Handbook of Clinical Neurology

Volume 151, 2018, Pages 185-206

Chapter 9 - Somatosensory deficits

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https://doi.org/10.1016/B978-0-444-63622-5.00009-7

Abstract

The analysis and interpretation of somatosensory information are performed by a complex network of brain areas located mainly in the parietal cortex. Somatosensory deficits are therefore a common impairment following lesions of the parietal lobe. This chapter summarizes the clinical presentation, examination, prognosis, and therapy of sensory deficits, along with current knowledge about the anatomy and function of the somatosensory system. We start by reviewing how somatosensory signals are transmitted to and processed by the parietal lobe, along with the anatomic and functional features of the somatosensory system. In this context, we highlight the importance of the thalamus for processing somatosensory information in the parietal lobe. We discuss typical patterns of somatosensory deficits, their clinical examination, and how they can be differentiated through a careful neurologic examination that allows the investigator to deduce the location and size of the underlying lesion. In the context of adaption and rehabilitation of somatosensory functions, we delineate the importance of somatosensory information for motor performance and the prognostic evaluation of somatosensory deficits. Finally, we review current rehabilitation approaches for directing cortical reorganization in the appropriate direction and highlight some challenging questions that are unexplored in the field.

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Keywords

somatosensory; thalamus; parietal lobe; pathways; deficits; rehabilitation

Introduction

Perception and interpretation of somatosensory information are general requirements in human life. Although the

impression of touch is considered as one of the five traditional senses, this impression is formed from a multitude of different somatosensory modalities, including touch, pressure, vibration, temperature, pain, and skin stretch. Information for each of these modalities is transmitted from the receptors via sensory nerves that ascend through the spinal tracts and thalamus to the parietal lobe.

The processing of somatosensory information is performed in a complex interplay between different parietal cortical regions and the thalamus (Klingner et al., 2015b). A lesion at any stage of the forwarding or interpretation of somatosensory information alters the processing in the parietal cortex, leading to somatosensory deficits. While impairments in the early stages of somatosensory transmission also change the interpretation at later stages of the cortical processing (Caselli, 1991), in this chapter we will discuss impairments at the thalamic and parietal lobe levels of the processing hierarchy. It is important to note that somatosensory information is mostly interpreted in conjunction with information from other sensory modalities. Hence, the somatosensory network is closely interlinked with all other sensory networks. Particularly, visual information can modify the somatosensory experience by altering the context in which somatosensory information is interpreted (Schaefer et al., 2006; Torta et al., 2015;Van der Biest et al., 2016). Likewise, impairments in other brain systems can alter the associative interpretation of somatosensory information (Mundinano and Martinez-Millan, 2010).

In clinical practice, the testing of somatosensory functions is one of the most important, but also most difficult, aspects of the neurologic examination and requires a detailed knowledge of the anatomy and functions of the somatosensory system. The multitude of somatosensory modalities and the complexity of their transmission and processing lead to very complex, but nevertheless, remarkably differentiable patterns of impairment. Therefore, the exact investigation of somatosensory deficits in the clinical examination provides important information to determine the anatomic location of the lesion causing the deficits and avoid unnecessary diagnostic tests (e.g., magnetic resonance imaging (MRI)/computed tomography from multiple body regions). Somatosensory deficits are a major complaint in most peripheral lesions, and somatosensory deficits due to central brain lesions have prevalence rates between 65% and 100% (Carey et al., 1993; Kim and Choi-Kwon, 1996; Rand et al., 2001; Connell et al., 2008). This variability is caused by differences in the definitions and assessment of somatosensory deficits. Outside of scientific studies, impaired sensory function is often underdiagnosed. Even in patients who were diagnosed with a pure motor stroke identified by a neurologic screening examination, sensory dysfunction was found in 88% of cases (Kim and Choi-Kwon, 1996).

The outstanding importance of somatosensory deficits in neurologic examinations, and their high prevalence, is accompanied by relatively minor importance in the neurologic rehabilitation of somatosensory deficits, as compared to motor deficits. However, impaired somatosensory input, and particularly proprioceptive information, was shown to severely impair motor performance, especially in goal-directed movement, grasping (Gentilucci et al., 1994), reaching (Gordon et al., 1995), balance (Sainburg et al., 1995), and locomotion (Dietz, 2002). Somatosensory deficits can severely exacerbate functional deficits, even in patients with good motor function (Doyle et al., 2010). Likewise, the ability to acquire new motor skills during rehabilitation depends heavily on the functioning of the somatosensory system (Vidoni and Boyd, 2009).

The purpose of this chapter is to provide a comprehensive overview of knowledge about somatosensory deficits along with the underlying anatomic and functional features of the somatosensory system.

We further summarize the clinical presentations, examination, differential diagnoses, and therapy of sensory syndromes. Herein, impairments and lesions located in the parietal lobe will be of particular importance. To integrate all findings within a theoretic framework, we briefly review the theory of cortical responses and predictive coding (Friston, 2005), and use this theory to explain the available experimental data.

Somatosensory processing in the parietal lobe

Principles of somatosensory learning and inference by the brain

This section considers the current knowledge about the processing of somatosensory information. Before we review the anatomic and functional properties of the somatosensory system, we will briefly discuss the broader framework of what the brain attempts to achieve.

The overall goal of somatosensory processing is to infer the cause of a stimulus. In this context, the cause is any process in the world that generates the somatosensory data, which can be another person who touches us, or the

movements of our fingers that touch the keyboard while we are writing these lines. We are able to obtain a conscious impression about specific features of a somatosensory stimulus, e.g., “the fabric is silky soft and pleasant to touch.” However, from an evolutionary perspective, the ability to sense the different somatosensory properties of an object is mainly used to improve the inference about the object itself. It is important to note that the ability to infer the correct cause of a stimulus requires the availability of a representation of the world and a representation of the process that caused the stimulus. Consider, for example, a light pressure on our back. The brain has to embed this new information into the existing representation of our surrounding (“Is someone standing behind us and are they touching us, or are we in a wood and have taken a step back?”). Often the information delivered by a simple somatosensory stimulus is not sufficient to infer its cause.

Current theories about how the brain infers the cause of a stimulus suggest that the brain has learned a generative model of our world (Friston, 2005). This includes representations of processes that generate specific somatosensory stimuli. To infer the cause of a stimulus, the brain tries to invert its generative model. However, the combinatorial explosion of how stochastic generative models can generate input patterns leads to a “many-to-one” relationship between causes and inputs. This indeterminacy renders the problem of inferring causes from sensory information ill posed, in that there is no uique solution. Because of the “many-to-one” relationship and the often insufficient sensory data, making inferences about the possible causes requires an internal representation of the probability of the specific causes given the data. Mathematically, this process can be described with empiric Bayes and hierarchic models that provide an account for many aspects of cortical organization and responses (Friston, 2005; Friston and Kiebel, 2009). In recent years, this theory of brain responses has become the leading theory under the term predictive coding. This name refers to the principle that the brain uses prediction error of responses to adjust the state of the internal generative model until the prediction error is minimized, thereby identifying the most likely cause of the sensory input.

The somatosensory pathway to the parietal lobe

The human body contains a multitude of somatosensory receptors that deliver a great spectrum of information about the current state and position of our body, and the state and properties of our environment. The acquired information contains information about the length, velocity, and tension of muscles, as well as the angle and torque of joints. Human skin has different receptors that sense vibration, skin stretch, touch, temperature, and pain. Pain and temperature sensation arise from free nerve endings in the skin. Light touch is sensed by free nerve endings, Merkel cells, and encapsulated mechanoreceptors. These encapsulated receptors provide information about touch, pressure, vibration, and cutaneous tension (Meissner, Pacinian, and Ruffini corpuscles) (Grunwald, 2008; Purves, 2008).

These receptors are further specialized to different types of stimulation, such as fast-adapting receptors (Pacinian corpuscles) for on/off inputs, and slow-adapting receptors for a constant monitoring of a stimulus (e.g., Meissner corpuscles). Receptor information is transmitted to the brain by nerves. Nerve fibers can be classified into three major size groups: large myelinated, small myelinated, and unmyelinated nerve fibers. The conduction velocity of these fibers is directly dependent on their size and whether they are myelinated. Large myelinated fibers have the fastest conduction velocities (exceeding > 100 m/s), whereas small unmyelinated fibers have the slowest conduction velocities (as slow as < 1 m/s). Afferent nerve fibers can be further classified into four types: I–IV. This terminology relies on axon conduction velocity, in which group I is the fastest and group IV is the slowest. Group I nerve fibers mediate information about tension, length, and velocity of muscles from Golgi tendon organs and muscle spindles. Group II nerve fibers mediate information from mechanoreceptors of the skin about touch, vibration, and skin stretch. Group III and IV nerve fibers mediate information about pain and temperature from free nerve endings in the skin (Grunwald, 2008; Purves, 2008).

Somatosensory information is mediated by four different pathways to the central nervous system, which are described below.

