CT scan from a more recent case demonstrating many aspects of this syndrome is illustrated in Fig. 14.4. In many cases, the location of lesion may appear to be predominantly posterior temporal. Such large posterior temporal lesions would certainly compromise the cortex and subcortical white matter of the adjacent inferior parietal area. In this case, marked deficits in perception of the cortical modalities of sensation were present. In other cases, as in the case demonstrated in Fig. 14.3, such involvement is much less marked. In some cases, involvement of the motor cortex is evident with an actual left hemiparesis accompanied by an increase in deep tendon reflexes and an extensor plantar response. At times in patients with neglect syndromes, there may be several indications in the clinical examination and in the laboratory studies that the involvement of the frontal lobe areas is more prominent than the parietal involvement. We have already indicated that the neglect components of this syndrome may also be noted in lesions of the anterior premotor area (area 8). The prefrontal and premotor areas as discussed earlier and in Chap. 10 receive projection fibers from the multimodal area of the posterior parietal area. It is possible that in some cases the posterior temporal–inferior parietal location of the lesion may also be critical in interrupting these association fibers.

For these several reasons, it is perhaps more appropriate to use the term, syndrome of the nondominant hemisphere, rather than the more localized designation, nondominant inferior parietal syndrome. have presented the concept of a network for directed attention – with right frontal lesions leading to left hemispatial neglect only for tasks that emphasize exploratory-motor components of directed attention whereas parietal lesions emphasize the perceptual-sensory
aspects of neglect. With lesions of the nondominant hemisphere, there is a significant alteration of the patient’s awareness of his environment. The behavior of an individual is in part determined by his own particular perception of the environment. If that perception is altered or disorganized, the behavioral responses of the patient may appear inappropriate to others. Obviously, not all individuals will respond in the same manner to a given environmental situation; part of the response will be determined by the past experience and personality of the individual. Thus, given the same lesion, one individual may be unaware of a hemiparesis, another may deny the hemiparesis but agree an illness is present, a third may claim to be healthy and claim that people are conspiring to keep him in the hospital.

**Parietal Lobe and Tactile Sensation from the Body**

**Basic principle of sensory system:** All sensory systems have three neurons:

*First-order neuron* found in the periphery – the dorsal root ganglion or ganglion of cranial nerves V, VII, IX, or X. Its axons are ipsilateral.

*Second-order neuron* within CNS in spinal cord or brain stem; its axon crosses to the contralateral side.

*Third-order neuron* within thalamus is the final subcortical neuron in the sensory systems and its axons synapse in the cerebral cortex.

**Tactile Sensation from the Body: Medial Lemniscus (Fig. 14.5)**

The ascending sensory fibers from the neck, trunk, and extremities for touch (posterior columns), from the face for touch and pain (trigeminothalamic), and from the viscera for general and special sensation ascend in the medial lemniscus to the thalamus.

**Posterior Columns: Tactile Sensation from the Neck, Trunk, and Extremities (Fasciculus Gracilis and Cuneatus)**

The posterior columns – the fasciculus gracilis and the fasciculus cuneatus (Fig. 14.5) – conduct proprioception (position sense), vibration sensation, tactile discrimination, object recognition, deep touch (pressure) awareness, and two-point discrimination from the neck, thorax, abdomen, pelvis, and extremities. The sensory receptors for the system are the Golgi tendon organs, muscle spindles, proprioceptors, tactile disks, and Pacinian corpuscles (deep touch or pressure). The primary cell body is located in the dorsal root ganglion. The well-myelinated fibers of this system enter the spinal cord as the medial division of the dorsal root and bifurcate into ascending and descending portions, which enter the dorsal column. In the spinal cord, the posterior columns are uncrossed and divided into the medial gracile fasciculus (lower extremity) and the lateral cuneate fasciculus (upper extremity). The fasciculus gracilis contains fibers from the sacral, lumbar, and lower thoracic levels, while the fasciculus cuneatus contains fibers from the upper thoracic and cervical levels. Fibers from the sacral levels lie most medial, followed by lumbar, thoracic, and finally, cervical fibers. Fibers from the upper extremity form 50% of the posterior columns with the lower extremity 25% and the remainder from the thorax and abdomen. The primary axons ascend in the dorsal columns of the spinal cord to the secondary cell body of this system located in the nucleus gracilis and the nucleus cuneatus in the spinomedullary junction. The secondary fibers cross in the sensory decussation and form the bulk of the medial lemniscus along with trigeminal and other fibers, and ascend in the contralateral medial lemniscus to

