AN OVERVIEW OF NEUROANATOMY
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REGIONAL NEUROANATOMY

THE CENTRAL NERVOUS SYSTEM (CNS)

The central nervous system contains two major structures, namely the brain and spinal cord. The brain, in turn, is divided into three major parts based on its embryological origins, namely the prosencephalon, mesencephalon and rhombencephalon.

Prosencephalon (Forebrain)

The prosencephalon is divided into two parts, the telencephalon and diencephalon.

Telencephalon (endbrain)

The telencephalon is the largest subdivision of the brain. It contains four major systems: the neocortex, the basal ganglia, the limbic system and the olfactory system.

Neocortex

The neocortex is phylogenetically the most recent acquisition of the nervous system and is that part of the brain responsible for initiating voluntary motor activity and for integrating and interpreting sensory information. Higher functions such as language, memory, abstraction and reasoning are controlled by the neocortex. It is also the source of an individual's personality.

The neocortex is a mantle of gray matter that contains six layers of cells. It is divided by sulci and primary fissures into four major lobes: frontal, parietal, temporal and occipital. The central sulcus separates the frontal and parietal lobes; the Sylvian fissure separates the frontal and parietal lobes from the temporal lobe; and the parieto-occipital fissure on the medial surface of the brain separates the parietal and occipital lobes.

The frontal lobe contains the primary motor strip (precentral gyrus), the secondary motor strip (pre-precentral gyrus), and the frontal eye fields that control voluntary eye movements. The parietal lobe contains the primary sensory strip (postcentral gyrus). The temporal lobe contains primary auditory cortex (transverse gyrus of Heschl). The occipital lobe contains primary visual cortex, located on both banks of the calcarine fissure.

Language is localized to the dominant hemisphere: Broca's area, on the lateral surface of the dominant frontal lobe, controls the production of language; Wernicke's area, near the supramarginal and angular gyri on the lateral surface of the dominant temporal lobe, controls the understanding of language.

Association cortex, where primary motor and sensory modalities are integrated, is located in all four lobes of the brain. Motor association cortex is located in the frontal
lobe, whereas sensory association cortex is located in the parietal, temporal and occipital lobes.

**Basal ganglia**

The basal ganglia are subcortical telencephalic structures that are integrally involved with the motor system. They allow the cortex to select wanted patterns of movement, and to suppress unwanted patterns of movement.

The three major components of the basal ganglia include the caudate nucleus, putamen and globus pallidus. The caudate nucleus and putamen together are called the striatum and function as one unit; the putamen and globus pallidus are anatomically close to one another and are collective called the lentiform nucleus because of their lens-like shape.

Specific details concerning the basal ganglia and their connections will be discussed in the motor system chapter.

**Limbic system**

The limbic system is a group of cortical and sub-cortical prosencephalic structures that control emotional responsiveness and affective behavior, resulting in an individualized interpretive response to external and internal stimuli. The major output of the limbic system is via the hypothalamus, which controls the autonomic nervous system and neuroendocrine secretions.

Cortical limbic structures include the hippocampus, parahippocampal gyrus, cingulate cortex and prefrontal cortex. Subcortical limbic structures include the amygdala, septum, nucleus basalis of Meynert, and the anterior perforated substance.

An important part of the limbic system is the Papez circuit, which is necessary for incorporating new memory. This circuit includes the following components:

hippocampus → fornix → mammillary bodies → anterior nucleus of the thalamus → cingulate cortex → hippocampus

Bilateral lesions to the Papez circuit result in an inability to incorporate new memory.

**Olfactory system**

The olfactory system consists of the olfactory bulb and tract, the olfactory striae, and the uncus (primary olfactory cortex). The olfactory system is a primitive system that has a major input into the limbic system. It is the only primary sensory system that bypasses the thalamus and projects directly to the cortex.
Diencephalon ("between" brain)

The diencephalon lies deep to the prosencephalon and rostral to the midbrain. It consists of four structures: the thalamus, hypothalamus, subthalamus and epithalamus.

**Thalamus**

The thalamus is the major sensory and motor relay station for the cortex, acting as a gateway, or "executive secretary". All primary sensory modalities (with the exception of olfaction), all motor relay information from the cerebellum and basal ganglia, and much autonomic information is processed by the thalamus before reaching the cortex. The thalamus is divided into many nuclei that each process one or more specific modalities, and these nuclei will be detailed below with their respective systems.

**Hypothalamus**

The hypothalamus lies ventral to the thalamus and surrounds the third ventricle. It is the major output structure for the limbic system by virtue of its two major functions, autonomic and neuroendocrine.

The hypothalamus is the head nucleus for the autonomic nervous system, influencing sympathetic and parasympathetic output with respect to limbic inputs. Its neuroendocrine function includes secreting oxytocin and vasopressin, as well as hormonal releasing and inhibiting factors into the hypophyseal portal system, which control the release of anterior pituitary hormones into the blood.

The hypothalamus also contains visceral regulatory areas that control various visceral activities, such as feeding, drinking, reproduction and thermoregulation.

**Subthalamus**

The subthalamus is a diencephalic structure that is integrally related to the basal ganglia and has direct connections with the globus pallidus. This structure will be discussed in greater detail with the basal ganglia below. Of note, a lesion to one of the subthalamic nuclei usually results in an involuntary movement disorder in the contralateral limbs, known as hemiballismus.

**Epithalamus**

The epithalamus consists of the pineal gland, habenular nuclei and habenular commissure. This subdivision of the diencephalon has significant limbic connections. The pineal gland calcifies in the majority of adults, and is an important radiologic midline marker on plain skull films and CT scans. It secretes melatonin, a hormone involved with diurnal rhythms and sleep. Tumors of the pineal gland compress the pretectum and cerebral aqueduct, resulting in paralysis of upgaze and obstructive hydrocephalus (Perinaud's syndrome).
Mesencephalon and Rhombencephalon (Brain stem)

The brain stem consists of the mesencephalon (midbrain) and the rhombencephalon (hindbrain). The rhombencephalon, in turn, is divided into two components, namely the metencephalon (pons and cerebellum) and the myelencephalon (medulla). The midbrain, pons and medulla, or brain stem proper, are integrally related and will be considered first. The cerebellum will be discussed afterwards.

The brain stem is a crucial part of the nervous system that contains ascending sensory and descending motor fiber tracts, cranial nerve nuclei, secondary motor and sensory nuclei, autonomic pathways and "centers", and the reticular formation. The brain stem may be divided into five functional components: motor, sensory, autonomic, cerebellar, and the reticular formation.

Motor components:

- **lower motor neurons** (cranial nerves III, IV, motor V, VI, VII, nucleus ambiguus [IX, X, XI], XI, and XII)

- **upper motor neurons** (red nucleus, superior and inferior colliculi, medial and lateral vestibular nuclei, and medial pontine and medullary reticular formation)

- **descending motor pathways** (corticospinal tract, rubrospinal tract, lateral and medial vestibulospinal tracts, pontine and medullary reticulospinal tracts and the tectospinal tract)

Sensory components:

- **secondary sensory nuclei** (nuclei gracilis and cuneatus, nucleus solitarius, descending nucleus of V, main sensory nucleus of V, dorsal and ventral cochlear nuclei, accessory auditory nuclei, superior, inferior, medial and lateral vestibular nuclei, and the superior and inferior colliculi)

- **ascending sensory pathways** (medial lemniscus, lateral lemniscus, trigeminal lemniscus, spinal lemniscus, and the solitario-thalamic tract)

Autonomic components:

- **parasympathetic preganglionic nuclei** (nucleus of Edinger-Westphal, superior and inferior salivatory nuclei, and the dorsal motor nucleus of X)

- **medullary autonomic "centers"** (respiratory, pressor, depressor and vomiting centers)

- **descending autonomic pathways** from the hypothalamus and limbic system

Cerebellar connections

The brain stem contains three pairs of peduncles that connect the cerebellum to each component of the brain stem, namely:
• superior cerebellar peduncle (brachium conjunctivum), connecting the midbrain with the cerebellum

• middle cerebellar peduncle (brachium pontis), connecting the pons with the cerebellum

• inferior cerebellar peduncle (restiform body), connecting the medulla with the cerebellum

Lesions to any of these peduncles, as well as to the cerebellum, can produce ataxia, a decomposition of movement with a coarse action tremor.

Reticular Formation:

The reticular formation is a phylogenetically ancient system that forms the core of the entire brain stem. It is responsible for maintaining consciousness, maintaining general muscle tone and posture, processing noxious stimuli, and regulating major visceral functions. The medial portion of the reticular formation has mainly motor function, and the lateral portion has mainly sensory function. Autonomic function is not as precisely localized and is found scattered throughout the entire reticular formation. The various components of the reticular formation can be summarized as follows:

• Motor components: Maintenance of muscle tone and posture by means of the pontine (extensor bias) and medullary (flexor bias) reticulospinal tracts

• Sensory components:
  • Processing of "slow pain" information via the spinoreticular tract
  • Maintenance of consciousness -- the brain stem reticular activating system "awakens" the cortex in response to noxious stimuli

• Autonomic components: Medullary "centers" control blood pressure, respirations, cardiac function and gastrointestinal function.

Cerebellum

The cerebellum is a "comparator", and as such coordinates and "smoothes out" motor activity by comparing the position of body parts in space with the intended movement of those body parts. The cerebellum consists of the midline vermis, the paravermis located lateral to the vermis, and the cerebellar hemispheres. The vermis coordinates the trunk and the cerebellar hemispheres coordinate appendicular (arm and leg) movements. This structure will be discussed in more detail in the motor systems chapter.

Spinal Cord

The spinal cord is located in the vertebral canal, and in the adult human extends from the foramen magnum to the L1 vertebral level. The reason for this is that during
development, the vertebral column grows more rapidly than the spinal cord that it encloses. Hence, vertebral levels and spinal levels do not correspond in the lumbar and sacral regions. The tapering end of the spinal cord at L1 is known as the conus medullaris.

The spinal cord is divided into 31 segments as follows:

- 8 cervical
- 12 thoracic
- 5 lumbar
- 5 sacral
- 1 coccygeal

Dorsal and ventral roots exit the spinal cord at each spinal level. The dorsal roots carry sensory information into the spinal cord, and the ventral roots carry motor and autonomic information away from the spinal cord. The dorsal and ventral roots join a short distance from the spinal cord to form a spinal nerve.

The lumbar region of the vertebral canal contains lumbar and sacral spinal nerves, which must travel caudally a certain distance to exit the vertebral canal, since the spinal cord ends at the L1 vertebral level in the adult. This collection of spinal nerves is known as the cauda equina (horse's tail).

The spinal cord consists of both gray matter that contains collections of cell bodies, and white matter, which contains myelinated ascending and descending fiber tracts. The gray matter is found centrally in the spinal cord, arranged in a "butterfly" or H-shaped pattern; the white matter surrounds the gray matter and thus is found peripherally in the spinal cord.

The gray matter is divided into a dorsal horn that contains mainly primary sensory axons and secondary sensory cell bodies, and a ventral horn that contains cell bodies of lower motor neurons (anterior horn cells). In addition, a lateral horn is present in the thoracic and upper lumbar cord, midway between the dorsal and ventral horns. This horn contains an intermediolateral cell column of preganglionic sympathetic cell bodies.

