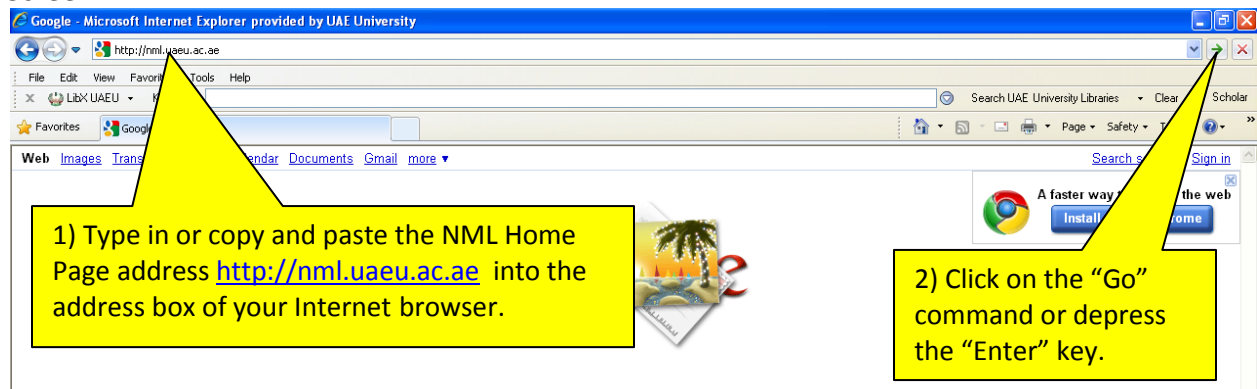


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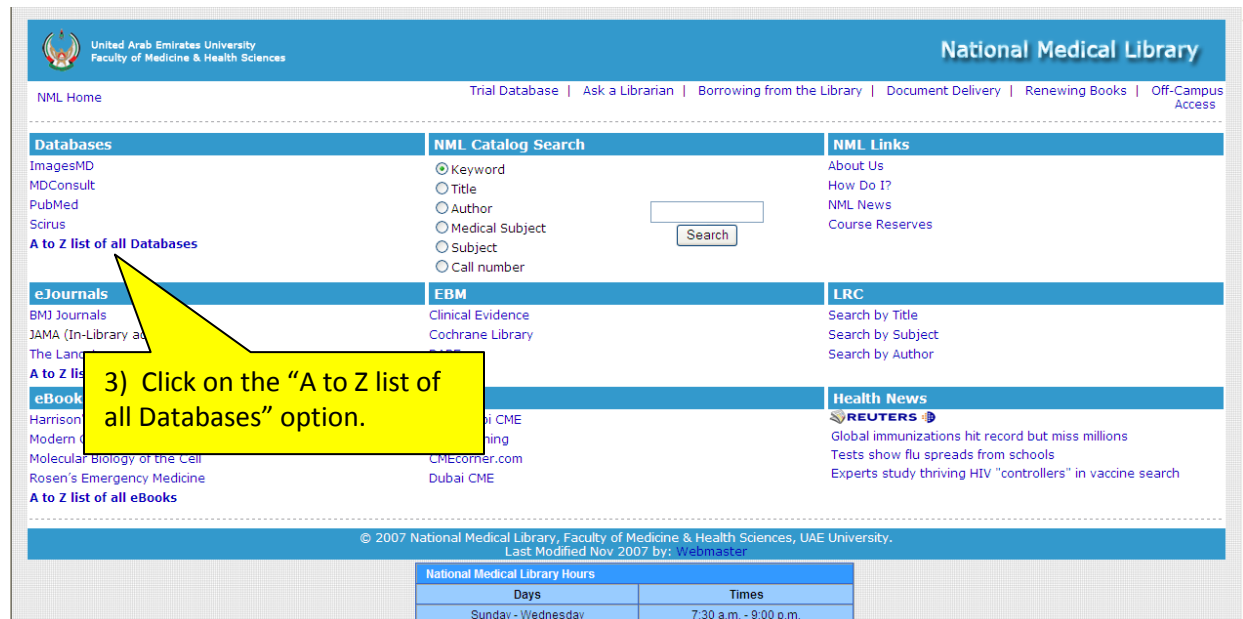
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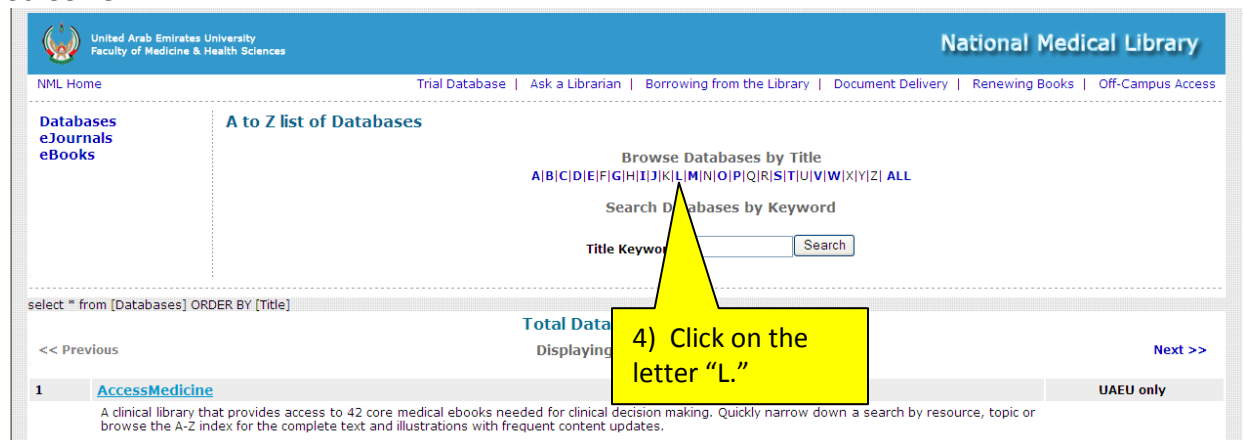
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