The medial lemniscal pathway: discriminative touch and proprioception

Somatosensory information is mediated by peripheral nerves that have their cell bodies in the dorsal root ganglia. Without synapsing or crossing the midline, the axons ascend further in the posterior column of the spinal cord up to the medulla. Here, the neurons synapse in the gracile nucleus (lower body) and the cuneate nucleus (upper body). The axons of these two nuclei cross the midline and form the medial lemniscus that ascends to the diencephalon and terminates in the ventral posterior lateral nucleus (VPL) of the thalamus. The axons of VPL neurons ascend in the posterior limb of the internal capsule and synapse mostly in the postcentral gyrus.

The neospinothalamic pathway: pain and temperature

Information about body temperature and pain is mediated by peripheral nerves that have their cell bodies in the dorsal root ganglia and synapse in the posterior marginal nucleus as they enter the spinal cord. The axons of the marginal nucleus cross the midline at the same level and then ascend through the spinal cord and the brainstem in the spinothalamic tract. Some afferents leave the spinothalamic tract within the brainstem, and synapse in the reticular formation and the periaqueductal gray of the midbrain. The other afferents ascend to the thalamus, where they synapse in the VPL and intralaminar nuclei of the thalamus. The thalamic axons convey information about touch and proprioception and ascend together within the posterior limb of the internal capsule, synapsing mostly in the postcentral gyrus.

The main somatosensory trigeminal pathway: discriminative touch and proprioception of the face

Somatosensory information from the face is transmitted by the trigeminal nerve to the trigeminal nucleus in the pons. Axons originating in the trigeminal nucleus cross the midline and ascend in the ventral trigeminal lemniscus to the ventral posterior medial (VPM) nucleus of the thalamus. The axons of the VPM ascend in the posterior limb of the internal capsule to the primary somatosensory cortex (SI).

The spinal trigeminal pathway: crude touch, pain, and temperature of the face

Somatosensory information from the face is transmitted by the trigeminal nerve to the principal nucleus of the trigeminal complex in the pons. Axons originating in the principal nucleus cross the midline, and ascend in the trigeminothalamic tract to the VPM nucleus of the thalamus. The axons of the VPM ascend in the posterior limb of the internal capsule to SI.

Although most information is transmitted directly to neurons of the SI area of the postcentral gyrus, recent findings indicate that some information is transmitted from the thalamus directly to the parietal operculum, where the secondary somatosensory cortex (SII) is located (Rowe et al., 1996; Klingner et al., 2015a).

Parietothalamic interactions

The thalamus was long considered a simple relay station for information transmitted to the cortex. This thinking has been replaced in the last two decades by increasing evidence indicating the involvement of the thalamus in nearly all aspects of cortical information processing. This major influence of the thalamus is attributed to two pieces of evidence. First, the thalamus can modulate the flow of information to the cortex (Sherman and Guillery, 2002). Second, the thalamus influences cortical processing by direct modulation of these brain areas, and the information transfer between brain areas by modulating the corticocortical information transfer (Sherman, 2012).

Sherman and colleagues suggest two classes of thalamocortical pathways: class I, carrying the information to be processed, and class II, modulating the information processing (Sherman and Guillery, 2011; Sherman, 2012). These two classes are separated by multiple properties, including the type of activated receptors, synapse properties, convergence of targets, and axon diameter. The corresponding thalamic subnuclei are also classified according to both classes. It was shown that the thalamic subnuclei in class I (also termed “driver” or “first-order”) receive subcortical input, whereas subnuclei in class II (also termed “modulator” or “higher-order”) receive input from layer 5 of the cortex (Reichova and Sherman, 2004; Lee and Sherman, 2008; Theyel et al., 2010). These higher-order thalamic nuclei relay information from one cortical area to another. Corticothalamic axons outnumber thalamocortical axons by a factor of 10 (Guillery, 1967; Liu et al., 1995; Temereanca and Simons, 2004). The modulatory role of the thalamus was further supported by demonstrating that cortical habituation originates to a large extent from altered information relayed through the thalamus (Klingner et al., 2011b). This combination of driving and modulatory pathways allows the cortex to regulate its own input from the thalamus, as well as the amount of information transmitted to other cortical areas.

The functional significance of these corticothalamocortical connections within the somatosensory network remains elusive, because of the parallel existence of direct corticocortical connections. However, increasing evidence suggests that thalamic neurons responsible for the processing and forwarding of information from one cortical area to another do not share the same characteristics as thalamic neurons that process and forward afferent sensory information from the body.

The corticothalamocortical pathway seems to provide driving input (input that drives cortical activity) to higher-order

cortical areas, but provides modulatory input (input that alters the transmission of sensory-driven activity) to the primary sensory cortices (Sherman and Guillery, 2011; Sherman, 2012). This architecture constitutes a hierarchic feedforward information transfer. In contrast, corticocortical connections have a similar architecture in both directions, providing no clear hierarchic order (Covic and Sherman, 2011; De Pasquale and Sherman, 2011). Accordingly, the VPL nucleus has connections with SII, the posterior parietal cortex (PPC), and the insula, as well as particularly strong connections with SI (Burton and Jones, 1976; Jones et al., 1979; Friedman and Murray, 1986; Friedman et al., 1986).

It has been shown that the processing and transmission of thalamic information are constantly adjusted by sensory experience (Herrero et al., 2002; Temereanca and Simons, 2004; Zhang et al., 2007; Klingner et al., 2011b, 2013). One underlying mechanism is the modulation of the information transfer through the transthalamic pathway (corticothalamocortical) (Sherman and Guillery, 2011). Parietal brain lesion should therefore not only alter the processing and interpretation of afferent sensory information but should also alter the thalamic influence on afferent information as well as the transthalamic information flow between cortical brain areas. Therefore, it can be assumed that the thalamus has an important role in the adaptation of somatosensory information processing due to parietal brain lesions.

Organization of the somatosensory cortex

Studies investigating the organization of the somatosensory brain network have long been dominated by anatomic studies. The rise of functional imaging techniques in the last two decades has greatly improved our understanding of the localization and function of brain areas that are involved in the processing of somatosensory information. Particularly, functional MRI (fMRI) is well suited to localizing the functions of brain areas with a high spatial accuracy. Studies utilizing fMRI, magnetoencephalography, or electroencephalography have revealed a complex network of cortical areas involved in the processing of somatosensory information. The synthesis of anatomic and functional data has revealed detailed knowledge about the network of responsible brain areas, involved in the processing of somatosensory information: they include the thalamus, SI and SII, PPC, insula, medial cingulate cortex, and ipsilateral cerebellum (Karhu and Tesche, 1999; Korvenoja et al., 1999; Backes et al., 2000; Boakye et al., 2000; Del Gratta et al., 2000; Kampe et al., 2000; Deuchert et al., 2002; Ferretti et al., 2003; Kanno et al., 2003; Nihashi et al., 2005; Arienzo et al., 2006; Dijkerman and de Haan, 2007; Klingner et al., 2010, 2011a, b, 2013, 2014, 2015b). Within this network, several aspects of somatosensory signals are processed, including detection, recognition, location, intensity, pleasantness, preparation for action, and affective interpretation (Del Gratta et al., 2000; Schnitzler and Ploner, 2000; Bingel et al., 2002; Ferretti et al., 2003; Arienzo et al., 2006; Henderson et al., 2007; Lamm et al., 2007).

Most somatosensory information enters the cerebral cortex in SI, through projections from the VPL nucleus of the thalamus. SI is located in the postcentral gyrus and includes Brodmann areas (BA) 3a, 3b, 1, and 2. These areas have different histologic characteristics. Each part of the body is represented according to its sensitivity; therefore, very sensitive body parts, such as the lips or the fingertips, have large cortical representations. It is thought that thalamic projections enter mainly BA 3b, but they also enter BA 1 and 2. These three tightly interconnected areas constitute a hierarchy that performs increasingly complex analyses from BA 3b over BA 1 to BA 2 (Depeault et al., 2013).

SII is found in the lower part (inferior parietal lobule) of the PPC (BA 40, supramarginal gyrus). The main source of input is transmitted from SI, though BA 40 also receives inputs from the thalamus and the contralateral SII (Rowe et al., 1996; Klingner et al., 2015a). This area integrates somatosensory information with other sensory modalities (e.g., vestibular and auditory input) and both sides of the body, and it performs higher-order somatosensory processing (see Chapter 7 for multisensory integration).

Posterior to the SI is the somatosensory association cortex (BA 5, 7), which is located in the superior parietal lobule of the PPC, and which is mainly involved in many higher-order perceptual, attentional, and motor-planning activities, as discussed in other chapters.

The insula can be divided into a granular posterior portion and an agranular anterior portion (Dupont et al., 2003). It has been shown that the anterior insula is more heavily involved in the processing of nociceptive stimuli, whereas the posterior insula is more involved in nonnociceptive stimuli (Klingner et al., 2011a; Allen et al., 2015; Hu et al., 2015). The insula is further thought to be involved in the integration of somatosensory stimuli with information from other sensory modalities.