The gray matter has been further divided into ten zones, known as Lamina of Rexed, based on cellular morphology. Lamina I-V contain secondary sensory cell bodies; lamina VI-VIII contain interneurons; lamina IX contains lower motor neurons; lamina X is the commissural gray found around the central canal.

The white matter is divided by the dorsal and ventral horns of gray matter into dorsal, lateral and ventral funiculi. The dorsal funiculus contains mainly ascending somatosensory tracts, and the lateral and ventral funiculi contain both ascending sensory and descending motor tracts.
PERIPHERAL NERVOUS SYSTEM (PNS)

General Organization of the PNS

The peripheral nervous system consists of all the neural elements not found in the brain or spinal cord, namely the peripheral nerves, primary sensory cell bodies found in the dorsal root ganglia, sensory receptors, neuromuscular junctions, and autonomic ganglion cells. The PNS contains motor, sensory and autonomic components, as follows:

- **Motor components:** axons of lower motor neurons and the neuromuscular junctions. Acetylcholine (nicotinic receptor) is the neurotransmitter at the neuromuscular junction.

- **Sensory components:** receptors, primary sensory axons and primary sensory cell bodies (which are found in dorsal root ganglia and cranial nerve ganglia).

- **Autonomic components:** sympathetic and parasympathetic preganglionic axons, ganglion cells, and postganglionic axons.

The Autonomic Nervous System

The autonomic nervous system is divided into two major divisions, namely the sympathetic nervous system and the parasympathetic nervous system. The peripheral components of these divisions will be discussed below:

**Sympathetic Nervous System**

The sympathetic nervous system "arouses" the organism to prepare for "fight or flight" activity, and its effects are widespread throughout the body. This system is described as a thoraco-lumbar system, since the preganglionic cell bodies are located in the intermediolateral cell column of spinal cord segments T1-L2.

The postganglionic cell bodies are found in two types of autonomic ganglia: sympathetic chain ganglia which are found at all spinal levels and are attached to spinal nerves by gray (proximal) and white (distal) rami communicantes; and sympathetic collateral ganglia which arise from splanchnic nerves that exit from the sympathetic chain ganglia. Acetylcholine (nicotinic receptor) is the preganglionic sympathetic neurotransmitter, and norepinephrine is the postganglionic sympathetic neurotransmitter.

**Parasympathetic Nervous System**

The parasympathetic nervous system is mainly homeostatic, allowing for maintenance and repair of the body. This system is described as cranio-sacral, since the preganglionic cell bodies are located in four brain stem cranial nerve nuclei and in the intermediate gray of spinal cord segments S2-4.

The postganglionic cell bodies for the cranial portion of the parasympathetic nervous system are found in specific ganglia, as noted in Table 1.
TABLE 1
PARASYMPATHETIC CRANIAL NERVE NUCLEI AND GANGLIA

<table>
<thead>
<tr>
<th>NUCLEUS</th>
<th>CRANIAL VERGE</th>
<th>GANGLION</th>
<th>TARGET</th>
</tr>
</thead>
<tbody>
<tr>
<td>Edinger-Westphal</td>
<td>III</td>
<td>Ciliary</td>
<td>Pupillary constriction, accommodation</td>
</tr>
<tr>
<td>Superior salivatory</td>
<td>VII</td>
<td>Pterygopalatine</td>
<td>Lacrimal glands</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Submandibular</td>
<td>Submandibular and sublingual salivary glands</td>
</tr>
<tr>
<td>Inferior salivatory</td>
<td>IX</td>
<td>Otic</td>
<td>Parotid gland</td>
</tr>
<tr>
<td>Dorsal motor nucleus of X</td>
<td>X</td>
<td>Intramural</td>
<td>Heart, lung, liver, gut to splenic flexure</td>
</tr>
</tbody>
</table>

The postganglionic cell bodies for the sacral portion of the parasympathetic nervous system are found in intramural ganglia located within the walls of the organ that is being innervated.

Acetylcholine (nicotinic receptor) is the preganglionic parasympathetic neurotransmitter, and acetylcholine (muscarinic receptor) is the postganglionic parasympathetic neurotransmitter.

Systems for classifying peripheral nerves

Peripheral nerves are frequently classified by the size of their axons. Two classification systems have been developed for this purpose. The Universal Classification (A, B, C) System is used for classifying all peripheral nerves, regardless of modality, while the Sensory Classification (I, II, III, IV) System is frequently used for further sub-classifying sensory nerves. Tables 2 through 5 detail the various systems used for classifying peripheral nerves.

TABLE 2
PERIPHERAL NERVE CLASSIFICATION SYSTEMS

<table>
<thead>
<tr>
<th>UNIVERAL CLASSIFICATION</th>
<th>DIAMETER</th>
<th>CONDUCTION VELOCITY</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>1-20 μ</td>
<td>5-120 m/sec</td>
</tr>
<tr>
<td>B</td>
<td>1-3 μ</td>
<td>3-15 m/sec</td>
</tr>
<tr>
<td>C</td>
<td>&lt;2 μ</td>
<td>0.5-2 m/sec</td>
</tr>
</tbody>
</table>
### TABLE 3
SENSORY NERVES

<table>
<thead>
<tr>
<th>SENSORY CLASSIFICATION</th>
<th>UNIVERSAL CLASSIFICATION</th>
<th>MODALITY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ia</td>
<td>Aα</td>
<td>muscle spindle - primary (annulospiral ending)</td>
</tr>
<tr>
<td>Ib</td>
<td>Aα</td>
<td>Golgi tendon organ</td>
</tr>
<tr>
<td>II</td>
<td>Aβ</td>
<td>muscle spindle - secondary (flower spray ending)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>epicritic mechanoreceptors (touch, pressure, joint afferent, vibration)</td>
</tr>
<tr>
<td>III</td>
<td>Aδ</td>
<td>fast pain, temperature, joint response, touch (small, myelinated)</td>
</tr>
<tr>
<td>IV</td>
<td>C</td>
<td>slow pain, painful temperature (unmyelinated)</td>
</tr>
</tbody>
</table>

### TABLE 4
MOTOR NERVES

<table>
<thead>
<tr>
<th>CLASSIFICATION</th>
<th>MOTOR NEURON</th>
<th>TYPE</th>
<th>CELL BODY SIZE</th>
<th>AXON DIAMETER</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aα</td>
<td>αMN</td>
<td>skeletomotor</td>
<td>30-100 μ</td>
<td>8-17 μ</td>
</tr>
<tr>
<td>Aγ</td>
<td>γMN</td>
<td>fusimotor</td>
<td>15-37 μ</td>
<td>3-8 μ</td>
</tr>
</tbody>
</table>

### TABLE 5
AUTONOMIC NERVES

<table>
<thead>
<tr>
<th>CLASSIFICATION</th>
<th>TYPE</th>
<th>MODALITY</th>
</tr>
</thead>
<tbody>
<tr>
<td>B</td>
<td>Small, myelinated</td>
<td>Preganglionics</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ciliary postganglionics</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Some visceral afferents (non-pain)</td>
</tr>
<tr>
<td>C</td>
<td>Small, unmyelinated</td>
<td>Postganglionics</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Visceral pain fibers</td>
</tr>
</tbody>
</table>
Columnar organization of cranial nerves

The cranial nerves III through XII are derived developmentally from the alar and basal plates of the neural tube. In general, the alar plate gives rise to sensory (afferent) neurons, while the basal plate gives rise to motor and autonomic (efferent) neurons. These plates subsequently divide into seven cellular columns, each located in a specific portion of the brain stem, and each processing a specific modality. Each of these columns gives rise to portions of one or more cranial nerves. Table 6 and 7 illustrate the components and columnar organization of the cranial nerves.

**TABLE 6**

**CRANIAL NERVE COMPONENTS**

<table>
<thead>
<tr>
<th>CRANIAL NERVE</th>
<th>COMPONENTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>III</td>
<td>SE, GVE</td>
</tr>
<tr>
<td>IV</td>
<td>SE</td>
</tr>
<tr>
<td>V</td>
<td>SVE, GSA</td>
</tr>
<tr>
<td>VI</td>
<td>SE</td>
</tr>
<tr>
<td>VII</td>
<td>SVE, GVE, SVA, GSA</td>
</tr>
<tr>
<td>VIII</td>
<td>SSA</td>
</tr>
<tr>
<td>IX, X</td>
<td>SVE, GVE, SVA, GVA, GSA</td>
</tr>
<tr>
<td>XI</td>
<td>SVE</td>
</tr>
<tr>
<td>XII</td>
<td>SE</td>
</tr>
</tbody>
</table>

SE       Somatic efferent  
SVE      Special visceral efferent  
GVE      General visceral efferent  
SVA      Special visceral afferent  
GVA      General visceral afferent  
GSA      General somatic afferent  
SSA      Special somatic afferent
<table>
<thead>
<tr>
<th>COLUMN</th>
<th>MODALITY</th>
<th>CENTRAL CONNECTIONS</th>
<th>BRAIN STEM NUCLEUS</th>
<th>CRANIAL NERVE</th>
<th>GANGLION</th>
<th>TARGET</th>
</tr>
</thead>
<tbody>
<tr>
<td>SE</td>
<td>LMN</td>
<td>Motor cortex</td>
<td>III, IV, VI</td>
<td>III, IV, VI</td>
<td></td>
<td>Extraocular muscles</td>
</tr>
<tr>
<td></td>
<td>Myotomes</td>
<td>Corticobulbar tract</td>
<td>XII</td>
<td>XII</td>
<td></td>
<td>Tongue</td>
</tr>
<tr>
<td>SVE</td>
<td>LMN</td>
<td>Motor Cortex</td>
<td>V</td>
<td>V</td>
<td></td>
<td>Muscles of Mastication</td>
</tr>
<tr>
<td></td>
<td>Branchial arches</td>
<td>Corticobulbar tract</td>
<td>VII, Nucleus ambiguus</td>
<td>VII, IX, X</td>
<td></td>
<td>Facial expression, Pharynx, larynx</td>
</tr>
<tr>
<td>GVE</td>
<td>Parasympathetic</td>
<td>Hypothalamus</td>
<td>Edinger-Westphal Superior Salivatory</td>
<td>III, VII</td>
<td>Ciliary, Pterygopalatine, Submandibular</td>
<td>Pupil constrictor Lacrimal glands, Submandibular &amp; sublingual glands, Parotid gland, Heart, lung, gut</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Inferior Salivatory Dorsal motor N. of X</td>
<td>IX, X</td>
<td>Otic Intramural</td>
<td></td>
</tr>
<tr>
<td>SVA</td>
<td>Taste</td>
<td>VPM via Solitario-thalamic tract</td>
<td>Nucleus solitarius-rostral portion</td>
<td>VII, IX</td>
<td>Geniculate, Petrosal, Nodose</td>
<td>Ant 2/3 tongue, Post 1/3 tongue, Epiglottis</td>
</tr>
<tr>
<td>GVA</td>
<td>Visceral sensory</td>
<td>Hypothalamus</td>
<td>Nucleus solitarius-caudal portion</td>
<td>IX, X</td>
<td>Petrosal Nodose</td>
<td>Carotid &amp; aortic body &amp; sinus, baroreceptors, chemoreceptors, gut sensation</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Parabrachial nuclei</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GSA</td>
<td>Facial sensation</td>
<td>VPM - Trigeminal Lem, VPM - Trigeminal Lem</td>
<td>Main sensory N. V Spinal nucleus V</td>
<td>V1,2,3 (V1,2,3) (VII, IX, X)</td>
<td>Trigeminal (Gen, Petr, Nod)</td>
<td>Epicritic-face, Protopathic-face, pharynx, larynx</td>
</tr>
<tr>
<td>SSA</td>
<td>Hearing Balance</td>
<td>Lat lem, MGB Ctx, MLF, Cbl, VST, Reticular formation</td>
<td>Cochlear N: D&amp;V Vestibular N: S, I, L, M</td>
<td>VIII, VIII</td>
<td>Spiral Scarpa's (Vestibular)</td>
<td>Cochlea, Vestibular apparatus</td>
</tr>
</tbody>
</table>

45
SYSTEMIC NEUROANATOMY

THE MOTOR SYSTEM

The motor system consists of all lower motor neurons, upper motor neurons and several specialized structures that help select and coordinate patterns of motor activity. This system can be divided into five parts, namely the pyramidal system, the extra-pyramidal system, the corticobulbar system, the cerebellum and the basal ganglia.

