Preface

In 2005 we published a complete revision of Duus’ textbook of topical diagnosis in neurology, the first new edition since the death of its original author, Professor Peter Duus, in 1994. Feedback from readers was extremely positive and the book was translated into numerous languages, proving that the concept of this book was a successful one: combining an integrated presentation of basic neuroanatomy with the subject of neurological syndromes, including modern imaging techniques. In this regard we thank our neuroradiology colleagues, and especially Dr. Kueker, for providing us with images of very high quality.

In this fifth edition of “Duus,” we have preserved the remarkably effective didactic concept of the book, which particularly meets the needs of medical students. Modern medical curricula require integrative knowledge, and medical students should be taught how to apply theoretical knowledge in a clinical setting and, on the other hand, to recognize clinical symptoms by delving into their basic knowledge of neuroanatomy and neurophysiology. Our book fulfills these requirements and illustrates the importance of basic neuroanatomical knowledge for subsequent practical work, as it includes actual case studies. We have color-coded the section headings to enable readers to distinguish at a glance between neuroanatomical (blue) and clinical (green) material, without disrupting the thematic continuity of the text.

Although the book will be useful to advanced students, also physicians or neurobiologists interested in enriching their knowledge of neuroanatomy with basic information in neurology, or for revision of the basics of neuroanatomy will benefit even more from it.

This book does not pretend to be a textbook of clinical neurology. That would go beyond the scope of the book and also contradict the basic concept described above. First and foremost we want to demonstrate how, on the basis of theoretical anatomical knowledge and a good neurological examination, it is possible to localize a lesion in the nervous system and come to a decision on further diagnostic steps. The cause of a lesion is initially irrelevant for the primary topical diagnosis, and elucidation of the etiology takes place in a second stage. Our book contains a cursory overview of the major neurological disorders, and it is not intended to replace the systematic and comprehensive coverage offered by standard neurological textbooks.

We hope that this new “Duus,” like the earlier editions, will merit the appreciation of its audience, and we look forward to receiving readers’ comments in any form.

Professor M. Baehr
Professor M. Frotscher
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Lesions of the anterior spinothalamic tract. As explained above, the central fibers of the first neurons of this tract ascend a variable distance in the ipsilateral posterior columns, giving off collaterals along the way to the second neurons, whose fibers then cross the midline and ascend further in the contralateral anterior spinothalamic tract. It follows that a lesion of this tract at a lumbar or thoracic level generally causes minimal or no impairment of touch, because many ascending impulses can circumvent the lesion by way of the ipsilateral portion of the pathway. A lesion of the anterior spinothalamic tract at a cervical level, however, will produce mild hypesthesia of the contralateral lower limb.

Lateral Spinothalamic Tract

The free nerve endings of the skin are the peripheral receptors for noxious and thermal stimuli. These endings constitute the end organs of thin group A fibers and of nearly unmyelinated group C fibers that are, in turn, the peripheral processes of pseudounipolar neurons in the spinal ganglia. The central processes pass in the lateral portion of the posterior roots into the spinal cord and then divide longitudinally into short collaterals that terminate within one or two segments in the substantia gelatinosa, making synaptic contact with funicular neurons (second neurons) whose processes form the lateral spinothalamic tract (Fig. 2.16d, p. 26). These processes cross the midline in the anterior spinal commissure before ascending in the contralateral lateral funiculus to the thalamus. Like the posterior columns, the lateral spinothalamic tract is somatotopically organized; here, however, the fibers from the lower limb lie laterally, while those from the trunk and upper limb lie more medially (Fig. 2.20).

The fibers mediating pain and temperature sensation lie so close to each other that they cannot be anatomically separated. Lesions of the lateral spinothalamic tract thus impair both sensory modalities, though not always to the same degree.