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



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Chapter 33

The Proprioceptive Sensations

The proprioceptive sensations arise from the deeper tissues of the body, principally from the muscles, ligaments, bones, tendons, and joints. Proprioception has both a conscious and an unconscious component. The conscious component travels with the fibers subserving fine, discriminative touch; the unconscious component forms the spinocerebellar pathways. The conscious proprioceptive sensations that can be tested clinically are motion, position, vibration, and pressure.


ANATOMY


The primary receptors for proprioception, or kinesthesia, are the muscle spindles. Other peripheral sense organs dealing with proprioception are located in the muscles, tendons, and joints, particularly Pacinian corpuscles. These respond to pressure, tension, stretching or contraction of muscles fibers, joint movement, changes in the position of the body or its parts, and related stimuli. Cutaneous afferents play a contributory role. Proprioceptors are essential for the normal coordination and grading of muscle contraction and the maintenance of equilibrium. Conscious proprioceptive impulses travel along large, myelinated fibers from the periphery to the first order neuron in the dorsal root ganglion (DRG) and then via the medial division of the posterior root (Figure 32.1). These fibers then enter, without a synapse, the ipsilateral fasciculus gracilis and cuneatus, and ascend to the nuclei gracilis and cuneatus in the lower medulla, where a synapse occurs. Axons of the second order neuron decussate as internal arcuate fibers, and then ascend in the medial lemniscus (ML) to the thalamus (Figure 33.1). The somatotopic organization in the posterior columns and lemniscal pathways is the same as for light touch (Figure 32.3). Other DRG fibers subserving kinesthesia synapse in the dorsal horn, and then ascend in the dorsolateral funiculus to the lateral cervical nucleus, where they join the ML. The thalamoparietal radiations then go through the posterior limb of the internal capsule, and the fibers are distributed to the cortex.

Proprioceptive impulses from the head and neck enter the central nervous system with the cranial nerves. Many terminate on the mesencephalic root of the trigeminal nerve; others accompany motor nerves from the muscles they supply. Impulses probably reach the thalamus through the ML.