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**Fig. 14.4** Nondominant parietal lobe syndrome. CT scans. This 68-year-old left-handed male with diabetes mellitus and hypertension had the sudden onset of left arm paralysis, loss of speech and ability to read and write all of which recovered rapidly. On examination 9 months after the acute episode he continued to have the following selective deficits: (1) He was vague in recalling his left hemiparesis. (2) In dressing, he reversed trousers and failed to cover himself on the left side. (3) He had extinction in the left visual field on bilateral simultaneous visual stimulation and over the left arm and leg on bilateral simultaneous tactile stimulation. (4) A left Babinski sign was present. CT scan now demonstrated an old cystic area of infarction in the right posterior temporal-parietal (territory of the inferior division of the right middle cerebral artery).
the ventral posterior lateral (VPL) nucleus in the thalamus. From this thalamic nucleus these fibers are projected to the postcentral gyrus (areas 1–3). Fibers also descend in the dorsal columns, but their functional significance is unknown. The fibers responsible for proprioception cross in the medial lemniscus. The fibers for vibration sensation and tactile discrimination ascend bilaterally in the medial lemniscus to the VPL nucleus. Consequently, a unilateral lesion can abolish proprioception, but tactile discrimination and vibration sensation will not be entirely lost.

**Clinical lesions.** Injury to the posterior column appears not to affect pressure sense, but vibration sense, two-point discrimination, and tactile discrimination are diminished or abolished, depending on the extent of the lesion. Interruption of the medial fibers (cervical-hand region) impairs the ability to recognize differences in the shape and weight of objects placed in the hand. Since the extremities are more sensitive to these modalities than any other body regions, position sense is impaired more severely in the extremities than elsewhere, and the person has trouble identifying small passive movements of the limbs. Consequently, performance of voluntary acts is impaired and movements are clumsy (sensory ataxia). Lesions in lateral fibers of the posterior column, gracilis, may be devastating due to the interruption of one of the most important sensory mechanisms the ability to detect the sole of the foot. This is a major handicap in walking in dim lighted or a dark room, in driving a car, etc.

**Tactile Sensation from the Head (Fig. 14.6)**

Cranial nerve V is the largest cranial nerve in the brain stem and it has three sensory nuclei (chief, spinal, and mesencephalic) and three divisions: Ophthalmic (V1), Maxillary (V2), and Mandibular (V3). The primary cells of the trigeminal
nerve are located in the trigeminal ganglion in Meckel’s cave in the middle cranial fossa. The second nuclei are in the brain stem and upper cervical spinal cord. Each of the three divisions brings in sensation of pain, temperature, touch, and pressure from receptors in the skin, muscles, and sinuses they innervate.

1. **Proprioception from the head** – Mesencephalic nucleus. These unique primary cell bodies are located not only in the trigeminal ganglion in the middle cranial fossa but also along the nerve rootlet within the pons and midbrain, where they form the mesencephalic nucleus of nerve V. The primary axons project to the motor nucleus of nerve V in the pond and the reticular formation. Axons are also projected to the cerebellum and inferior olive.

2. **Pain and temperature-spinal/descending nucleus of V**. The origin of the second neuron is the descending nucleus of nerve V. The secondary axons ascend in the dorsal portion of the medial lemniscus to the VPM nucleus of the thalamus (see Chap. 4).

3. **Tactile sensation-chief/main nucleus of V**. The primary tactile fibers synapse within the chief sensory nucleus of V the second-order nucleus of V in the pons. The proprioceptive fibers arise from the muscles in the face including the muscles of mastication, facial expression, ears, and the muscles of the eye. From this second-order nucleus, axons enter the brain stem, ascend bilaterally through the pons, and synapse in the midbrain on the mesencephalic nucleus of V from which fibers then descend onto the motor nucleus of V in the pons for the “jaw jerk.” The primary tactile and proprioceptive axons synapse in the chief nucleus and from this second-order neuron the information ascends in the dorsal most portions of the medial lemniscus, the trigeminal lemniscus and synapse in the VPM nucleus of the thalamus. This information is then sent onto the lower 1/3 of the postcentral gyrus, the region that contains the somatotopic representation for the head and neck including the face (Fig. 14.1).

**References**


Suggested Readings


References


Olfactory References