Central continuation of the lateral spinothalamic tract. The fibers of the lateral spinothalamic tract travel up through the brainstem together with those of the medial lemniscus in the spinal lemniscus, which terminates in the ventral posterolateral nucleus of the thalamus (VPL, pp. 172, 173; see Fig. 6.4, p. 174, and Fig. 2.19). The third neurons in the VPL project via the thalamocortical tract to the postcentral gyrus in the parietal lobe (Fig. 2.19). Pain and temperature are perceived in a rough manner in the thalamus, but finer distinctions are not made until the impulses reach the cerebral cortex.

Lesions of the lateral spinothalamic tract. The lateral spinothalamic tract is the main pathway for...
pain and temperature sensation. It can be neurosurgically transected to relieve pain (cordotomy); this operation is much less commonly performed today than in the past, because it has been supplanted by less invasive methods and also because the relief it provides is often only temporary. The latter phenomenon, long recognized in clinical experience, suggests that pain-related impulses might also ascend the spinal cord along other routes, e.g., in spinospinal neurons belonging to the fasciculus proprius.

If the lateral spinothalamic tract is transected in the ventral portion of the spinal cord, pain and temperature sensation are deficient on the opposite side one or two segments below the level of the lesion, while the sense of touch is preserved (dissociated sensory deficit).

Other Afferent Tracts of the Spinal Cord

In addition to the spinocerebellar and spinothalamic tracts discussed above, the spinal cord contains yet other fiber pathways ascending to various target structures in the brainstem and deep subcortical nuclei. These pathways, which originate in the dorsal horn of the spinal cord (second afferent neuron) and ascend in its anterolateral funiculus, include the spinoreticular, spinotectal, spinovestibular, and spinovestibular tracts. The spinovestibular tract is found in the cervical spinal cord, from C4 upward, in the area of the (descending) vestibulospinal tract and is probably a collateral pathway of the posterior spinocerebellar tract.

Figure 2.20 is a schematic drawing of the various sensory (ascending) tracts, as seen in a cross section of the spinal cord. The motor (descending) tracts are also indicated, so that the spatial relationships between the various tracts can be appreciated. Finally, in addition to the ascending and descending tracts, the spinal cord also contains a so-called intrinsic apparatus, consisting of neurons that project upward and downward over several spinal segments in the fasciculus proprius (Fig. 2.9, p. 20).
Central Processing of Somatosensory Information

Figure 2.17 traces all of the sensory pathways discussed above, in schematically simplified form and in spatial relation to one another, as they ascend from the posterior roots to their ultimate targets in the brain. The sensory third neurons in the thalamus send their axons through the posterior limb of the internal capsule (posterior to the pyramidal tract) to the primary somatosensory cortex, which is located in the postcentral gyrus (Brodmann cytoarchitectural areas 3a, 3b, 2, and 1). The third neurons that terminate here mediate superficial sensation, touch, pressure, pain, temperature, and (partly) proprioception (Fig. 2.19, p. 29).

Sensorimotor integration. In fact, not all of the sensory afferent fibers from the thalamus terminate in the somatosensory cortex; some terminate in the primary motor cortex of the precentral gyrus. Thus, the sensory and motor cortical fields overlap to some extent, so that the precentral and postcentral gyri are sometimes together designated the sensorimotor area. The integration of function occurring here enables incoming sensory information to be immediately converted to outgoing motor impulses in sensorimotor regulatory circuits, about which we will have more to say later. The descending pyramidal fibers emerging from these circuits generally terminate directly—without any intervening interneurons—on motor neurons in the anterior horn. Finally, even though their functions overlap, it should be remembered that the precentral gyrus remains almost entirely a motor area, and the postcentral gyrus remains almost entirely a (somato)sensory area.

Differentiation of somatosensory stimuli by their origin and quality. It has already been mentioned that somatosensory representation in the cerebral cortex is spatially segregated in somatotopic fashion: the inverted sensory homunculus has been encountered in Figure 2.19 and will be seen again in Figure 9.19, p. 240. But somatosensory representation in the cerebral cortex is also spatially segregated by modality: pain, temperature, and the other modalities are represented by distinct areas of the cortex.