General Scheme

The motor system is a two-neuron system, containing upper motor neurons (UMNs) and lower motor neurons (LMNs). The UMNs "talk" to LMNs directly, or through interneurons. The UMNs are located either in the cerebral cortex or in the brain stem. The LMNs are located in the ventral horn of the spinal cord and in several brain stem cranial motor nerve nuclei.

The cerebellum and the basal ganglia influence motor activity, principally by projecting to the thalamus that, in turn, projects to the motor and premotor cortex. Note that the cerebellum and basal ganglia do not talk to LMNs directly.

The Pyramidal (Corticospinal) system

The pyramidal system is clinically one of the most important UMN tracts, and understanding the exact pathway of this tract will help one to understand clinical neurologic lesions. The cells of origin of this tract are located in three areas of cerebral cortex: primary motor cortex (pre-central gyrus), premotor cortex (pre-pre-central gyrus) and primary sensory cortex (post-central gyrus). These three areas of cortex contribute more-or-less an equal number of fibers to this tract.

This tract has a long course throughout the brain, brain stem and spinal cord, and synapses primarily on cervical, and to a less extent lumbosacral LMNs and interneurons, providing fine motor control of the distal limbs. It has a flexor bias (preferentially synapsing on flexor LMNs).

This tract changes names frequently, depending upon which region of the CNS is being discussed, and this may confuse the uninitiated. Table 8 lists the different names this tract assumes. Remember that the entire tract is only one axon in length, despite the name changes.
TABLE 8
CORTICOSPINAL (PYRAMIDAL) TRACT

<table>
<thead>
<tr>
<th>BRAIN REGION</th>
<th>TRACT NAME</th>
</tr>
</thead>
<tbody>
<tr>
<td>Telencephalon</td>
<td>Corona radiata</td>
</tr>
<tr>
<td>Diencephalon</td>
<td>Internal capsule (posterior limb)</td>
</tr>
<tr>
<td>Midbrain</td>
<td>Cerebral peduncle</td>
</tr>
<tr>
<td>Pons</td>
<td>Basis pontis</td>
</tr>
<tr>
<td>Medulla</td>
<td>Pyramids (Decussation)</td>
</tr>
<tr>
<td>Spinal cord</td>
<td>Lateral funiculus of spinal cord</td>
</tr>
</tbody>
</table>

The Extrapyramidal System

The extrapyramidal system consists of all the other descending UMN tracts that control spinal LMNs. This system can be divided into two groups: those tracts that are directed at all spinal LMNs, and those tracts directed at cervical LMNs only.

**UMN tracts directed toward all spinal LMNs:**

1. **Rubrospinal tract**

   The cells of origin for this tract are located in the red nucleus, which receives input from the cerebral cortex and cerebellum. Rubrospinal tract fibers exit the red nucleus, decussate immediately upon exiting, and travel through the brain stem and lateral funiculus of the spinal cord, synapsing indirectly via interneurons on spinal LMNs.

   The rubrospinal tract has a flexor bias and assists the pyramidal tract in providing fine motor control to distal extremities. Cortical input to the red nucleus is necessary for normal functioning of this tract, and lesions that interfere with this input to the red nucleus produce flexion of the upper extremities and extension of the lower extremities, a condition known as **deorticuate posturing**.

2. **Lateral vestibulospinal tract**

   The cells of origin for this tract are located in the lateral vestibular nucleus, which receives input from the vestibular apparatus. This tract descends ipsilaterally through the brain stem and ventral funiculus of the spinal cord, and synapses directly and via interneurons on spinal LMNs.

   The lateral vestibulospinal tract has a very powerful extensor bias, and is important in the maintenance of antigravity tone and upright posture. It is held in check (inhibited) by the red nucleus, and lesions to the red nucleus produce uninhibited extension of all four extremities, a condition known as **decerbrate posturing**.
3. **Pontine reticulospinal tract**

The cells of origin for this tract are located in the pontine reticular formation, which receives input from spino-reticular sensory fibers. This tract descends ipsilaterally through the brain stem and ventral funiculus of the spinal cord, and synapses via interneurons on spinal LMNs.

The pontine reticulospinal tract has an extensor bias, and assists the lateral vestibulospinal tract in maintaining extensor tone, particularly in response to cutaneous noxious stimuli.

4. **Medullary reticulospinal tract**

The cells of origin for this tract are located in the medullary reticular formation, which depends critically upon cortical input for its function. This tract descends ipsilaterally through the brain stem and ventral funiculus of the spinal cord, and synapses via interneurons on spinal LMNs.

The medullary reticulospinal tract has a flexor bias, augmenting the effects of the corticospinal and rubrospinal tracts.

**UMN tracts directed toward cervical LMNs only:**

5. **Medial vestibulospinal tract**

The cells of origin for this tract are located in the medial vestibular nucleus, which receives input from the vestibular apparatus. This tract descends ipsilaterally through the brain stem and ventral funiculus of the cervical spinal cord, synapsing directly on cervical LMNs.

The medial vestibulospinal tract helps coordinate head and neck motion with vestibular input for maintenance of head position.

6. **Tectospinal tract**

The cells of origin of this tract are located in the superior colliculus, and to a lesser extent the inferior colliculus, which receive visual and auditory input, respectively. Tectospinal tract fibers exit the superior and inferior colliculi, decussate immediately, and descend in the medial-dorsal brain stem and ventral cervical spinal cord, synapsing via interneurons on cervical LMNs.

The tectospinal tract helps coordinate head and neck motion with visual, and to a lesser extent auditory stimuli for maintenance of head position.

**Corticobulbar System**

The corticobulbar tract is the major UMN tract controlling brain stem LMNs. It differs from the corticospinal tract in three ways:

- it innervates brain stem and not spinal LMNs;
- it travels in the **genu** of the internal capsule;

- it provides **both ipsilateral and contralateral innervation** to all brain stem LMNs with the exception of LMNs supplying the lower half of the face.

These lower facial LMNs receive only **contralateral UMN innervation** via the corticobulbar tract, and hence unilateral lesions to this tract result in **contralateral lower facial weakness**; upper facial strength remains normal because the ipsilateral projection of the corticobulbar tract remain intact.

Bilateral cortical lesions produce bilateral corticobulbar tract dysfunction, manifesting as generalized facial and bulbar weakness with difficulty speaking and swallowing. This condition is known as **pseudobulbar palsy**.

**Cerebellum**

The cerebellum is a "comparator", and as such coordinates and "smoothes out" motor activity by comparing the position of body parts in space with the intended movement of those body parts. Its macroscopic organization, microscopic organization, inputs and outputs, and the spinocerebellar tracts will be discussed below:

**Macroscopic organization**

The cerebellum consists of the midline **vermis**, the **paravermis** located lateral to the vermis, and the **cerebellar hemispheres**. The vermis coordinates the trunk; the paravermis coordinates proximal appendicular (arm and leg) movements; and the cerebellar hemispheres coordinate distal appendicular movements.

Cerebellar lesions result in uncoordinated movements (**ataxia**), difficulty in judging distances when reaching for objects (**dysmetria**), and truncal instability.

Most sensory inputs to the cerebellum are ipsilateral, and hence a lesion to a cerebellar hemisphere produces **ipsilateral ataxia**.

**Microscopic Organization**

The cerebellum has two layers: the cortex, containing gray matter, and the deeper white matter. The deep cerebellar nuclei, located deep in the white matter, are the major source of output for the cerebellum.

The **cerebellar cortex** has three layers, as follows:

- **Molecular cell layer**: This is the outermost layer of the cerebellum, which contains dendrites of Purkinje cells, parallel fibers that arise from granule cells, climbing fibers that arise from the inferior olivary nucleus, and basket and stellate cells.

- **Purkinje cell layer**: This layer contains Purkinje cells, the major output cells of the cerebellar cortex.
• **Granule cell layer**: This layer contains granule cells, mossy fibers and Golgi II cells.

The **deep cerebellar nuclei** are the major source of output for the cerebellum. There are four pair of deep cerebellar nuclei, each associated with a different anatomical region of the cerebellum, as follows:

• **Fastigial nucleus**, the source of outflow from the vermis;

• **Globose and emboliform nuclei**, the source of outflow from the paravermis, and

• **Dentate nucleus**, the source of outflow from the cerebellar hemispheres.

The cerebellar circuitry is quite complex. Inputs to the cerebellum come primarily from two sources: (1) spinal cord, containing mainly proprioceptive information; and (2) cerebral cortex, containing information about intended motor activity. The cerebellum then compares these two inputs and feeds this information back to the cerebral cortex UMNs via the thalamus, resulting in a smooth and coordinated movement.

Input to the cerebellum comes via two types of fibers: **climbing fibers** carrying information from the inferior olive only; and **mossy fibers** carrying information from all other sources. Both of these fiber systems send collaterals to the deep cerebellar nuclei, and eventually synapse on Purkinje cell dendrites: the climbing fibers synapse directly; while the mossy fibers first synapse on **granule cells**, whose axons, called **parallel fibers**, synapse on Purkinje cell dendrites. Complex local cells, including **stellate cells**, **basket cells** and **Golgi II cells**, modulate the climbing fiber, mossy fiber and parallel fiber input into the cerebellar cortex.

The **Purkinje cell** is the major source of output from the cerebellar cortex, and this output is inhibitory. Axons from Purkinje cells synapse directly on climbing fiber and mossy fiber collaterals in the four deep cerebellar nuclei, resulting in inhibition of these fiber collaters.

**In summary**, the cerebellum smoothes and adjusts motor activity by comparing the position of the trunk and limbs in space with the intended movement of these body parts. The **coarse adjust** occurs in the deep cerebellar nuclei, while the **fine adjust** occurs in the cerebellar cortex.

Of note, **all of the cells in the cerebellar cortex**, including the Purkinje cell, are inhibitory with the sole exception of the **granule cell** that is excitatory.