Current evidence indicates the existence of at least two different processing streams for nonpainful somatosensory stimuli. Both streams originate in the thalamus and project to SI. One stream is further transmitted to SII and then the

posterior insula, whereas the other stream terminates in the PPC (Dijkerman and de Haan, 2007). The sequence of brain areas along these pathways reflects a hierarchy in the processing of somatosensory information. It was proposed that both streams participate in the recognition and perception of somatosensory information, whereas action-related processing occurs mainly in the PPC (Dijkerman and de Haan, 2007). Both streams transmit information to homologous brain areas in the contralateral hemisphere at later stages of the processing hierarchy.

In conclusion, there is currently a good understanding of which brain areas are involved in the processing of somatosensory information. There is additional detailed knowledge indicating where these areas are located, how they are connected anatomically, and in which tasks they are involved. However, we know less about the functional properties of the connections among cortical areas and how information is processed within each brain area. Moreover, it remains elusive how we become aware of a stimulus (see Chapter 14 for the role of unilateral spatial neglect in impairing awareness of somatosensory stimuli), and how somatosensory stimuli and information are stored and recognized. Despite major progress in understanding the cerebral processing of somatosensory information, these unanswered points lead us to conclude that there is a major lack of understanding of even the basic functions of somatosensory brain networks.

Clinical examination of somatosensory deficits

In clinical examinations of somatosensory function, one has to distinguish between primary and cortical sensory modalities (Campbell et al., 2005). The primary sensory modalities include touch, pressure, vibration, pain, temperature, and position sense of joints. The cortical sensory modalities are the sensory experiences that are fused from the primary sensory modalities by the parietal cortex. For example, the ability to recognize an object using tactile information alone (stereognosis) utilizes different primary modalities, including touch, pressure, position sense, and temperature. Other examples of cortical sensory modalities are two-point discrimination, graphesthesia, and tactile localization. Testing primary and cortical sensory modalities provides an indication of where a possible lesion is located in the sensory system and whether the parietal cortex is affected. Abnormal sensory sensation can be characterized relative to normal sensation as a decrease, increase, absence, or distortion. An increase in sensation is particularly important for pain perception where an increased excitation of receptors, neurons, or fibers causes an unpleasant feeling. Examples of a distorted sensory sensation are paresthesia, dysesthesia, and phantom sensation.

The primary goal of every examination of somatosensory function is to reveal abnormal functions and their distributions. The description of abnormal functions includes the affected modality, as well as its degree and distribution. The multitude of sensory modalities and their complex cortical interactions make the examination of sensory function undoubtedly the most complex part of the neurologic investigation. In clinical practice, this complexity often creates the need to shorten this investigation to a sensory screening in the absence of sensory deficits. Particularly, an altered mental status, decreased vigilance, or extensive deficits, which can occur with major strokes, can greatly reduce the potential for a conclusive examination of sensory function down to a simple reaction test for painful stimuli at different parts of the body. However, the observation of sensory deficits, diseases that cause sensory deficits, or other symptoms, such as ataxia, should always trigger a complete sensory examination when possible. An additional difficulty is the fact that the somatosensory examination relies heavily on the responsiveness and cooperation of the patient. Furthermore, the patient has to understand the procedure. Although there are techniques to uncover unreliable declarations of sensory deficits from the patient (Stone et al., 2002; Campbell et al., 2005), sensory examinations are still the least objective part of a neurologic examination and require the greatest amount of knowledge and experience in neurology.

As in most areas of neurology, a patient's description and history of sensory symptoms are of utmost importance. Patients should always be asked whether they have experienced changes in sensation and whether there are abnormal or spontaneous sensory experiences. When sensory changes are reported, they should be further investigated to determine the affected modality and whether there is an increase, decrease, absence, or distortion in the affected modalities. Moreover, cortical sensory function should also be examined.

Next, one has to inquire about the time course and distribution of the sensory changes. A description of symptoms is also helpful to differentiate between the organic and the nonorganic causes of disability (Stone et al., 2002). Patients suffering from nonorganic causes often describe their symptoms according to the point of view of their own body; for example, patients with nonorganic causes may report symptoms and show signs affecting one arm, one leg, all limbs, or the whole body. These patients describe their deficits using mostly short and often interspersed words, such as “simply deaf.” The symptoms mostly affect all modalities, and the report is associated with inappropriate affect

(indifference or excessive emotionality). Descriptions of sensory deficits are often vague in character and location. However, sensory deficits with organic causes are often difficult to localize, and extreme caution should be exercised in interpreting unsuspected, imprecise, or confusing results.

All results of sensory testing should be interpreted in the context of the behavior and responsiveness of the patient. For example, it is unlikely that a patient who is indifferent to other obvious neurologic symptoms would report slight sensory deficits during history taking. On the other hand, the overly cooperative and concerned patient is likely to overreact to the slightest changes in sensation, and report variations that are not actually present. Particularly, suggestive questioning can alter the examination results in the latter group of patients. The suspicion of a nonorganic cause of sensory deficits should result only from a summary of the history and examination, and not from a single, confusing symptom.

There are two different sensory testing patterns that should be used during screening examinations. First, a distal-to-proximal examination should be performed to reveal any bilateral or symmetric sensory loss, such as polyneuropathy. Second, a side-to-side investigation should compare the major dermatomes and peripheral nerve distributions. When sensory deficits are present, their distribution should be drawn on a dermatome chart. This is helpful for comparing subsequent investigations in the course of the disease, uncovering unreliable statements, and comparing investigations between examiners (Campbell et al., 2005).

Examination techniques for primary somatosensory modalities

Tactile sensation

Light touch can be investigated by a multitude of instruments such as tissue paper, a feather, or a soft brush. As the name suggests, this modality also can be investigated by light touch with the fingertips. We suggest the latter method for sensory screening in clinical practice. One must avoid heavy pressure that affects the subcutaneous tissue, as the patient should recognize and localize the stimulus. However, no quantitative information of impairment will be acquired with these methods. The most widely used method for quantitative testing of light touch involves filaments of different thickness delivered at a varying but graded intensity (e.g., Semmes–Weinstein monofilaments: Mayfield and Sugarman, 2000).

Motion and position sense

The clinical examination should test the patient's ability to sense the movement of a body part and its position in space. Motion and position sense are tested together by passively moving a body part. The examiner inquires about the direction and angle of the movement while the patient's eyes are closed.

The testing starts at the most distal joints, such as the interphalangeal joints of the upper extremity. The examiner should use one hand to hold the patient's finger on both sides proximal to the examined joint and use the other hand to passively move the distal part of the patient's finger. The patient should be able to report the direction of movement in the absence of a sensory deficit. The ability of healthy, young individuals for sensing finger movements is normally higher than the visual resolution of the examiner. Therefore, every movement that can be visually detected should also be reported by the patient. More proximal joints only have to be tested for impairments of distal motion and position sense. An inability to sense motion and position of large joints is always accompanied by a sensory ataxia. There are more reliable assessments of position sense available (Dukelow et al., 2010; Cappello et al., 2015); however, they are currently used mainly for scientific purposes.

Vibration sense

Vibration sense is tested with a tuning fork (normally 128 Hz) placed on predefined bony prominences. Sensory receptors (mostly Pacinian and Meissner corpuscles) convert the vibration into a neural signal. The temporal resolution of the neural information transfer (action potentials) should be at least equal to the frequency of the vibration.

Demyelinating diseases can prolong the nerve refractory period, and impair the ability to code higher frequencies (McDonald, 1974). Therefore, the testing of vibration sense is a very sensitive parameter of polyneuropathy. Vibration sense testing should be performed for screening at the malleoli and the styloid processes of the radius. Further testing should be performed in case of an impaired vibration sense. Vibration sense can by tested quantitatively by noting the duration and intensity of the stimulation.

Pressure sensation

The examiner should press the fingers firmly on the patient's skin to engage subcutaneous structures, such as muscles and tendons. This test involves touching the skin and movements of muscles, which explains its close relationship to position sense and light touch. Due to this redundancy, the testing of pressure sensation is of minor importance in clinical practice.

Superficial pain

Although specialized equipment exists, it is mostly sufficient to break a wooden applicator stick to create sharp shards. Contact with the skin should elicit a mildly painful sensation. Whatever instrument is used to test superficial pain, it has to be discarded after testing due to the potential infectious risk for other patients from possible skin puncture.

The difference between sharp and dull should not be evaluated by asking: “Is this dull or sharp?” Also questions such as, “Does this feel different?” or “Which feels sharper?” should be avoided, because they encourage patients to overanalyze the stimulus. A good question is: “Does it feel the same?” If the clinician is interested in quantification, the patient can be asked afterwards to indicate on a percentage scale how sharp was the object felt.

Temperature

It is widely suggested to use test tubes with warm and cold water to test temperature sensation. However, in our experience, this is a time-consuming procedure that should be reserved for patients with impaired temperature sense.