Although the different sensory modalities are already spatially segregated in the thalamus, conscious differentiation among them requires the participation of the cerebral cortex. Higher functions, such as discrimination or the exact determination of the site of a stimulus, are cortex-dependent.

A unilateral lesion of the somatosensory cortex produces a subtotal impairment of the perception of noxious, thermal, and tactile stimuli on the opposite side of the body; contralateral discrimination and position sense, however, are totally lost, as they depend on an intact cortex.

Stereognosis. The recognition by touch of an object laid in the hand (stereognosis) is mediated not just by the primary sensory cortex, but also by association areas in the parietal lobe, in which the individual sensory features of the object, such as its size, shape, consistency, temperature, sharpness/dullness, softness/hardness, etc., can be integrated and compared with memories of earlier tactile experiences.

Astereognosis. Injury to an area in the inferior portion of the parietal lobe impairs the ability to recognize objects by touch with the contralateral hand. This is called astereognosis.

Somatosensory Deficits due to Lesions at Specific Sites along the Somatosensory Pathways

Figure 2.21 shows some typical sites of lesions along the somatosensory pathways; the corresponding sensory deficits are discussed below.

- A cortical or subcortical lesion in the sensorimotor area corresponding to the arm or leg (a and b, respectively, in Fig. 2.21) causes paresthesia (tingling, etc.) and numbness in the contralateral limb, which are more pronounced distally than proximally. An irritative lesion at this site can produce a sensory focal seizure when spontaneous (epileptic) discharge of the inflamed/damaged nerve cells occurs; because the motor cortex lies directly adjacent, there are often motor discharges as well (jacksonian
seizure, see textbooks of neurology for the classification of epileptic seizures).

- **A lesion of all sensory pathways below the thalamus** (c) eliminates all qualities of sensation on the opposite side of the body.

- If all somatosensory pathways are affected except the pathway for pain and temperature (d), there is hypesthesia on the opposite side of the body and face, but pain and temperature sensation are unimpaired.

- Conversely, a **lesion of the trigeminal lemniscus** and of the lateral spinothalamic tract (e) in the brainstem impairs pain and temperature sensation on the opposite side of the body and face, but does not impair other somatosensory modalities.

- If the **medial lemniscus and anterior spinothalamic tract** (f) are affected, all somatosensory modalities of the contralateral half of the body are impaired, except pain and temperature.

- **Lesions of the spinal nucleus and tract of the trigeminal nerve** and of the **lateral spinothalamic tract** (g) impair pain and temperature...
sensation on the ipsilateral half of the face and the contralateral half of the body.

- **Posterior column lesions (h)** cause loss of position and vibration sense, discrimination, etc., combined with ipsilateral ataxia (see Case Presentation 1).

- If the **posterior horn of the spinal cord** is affected by a lesion (i), ipsilateral pain and temperature sensation are lost, but other modalities remain intact (dissociated sensory deficit).

- A lesion affecting **multiple adjacent posterior roots (j)** causes radicular pain and paresthesiae, as well as impairment or loss of all sensory modalities in the affected area of the body, in addition to hypotonia or atonia, areflexia, and ataxia if the roots supply the upper or lower limb.

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### Case Presentation 1: Subacute Combined Degeneration

An 80-year-old woman was hospitalized because of marked shortness of breath with dyspnea. The patient reported that she had been suffering from an increasingly unsteady gait and burning sensations throughout her body for about a year and a half. The shortness of breath had developed in the previous month and had worsened dramatically in the past few weeks. The only previous disease reported by the patient was “stomach inflammation.”