**Inputs and outputs**

The cerebellar inputs and outputs enter and leave the cerebellum via the three pairs of cerebellar peduncles. In general, **most inputs enter the cerebellum via the inferior and middle cerebellar peduncles**, while **most outputs exit the cerebellum via the superior cerebellar peduncle**. Table 9 lists the specific cerebellar inputs and outputs.
<table>
<thead>
<tr>
<th>PEDUNCLE</th>
<th>INPUT</th>
<th>OUTPUT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inferior</td>
<td>Dorsal spinocerebellar tract</td>
<td>Vestibular nuclei</td>
</tr>
<tr>
<td></td>
<td>Cuneocerebellar tract</td>
<td>Reticular formation</td>
</tr>
<tr>
<td></td>
<td>Rostral spinocerebellar tract</td>
<td>Trigeminal system</td>
</tr>
<tr>
<td></td>
<td>Vestibular nuclei</td>
<td>Inferior olive</td>
</tr>
<tr>
<td>Middle</td>
<td>Cerebral cortex (via pontine nuclei)</td>
<td>Nucleus VL of thalamus</td>
</tr>
<tr>
<td>Superior</td>
<td>Ventral spinocerebellar tract</td>
<td>Red nucleus</td>
</tr>
<tr>
<td></td>
<td>Reticular formation</td>
<td>Reticular formation</td>
</tr>
<tr>
<td></td>
<td>Trigeminal system</td>
<td></td>
</tr>
</tbody>
</table>

Spinocerebellar tracts

Proprioceptive input from the spinal cord enters the cerebellum via the four spinocerebellar tracts. These four tracts differ with respect to the type of proprioceptive information carried and the region of the body represented by each tract.

The dorsal and ventral spinocerebellar tracts carry proprioceptive information from spinal level T6 and below, while the cuneocerebellar and rostral spinocerebellar tracts carry proprioceptive information from above spinal level T6. Table 10 summarizes the four spinocerebellar tracts.

<table>
<thead>
<tr>
<th>TRACT</th>
<th>BODY REGION</th>
<th>MODALITY</th>
<th>FIBERS</th>
<th>RELAY NUCLEUS</th>
<th>PEDUNCLE</th>
<th>CROSSING</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dorsal</td>
<td>Below T6</td>
<td>Muscle spindles</td>
<td>Ia, II</td>
<td>Clarke</td>
<td>Inferior</td>
<td>No</td>
</tr>
<tr>
<td>Ventral</td>
<td>Below T6</td>
<td>Golgi tendon organs</td>
<td>Ia, II</td>
<td>Border cells</td>
<td>Superior</td>
<td>Twice</td>
</tr>
<tr>
<td>Cuneo</td>
<td>Above T6</td>
<td>Muscle spindles</td>
<td>Ia, II</td>
<td>Lateral Cuneate</td>
<td>Inferior</td>
<td>No</td>
</tr>
<tr>
<td>Rostral</td>
<td>Above T6</td>
<td>Golgi tendon organs</td>
<td>Ia, II</td>
<td>Intermediate gray of spinal cord</td>
<td>Inferior</td>
<td>No</td>
</tr>
</tbody>
</table>

The dorsal spinocerebellar and cuneocerebellar tracts process muscle spindle information, and this information is carried into the spinal cord by group Ia and II sensory fibers; the ventral and rostral spinocerebellar tracts process Golgi tendon organ information.
information, and this information is carried into the spinal cord by group Ib sensory fibers.

The spinocerebellar tracts are two neuron chains, with the primary sensory neuron synapsing on a relay nucleus located in either the spinal cord or brain stem. These relay nuclei are listed in the table above. The lateral cuneate nucleus is located lateral to the nucleus cuneatus in the medulla. Clarke's nucleus, border cells and intermediate gray are found in the dorsal horn of the spinal cord.

The ventral spinocerebellar tract is peculiar on two counts: it is the only tract that enters the cerebellum via the superior cerebellar peduncle; it also crosses twice: at the spinal cord level of entry, and in the midbrain at the decussation of the superior cerebellar peduncle.

Parallel Cerebellar Systems

The cerebellum may be considered as having three parallel systems, each consisting of a specific cerebellar region, with its own inputs, deep cerebellar nuclei, outputs and specific targets. These are detailed in Table 11.

<table>
<thead>
<tr>
<th>SYSTEM</th>
<th>EMBRYOLOGIC NAME</th>
<th>INPUTS</th>
<th>CEREBELLAR REGION</th>
<th>DEEP CEREBELLAR NUCLEI</th>
<th>OUTPUT</th>
<th>TARGET</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vestibulo-</td>
<td>Archicerebellum</td>
<td>Vestibular</td>
<td>Vermis and</td>
<td>Fastigial</td>
<td>Vestibular nucleus</td>
<td>Vestibulo-spatial tracts</td>
</tr>
<tr>
<td>cerebellum</td>
<td>apparatus</td>
<td>apparatus</td>
<td>flocculonodular lobe</td>
<td></td>
<td>Reticular formation</td>
<td>Reticulo-spatial tracts</td>
</tr>
<tr>
<td>Spino-</td>
<td>Paleocerebellum</td>
<td>Spino-cerebellar</td>
<td>Paravermis</td>
<td>Globose</td>
<td>Red nucleus</td>
<td>Red nucleus</td>
</tr>
<tr>
<td>cerebellum</td>
<td>tracts</td>
<td>tracts</td>
<td></td>
<td>Emboliform</td>
<td></td>
<td>Rubrospinal tract</td>
</tr>
<tr>
<td>Ponto-</td>
<td>Neocerebellum</td>
<td>Cortico-pontine</td>
<td>Lateral hemispheres</td>
<td>Dentate</td>
<td>VL of thalamus</td>
<td>Cerebrum and corticospinal</td>
</tr>
<tr>
<td>cerebellum</td>
<td>fibers</td>
<td>fibers</td>
<td></td>
<td></td>
<td></td>
<td>tract</td>
</tr>
</tbody>
</table>

Basal ganglia

The basal ganglia are subcortical telencephalic structures that are integrally involved with the motor system. They allow the cortex to select wanted patterns of movement, and suppress unwanted patterns of movement. Disorders of the basal ganglia typically produce movement disorders, characterized by an excess or a paucity of movement. Parkinson's disease, Huntington's disease and Tourette's syndrome are all examples of basal ganglia disorders.

Components

The basal ganglia consist of five structures, as follows: caudate nucleus, putamen, globus pallidus, substantia nigra and subthalamus. The caudate nucleus and putamen together are called the striatum and function as one unit; the putamen and globus
pallidus are anatomically close to one another and are collective called the **lentiform nucleus**, because of their lens-like shape.

**Circuitry**

The basal ganglia can be considered to have a **sensory component**, the **striatum** (caudate and putamen), and a **motor component**, the **globus pallidus**. The striatum, like all sensory components, receives **input**, in this case from four structures: cerebral cortex, thalamus (mainly nucleus CM), brain stem raphe nuclei, and the substantia nigra, with which it has reciprocal connections. The striatum, in turn, projects to the **globus pallidus**, which **projects to primary motor and premotor cortex via nuclei VL and VA of the thalamus**, respectively. The globus pallidus also has a reciprocal connection with the **subthalamus**.

**Lower motor neurons**

Lower motor neurons are the final common pathway for all motor activity. Lower motor neurons are located in the ventral horn of the spinal cord (anterior horn cells) and in brain stem cranial motor nerve nuclei. Table 12 summarizes the nuclei, nerves and targets for the lower motor neurons.

<table>
<thead>
<tr>
<th>NUCLEUS</th>
<th>NERVE</th>
<th>TARGET</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ventral horn of SC</td>
<td>Spinal nerve</td>
<td>Limb and trunk musculature</td>
</tr>
<tr>
<td>Oculomotor</td>
<td>CN III</td>
<td>Extraocular muscles: SR, IR, MR, IO, Levator palpebrae</td>
</tr>
<tr>
<td>Trochlear</td>
<td>CN IV</td>
<td>Superior oblique muscle</td>
</tr>
<tr>
<td>Abducens</td>
<td>CN VI</td>
<td>Lateral rectus muscle</td>
</tr>
<tr>
<td>Trigeminal motor</td>
<td>CN V</td>
<td>Muscles of mastication</td>
</tr>
<tr>
<td>Facial</td>
<td>CN VII</td>
<td>Muscles of facial expression</td>
</tr>
<tr>
<td>Ambiguus</td>
<td>CN IX, X, XI</td>
<td>Pharyngeal and laryngeal muscles</td>
</tr>
<tr>
<td>Spinal accessory</td>
<td>CN XI</td>
<td>Sternocleidomastoid and trapezius muscles</td>
</tr>
<tr>
<td>Hypoglossal</td>
<td>CN XII</td>
<td>Tongue musculature</td>
</tr>
</tbody>
</table>

**Muscle spindles**

The muscle spindle is a sophisticated sensory receptor that reports information to the CNS concerning the length of and amount of stretch on each individual muscle fiber. Since muscle spindles are connected in parallel with muscle fibers, the amount of stretch on each spindle varies with the state of contraction of each muscle fiber. In order for the muscle spindles to maintain responsiveness throughout the entire range of contraction of the skeletal muscle fiber, they have developed an independent **intrafusal** contractile element that alters the length of each muscle spindle with respect to the state of contraction of the **extrafusal** muscle fiber. These intrafusal muscle fibers are innervated by separate efferent nerves, the **gamma motor neurons**, which adjust the sensitivity of the muscle spindles to the amount of stretch on the muscle fiber.
There are two different types of muscle spindle fibers, **nuclear bag** and **nuclear chain fibers**, each of which having its own afferent and efferent innervation and nerve ending. The nuclear bag fibers report information concerning both velocity of movement (dL/dt) and length (L) of the extrafusal muscle fiber, while the nuclear chain fibers primarily report information concerning muscle fiber length. Table 13 details the two different types of muscle spindle fibers.

**TABLE 13**

**MUSCLE SPINDLE TYPES**

<table>
<thead>
<tr>
<th>SPINDLE TYPE</th>
<th>AFFERENT FIBER</th>
<th>AFFERENT ENDING</th>
<th>MODALITY</th>
<th>EFFERENT FIBER</th>
<th>EFFERENT ENDING</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nuclear bag</td>
<td>Ia</td>
<td>Primary (Annulospiral)</td>
<td>dL/dt, L</td>
<td>$\gamma_1$</td>
<td>Gamma plate</td>
</tr>
<tr>
<td>Nuclear chain</td>
<td>II</td>
<td>Secondary (Flower spray)</td>
<td>L</td>
<td>$\gamma_2$</td>
<td>Gamma trail</td>
</tr>
</tbody>
</table>
SENSORY SYSTEMS

Sensory Channels

Primary sensory information entering the CNS forms three types of functional secondary sensory connections, called channels: reflex channels, cerebellar channels and lemniscal (ribbon) channels.

1. Reflex channels: There are two types of reflex channels: monosynaptic and polysynaptic.

   • **Monosynaptic channels**: These are the simplest of all reflex channels. The monosynaptic muscle stretch (deep tendon) reflex is the best example, in which a primary sensory axon synapses directly on an anterior horn cell in the ventral horn of the spinal cord. Stretch of a muscle spindle excites group Ia sensory afferent axons, which then synapse on anterior horn cells innervating the muscle that was stretched, resulting in contraction of that muscle.

   • **Polysynaptic channels**: These are more complex reflex channels in which interneurons participate. The withdrawal (nociceptive) reflex is an example, in which a pain stimulus causes a complex withdrawal response involving many myotomal segments on both sides of the spinal cord.

2. Cerebellar channels: This is a two-neuron system that carries unconscious proprioceptive information to the cerebellum via the four spino-cerebellar tracts (see table 10). In addition, two indirect pathways utilize the inferior olive and the reticular formation to provide additional unconscious proprioceptive information to the cerebellum: the spino-olivo-cerebellar system; and the spino-reticulo-cerebellar system.