For temperature sensitivity screening we suggest using commercial devices in symmetric form but with different alloys on both sides (polymer/metal), so that the polymer alloy side feels warmer and the metal alloy side feels cooler due to the different thermal conductivity properties of the materials. For a general screening, it is sufficient to determine whether the patient can distinguish between the warm and the cold stimuli. A detailed examination should be performed only if there are pathologic findings during the screening examination. In clinical practice, impairments of temperature sensation are mostly combined with impaired pain sensation. Therefore, it is often sufficient to test only one of these modalities.

Examination techniques for cortical somatosensory modalities

The cortical somatosensory network receives and interprets information for primary somatosensory modalities. This afferent information will be correlated, synthesized, and put into context in different stages of the somatosensory processing hierarchy. All higher functions that require information from more than one primary modality, involving discriminative functions within one primary modality, or utilizing other brain functions, such as memory, are called cortical somatosensory modalities.

Accordingly, these primary modalities and the other involved brain functions have to be preserved to form conclusions about a lesion in the parietal lobe as a cause for an impaired cortical somatosensory function. Therefore, the primary somatosensory modalities should be examined prior to cortical somatosensory modalities. The following section summarizes the clinical examination of some important cortical somatosensory modalities.

Stereognosis

Stereognosis is the ability to “understand” an object by touch. This understanding involves multiple functions, including perception, recognition, and identification of multiple object properties, such as size, texture, weight, and shape. Impairments in stereognosis (astereognosis) can only be diagnosed if the perception for touch and proprioception is intact. Object recognition can be tested by having the patient identify objects by touch (e.g., a key). Submodalities can also be tested by using an object that differs only in the tested modality (e.g., same object, different size). The testing of stereognosis compares the patient's two hands. Indications for impaired stereognosis are the inability to recognize or differentiate between objects and unilateral delays in performance. The inability to recognize objects with either hand is called tactile agnosia.

Graphesthesia

Graphesthesia is one of the most sensitive measures for somatosensory function (Julkunen et al., 2005). It is tested by having the patient recognize a number or letter that is written on the patient's skin. Interestingly, the orientation of the letter is unimportant. Even slight impairments of primary somatosensory modalities can cause agraphesthesia. A

related function is the ability to recognize the direction of a movement of a light scratch.

Two-point discrimination

Two-point (or spatial) discrimination is the ability to distinguish the cutaneous stimulation of one point from the stimulation of two points. There are commercial two-point discrimination devices available that are best suited for this examination. If such an instrument is unavailable, a paper clip bent to a “V” is appropriate. The two-point discrimination is expressed in the minimal distance at which the two stimuli are felt consistently as separate. The testing should randomly vary between the presentation of one and two stimuli.

There is a great difference in the spatial discrimination ability among body regions, with the best results achieved at the tip of the tongue (1–2 mm), followed by lips and fingertips (2–4 mm), whereas at the back of the hand a distance of 20–30 mm is necessary to distinguish one from two stimuli.

Two-point discrimination can be tested by static or moving stimuli. The results should be compared between homologous body sides. The ability to discriminate two points requires precise somatosensory sensibility and is probably the most subtle sign of a parietal lesion.

Somatosensory extinction

Sensory extinction describes the inability to perceive two simultaneous stimuli. The testing is performed by stimulating homologous body regions simultaneously. A consistent inability to feel stimuli on one side of the body is considered somatosensory extinction. The severity can be roughly investigated by increasing the intensity of the stimulus of the impaired side. The deficit is considered to reflect higher-order impairments of spatial attention, and has been related to unilateral spatial neglect (see Chapters 8 and 14; Brozzoli et al., 2006; Gallace and Spence, 2008).

Autotopagnosia

Autotopagnosia, first described by Pick (1908), is the selective inability to point, both on verbal command and in imitation, to body parts that can, however, be recognized and named correctly by the patient (Denes, 1999; Ardila, 2016). Autotopagnosia is not due to a primary somatosensory deficit, but the examiner should be aware of it, since defective behavior suggesting autotopagnosia may be observed during the neurologic examination. There have been few reported cases, with lesions involving the left PPC (Buxbaum and Coslett, 2001; Schwoebel et al., 2001).

Somatosensory evoked potentials

Somatosensory evoked potentials (SEPs) are electric signals generated by the nervous system following a somatosensory stimulus. SEPs can be recorded at different levels of the somatosensory pathway and elicited by almost any somatosensory stimulus. In clinical practice, SEPs are mostly generated by transcutaneous electrical nerve stimuli of 0.2–2-ms duration and are applied at the median and posterior tibial nerves. The elicited potentials are recorded at the scalp and the cervical spine. The latency and amplitude of these potentials are analyzed, allowing the identification and monitoring of impairments of the somatosensory pathway (Mauguiere, 1999; Passmore et al., 2014).

Standardized somatosensory assessments

Here, we have included a list of standardized assessments of somatosensory functions that are often used in studies reporting quantitatively about somatosensation. The Nottingham sensory assessment is used most frequently and is easy to complete, although it lacks depth in testing of somatosensory functioning. A summary of these tests along with the tested primary and cortical modalities is given in Table 9.1.

- (Revised) Nottingham sensory assessment (Gaubert and Mockett, 2000; Stolk-Hornsveld et al., 2006);
- Quantitative sensory testing (Siao and Cros, 2003);
- Byl–Cheney Boczai stereognosis test (Byl et al., 2002);
- Fugl-Meyer assessment (Fugl-Meyer et al., 1975; Gladstone et al., 2002);
- Tactile extinction test (Schwartz et al., 1977);
- Rivermead ASSESSMENT OF SOMATOSENSORY PERFORMANCE (Winward et al., 2002);

- Semmes–Weinstein monofilaments (Mayfield and Sugarman, 2000).

Table 9.1. Standardized somatosensory assessments with the tested primary- and cortical modalities

	Primary sensory modalities						Cortical somatosensory modalities			
Test	**Touch**	**Pressure**	**Vibration**	**Pain**	**Temp.**	**Pos. sense**	**Stereo-gnosis**	**Graph-esthesia**	**Two-point discrimination**	**Somato extinctio**
rNSA	X	X	-	-	X	X	X	-	-	X
QST	X	X	X	X	X	-	-	-	-	-
BCBST	-	-	-	-	-	-	X	(X)	-	-
FMA	X	-	-	-	-	X	-	-	-	(X)
TET	(X)	-	-	-	-	-	-	-	-	X
RASP	X	X	-	-	X	X	-	-	X	X
SWM	X	-	-	-	-	-	-	-	-	-

rNSA: revised Nottingham Sensory Assessment; QST: Quantitative Sensory Testing; BCBST: Byl-Cheney Boczai Stereognosis Test; FMA: Fugl Meyer Assessment; TET: Tactile Extinction Test; RASP: Rivermead Assessment of Somatosensory Performance; SWM: Semmes-Weinstein Monofilaments.

Clinical presentations of somatosensory deficits

Somatosensory deficits are a common symptom due to impairments of the peripheral and central nervous systems. Particularly, peripheral nerves almost always include sensory afferent fibers that are more sensitive to any type of disturbance than are motor fibers, and often are the cause of consultation. Exceptions to this rule are the facial and hypoglossal nerves, which do not include somatosensory fibers (Guntinas-Lichius and Schaitkin, 2016).

Somatosensory deficits due to lesions in the central nervous system are mostly combined with impairments of other functions. However, pure sensory deficits are reported after lesions in ~ 10% of acute stroke patients (Arboix et al., 2005). These pure somatosensory deficits can by caused by lesions in all parts of the central somatosensory system: the medulla (Blitshteyn and Rubino, 2005), pons (Araga et al., 1987; Shintani et al., 1994), and mesencephalon (Tuttle and Reinmuth, 1984; Alvarez-Sabin et al., 1991), not discussed in this chapter; the thalamus (Landi et al., 1984; Bogousslavsky et al., 1988; Chen et al., 1998; Paciaroni and Bogousslavsky, 1998), internal capsule (Kim, 1999), and cortex (Derouesne et al., 1984; Arboix et al., 2005)).

Thalamic lesions/thalamic somatosensory syndrome

Although the thalamus is deeply involved in the cortical processing of somatosensory information, it is also responsible for forwarding somatosensory information. Therefore, even small lesions in the thalamus can lead to deafferentation and a loss of a substantial amount of somatosensory information. It was found that ~ 35% of patients suffering from somatosensory deficits due to a thalamic lesion also had a loss of all primary modalities of somatosensation with a face–arm–leg distribution (Paciaroni and Bogousslavsky, 1998). The other patients in this study suffered from a dissociated sensory loss with a partial (45%) or face–arm–leg distribution (20%). It is important to note that subjects with a clinically suggestive thalamic infarct, but without evidence of lesions on computed tomography or MRI, were excluded in this study (Paciaroni and Bogousslavsky, 1998). This might explain the relatively low number of patients suffering from a partial distribution of symptoms, which contradicts somewhat our own clinical experience.