On detailed examination by the admitting neurologist, the patient was in obviously poor condition, dehydrated and with marked dyspnea. Neurological examination revealed spastic tetraparesis, which was more marked in the legs, with increased intrinsic muscle reflexes despite the presence of obvious muscle atrophy, especially on the trunk. There was also evidence of severe spinal ataxia, severely disturbed position sense, and hypesthesia and hypalgesia that increased distally below about T8. Vibration sense in the legs was almost absent (pallanesthesia). The neurologist ordered pulmonary function tests because of the dyspnea, along with MRI of the cervical and thoracic spine because of the neurological abnormalities.

The pulmonary function tests revealed markedly diminished expiratory volume and reduced vital capacity. Blood gases confirmed global respiratory insufficiency with reduced O₂ and elevated CO₂ levels. Blood chemistry revealed a markedly reduced vitamin B₁₂ level, and vitamins B₆, C, D, and folic acid were also low. MRI of the cervical and thoracic spine showed marked signal enhancement in the posterior and anterolateral columns and also in the anterior horns (Fig. 2.22).

Discussion with her family physician revealed that the patient had known chronic atrophic gastritis with intrinsic factor deficiency but that she had obtained vitamin B₁₂ replacement therapy very irregularly in recent years. All of the findings together confirmed the diagnosis of advanced subacute combined degeneration, which involved not only the classical posterior and pyramidal tracts but also the anterior horns (quadriplegic syndrome).

The patient’s respiratory insufficiency was the result of paresis of the respiratory muscles (destruction of the innervating motor neurons). Because of the poor blood gases, the patient required controlled ventilation for several weeks. After correction of the dehydration, electrolyte disturbances, and hypovitaminosis, the patient recovered slowly and was transferred to a geriatric rehabilitation clinic 2 months following her initial hospitalization.

![Fig. 2.22 Advanced subacute degeneration (funicular myelosis) with symptoms of paraplegia. On MRI of the cervical spine (C6 level), signal enhancement is seen in the posterior and anterolateral columns. This appearance is typical of advanced funicular myelosis.](image)
thalami to the habenular nuclei, which emit efferent projections to the autonomic (salivatory) nuclei of the brainstem, thus playing an important role in nutritional intake.

The epiphysis (pineal gland) contains specialized cells, called pinealocytes. Calcium and magnesium salts are deposited in the epiphysis from approximately age 15 years onward, making this structure visible in plain radiographs of the skull (an important midline marker before the era of CT and MRI). Epiphyseal tumors in childhood sometimes cause precocious puberty; it is thus presumed that this organ inhibits sexual maturation in some way, and that the destruction of epiphyseal tissue can remove this inhibition. In lower vertebrates, the epiphysis is a light-sensitive organ that regulates circadian rhythms. In primates, light cannot penetrate the skull, but the epiphysis still indirectly receives visual input relating to the light–dark cycle. Afferent impulses travel from the retina to the suprachiasmatic nucleus of the hypothalamus, from which, in turn, further impulses are conducted to the intermediolateral nucleus and, via postganglionic fibers of the cervical sympathetic chain, to the epiphysis.

**Subthalamus**

**Location and components.** The subthalamus is found immediately caudal to the thalamus at an early stage of embryological development and then moves laterally as the brain develops. It comprises the subthalamic nucleus, part of the globus pallidus (cf. p. 217), and various fiber contingents that pass through it on their way to the thalamus, including the medial lemniscus, the spinothalamic tract, and the trigeminothalamic tract. All of these tracts terminate in the ventroposterior region of the thalamus (Fig. 6.4, p. 174). The substantia nigra and red nucleus border the subthalamus anteriorly and posteriorly. Fibers of the dentatothalamic tract travel in the prerubral field H1 of Forel to terminate in the ventro-oral posterior nucleus of the thalamus (a part of the ventral lateral nucleus, VL); fibers from the globus pallidus travel in the lenticular fasciculus (Forel’s fasciculus H2) to the ventro-oral anterior nucleus (another part of VL) and the ventral anterior nucleus (VA). These tracts are joined more rostrally by the ansa lenticularis. The subthalamus also contains the zona incerta, a rostral continuation of the midbrain reticular formation. The major connections of the putamen, pallidum, subthalamus, and thalamus are depicted in Fig. 6.7.