3. Lemniscal channels: Lemniscal channels carry conscious sensory information to the cortex. The lemniscal sensory system, in a broad sense, is a three-neuron system. Primary sensory cell bodies are located in peripheral ganglia: dorsal root ganglia in the case of spinal nerves, and specialized sensory cranial nerve ganglia in the case of cranial nerves. Primary sensory axons synapse on secondary sensory axons, located in secondary sensory nuclei in the brain stem or spinal cord. Secondary sensory axons, in turn, synapse on tertiary sensory axons located in the thalamus, and these axons ultimately project to the cerebral cortex.

Somatosensory System

The somatosensory system carries and processes somatosensory information from the trunk and limbs. This form of sensation can be divided into two types, epicritic and protopathic, and each to these types of sensory information has its own set of receptors and its own projection channels (lemnisci) within the CNS. Epicritic modalities include fine, discriminative touch, position sense and vibration; protopathic modalities include poorly localized touch, and pain and temperature perception.
Epicritic (Dorsal column) system

The sensory receptors for the epicritic system include encapsulated nerve endings, such as Pacinian corpuscles and Meissner's corpuscles. The primary sensory cell bodies are located in dorsal root ganglia.

The primary sensory axons enter the dorsal horn of the spinal cord, immediately enter the dorsal funiculus (dorsal column), and ascend without synapsing in the fasciculi gracilis and cuneatus to nuclei gracilis and cuneatus located in the medulla. The nucleus and fasciculus gracilis are located medially and carry epicritic information from spinal level T6 and below; the nucleus and fasciculus cuneatus are located lateral to nucleus and fasciculus gracilis and carry epicritic information from spinal level T6 and above.

Secondary epicritic sensory afferents exit nuclei gracilis and cuneatus, decussate immediately in the medulla as the internal arcuate fibers, and ascend through the brain stem as the medial lemniscus to nucleus VPL of the thalamus. Tertiary epicritic sensory afferents exit nucleus VPL and ascend through the corona radiata to the primary sensory cortex (post-central gyrus).

Protopathic (Spinothalamic) system

The protopathic system is divided into two systems: one for processing fast (well-localized) pain and temperature; and the other for processing slow (poorly localized and excruciating) pain.

Fast pain and temperature:

The sensory receptors for this system include small, myelinated fibers (Aδ fibers) for fast pain, and (perhaps) Krause end bulbs and Ruffini corpuscles for temperature. Primary sensory cell bodies are located in dorsal root ganglia.

Primary sensory axons enter the dorsal horn of the spinal cord and synapse in Rexed laminae I and V. Secondary sensory axons then exit these laminae, decussate in the anterior white commissure of the spinal cord, and ascend in the contralateral lateral funiculus and brain stem as the spinothalamic tract to nucleus VPL of the thalamus. Tertiary sensory afferents exit nucleus VPL and ascend through the corona radiata to the primary sensory cortex (post-central gyrus).

Slow pain

The sensory receptors for this system are bare nerve endings (C fibers). Primary sensory cell bodies are located in dorsal root ganglia.

Primary sensory axons enter the dorsal horn of the spinal cord and synapse in Rexed laminae II and III (substantia gelatinosa). Axons from substantia gelatinosa project to cells in Rexed laminae IV and V (nucleus proprius) and lamina VII (intermediate gray). Axons from these laminae then decussate in the anterior white commissure of the spinal cord and ascend in the contralateral lateral funiculus as the spinothalamic tract to the brain stem reticular formation and to nucleus CM of the thalamus where most slow pain is processed. In addition, nucleus CM of the thalamus may send some axonal
projections to secondary sensory cortex (SII) and to non-specific areas of cortex for further slow pain processing.

Trigeminal Sensory System

The trigeminal sensory system carries and processes sensory information from the face. It is analogous to the somatosensory system, in that separate channels exist for processing epicritic information, and for processing both the slow pain and fast pain components of the protopathic system.

Epicritic system

The primary sensory cell bodies for this system are located in the trigeminal (semilunar or Gasserian) ganglion. The primary sensory axons enter the lateral pons via the trigeminal nerve and synapse in the main (chief or principal) sensory nucleus of V. Secondary sensory afferents exit this nucleus, immediately decussate, and ascend in the trigeminal lemniscus to nucleus VPM of the thalamus. Tertiary epicritic sensory afferents exit nucleus VPM and ascend through the genu of the internal capsule to the lateral portion of the primary sensory cortex (post-central gyrus).

Protopathic system

The primary sensory cell bodies for this system are located in the trigeminal (semilunar or Gasserian) ganglion. The primary sensory axons enter the lateral pons via the trigeminal nerve and synapse in the descending (spinal) nucleus of V. This nucleus extends from the mid pons caudally through the medulla into upper cervical levels of the spinal cord.

Secondary sensory afferents for the fast pain system exit the descending nucleus of V, immediately decussate, and ascend in the trigeminal lemniscus to nucleus VPM of the thalamus. Tertiary epicritic sensory afferents exit nucleus VPM and ascend through the genu of the internal capsule to the lateral portion of the primary sensory cortex (post-central gyrus).

Secondary sensory afferents for the slow pain system exit the descending nucleus of V and enter the brain stem reticular formation. Some fibers also ascend to nucleus CM of the thalamus. As with the somatosensory slow pain system, nucleus CM of the thalamus may send some axonal projections to secondary sensory cortex (SII) and to non-specific areas of cortex for further slow pain processing.

Visceral sensory

The visceral sensory system processes two modalities: taste and visceral sensation, the latter including baroreceptor and chemoreceptor information from aortic and carotid arches and bodies, respectively.
Taste System

Taste sensation is carried by three cranial nerves, depending upon which portion of the tongue or epiglottis is stimulated. In addition, each cranial nerve has its own primary sensory ganglion associated with it. Table 14 lists the respective cranial nerves, ganglia and body regions subserving taste.

**TABLE 14**

CRANIAL NERVES INVOLVED WITH TASTE

<table>
<thead>
<tr>
<th>CRANIAL NERVE</th>
<th>GANGLION</th>
<th>REGION OF INNERVATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>VII</td>
<td>Geniculate</td>
<td>Anterior 2/3 of tongue</td>
</tr>
<tr>
<td>IX</td>
<td>Petrosal</td>
<td>Posterior 1/3 of tongue</td>
</tr>
<tr>
<td>X</td>
<td>Nodose</td>
<td>Epiglottis</td>
</tr>
</tbody>
</table>

Primary sensory afferents carrying taste information enter the brain stem via CN VII, IX and X, enter the solitary tract located in the dorsal medulla, and synapse in the rostral portion of the nucleus solitarius. Secondary sensory afferents exit this nucleus and ascend ipsilaterally in the solitario-thalamic tract to nucleus VPM of the thalamus. Tertiary sensory afferents exit nucleus VPM and ascend through the genu of the internal capsule to the lateral portion of the post-central gyrus and to portions of the insular cortex.

The nucleus solitarius also sends projections to the parabrachial nuclei of the pons, and these nuclei subsequently project to the hypothalamus and other limbic areas, thus providing gustatory input to areas of the brain that regulate visceral behavior.

Visceral sensation

The primary sensory cell bodies are located in the petrosal ganglion (CN IX) and in the nodose ganglion (CN X). Primary sensory afferents carrying visceral information enter the brain stem via CN IX and X, enter the solitary tract located in the dorsal medulla, and synapse in the caudal portion of the nucleus solitarius. This nucleus subsequently relays this visceral information to the dorsal motor nucleus of X and other medullary autonomic centers, allowing for reflex regulation of cardiovascular and respiratory responses.

Auditory System

The auditory system converts mechanical energy of sound into electrical signals, analyzes these for tone, loudness and timbre, and interprets these properties with respect to the understanding of speech.

Peripheral apparatus:

The peripheral apparatus includes the outer ear, middle ear and inner ear:

- **Outer ear**: Pinna and external ear canal
- **Middle ear**: Typanic membrane, ossicles (malleus, incus and stapes), muscles (tensor tympani [CN V], stapedius [CN VII]).

- **Inner ear**: Oval window, round window, cochlea (scala vestibuli, scala tympani, scala media, helicotrema, basilar membrane, Meissner's membrane, tectorial membrane, Organ of Corti, inner and outer hair cells, perilymph, endolymph).

Sound waves are funneled by the outer ear, strike the tympanic membrane, are amplified by the three ossicles, and cause the oval window to vibrate. This produces a fluid wave in the perilymph-filled scala vestibuli that then sets up a standing wave in the basilar membrane. Each specific portion of the basilar membrane responds best to a certain frequency of sound, with the base responding best to high-frequency sounds. Vibration of the basilar membrane excites the inner and outer hair cells that are located on it in an organized fashion, and this results in action potentials that enter the brain stem via the cochlear nerve.

**Central connections**

The primary sensory cell bodies for the cochlear nerve are located in the spiral (auditory) ganglion located in the spiral of the cochlea. Axons from these bipolar cells synapse on the dorsal and ventral cochlear nuclei in the pons. Secondary sensory axons from these nuclei then project bilaterally through the lateral lemniscus to the inferior colliculus in the midbrain. The inferior colliculus projects bilaterally through the brachium of the inferior colliculus to the medial geniculate body in the thalamus, which in turn projects to the transverse gyrus of Heschl (primary auditory cortex) in the temporal lobe of the brain.

The following figure summarizes the central auditory connections:

\[
\text{CN VIII} \rightarrow \text{Cochlear nucleus} \rightarrow \text{Lateral lemniscus} \rightarrow \text{Inferior colliculus} \rightarrow \\
\text{Medial geniculate body} \rightarrow \text{Transverse gyrus of Heschl}
\]

Several accessory auditory nuclei are interposed between the cochlear nuclei and the lateral lemniscus in the pons. These include the nucleus of the lateral lemniscus, trapezoid body, and superior olivary nucleus.

The superior olivary nucleus is important in sound localization, since each of its cells receives auditory input from both the right and left ears and integrates the temporal sequence of the sound striking each ear at a slightly different time.

It is important to realize that all brain stem auditory nuclei and tracts carry auditory information originating from both ears, with the exception of the cochlear nuclei. This means that unilateral brain stem lesions do not produce unilateral hearing loss; such lesions only produce a generalized, bilateral diminution in auditory acuity. Unilateral hearing loss implies a lesion to the peripheral auditory apparatus, cochlear nerve or cochlear nuclei.
Vestibular System

The vestibular system senses changes in head and body position in space, and uses this information in numerous ocular and somatic reflex circuits.

Peripheral apparatus

The peripheral apparatus has two types of receptors: those that sense angular acceleration, and those that sense linear acceleration.

Angular acceleration

The receptors for angular acceleration are the semicircular canals. There are three pairs of canals, and all pairs are mutually orthogonal (all at right angles to one other). Each canal is filled with endolymph and has a dilated portion at one end, called the ampulla. The ampulla contains hair cells that protrude into a gelatinous substance known as the cupula.

The hair cells for each of the semicircular canals have a tonic rate of electrical firing, which means that they all fire at a baseline rate when the head is not moving. Rotating the head in a certain direction causes endolymph to deflect the hair cells in a specific direction in one or more of the semicircular canals. This deflection results in a change in the baseline electrical firing rate, which then propagates down the vestibular nerve into the brain stem.