Sensory hemiataxia is also a common symptom due to lateral thalamic infarcts (Caplan et al., 1988; Melo et al., 1992; Paciaroni and Bogousslavsky, 1998). The relationship between the symptoms and locations of lesions suggests a somatotopic organization of somatosensory neurons in the thalamus (Paciaroni and Bogousslavsky, 1998; Chen et al.,

2008). Clinical as well as cytoarchitectural studies in monkeys demonstrate a relative separation of inputs from the spinothalamic tract and the medial lemniscus (Asanuma et al., 1983a, b; Paciaroni and Bogousslavsky, 1998).

A further study reported decreased sensation of pressure and touch on one side of the body due to thalamic lesions, whereas position and vibration were spared (Tong et al., 2010). This report, as well as other data, suggests a possible separation of all six sensory modalities (tactile sensation, motion and position sense, vibration sense, pressure sensation, superficial pain, and temperature) in the thalamus (Bowsher, 2005).

Approximately 15–20% of patients with a thalamic lesion develop thalamic central pain (Bogousslavsky et al., 1988; Paciaroni and Bogousslavsky, 1998). In these patients, the lesion is located mostly in the caudal part of the nucleus ventralis (Paciaroni and Bogousslavsky, 1998; Krause et al., 2012). In the studies available, there is no clear relationship between the degree of sensory deficiency and the appearance of pain (Paciaroni and Bogousslavsky, 1998).

The mechanism underlying the development of thalamic pain is not known. At least partial cortical deafferentation seems to be necessary for the appearance of pain (Willis and Westlund, 1997). However, a reduced inhibition exerted by the medial lemniscal pathways on thalamocortical neurons was also discussed (Boivie et al., 1989). Lesions in the thalamus that cause pain were particularly found in the VPL nucleus. Compared with controls, these lesions are more posterior, inferior, and lateral in the VPL, but thalamic lesions in the anterior pulvinar and ventral medial nucleus also can cause central pain (Krause et al., 2012). There are reports that central poststroke pain can occur in the absence of other somatosensory deficits (Kumar et al., 2016). This finding, as well as the fact that the development of thalamic central pain does not depend on the severity of the deafferentation, provides arguments against the deafferentation hypothesis as a mechanism of thalamic central pain. Pain due to thalamic stroke is not present directly after the occurrence of the lesion, but it develops in the weeks and months to come (Caplan et al., 1988; Paciaroni and Bogousslavsky, 1998).

In conclusion, a pure sensory deficit with a face–arm–leg distribution is strongly suggestive of a lateral thalamic lesion. However, in particular incomplete somatosensory deficits are difficult to distinguish from lenticulocapsular or cortical lesions (Kim, 1999).

Corticoparietal somatosensory syndromes

The processing of nonpainful somatosensory stimuli is mainly performed in the parietal cortex. Therefore, somatosensory deficits due to a central brain lesion are mostly caused by lesions of the parietal cortex. These somatosensory deficits are mostly combined with other functional deficits (particularly motor weakness). Particularly, enlarged ischemic lesions of the territory of the middle cerebral artery cause somatosensory deficits of all modalities. Here, we will focus on smaller lesions with more focused somatosensory deficits that reveal the importance of the responsible brain area in the parietal cortex.

When concluding about patients with sensory deficits, one has to keep in mind the task of the parietal cortex: to integrate the primary modalities, such as vibration, touch, position, motion, and pressure. Until this information arrives at the cerebrum, they are simply trains of action potentials. Each form of perception, discrimination, and recognition requires the integration of simple information into concrete concepts of the world. Moreover, all functions that rely on a combination of these modalities, such as two-point discrimination, graphesthesia, or stereognosis, are cortical functions (cortical modalities). Different cortical functions require different grades of higher-order cortical processing. Therefore, the pattern of impairment allows us to conclude about which level of cortical or subcortical processing is impaired. It is mandatory to ensure that the primary modalities are mainly preserved before concluding about the role of cortical lesions in the parietal cortex in bringing about an impairment of higher cortical modalities. For example, a patient presenting with sulcus ulnaris or cubital tunnel syndrome (Assmus et al., 2011) will have a greatly reduced two-point discrimination at the fifth finger, which is not caused by a cortical dysfunction but is simply due to a reduced forwarding of information regarding the primary modalities due to the impairment of the ulnar nerve.

There is no consistent terminology available on how to categorize somatosensory syndromes. There have been attempts to classify somatosensory impairments into clinical somatosensory syndromes, e.g., as pseudothalamic, cortical, or atypical sensory syndrome (Bassetti et al., 1993), described later. Although this classification might be helpful, we believe that it is more appropriate to analyze the individual somatosensory impairments according to the lesions of the underlying cerebral network necessary to perform a specific type of analysis. We will describe somatosensory impairments due to localized lesions of specific areas of the parietal somatosensory brain network and

then discuss the implications and clinical findings from lesions affecting multiple areas.

Clinical classification of somatosensory syndromes in the parietal cortex

Studies analyzing case series of somatosensory impairments after cortical lesions in the parietal lobe have found three main sensory syndromes: pseudothalamic, cortical, and atypical (Bassetti et al., 1993). The pseudothalamic syndrome, first defined by Roussy and Foix (Foix and Levy, 1927), consists of an impairment in all primary somatosensory modalities with a face–arm–leg distribution. As the name indicates, the pattern of sensory loss is similar to that caused by a thalamic lesion. A pure sensory impairment without other symptoms is rare, with the sensory deficit being often combined with motor symptoms and/or conduction aphasia (Bogousslavsky et al., 1989). This syndrome is attributed to a lesion of SI and SII (Bassetti et al., 1993; Horiuchi et al., 1996; Shintani et al., 2000; Wolk et al., 2002), but in all cases there is a considerable involvement of white-matter tracts.

The cortical somatosensory syndrome is characterized by a loss of cortical somatosensory modalities (e.g., discriminative touch, graphesthesia, stereognosis) involving specific parts of the body due to a superior posterior parietal stroke (Bassetti et al., 1993). Primary somatosensory modalities are mainly spared. The atypical somatosensory syndrome is characterized by a loss of some or all somatosensory modalities in a partial distribution. The lesion causing the atypical somatosensory syndrome is more inconsistently localized in the parietal lobe and often involves gray and white matter (Bassetti et al., 1993).

Subcortical lesion of thalamocortical projections

All primary somatosensory modalities are located near each other, so that a lesion of the somatosensory fibers between the thalamus and parietal cortex mostly involves all primary modalities of one body side and includes the face. The impairments are pronounced at the face and arm. Mostly, these sensory symptoms are associated with at least minor motor impairments, but pure sensory impairments are also described (Arboix et al., 1990, 2005). In the absence of motor symptoms, it is not possible to differentiate these lesions from a thalamic lesion. The closer a lenticulocapsular lesion is located to the thalamus, the more likely a pure sensory stroke becomes. This type of impairment is also known as pseudothalamic syndrome (Bassetti et al., 1993). Lenticulocapsular strokes are often evaluated as pure motor strokes by clinical screening, but a more precisely performed examination may find point localization and stereognosis to be particularly affected (Kim and Choi-Kwon, 1996).

Lesions of the primary somatosensory cortex

The part of the body that is affected by sensory loss depends on the location of the lesion within the postcentral gyrus (top to bottom: leg–arm–face). The somatosensory representations of the face and arm are supplied by the middle cerebral artery, which is most often affected by stroke, and both areas are often impaired together. The representation of the leg in SI is supplied by the anterior cerebral artery, and pure somatosensory deficits are also reported here (Nishida et al., 2010).

SI receives most of the somatosensory inputs from the thalamus. These inputs are preprocessed and forwarded mainly to the PPC and to SII. Lesions in SI will therefore affect the preprocessing and forwarding of primary somatosensory modalities, but they will also affect cortical somatosensory modalities. Accordingly, lesions of SI are reported to cause somatosensory deficits of primary as well as of cortical modalities (Bassetti et al., 1993; Kim and Choi-Kwon, 1996; Shintani et al., 2000). However, there is a report that describes a patient with a cortical lesion mainly involving SI that affects all primary somatosensory modalities, while cortical functions remained intact (Wolk et al., 2002). Animal studies have found an impairment in categorizing tactile speed due to lesions of SI (Zainos et al., 1997). However, the ablation of SI does not affect the ability to detect tactile stimuli (Zainos et al., 1997).

Lesions of the secondary somatosensory cortex and insula

Lesions are often due to strokes of the middle cerebral artery, which are mostly not restricted to functionally defined brain areas. Due to the close spatial relationship, most lesions involve parts of both areas (SII and the posterior insula). Moreover, these regions are also closely related spatially to the thalamocortical projections, which are also often involved.

Studies investigating lesions of these areas have mainly found a pseudothalamic pattern of somatosensory impairments (Bassetti et al., 1993). This result is possibly due to the involvement of the thalamocortical projections. A further study reported that an isolated lesion of the SII leads to an impairment of primary somatosensory modalities,

whereas cortical functions are not involved (Horiuchi et al., 1996). However, most studies also report somatosensory impairments of cortical functions due to lesions in these areas (Bassetti et al., 1993; Kim and Choi-Kwon, 1996; Arboix et al., 2005). The impairments normally affect all cortical somatosensory functions, while isolated loss of one cortical function is uncommon (Kim and Choi-Kwon, 1996). After a lesion that affects these areas, somatosensory deficits are most frequently combined with motor symptoms. Indeed, in clinical examination, these lesions are often considered pure motor strokes, and only more detailed somatosensory testing reveals somatosensory impairments (Kim and Choi-Kwon, 1996).