**Function.** The subthalamic nucleus (corpus Luysii) is, functionally speaking, a component of the basal ganglia and has reciprocal connections with the globus pallidus (p. 217). Lesions of the subthalamic nucleus produce contralateral hemiballism (p. 223 f.).

**Hypothalamus**

**Location and Components**

The hypothalamus (Fig. 6.8) is composed of gray matter in the walls of the third ventricle from the hypothalamic sulcus downward and in the floor of the third ventricle, as well as the infundibulum and the mamillary bodies. The posterior pituitary lobe, or neurohypophysis, is also considered part of the hypothalamus; this structure is, in a sense, the enlarged caudal end of the infundibulum. The anterior pituitary lobe, on the other hand, is not derived from the neuroectoderm at all, but rather
from Rathke’s pouch, an outcropping of the rostral end of the primitive alimentary tract. The two pituitary lobes, though adjacent to each other, are not functionally connected. Remnants of Rathke’s pouch in the sellar region can grow into tumors, e.g., craniopharyngioma.

The columns of the fornix, as they descend through the hypothalamus to the mamillary bodies on either side, divide the hypothalamus of each side into a **medial** and a **lateral segment** (Fig. 6.8). The lateral segment contains various groups of fibers, including the **medial forebrain bundle**, which runs from basal olfactory areas to the midbrain. It also contains the lateral tuberal nuclei (see p. 180).

The medial segment, in contrast, contains a number of more or less clearly distinguishable nuclei (Fig. 6.8a–c), which are divided into an **anterior (rostral)**, a **middle (tuberal)**, and a **posterior (mamillary) nuclear group**.

### Hypothalamic Nuclei

**Anterior nuclear group.** The important members of this group are the **preoptic, supraoptic, and paraventricular nuclei** (Fig. 6.8). The latter two nuclei project, by way of the supraoptico-hypophyseal tract, to the neurohypophysis (see Figs. 6.10 and 6.11).
Middle nuclear group. The important members of this group are the infundibular nucleus, the tuberal nuclei, the dorsomedial nucleus, the ventromedial nucleus, and the lateral nucleus (or tuberomamillary nucleus) (Fig. 6.8).

Posterior nuclear group. This group includes the mamillary nuclei (the supramamillary nucleus, the mamillary nucleus, the intercalate nucleus, and others) and the posterior nucleus (Fig. 6.8). This area has been termed a dynamogenic zone (Hess), from which the autonomic nervous system can be immediately called into action, if necessary.

Afferent and Efferent Projections of the Hypothalamus

The neural connections of the hypothalamus (Figs. 6.9 and 6.10) are multifarious and complex. In order to carry out its function as the coordinating center of all autonomic processes in the body (p. 190), the hypothalamus must communicate via afferent and efferent pathways with very many different areas of the nervous system. Information from the outside world reaches it through visual, olfactory, and probably also auditory pathways. The presence of cortical afferents implies that the hypothalamus can also be influenced by higher centers. The major connections of the hypothalamus are to the cingulate gyrus and frontal lobe, the hippocampal formation, the thalamus, the basal ganglia, the brainstem, and the spinal cord.

Some of the more important afferent connections (Fig. 6.9) will be described in the following section.

Afferent Pathways

The medial forebrain bundle originates in the basal olfactory areas and the septal nuclei and runs as a chain of neurons through the hypothalamus (lateral area) until it arrives in the midbrain reticular formation. Along the way, it gives off collateral fibers to the preoptic nucleus, the dorsomedial nucleus, and the ventromedial nucleus. The medial forebrain bundle constitutes a reciprocal connection between olfactory and preoptic nuclear areas and the midbrain. It has olfacto-visceral and olfacto-somatic functions.