Linear acceleration (gravity)

The receptors for linear acceleration are the utricle and saccule. These structures contain hair cells that have calcium carbonate crystals sitting on them. Moving the head in any direction causes gravity to deflect the calcium crystals and the attached hair cells, and this deflection results in an electrical potential that propagates down the length of the vestibular nerve into the brain stem.

Central connections

The primary sensory cell bodies for the vestibular system are located in the vestibular (Scarpa's) ganglion. Axons from these bipolar cells synapse on the superior, inferior, lateral and medial vestibular nuclei in the pons. Secondary sensory axons from these nuclei project to five areas of the CNS, as follows:

- Spinal cord, via the lateral and medial vestibulospinal tracts.
- Cerebellum, particularly the vermis.
- Reticular formation, particularly the vomiting center in the medulla.
- Extraocular muscles via the medial longitudinal fasciculus (MLF) and CN nuclei III, IV and VI.
• **Medial geniculate body and cortex**, perhaps providing conscious perception of orientation of the body in space.

**Visual System**

The visual system transforms light energy into electrical energy by means of specialized cells located in the retina.

**Peripheral Apparatus:**

Light enters the eye by passing through the transparent cornea, the anterior and posterior chambers that are filled with aqueous humor, the lens and the vitreous humor. The cornea and lens refract the light and focus it on the fovea centralis, which is located in the center of the macula at the posterior pole of the retina. Light energy is then transduced into electrical energy by two sets of photoreceptors: rods that sense black and white images; and cones that sense color vision. The fovea centralis contains only cones, and the peripheral retina contains primarily rods.

Rods and cones synapse on bipolar cells, and these in turn project to ganglion cells, which are the primary visual sensory afferent cells. **Horizontal cells** and **amacrine cells** aid in local processing of visual information in the retina.

**Optic Nerve, Optic Chiasm, and Optic Tract**

Axons from retinal ganglion cells form the optic nerves that exit the posterior pole of the eyeball. Fibers carrying visual information from the nasal retina cross in the optic chiasm and enter the contralateral optic tract. Fibers carrying visual information from the temporal retina do not cross but enter the ipsilateral optic tract directly.

It is important to remember that the eye works more or less like a pinhole camera, whereby images are inverted: temporal images strike the nasal retina, and superior images strike the inferior retina. If this is understood, and if one recalls which retinal fibers cross in the optic chiasm, one can easily understand the visual field deficits that occur when different parts of the optic pathway are lesioned. These visual field deficits are summarized in table 15.

**TABLE 15**

**VISUAL FIELD DEFECTS**

<table>
<thead>
<tr>
<th>LESION</th>
<th>VISUAL FIELD DEFECT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Optic nerve</td>
<td>Ipsilateral monocular blindness</td>
</tr>
<tr>
<td>Optic chiasm</td>
<td>Bitemporal hemianopsia</td>
</tr>
<tr>
<td>Optic tract</td>
<td>Contralateral homonymous hemianopsia</td>
</tr>
</tbody>
</table>
Central connections

Axons from retinal ganglion cells enter the brain and project to four different areas: the lateral geniculate body, the pretectum, the superior colliculus, and the hypothalamus. These projections will be discussed below.

Lateral geniculate body

Retinal ganglion cell axons destined for conscious perception of vision synapse in the lateral geniculate body (LGB) of the thalamus. The LGB then projects through the optic radiations in the posterior limb of the internal capsule to primary visual (striate) cortex, located on both banks of the calcarine fissure on the medial surface of the occipital lobe.

Pretectum

Retinal ganglion cell axons involved with the pupillary light reflex bypass the lateral geniculate body and synapse via interneurons on the nucleus of Edinger-Westphal in the midbrain. This nucleus subsequently projects to the ciliary ganglion, and then to the pupillary constrictor muscle via CN III. Remember that the pupillary light reflex is a direct and consensual reflex; fiber crossing occurs in the pretectum and in the posterior commissure.

Superior colliculus

Retinal ganglion cell axons involved with visual reflexes synapse in the superior colliculus. Fibers from the superior colliculus project to three areas:

- Spinal cord, via the tectospinal tract.
- Cerebellum, via the superior cerebellar peduncle.
- Pulvinar of the thalamus, which in turn projects to association visual cortex.

These projections are thought to tell where an object is, as opposed to what the object is.

Hypothalamus

Retinal ganglion cell axons project to the suprachiasmatic nucleus of the hypothalamus, and this projection is felt to be important in setting circadian light-dark rhythms, which are important for the secretion of many pituitary hormones.
**Thalamic Nuclei**

Table 16 summarizes the inputs and projections of the more important thalamic nuclei.

**TABLE 16**  
**THALAMIC NUCLEI**

<table>
<thead>
<tr>
<th>INPUT</th>
<th>NUCLEUS</th>
<th>OUTPUT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sensory relay nuclei:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Somatosensory</td>
<td>VPL</td>
<td>Primary sensory cortex (medial portion)</td>
</tr>
<tr>
<td>Facial sensory and taste</td>
<td>VPM</td>
<td>Primary sensory cortex (lateral portion)</td>
</tr>
<tr>
<td>Vision</td>
<td>LGB</td>
<td>Primary visual cortex</td>
</tr>
<tr>
<td>Audition</td>
<td>MGB</td>
<td>Primary auditory cortex</td>
</tr>
<tr>
<td>Motor relay nuclei:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Basal ganglia</td>
<td>VA</td>
<td>Premotor cortex</td>
</tr>
<tr>
<td>Basal ganglia and cerebellum</td>
<td>VL</td>
<td>Primary motor cortex</td>
</tr>
<tr>
<td>Limbic relay nuclei:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mammillary bodies</td>
<td>ANT</td>
<td>Cingulate cortex</td>
</tr>
<tr>
<td>Amygdala, hypothalamus, temporal neocortex</td>
<td>DM</td>
<td>Prefrontal cortex</td>
</tr>
<tr>
<td>Association cortex relay nuclei:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Superior and inferior colliculi, LGB, MGB</td>
<td>Pulvinar</td>
<td>Parietal and temporal association cortex</td>
</tr>
<tr>
<td>Nonspecific cortex relay nuclei:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Slow pain</td>
<td>CM</td>
<td>Non-specific areas of cortex</td>
</tr>
</tbody>
</table>
THE NEUROLOGIC EXAMINATION
Ralph F. Józefowicz, MD

NEUROLOGIC DIAGNOSIS

The neurologic history and physical examination are the most important tools in neurologic diagnosis. Although confirmatory laboratory data, including modern imaging techniques such as CT scanning and magnetic resonance imaging, have provided further accuracy in neurologic diagnosis, the history and physical examination remain the mainstays.

Neurologic diagnosis can be divided into two types, anatomic and etiologic:

The Anatomic Diagnosis localizes the lesion within a specific area of the neuraxis, i.e. cerebral hemispheres, diencephalon, brain stem, spinal cord, or the peripheral nervous system. Findings on neurologic examination are obviously most important in making an anatomic diagnosis.

The Etiologic Diagnosis specifies the cause of the lesion, and is mainly obtained from information provided by the neurologic history. The time course of the illness often helps define the etiologic agent responsible for causing the anatomic lesion. Several examples follow:

- **Lesions of Sudden Onset** are typically due to vascular accidents, such as stroke.

- **Slowly Progressive Lesions** are typically due to expanding mass lesions, such as a tumor or abscess.

- **Lesions With Exacerbating and Remitting Courses** are frequently due to demyelination, such as can be seen with multiple sclerosis.

- **Relentlessly Progressive Lesions Involving Diffuse Areas of the Nervous System** are typically due to nutritional deficits or to degenerative disorders of the brain and nervous system.

The Neurologic History

The neurologic history is the most important component of neurologic diagnosis. A careful history frequently determines the etiology and allows one to begin localizing the lesion(s), aiding in the determination if the disease is diffuse or focal. Symptoms of acute onset suggest a vascular etiology or seizure; symptoms that are subacute in onset suggest a mass lesion such as a tumor or abscess; symptoms that have a waxing and waning course with exacerbations and remissions suggest a demyelinating etiology; while symptoms that are chronic and progressive suggest a degenerative disorder.

The history is often the only way of diagnosing neurologic illnesses that typically have normal or non-focal findings on neurologic examination. These illnesses include many seizure disorders, narcolepsy, migraine and most other headache syndromes, the various causes of dizziness, and most types of dementia. The neurologic history may
often provide the first clues that a symptom is psychological in origin. Points to consider when obtaining a neurologic history:

- **Carefully identify the chief complaint or major problem.** Not only is the chief complaint important in providing the first clue to the physician as to the differential diagnosis, it is also the reason why the patient is seeking medical advice and treatment. If the chief complaint is not properly identified and addressed, the proper diagnosis may be missed and an inappropriate diagnostic work-up may be undertaken. Establishing a diagnosis that does not incorporate the chief complaint frequently focuses attention on a coincidental process irrelevant to the patient’s concerns.

- **Listen carefully to the patient for as long as is necessary.** A good rule of thumb is to listen initially for at least 5 minutes without interrupting the patient. The patient often volunteers the most important information at the start of the history. During this time, the examiner can also assess mental status including speech, language, fund of knowledge, and affect, and observe the patient for facial asymmetry, abnormalities of ocular movement, a paucity of spontaneous movements as seen with movement disorders.

- **Steer the patient away from discussions of previous diagnostic tests and of the opinions of previous caregivers.** Abnormalities on laboratory studies may be incidental to the patient’s primary problem or may simply represent a normal variant.

- **Take a careful medical history, medication history, psychiatric history, family history, and social and occupational history.** Many neurologic illnesses are complications of underlying medical disorders or due to adverse effects of drugs. For example, parkinsonism is a frequent complication of metoclopramide and most neuroleptic agents. A large number of neurologic disorders are hereditary, and a positive family history may establish the diagnosis in many instances. Occupation plays a major role in various neurologic disorders such as carpal tunnel syndrome (computer keyboard operators), and peripheral neuropathy (exposure to lead or other metals).

- **Interview surrogate historians.** Patients with dementia or altered mental status are usually unable to provide exact details of the history, and a family member may provide key details needed to make an accurate diagnosis. This is especially true for patients with dementia and certain right hemispheric lesions with variousagnosias (unawareness of disease) that may interfere with their ability to provide a cogent history. Surrogate historians also provide missing historical details for patients with episodic loss of consciousness, such as syncope, epilepsy, and narcolepsy.

- **Summarize the history for the patient.** Summarizing the history is an effective way to insure that all details were covered in sufficient detail to make a tentative diagnosis. Summarizing will also allow the physician to fill in historical gaps that may not have been apparent when the history was initially taken. In addition, the patient or surrogate may correct any historical misinformation at this time.

- **End by asking the patient what he thinks is wrong with him.** This allows the physician to evaluate the patient’s insight into his condition. Some patients have a specific diagnosis in mind that brings them to seek medical attention. Multiple sclerosis, amyotrophic lateral sclerosis, Alzheimer’s disease and brain tumors are diseases that patients often suspect may be the cause of their neurologic symptoms.
The neurologic history has several components, including the history of present illness, review of systems, past medical history, medication history, family history and social history.

The History of the Present Illness consists of an accurate, chronological description of the patient's presenting illness.

The Neurologic Review of Systems questions the patient about dysfunction affecting the various components of the nervous systems. Typical questions asked would include:

- **Mental Status**: Changes in memory or mood, ability to care for oneself, ability to balance a checkbook, difficulty with language, geographical orientation, etc.