Bilateral sensory impairment due to unilateral lesions

There are multiple studies demonstrating a unilateral somatosensory impairment after brain lesions (Semmes and Mishkin, 1965; Carmon and Benton, 1969; Corkin et al., 1970, 1973; Kim and Choi-Kwon, 1996). In a very early study, somatosensory impairments of discriminative touch and pressure were demonstrated ipsilateral to an experimentally induced sensorimotor lesion in monkeys, while position sense remained intact (Semmes and Mishkin, 1965). Ipsilateral somatosensory deficits could also be observed in human subjects after brain lesions (Carmon and Benton, 1969; Corkin et al., 1970, 1973; Kim and Choi-Kwon, 1996). These ipsilateral impairments were found particularly after right-sided brain lesions in one study (Carmon and Benton, 1969): the ipsilateral impairment involved the tactile perception of the orientation of a pattern, but not of the number of stimuli constituting it, suggesting a spatial deficit. This might reflect spatial attentional factors, in line with the evidence for a role of the right hemisphere for somatosensory attention also for the ipsilateral side of the body (Bottini et al., 2005). However, other studies found an equal frequency of ipsilateral somatosensory deficits in patients with left- or right-sided lesions (Corkin et al., 1973; Kim and Choi-Kwon, 1996).

In all studies the ability for discriminative touch was most affected (Carmon and Benton, 1969; Corkin et al., 1970, 1973; Kim and Choi-Kwon, 1996). One study, however, also found an impaired position sense in some subjects (Corkin et al., 1973). To explain the bilateral sensory impairments after a unilateral brain lesion, it was suggested that discriminative sensations are not entirely mediated by the medial lemniscal tract, but they might also be mediated by other bilaterally represented tracts, such as the anterolateral system (Semmes and Mishkin, 1965).

Lesions involving multiple cortical areas

Proximal occlusion of the middle cerebral artery often causes lesions involving the major part of the parietal cortex. Somatosensory deficits are mostly present in these patients, but they are difficult to investigate due to the typical combination of additional symptoms, such as contralateral spatial neglect (see Chapter 14), that may present with contralateral somatosensory hemi-inattention, mimicking somatosensory deficits (Vallar et al., 1991a, b). Other factors preventing an accurate investigation include contralateral motor palsy, anarthria, and often aphasia after damage to the left hemisphere.

It is well known that the more spatially extended a lesion is, the more functional brain areas are affected, and the more severe the clinical symptoms are (Bassetti et al., 1993; Kim and Choi-Kwon, 1996; Arboix et al., 2005). However, it is difficult to investigate specific patterns in humans due to the great heterogeneity of patterns resulting from such lesions. By reviewing the excellent study of Bassetti and colleagues (1993), we identified 3 patients in their study who might have suffered from a combined lesion of SI and SII without motor symptoms. All of these patients showed a pseudothalamic syndrome, indicating a loss of the ability to process primary somatosensory modalities. The available data suggest that a lesion of SI or SII can be partly compensated for (Zainos et al., 1997), whereas a lesion that comprises both areas severely affects the ability to process primary as well as cortical somatosensory modalities.

Interestingly, position sense is the primary modality that was found to be least affected by subcortical or cortical strokes (Kim and Choi-Kwon, 1996). This suggests that the positional sense is highly redundant in the parietal cortex, and least sensitive to a partial loss of sensory input.

Lesions of the posterior parietal cortex

It is well known that lesions of the PPC cause deficits in attention to and awareness of stimuli of the contralateral side of the body and limb apraxia after left-hemispheric damage (see Chapter 17). Particularly, hemispatial neglect is more frequently observed after parietal lesions of the right hemisphere than those of the left hemisphere (Stone et al., 1993) (see Chapter 14). In the context of the clinical examination of somatosensory deficits, higher-order attentional pathologic factors are suggested by the presence of extinction of the contralateral touch under conditions of double

simultaneous stimulation (see Chapter 8; Vallar et al., 1994).

In clinical practice, the examiner should consider that most patients suffering from an isolated cortical stroke of the PPC will report normal somatosensory function, being unaware (anosognosic) of the sensory deficits (Vallar et al., 2003). Interestingly, if they are confronted with their inabilities, they will normally ascribe them to other causes or may produce elaborate confabulations. The unawareness of somatosensory deficits indicates to the clinician the involvement of higher-order cortical parietal areas, such as the PPC, and the possibility that spatial neglect may contribute to the clinical deficit of somatic sensation.

Somatosensory–motor interaction

In clinical practice, most patients with brain lesions suffer from a combination of somatosensory and motor impairments. In particular, sensory deficits are often reported even if seemingly only motor structures are affected. However, it is difficult to conclude whether some sensory deficits are caused by the motor deficit itself or by a loss of some somatosensory tracts. Animal experiments have demonstrated that somatosensory information and the anatomic connection between the motor and the somatosensory cortex are important for the acquisition of new motor skills (Bornschlegl and Asanuma, 1987; Nudo et al., 2000). Other data suggest that motor impairments after pure motor stroke are at least partly due to a loss of sensory information caused by sensorimotor disconnection (Nudo et al., 2000). Moreover, somatosensory input increases motor cortex excitability (Asanuma and Arissian, 1984; Hamdy et al., 1998). Also, in stroke patients, the ability for motor learning is related to the degree of proprioceptive deficits: the greater the deficit, the lesser the learning (Vidoni and Boyd, 2009). Even an unspecific sensory stimulation of a peripheral nerve facilitates training effects on motor function after subacute stroke (Conforto et al., 2010). Moreover, it is well known that a peripheral facial nerve palsy (pure deafferentation of the face while the somatosensory afference from the face is intact) causes also somatosensory impairments (Vanopdenbosch et al., 2005).

Although the exact mechanisms of sensorimotor interactions remain largely unknown, there is increasing evidence that both functional systems share and communicate some information and are tightly linked on an anatomic basis, which causes functional impairments in either system due to a lesion in the other that greatly exceeds those impairments that could be expected due to a simple loss of information from one system.

Inferring the anatomic location of lesions from the pattern of somatosensory deficits

Somatosensory deficits can be caused by lesions in all parts of the nervous system (peripheral nerves, nerve roots, spinal cord, medulla, pons, thalamus, thalamocortical projections, and cortex). The anatomic location of the lesion can be inferred from the pattern and distribution of somatosensory deficits. Herein, it cannot be overemphasized that a careful clinical examination of these deficits is the key to concluding about the location and a differential diagnosis (see Fig. 9.1 for a visual impression of common patterns of somatosensory deficits).

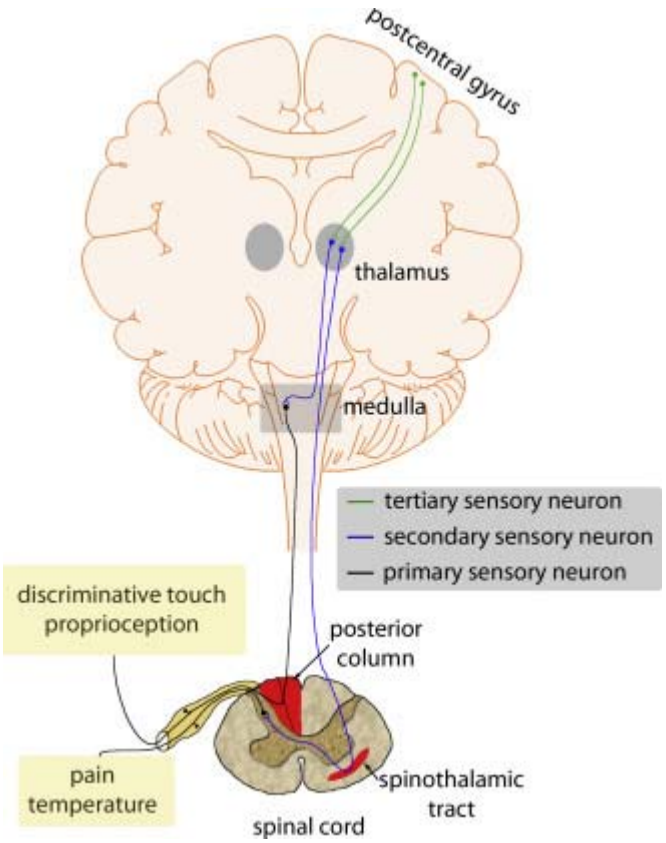

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Fig. 9.1. Schematic illustration of somatosensory projections.