The striae terminales originate in the amygdala in the temporal lobe, then form an arch over the thalamus, terminating in the preoptic area and to
the anterior hypothalamic nuclei. These fiber bundles are thought to transmit olfactory information, as well as impulses relating to mood and drive.

The fornix transmits corticomamillary fibers originating in the hippocampus and subiculum and traveling to the mamillary body, with collaterals to the preoptic nucleus, the anterior nucleus of the thalamus, and the habenular nucleus. The fornix is an important pathway in the limbic system (p. 203). As it passes over the dorsal surface of the pulvinar, some of its fibers cross the midline to join the contralateral fornix (commissure of the fornices, psalterium).

At the level of the psalterium, the two fornices lie under the splenium of the corpus callosum, where they are usually not directly visible in an uncut brain specimen. Lesions in the area of the psalterium often affect both fornices, because these two thin structures are close together at this point. The serious functional deficits produced by bilateral limbic lesions are discussed below on p. 208 ff.

Ascending visceral impulses from the peripheral autonomic nervous system, and from the nucleus of the tractus solitarius (taste), reach the hypothalamus along various pathways: through relay nuclei in the brainstem reticular formation, from tegmental and interpeduncular nuclei, through reciprocal connections in the medial forebrain bundle, through the dorsal longitudinal fasciculus, and through the peduncle of the mamillary body (Figs. 6.9 and 6.10). Somatosensory information from the erogenous zones (genitalia and nipples) also reaches the hypothalamus by these pathways and induces autonomic reactions.

Finally, further afferent input comes to the hypothalamus from the medial nucleus of the thalamus, the orbitofrontal neocortex, and the globus pallidus.

**Efferent Pathways**

Efferent fibers to the brainstem. The most important efferent projections from the hypothalamus to the brainstem are the dorsal longitudinal fasciculus (of Schütz), which contains fibers traveling in both directions, and the medial forebrain bundle (Figs. 6.9 and 6.10). Hypothalamic impulses traveling in these pathways pass through multiple synaptic relays, mainly in the reticular formation, until they terminate in parasympathetic nuclei of the brain-
stem, including the oculomotor nucleus (miosis), the superior and inferior salivatory nuclei (lacrimation, salivation), and the dorsal nucleus of the vagus nerve. Other impulses travel to autonomic centers in the brainstem that coordinate circulatory, respiratory, and alimentary function (etc.), as well as to motor cranial nerve nuclei that play a role in eating and drinking: the motor nucleus of the trigeminal nerve (mastication), the nucleus of the facial nerve (facial expression), the nucleus ambiguus (swallowing), and the nucleus of the hypoglossal nerve (licking). Yet other impulses derived from the hypothalamus, relayed to the spinal cord through reticulospinal fibers, affect the activity of spinal neurons that participate in temperature regulation (shivering).

The mamillotegmental fasciculus (Fig. 6.10) runs from the mamillary body to the midbrain tegmentum, and then onward to the reticular formation.

The mamillothalamic tract (of Vicq d’Azyr) reciprocally connects the hypothalamus with the anterior nucleus of the thalamus, which, in turn, is reciprocally connected with the cingulate gyrus (Fig. 6.6). The anterior thalamic nucleus and the cingulate gyrus are important components of the limbic system. The main function of the limbic system is said to be the regulation of affective behavior so as to promote the survival of the individual and of the species (MacLean 1958; cf. p. 202).

The supraoptico-hypophyseal tract has already been mentioned as an efferent pathway to the neurohypophysis. Neurons in the supraoptic and paraventricular nuclei produce the hormones oxytocin and vasopressin (antidiuretic hormone), which are transported along the axons of the supraoptico-hypophyseal tract to the neurohypophysis, and are then released there, from the axon terminals, into the bloodstream (Figs. 6.10 and 6.11). The neurons in these nuclei are thus comparable to the hormone-producing cells of other organs, and are referred to as neurosecretory cells. Oxytocin and vasopressin mainly exert their effects on cells outside the nervous system: oxytocin induces con-
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