- **Skull, Spine and Meninges**: History of head trauma, neck injury, back injury, headache or stiff neck.

- **Cranial Nerves**: Abnormalities in vision, hearing, smell, taste, speech or swallowing. Facial weakness or numbness.

- **Motor Function**: History of muscular weakness, tremor, difficulty in initiating movements, loss of muscle bulk.

- **Sensory Function**: Numbness, tingling, or altered sensation in any limbs.

- **Coordination**: Clumsiness, difficulty with hand writing or carrying out coordinated tasks.

- **Gait and Station**: Abnormalities of gait, frequent falling, difficulty maintaining balance.

- **General Symptoms**: History of seizures, vertigo, loss of consciousness, bowel or bladder difficulty.

**Past Medical History**: Many pre-existing medical conditions are significant risk factors for neurologic illness, including diabetes mellitus, hypertension, heart disease, systemic malignancy, immunologic or vasculitic disorders, or a history of cigarette smoking or alcohol abuse.

**Medication History**: Numerous medications can affect the nervous system. A careful medication history should be obtained in all patients.

**Family History**: Many neurologic disorders are hereditary. A careful family history should be taken in all patients.

**Social History**: Many occupations predispose certain individuals to neurologic illness. Repetitive hand motion, such as that which can occur on the assembly line, in butchers or in keyboard operators, can lead to entrapment of the median nerve across the carpal tunnel at the wrist (carpal tunnel syndrome). Exposure to heavy metals or toxic fumes is a frequent cause of peripheral neuropathy. Lastly, emotional stress at work or at home can cause or significantly affect an underlying neurologic illness.
MENTAL STATUS TESTING

The neurologic examination is typically divided into eight components: mental status; skull, spine and meninges; cranial nerves; motor examination; sensory examination; coordination; reflexes; and gait and station.

The mental status is an extremely important part of the neurologic examination that is often overlooked. It should be assessed first in all patients. Mental status testing can be divided into five parts: level of alertness; focal cortical functioning; cognition; mood and affect; and thought content.

Level of Alertness
(Level of Consciousness)

Level of alertness is defined as the best verbal or motor response that can be elicited from the patient in response to a specific stimulus. Many physicians label the level of alertness using such non-specific terms as “awake”, “lethargic”, “stuporous”, or “comatose”. Since not all physicians agree on the exact definitions of each of these terms, it is preferable to describe the response of the patient to a specific stimulus.

Structures Required for Consciousness

Two neural structures are required for consciousness: the brain stem reticular activating system; and one cerebral hemisphere. Thus, a patient is unconsciousness if injury has occurred to both cerebral hemispheres or to the brain stem reticular activating system.

Focal Cortical Functioning

Aphasia, apraxia and agnosia are three examples of focal cortical dysfunction.

Aphasia

Aphasia is an acquired disorder in the production or understanding of language due to a lesion involving the dominant cerebral hemisphere. In general, aphasias are of two types, namely expressive or receptive.

An expressive aphasia (front, motor, non-fluent, Broca) is usually seen following a lesion involving Broca's area (lateral pre-motor cortex). An expressive aphasia is marked by significant difficulty producing language, but with preserved understanding. Patients with this form of aphasia typically have a right hemiparesis, due to involvement of the adjacent motor cortex.

A receptive aphasia (back, sensory, fluent, Wernicke) is seen with a lesion involving the supramarginal and angular gyri in the temporal lobe (Wernicke's area). This aphasia is characterized by fluent, nonsensical speech with numerous paraphasic errors, and markedly impaired understanding. Patients with a receptive aphasia frequently have a contralateral homonymous hemianopia due to involvement of the adjacent optic radiations.
There are several other types of aphasias, including conduction, isolation, anomic, and global. The characteristics of these aphasias are detailed in table 1.

<table>
<thead>
<tr>
<th></th>
<th>BROCA</th>
<th>WERNICKE</th>
<th>CONDUCTION</th>
<th>ISOLATION</th>
<th>ANOMIC</th>
<th>GLOBAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fluency</td>
<td>↓</td>
<td>OK</td>
<td>↓</td>
<td>↓</td>
<td>OK</td>
<td>↓</td>
</tr>
<tr>
<td>Comprehension</td>
<td>OK</td>
<td>↓</td>
<td>OK</td>
<td>↓</td>
<td>OK</td>
<td>↓</td>
</tr>
<tr>
<td>Repetition</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
<td>OK</td>
<td>↓</td>
</tr>
<tr>
<td>Naming</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
<td>OK</td>
<td>↓</td>
</tr>
<tr>
<td>Reading</td>
<td>↓</td>
<td>↓</td>
<td>OK</td>
<td>↓</td>
<td>OK</td>
<td>↓</td>
</tr>
<tr>
<td>Writing</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
<td>OK</td>
<td>↓</td>
</tr>
<tr>
<td>Lesion location</td>
<td>post inferior frontal lobe</td>
<td>post superior temporal lobe</td>
<td>arcuate fasciculus</td>
<td>border zone</td>
<td>post inferior temporal lobe</td>
<td>large portion of left hemisphere</td>
</tr>
</tbody>
</table>

Aphasia Testing:

Six language functions are routinely tested to evaluate the patient for the presence of aphasia:

- **Fluency**: The amount and ease of speech production.
- **Naming**: The ability to name objects and parts of objects.
- **Comprehension**: The ability to understand simple and complex commands.
- **Repetition**: The ability to repeat a spoken phrase, such as "no ifs, ands or buts about it".
- **Reading**: The ability to read and understand a written sentence.
- **Writing**: The ability to write to dictation.

Agnosia

Agnosia is a defect in recognizing a complex sensory stimulus. Normal primary sensory function is assumed. Agnosias are due to lesions involving "association cortex", primarily located in parietal and temporal lobes in either the dominant or non-dominant hemispheres. Several examples of agnosia include the following:

- **Anosognosia**: Denial of illness.
- **Asomatognosia**: Denial of half of one's body.
- **Prosopagnosia**: Inability to recognize faces.
- **Extinction** to double simultaneous stimulation.
- Geographic disorientation.
Apraxia

Apraxia is a defect in the performance of a complex motor task. Normal primary motor function is assumed. Apraxias are also due to lesions involving "association cortex", primarily in the frontal lobes of the dominant or non-dominant hemispheres. Several examples of apraxia include the following:

- **Ideomotor Apraxia**: Inability to perform motor tasks on command ("Show me how you would salute", etc.).
- **Ideational Apraxia**: Inability to plan a series of complex tasks ("How would you set the table for dinner?")
- **Constructional Apraxia**: Inability to copy complex figures.
- **Dressing Apraxia**: Inability to dress oneself.

**Cognition**

Assessing cognition implies evaluating higher cortical functions. These usually reside in diffuse areas of cortex and subcortical white matter, and damage to large areas of the cerebral hemispheres is required to produce abnormalities in cognition. Five components of cognition that can easily be tested include the following:

- **Orientation**: To person, place, time and situation.
- **Memory**: Including immediate recall, recent and remote memory. Typically, memory is assessed by giving the patient a learning trial: the patient is asked to remember 3 objects, and after five minutes of distraction, is asked to recall the objects.
- **Intellect**: This can be assessed by asking the patient to perform simple calculations, such as serial 7's (subtracting seven serially from 100), or by asking the patient to recall historical facts, such as the recent presidents or current world events. Asking the patient to spell a five-letter word forwards and backwards is another test of intellect.
- **Abstraction**: This can be assessed by asking the patient to interpret a simple proverb. Alternatively, the patient can be asked similarities. ("How are an apple and orange alike?")
- **Judgment**: This can be assessed by describing an ambiguous situation to the patient and asking for an appropriate response. ("What would you do if you found a stamped, addressed envelope on a sidewalk?")
Mood and Affect

*Mood* refers to how the patient feels; *affect* refers to how the patient comes across to others. Both of these should be carefully assessed. The patient should be asked specifically about depression and manic behavior.

Thought Content

Abnormal thought content should be noted, including hallucinations, paranoid behavior, loss of reality testing, and evidence for psychosis. Abnormal thought content is seen with delirium or with schizophrenia.
SKULL, SPINE AND MENINGES

The skull, spine and meninges are the protective covering of the central nervous system. Lesions affecting any of these structures are often associated with neurologic signs and symptoms. Hence, detailed evaluation of these structures should be part of every neurologic examination.

Skull

The skull is palpated to the detect defects secondary to trauma or surgery. It is important to palpate for burr holes, since these frequently indicate surgery for previous subdural or epidural hematomas. Inspection for hematomas, particularly below the eyes (raccoon eyes) and behind the ears (battle sign), is also important, since these hematomas frequently signify the presence of a basilar skull fracture. CSF otorrhea or rhinorrhea imply leakage of spinal fluid into the auditory canals and nasal cavities, respectively, and are also sequelae of skull fractures. The skull should also be auscultated for bruits over the orbits, mastoid processes, and temporal bones. Bruits in these areas are highly suggestive of arteriovenous malformations.

Spine

The spine is inspected for scoliosis, which may indicate an underlying weakness of paraspinal muscles. Palpation of the spine is performed to detect any tenderness. Range-of-motion in the six cardinal directions is evaluated in the cervical and lumbar regions. Limitations in cervical or lumbar range-of-motion may reflect osteoarthritis, increased muscle tone due to paratonic muscle rigidity (see below), or meningismus that reflects inflammation of the meninges (see below).

Straight Leg Raising Test

This test allows one to evaluate for lower lumbar or sacral nerve root irritation, as can occur with herniated lumbar disks. To perform this test, the patient lies supine and the thigh is flexed at the hip, with the leg extended at the knee, and the patient is observed for the development of lumbar pain that radiates down the involved leg in a dermatomal pattern (sciatica). This maneuver stretches the sciatic nerve, including all of the nerve roots that constitute this nerve (L4-S2). Hence, a positive straight leg-raising test implies compression or irritation of any of these nerve roots. Dorsiflexion of the foot, while the thigh is flexed and lower leg extended, increases the amount of stretch on the sciatic nerve, and hence may increase the pain felt by the patient.

Meninges

The meninges completely encircle the central nervous system and protect it from infection and other injury. Meningeal inflammation can be seen with infection (meningitis) or with a subarachnoid hemorrhage due to a ruptured saccular aneurysm. Meningeal inflammation is manifested as severe neck pain that is made profoundly worse with neck flexion (meningismus).

The Brudzinski sign (spontaneous flexion of the legs at the hips and knees following neck flexion) and the Kernig sign (resistance to knee extension when the hips are flexed)
are two other signs indicative of meningeal inflammation, and are often helpful in evaluating for meningismus. As noted above, neck stiffness due to meningeal inflammation should be differentiated from limited neck range of motion in all directions which can be seen with degenerative arthritis of the cervical spine or with increased muscular tone as is seen with paratonic muscle rigidity. This distinction is not difficult to make, since meningeal inflammation primarily limits neck range of motion with flexion only.
CRANIAL NERVES

The cranial nerve examination allows one to examine the brain stem. Recall that cranial nerves III through XII exit the CNS at all three levels of the brain stem: midbrain (CN III and IV); pons (CN V - VIII); and medulla (CN IX - XII). The twelve cranial nerves are usually evaluated sequentially.