Sensory impairments caused by peripheral nerve damage are the easiest to recognize because the sensory loss equates to the sensory innervation of the corresponding nerve. A sensory loss due to a radiculopathy is also clinically easy to identify. The sensory impairments are restricted to a body area consistent with the distribution of the affected nerve root. Moreover, sensory impairments are mostly combined with pain. Somatosensory deficits due to lesions in the spinal cord are characterized by a clear level between impaired and normal somatosensory function. Moreover, the spinothalamic tract and the medial lemniscus are located in spatially different sites in the spinal cord. Therefore, in most cases, only one of these tracts is affected. Moreover, the spinothalamic tract decussates at the level of entry to the spinal cord, whereas the posterior column decussates in the brainstem and forms the medial lemniscus. Lesions involving both tracts below the medulla produce dissociated sensory loss (ipsilateral touch, pressure, vibration, proprioception; contralateral temperature, and pain). Lesions above the decussation in the medulla cause somatosensory deficits on the contralateral side, but they might also elicit somatosensory deficits in the ipsilateral face (Fig. 9.2).

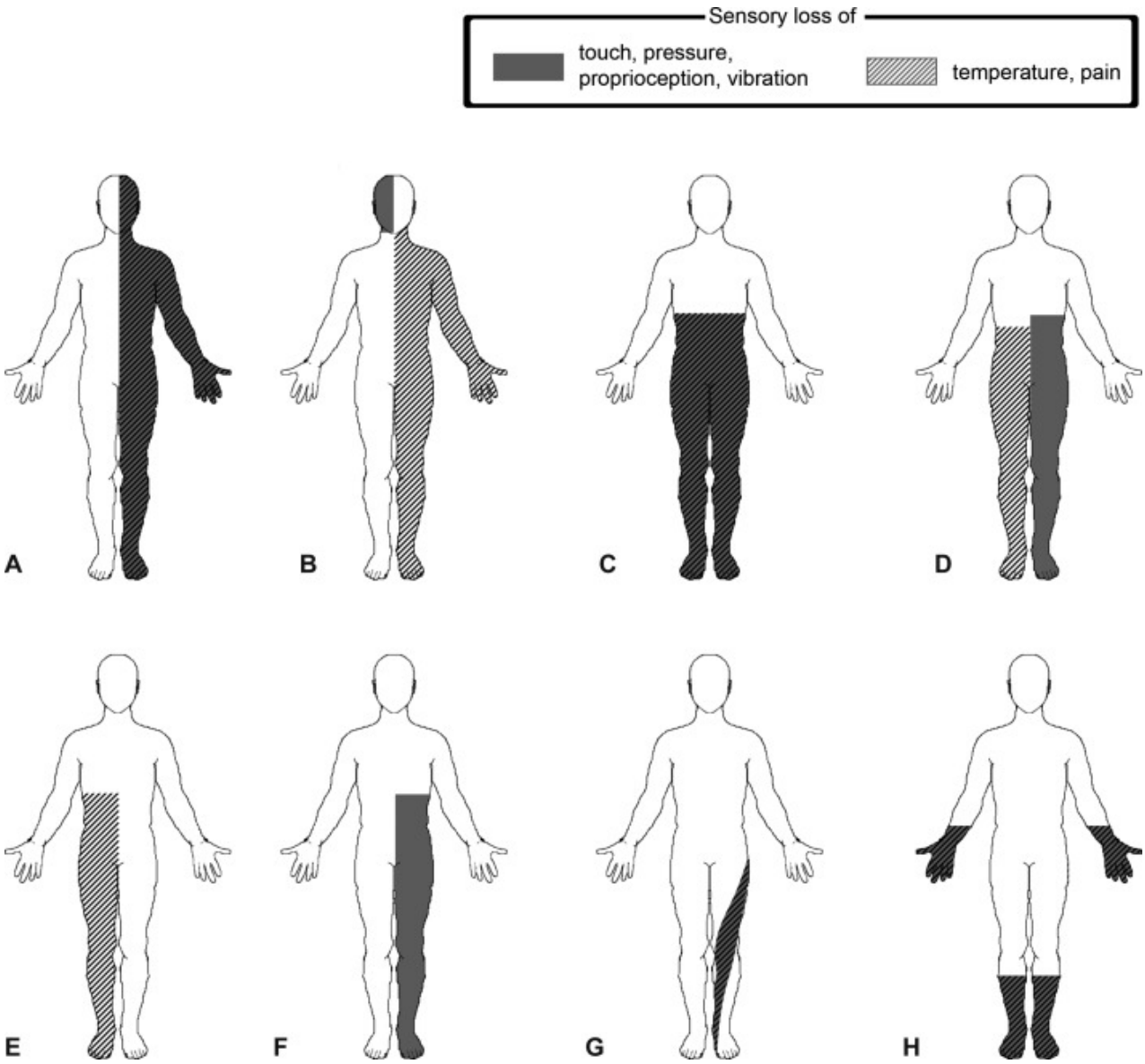

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Fig. 9.2. Common patterns of somatosensory deficits. (**A**) Somatosensory deficit of all primary modalities of one side of the body caused by a contralateral thalamic or major parietal lesion; (**B**) lesion of the dorsolateral medulla (Wallenberg syndrome); (**C**) lesion of the whole spinal cord; (**D**) lesion of one-half of the spinal cord (Brown-Séquard syndrome); (**E**) lesion of the spinothalamic tract; (**F**) lesion of the posterior column; (**G**) somatosensory deficit of all primary modalities due to lumbar radiculopathy (L4 root); (**H**) polyneuropathy.

Small lesions within the thalamus often cause a sensory loss over an entire half of the body (Fisher, 1965, 1982). Due to the tightly packed fibers in the thalamus, the impairments mostly affect all primary somatosensory modalities to an equal degree. Cortical lesions causing more diverse somatosensory deficits range from pure cortical somatosensory deficits to deficits of single modalities and pseudothalamic syndromes. The symptoms are dependent on the location of the lesion, but they also depend on the spatial extent of the lesion. Small lesions of the parietal cortex usually cause sensory impairments that are limited to one or two parts of the body (Bogousslavsky et al., 1989), and can even be restricted to single fingers (Fisher, 1982). Larger lesions are mostly combined with other symptoms and motor symptoms, whereas pure somatosensory deficits are rare (Bogousslavsky et al., 1989). Often, somatosensory deficits are more pronounced in the upper limp and spare the trunk in ~ 50% of cases (Fisher, 1982; Bogousslavsky et al., 1989; Bassetti et al., 1993). However, it is sometimes difficult to distinguish a cortical lesion with a pseudothalamic somatosensory pattern of impairment from a thalamic stroke.

By reviewing the literature, there is no study available that identifies a clinical parameter that distinguishes these two lesions. However, motor impairments are present in more than 90% of these cortical lesions (Bogousslavsky et al., 1989). Moreover, from our own clinical experience, it can be stated that a sensory loss due to thalamic lesions strongly affecting proprioception is very uncommon. An inability to sense motion and position of joints is always accompanied by a sensory ataxia. The patient is not able to move the corresponding limbs accurately during goal-directed movements. Such a loss of proprioception combined with a sensory loss in other modalities without an impairment in strength is very suggestive of a thalamic lesion.

Prognosis of somatosensory deficits

By far the most important cause of cerebral lesions is stroke (Mozaffarian et al., 2016). Multiple studies have investigated the percentage of patients affected by somatosensory impairments of different modalities. These studies found a loss of touch in 65–94% (Carey et al., 1993; Tyson et al., 2008), impaired proprioception in ~ 50% (Smith et al., 1983; Dukelow et al., 2010), impaired vibration sense in 44%, and loss of pinprick sensation in 35–71% (Hunter and Crome, 2002; Acerra et al., 2005; Tyson et al., 2008). Impairments of upper-limb function most seriously affect patient quality of life; this is a symptom in approximately 70% of stroke survivors (Nakayama et al., 1994; Tennant et al., 1997).

Coordinated motor function relies heavily on proprioceptive information, which is affected in ~ 50% of stroke patients (Smith et al., 1983; Dukelow et al., 2010). Accordingly, proprioceptive function strongly correlates with motor recovery (Kusoffsky et al., 1982; de Weerdt et al., 1987), and the degree of proprioceptive impairment predicts the necessary duration of the hospital stay, the duration of rehabilitation, the ability for self-care, the likelihood of discharge, and long-term rehabilitation outcome (Smith et al., 1983; de Weerdt et al., 1987; Carey et al., 1996). Therefore, the extent of proprioception impairment can be regarded as the best somatosensory parameter for predicting the overall functional outcome of patients.

Tactile extinction was also found to be an important predictor for functional outcome (Rose et al., 1994), suggesting a role for higher-order attentional factors. In addition, impairments of sensation were associated with long-term participation of stroke survivors in social life (Desrosiers et al., 2006). A particularly good prognosis was observed in patients with an isolated impairment of only one functional system. Particularly, somatosensory deficits without other symptoms are reported after stroke in ~ 10% of cases (Arboix et al., 2005). These patients have a very good prognosis overall, with symptom-free patients discharged in ~ 40% (Arboix et al., 2005). It is obvious that patients with only slight symptoms have a better prognosis compared to patients with severe deficits. Accordingly, the location and volume of the brain lesion can be used to predict the outcome in some functional systems (Munsch et al., 2016). However, some patients recover far better than others. Therefore, the question arises as to whether there are parameters that allow one to predict the degree of individual recovery.