SENSES OF MOTION AND POSITION

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Chapter 33

The Proprioceptive Sensations

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SENSES OF MOTION AND POSITION

The sense of motion, also known as the kinetic or kinesthetic sense, or the sensation of active or passive movement, consists of an awareness of motion of various parts of the body. The sense of position, or posture, is awareness of the position of the body or its parts in space. These sensations depend on impulses arising as a result of motion of the joint and of lengthening and shortening of the muscles. Motion and position sense are usually tested together, by passively moving a part and noting the patient's appreciation of the movement and recognition of the direction, force, and range of movement; the minimum angle of movement the patient can detect; and the ability to judge the position of the part in space. In the lower extremity, testing usually begins at the metatarsophalangeal joint of the great toe, in the upper extremity at one of the distal interphalangeal joints. If these distal joints are normal there is no need to test more proximally. Testing is done with the patient's eyes closed. It is extremely helpful to instruct the patient, eyes open, about the responses expected before beginning the testing. No matter the effort, nonsensical replies are frequent. The examiner should hold the patient's completely relaxed digit on the sides, away from the neighboring digits, parallel to the plane of movement, exerting as little pressure as possible to eliminate clues from variations in pressure.

If the digit is held dorsoventrally, the grip must be firm and unwavering so that the pressure differential to produce movement provides no directional clue. The patient must relax, and not attempt any active movement of the digit that may help to judge its position. The part is then passively moved up or down, and the patient is instructed to indicate the direction of movement from the last position (Figure 33.2). Even when instructed that the response is two alternative, forced choice, up or down, some patients cannot be dissuaded from reporting the absolute position (e.g., down), even if the movement was up.

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up and down in tiny increments, gradually increasing the excursion until the patient is aware of the motion. Quick movements are more easily detected than very slow ones. Healthy young individuals can detect great toe movements of about 1 mm; in the fingers virtually invisible movements at the distal interphalangeal joint are accurately detected. There is some rise in the threshold for movement and position sense with advancing age.

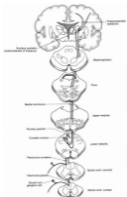


FIGURE 33.1 • The pathways for position sense and fine discriminative touch through the posterior columns and medial lemniscus.

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FIGURE 33.2 • Method of testing position sense; done similarly with toe.

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SENSE OF VIBRATION (PALLESTHESIA)

Vibratory sensation is the ability to perceive the presence of an oscillating tuning fork is placed over certain bony prominences. For clinical purposes, it can be considered a combination of other sensations. Bone may act largely as a resonator. The receptors for vibratory sensation are the Pacinian corpuscles, located deep in the skin, subcutaneous tissues, muscles, periosteum, and other tissues of the body; and Meissner's corpuscles, located in the dermis.

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When an oscillating tuning fork is placed over certain bony prominences, the fork invoke impulses that are coded so that the brain can interpret the signals: the vibration frequency. The impulses travel through the dorsal columns, but likely other pathways are involved in the ipsilateral dorsolateral funiculus, and ascend to the medulla where they join the other sensory pathways. This divergence of the position sense and vibration sense pathways may partially explain why position sense is more affected than vibration sense in tabes dorsalis. Thalamocortical fibers from the ventral posterior lateral (VPL) and the ventral posterior medial (VPM) nuclei project to the primary somatosensory areas in the postcentral gyrus, and terminate on vibration responsive neurons.

A tuning fork of 128 Hz with weighted ends is most frequently used. Sensation may be tested on the great toes, the metatarsal heads, the malleoli, the tibia, anterior

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1. Passage: ... due to increased intracranial pressure is referred to as **papilledema**; under all other circumstances, the noncommittal terms **disc edema** or **disc swelling** are preferred. Visual function provides a critical clue to the nature of disc abnormalities. Patients with acute **papilledema** and those with disc anomalies have normal visual acuity, visual fields, and color perception. Impairment of these functions is the rule in patients suffering from **optic** neuropathies of any etiology. The first step in evaluating a questionably abnormal disc is therefore a careful assessment of vision. **Papilledema**.