Multiple studies have investigated different methods for predicting the individual functional outcome of somatosensory deficits. One very popular parameter is the SEP (Keren et al., 1993; Pereon et al., 1995; Feys et al., 2000; Al-Rawi et al., 2009). It was found that the SEP has prognostic value for the overall functional outcome, including motor function (especially peak-to-peak amplitude) (Feys et al., 2000; Al-Rawi et al., 2009). Particularly, for the somatosensory domain, it was shown that an initially normal SEP predicted a good sensory recovery (Julkunen et al., 2005), whereas the prognostic value of an absent SEP in the early phase after stroke is disputed (Pereon et al., 1995; Julkunen et al., 2005). Improvements in two-point discrimination ability have been found to correlate with SEP improvements (Wikstrom et al., 2000).

It has to be concluded that, until now, it has been difficult to determine specific factors that provide reliable predictions of individual outcome and recovery from somatosensory deficits. In discussing the possible roles of different parameters, one has to keep in mind that the age of the patient is by far the best predictor of functional recovery and outcome, especially after cortical lesions (Ween et al., 1996; Knoflach et al., 2012). Therefore, the possible predictive factors have to be interpreted together with the age of the patient, along with the location and size of the lesion and the severity of symptoms.

Rehabilitation therapy of somatosensory deficits

It is known that somatosensory deficits after brain lesions recover at least partially after stroke, with most progress observed in the first 3 months; however, this shows strong interindividual divergence (Julkunen et al., 2005). Accordingly, a re-emergence of activity in the somatosensory cortex was found during poststroke recovery (Carey et

al., 2002). Rehabilitation therapy aims to reinforce desired adaptive mechanisms to restore function.

One difficulty in selecting the appropriate therapy is the divergence of the affected sensory modalities due to brain lesions and their different needs for rehabilitation strategies. For example, an impairment of a cortical function, such as graphesthesia, requires a completely different therapeutic approach as impaired proprioception. Impairments in more than one somatosensory modality can greatly increase the functional impairment (Tyson et al., 2008). Therefore, rehabilitation of somatosensory deficits is an individually tailored therapy that requires a precise analysis of the impairments in all somatosensory modalities. However, one should always keep in mind that the overall goal is not to improve somatosensory function but to improve functional recovery and quality of life.

Certain types of somatosensory impairments might heavily affect function and quality of life, whereas others did not. For example, a slight numbness for touch in one body region should not seriously affect quality of life, whereas impaired proprioceptive feedback from one limb might seriously degrade motor function and quality of life. Therefore, the rehabilitation approach requires extensive knowledge about the importance of different somatosensory modalities and has to be well balanced with the goal of improving function.

The available studies investigating the effects of therapeutic interventions on somatosensory function can be categorized as active and passive somatosensory training. Passive somatosensory training involves the application of an external stimulus that can be electric or tactile. Active somatosensory training involves the active participation of the patient in performing specific exercises that aim to train somatosensory function (e.g., mirror training or practice with localizing position of body parts).

There are studies reported in the literature that have investigated these interventions, in addition to conventional or stand-alone therapies. Overall, multiple studies are available investigating the efficiency of somatosensory training. However, most of these studies that have investigated pure somatosensory interventions and improvements lack high methodologic quality or are insufficiently controlled (Doyle et al., 2010).

Passive somatosensory training

Passive somatosensory stimulation approaches have been frequently used in order to help patients regain lost function. These studies have used transcutaneous electric nerve stimulation (Cuypers et al., 2010), cutaneous electric stimulation (Peurala et al., 2002; Yozbatiran et al., 2006), neuromuscular stimulation (Mann et al., 2005), intermittent pneumatic stimulation (Cambier et al., 2003), thermal stimulation (Chen et al., 2005), and repetitive peripheral magnetic stimulation (Heldmann et al., 2000). Results are heterogeneous. For example, one study found no improvement of kinesthesia and position sense by an electric stimulation combined with a conventional rehabilitation compared to conventional rehabilitation alone (Yozbatiran et al., 2006). Burridge and colleagues (1990) found no effect on somatosensory function by comparing electric stimulation with a placebo. Another study that compared an inflatable pressure splinting intervention with a nonsplinting therapy reported no effect on light touch and position sense but a positive effect on pain scores (Poole et al., 1990). Chen and colleagues (2005) investigated the effects of repetitive thermal stimulation on the hand in addition to standard rehabilitation therapy. They found an increase in sensory function when tested with Semmes–Weinstein monofilaments. There are also other studies that mostly demonstrate an improvement in at least one somatosensory parameter. The analysis of their effects is difficult to interpret due to different measures of somatosensory outcome, study design, duration of training, and partly a mixing with active movement elements.

In conclusion, there is little evidence that passive somatosensory stimulation significantly improves somatosensory function (Doyle et al., 2010).

Active somatosensory training

Task-oriented training with active assistance of sensory feedback improves manipulation capability, even without recovery of the sensory pathway (Kita et al., 2013). Most of these studies use a sensory retraining approach that aims to restore or maintain sensory brain areas through the use of alternate senses (particularly vision) or cognitive learning techniques. For sensory retaining approaches, some programs use increasingly complex stimuli for tactile recognition (Posteraro et al., 2001), hand mirror therapy (Acerra, 2007), and a training of sequences of repetitive sensor discrimination tasks (Byl et al., 2003). Similarly to passive somatosensory training, there is also only limited evidence that active somatosensory training improves somatosensory rehabilitation compared to classic therapy (Oud et al., 2007; Doyle et al., 2010). Particularly, there is a lack of well-controlled studies (Doyle et al., 2010).

Proprioceptive training

Due to the importance of proprioceptive information for the neural control of movements, there are multiple studies available that investigated the effects of proprioceptive training. We describe these studies in a separate section because, in most of these studies, motor function improvement, and not somatosensory function, was the primary outcome parameter. The available studies used active movement or balance training (Wong et al., 2012), passive movement training (Beets et al., 2012), somatosensory stimulation training (mostly vibration training, targeting patients with Parkinson disease (Chouza et al., 2011), somatosensory discrimination training (Lynch et al., 2007), or a combination of these (McKenzie et al., 2009)). Although interventions and outcome measures vary greatly, there is converging evidence that proprioceptive training improves somatosensory and sensorimotor function (Aman et al., 2014).

Implications for practice

Studies investigating primarily somatosensory training suffer from the heterogeneity of interventions, a small number of available studies, and low quality with a particularly high risk of bias in the available studies. These facts make it difficult to draw significant conclusions. Even the available meta-analyses and specific reviews arrive at different conclusions. Schabrun and Hillier (2009) conclude that there is some support for the effectiveness of passive somatosensory (electric) training, but insufficient support for active somatosensory training (Schabrun and Hillier, 2009). Doyle et al. (2010) found insufficient evidence to support or refute the effectiveness of both active and passive interventions (Doyle et al., 2010). A recent review concerning multisensory stimulation found in 20 out of 21 included studies beneficial effects on sensory deficits after stroke (Tinga et al., 2016). However, the quality of these studies was regarded as insufficient for the conclusion that multisensory stimulation can be successfully applied as effective intervention (Tinga et al., 2016).

Currently, in our opinion, there is no sufficient evidence that recommends pure sensory interventions. More high-quality studies are needed to determine the effectiveness of somatosensory therapeutic interventions. However, there are some active interventions that jointly train somatosensory and motor abilities; in particular, joint position, target reaching, and balancing training bring about improvements in somatosensory and sensorimotor functions (Aman et al., 2014). Overall, there is a clear trend in favor of interventions that use passive and active elements, combining somatosensory and motor training (Aman et al., 2014).

Future perspectives

This chapter has highlighted the need for a better understanding of the processing of somatosensory information in the brain. In particular, there are important gaps in our current knowledge regarding the connections and communication within and between brain areas of the somatosensory network. In addition, the interaction between functional systems needs to be further explored. Although clinical studies demonstrate that multidimensional rehabilitation strategies are superior compared to pure somatosensory training, the underlying mechanisms that drive cortical reorganization between functional systems are mostly unknown.

A large number of nonrandomized studies report different somatosensory interventions that show promising preliminary results for rehabilitation. The effectiveness of these interventions should be investigated by high-quality studies combining neurophysiologic and clinical somatosensory measures of different modalities. There is a clear trend in favor of therapeutic interventions that combine passive and active somatosensory and motor training elements, particularly in the rehabilitation of proprioception. However, the question of which interventions should be combined and in which patients remains unclear. Therefore, the major challenge in clinical neurology and rehabilitation lies in the optimization of rehabilitation strategies with reference to the impairments of the individual patient. However, more evidence from high-quality studies is desperately needed to implement evidence-based treatment options on the individual level.

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