Authors: Campbell, William W.

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☐ Chapter: The Optic Nerve

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☐ Chapter: The Optic Nerve

2. Passage: ... alone are not enough to diagnose **papilledema**. There is no alteration of the physiologic cup with early **papilledema**. With further evolution, the patient with early **papilledema** exudates, and venous engorgement. Frank disc elevation (Figure 13.21). In chronic **papilledema**, hemorrhage bulging up from the plane of the retina. If unrelieved, impairment, which evolves into the stage of atrophic over days to weeks. With acutely ...

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Chapter 13 - The Optic Nerve and Optic Disc

Disorders of the Optic Disc > Papilledema

Pressure exerts pressure on the optic nerves, which impairs axoplasmic flow and produces axonal edema and an increased volume of axoplasm at the optic disc. This pressure impairs venous return from the retina, engorging first the capillaries on the disc surface, then the retinal veins, and ultimately causing splinter- and flame-shaped hemorrhages as well as cotton wool exudates in the retinal nerve fiber layer. Further axonal swelling eventually leads to elevation of the disc above the normal level. Transient visual obscurations, momentary graying out or blacking out of vision, often precipitated by postural changes, are classical symptoms of papilledema, especially in pseudotumor cerebri. Obscurations may be due to microvascular compromise at the nerve head.

The four stages of papilledema are early, fully developed, chronic, and atrophic. Fully developed papilledema is obvious, with elevation of the disc surface, humping of vessels crossing the disc margin, obliteration of disc margins, peripapillary hemorrhages, cotton wool exudates, engorged and tortuous retinal veins, and marked disc hyperemia. The recognition of early papilledema is much more problematic (Figure 13.20). Occasionally, the only way to resolve the question of early papilledema is by serial observation. The earliest change is loss of previously observed spontaneous venous pulsations (SVPs). Venous pulsations are best seen where the large veins dive into the disc centrally. The movement is a back-and-forth rhythmic oscillation of the tip of the blood column, which resembles a slowly darting snake's tongue. Side-to-side expansion of a vein is much more difficult to see. The presence of SVPs indicates an intracranial pressure less than approximately 200 mm H₂O. However, since they are absent in 10% to 20% of normals, only the disappearance of previously observed SVPs is clearly pathologic.

TABLE 13.1 Clinical Characteristics of Acute Lesions Involving Different Parts of the Afferent Visual Pathway

| | Visual Acuity | Color Vision | Visual field Defect | Pupillary Function | Disc Appearance | Comment |
|--------|---------------|--------------|-----------------------------|--------------------|-----------------|--|
| Macula | Decreased | Decreased | Ipsilateral central scotoma | Possible mild APD | Normal | May have metamorphopsia; macula may be abnormal on ophthalmoscopy; common etiologies: age-related macular degeneration, central serous retinopathy, macular hole, cystoid macular edema, trauma, toxic retinopathy |

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Pressure is referred to as papilledema; under all other circumstances, the noncommittal term "disc edema" is used. Visual function provides a critical clue to the nature of disc abnormalities. Patients with disc anomalies have normal visual acuity, visual fields, and color perception. Impairment of these functions is the rule in patients suffering from optic neuropathies of any etiology. The first step in evaluating a questionably abnormal disc is therefore a careful assessment of vision. Papilledema.

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☐ Chapter: The Optic Nerve

2. Passage: ... alone are not enough to diagnose papilledema. There is no alteration of the physiologic cup with early papilledema. With further evolution, the patient with early papilledema will develop diffuse disc edema, cup obscuration, hemorrhages, exudates, and venous engorgement. Frank disc elevation then ensues as the fundus ripens into fully developed papilledema (Figure 13.21). In chronic papilledema, hemorrhages and exudates resolve and leave a markedly swollen "champagne cork" disc bulging up from the plane of the retina. If unrelieved, impaired axoplasmic flow eventually leads to death of axons and visual impairment, which evolves into the stage of atrophic papilledema, or secondary optic atrophy. Papilledema ordinarily develops over days to weeks. With acutely

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