Olfactory Nerve

This nerve is tested by occluding one nostril and presenting a non-volatile stimulus (e.g. spices, coffee) to the other nostril. This is then repeated on the opposite side. Smell should always be evaluated after head trauma, because the olfactory nerve may be sheared off as it penetrates the cribiform plate. Basal meningiomas also cause neurologic loss of smell by invading the cribiform plate. Remember that the most common cause of loss of smell is non-neurologic, and is due to inflammation of the nasal mucosa as seen with upper respiratory infections.

Optic Nerve

Three components of the optic nerve are typically evaluated: visual acuity, visual fields and the funduscopic examination.

Visual Acuity

For neurologic purposes, corrected visual acuity is tested (with eyeglasses or contact lenses). Each eye is checked individually. Distance vision is checked by means of the Snellen chart, and near vision is tested by means of the Jeager chart. Visual acuity is a reflection of the integrity of the entire visual system, including the refractive components (cornea, lens, vitreous humor, retina, optic nerve, optic chiasm, optic tract, lateral geniculate nucleus, optic radiations and the occipital cortex). It is important to remember that visual acuity evaluates only macular vision, which is the central 5° of the visual field.

Visual Fields

These are evaluated by the confrontation method. In this method, the examiner stands directly in front of the patient, usually 2-3 feet away. The patient closes one eye and looks at the examiner’s nose with the other eye. The examiner does the same. A target (usually the examiner’s finger) is then introduced from the periphery of each visual quadrant and the visual field is assessed in this quadrant, using the examiner’s visual field as the control. Each eye is checked individually.

To evaluate for visual neglect, the patient keeps both eyes open and looks at the examiner’s nose. The examiner then presents bilateral simultaneous stimuli and the patient is asked to localize the stimuli. Visual neglect often implies parietal lobe lesions.

Various visual field defects can be seen, depending on the location of the lesion within the visual pathway. These are listed in figure 1.
**Figure 1**
Visual Field Defects

<table>
<thead>
<tr>
<th>Structure</th>
<th>Lesion of Right Side Produces</th>
</tr>
</thead>
<tbody>
<tr>
<td>Retina</td>
<td>L and R Fields</td>
</tr>
<tr>
<td>Nerve</td>
<td></td>
</tr>
<tr>
<td>Chiasm</td>
<td></td>
</tr>
<tr>
<td>Tract</td>
<td></td>
</tr>
<tr>
<td>Lateral Geniculate</td>
<td></td>
</tr>
<tr>
<td>Radiations</td>
<td></td>
</tr>
<tr>
<td>Cortex</td>
<td></td>
</tr>
</tbody>
</table>

(hatched area is defective)

<table>
<thead>
<tr>
<th>Lesion</th>
<th>Most Common Etiology</th>
<th>L and R Fields</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Optic nerve head</td>
<td>Early papilledema</td>
<td>○○</td>
<td>enlarged blind spot</td>
</tr>
<tr>
<td></td>
<td>Late papilledema</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Retrobulbar neuritis</td>
<td>Multiple sclerosis</td>
<td>○○</td>
<td>central scotoma</td>
</tr>
<tr>
<td>Optic neuritis</td>
<td>Neuromyelitis optica</td>
<td>○○</td>
<td>cecocentral scotoma</td>
</tr>
<tr>
<td>Mid-chiasm</td>
<td>Pituitary tumor</td>
<td>○○</td>
<td>bitemporal hemianopia</td>
</tr>
</tbody>
</table>
Funduscopic Examination

The fundus is evaluated with the ophthalmoscope. The optic disk, surrounding retina, blood vessels and macula can be visualized. In addition, by changing the plane of focus on the ophthalmoscope, one can visualize the cornea and lens. With this technique, one can see evidence for optic disk swelling (papilledema), optic disk atrophy, retinal hemorrhages, retinal vascular changes of hypertension and diabetes, as well as corneal scarring and cataracts. The ophthalmoscopic examination is difficult and requires many years of practice, but, once mastered, can provide a great deal of information about the central nervous system.

Oculomotor, Trochlear, and Abducens Nerves

These nerves are examined together since they have similar functions. There are three parts to the examination of these nerves: pupillary light response, ocular movements and ptosis.

Pupillary Light Response

Pupillary size depends on the balance between the parasympathetic nervous system which causes constriction via CN III (figure 2), and the sympathetic nervous system which causes dilatation via the sympathetic pathway originating in the hypothalamus, traversing the brain stem, cervical and upper thoracic spinal cord, and forming the peripheral sympathetic pathway passing through the superior cervical sympathetic ganglion, and traveling along the external carotid artery and with the ophthalmic division of the trigeminal nerve (figure 2).

![Diagram of oculomotor, trochlear, and abducens nerves](image)

**Figure 7.** A. The parasympathetic pupilloconstrictor pathway. B. The sympathetic pupillodilator pathway.
Pupillary size is first observed and measured in dim light: small pupils are termed miotic, large pupils are termed mydriatic, and unequal pupils are termed anisocoric.

The pupillary light response is next tested. To test this reflex, a bright light is shone on each eye individually, and the pupils are examined for direct and consensual pupillary constriction. The afferent information for this reflex is carried by the optic nerve (CN II) and the efferent response is carried by the oculomotor nerve (CN III).

In addition to constricting to light, pupils also constrict when shifting from far to near gaze, and this response is known as accommodation. The stimulus for this response originates in the optic pretectum.

Specific pupillary lesions are listed in table 2.

<table>
<thead>
<tr>
<th>TYPE</th>
<th>CLINICAL FINDINGS</th>
<th>ANISOCORIA</th>
<th>LESION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Marcus-Gunn pupil</td>
<td>A deafferented pupil which constricts to consensual but not to direct light</td>
<td>Absent</td>
<td>CN II</td>
</tr>
<tr>
<td>Hutchinson pupil</td>
<td>A dilated pupil that does not respond to direct or consensual light</td>
<td>Present</td>
<td>CN III</td>
</tr>
<tr>
<td>Horner's syndrome</td>
<td>A small pupil with associated ipsilateral ptosis and decreased facial sweating</td>
<td>Present</td>
<td>Sympathetics</td>
</tr>
<tr>
<td>Adie's tonic pupil</td>
<td>A dilated pupil with an impaired light response and slow constriction to near vision</td>
<td>Present</td>
<td>Parasympathetics</td>
</tr>
<tr>
<td>Argyll Robertson pupil</td>
<td>A small, irregular pupil that constricts to near vision but not to light</td>
<td>Absent</td>
<td>Pretectum</td>
</tr>
</tbody>
</table>

Ocular Movements

Voluntary and reflex eye movements are coordinated by the cortical connections (frontal eye fields and occipital cortex), vestibular apparatus, medial longitudinal fasciculus and CNs III, IV and VI.

Eye movements may be dysconjugate or conjugate. Convergence is a normal dysconjugate eye movement that is part of the near response.

There are three types of conjugate eye movements. Two of these fixate the image on the retina (one of these with respect to head and neck motion, and the other with respect to image motion), and one redirects the line of sight. These eye movements are detailed below.
Ocular movements that fixate the image on the retina:

1. **Vestibulo-Ocular Reflex (VOR) (oculocephalic reflex)** ("doll’s eyes" reflex): This reflex fixates the image on the retina with respect to head and neck motion. Head rotation is a form of angular acceleration that stimulates the semicircular canals in the inner ear. These canals sense and convert this angular acceleration into electrical impulses that then are conveyed to the four vestibular nuclei in the brain stem via CN VIII. The vestibular nuclei subsequently project to CNs III, IV, and VI via the medial longitudinal fasciculus (MLF), maintaining a stable visual field despite head motion.

2. **Visual Pursuit**: This reflex fixates the image on the retina with respect to image motion. Image motion is sensed by the occipital cortex that then relays this information in a crossed fashion to the lateral gaze center in the pons (paramedian pontine reticular formation [PPRF]), and then via the MLF to CNs III, IV, and VI.

Ocular movements that re-direct the line-of-sight:

3. **Visual Saccade**: The stimulus for this ocular movement originates in the frontal eye fields located in the frontal lobes of the cerebral hemispheres. The information then travels in a crossed fashion to the lateral gaze center in the pons (PPRF), and then is relayed via the MLF to CNs III, IV, and VI, in an analogous fashion as for the other two eye movements noted above (figure 3).
Figure 3

Schematic Diagram for Mechanisms of Horizontal Conjugate Gaze

Frontal Lobe (saccades)

Occipital Lobe (pursuit)

M.L.F.

VI

PPRF

Cerebral Hemispheres

Occipital Lobe (pursuit)

Midbrain

Pons
In an awake individual, eye movements are assessed by having the patient look in the six primary directions of gaze. These directions correspond to the directions of action of the extra-ocular muscles, and are detailed in figure 4.

**Figure 4**

**Principal Directions of Action of Extraocular Muscles**

In a comatose individual, eye movements can be evaluated by means of the oculocephalic (Doll's eyes) reflex and by means of caloric testing. These are detailed in figure 5.
Figure 5

<table>
<thead>
<tr>
<th>CONDITION:</th>
<th>OCULAR REFLEXES IN UNCONSCIOUS PATIENTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brainstem Intact</td>
<td><img src="image" alt="Diagram of ocular reflexes" /></td>
</tr>
<tr>
<td>MLF (bilateral)</td>
<td><img src="image" alt="Diagram of ocular reflexes" /></td>
</tr>
<tr>
<td>Low Brainstem Lesion</td>
<td><img src="image" alt="Diagram of ocular reflexes" /></td>
</tr>
</tbody>
</table>

Figure 12. Ocular reflexes in unconscious patients. The upper section illustrates the oculocephalic (above) and oculovestibular (below) reflexes in an unconscious patient whose brainstem ocular pathways (see Fig. 11) are intact. Horizontal eye movements are illustrated on the left, and vertical eye movements on the right. Lateral conjugate eye movements (upper left) to head turning are full and opposite in direction to the movement of the face. A stronger stimulus to lateral deviation is achieved by douching cold water against the tympanic membrane(s). There is tonic conjugate deviation of both eyes toward the stimulus; the eyes usually remain tonically deviated for 1 or more minutes before slowly returning to the midline. Because the patient is unconscious, there is no nystagmus. Extension of the neck in a patient with an intact brainstem produces conjugate deviation of the eyes in the downward direction, and flexion of the neck produces deviation of the eyes upward. Bilateral cold water against the tympanic membrane likewise produces conjugate downward deviation of the eyes, whereas hot water (no warmer than 44°C) causes conjugate upward deviation of the eyes.

In the middle portion of the drawing, the effects of bilateral medial longitudinal fasciculus lesions on oculocephalic and oculovestibular reflexes are shown. The left portion of the drawing illustrates that oculocephalic and oculovestibular stimulation deviates the appropriate eye laterally and brings the eye, which would normally deviate medially, only to the midline, since the medial longitudinal fasciculus, with its connections between the abducens and oculomotor nuclei, is interrupted. Vertical eye movements often remain intact. The lower portion of the drawing illustrates the effects of a low brainstem lesion. On the left, neither oculovestibular nor oculocephalic movements cause lateral deviation of the eyes because the pathways are interrupted between the vestibular nucleus and the abducens area. Likewise, in the right portion of the drawing, neither oculovestibular nor oculocephalic stimulation causes vertical deviation of the eyes. On rare occasions, particularly with low lateral brainstem lesions, oculocephalic responses may be intact even when oculovestibular reflexes are abolished (see Patient 1–3).
One also observes the patient for nystagmus. **Nystagmus** is a rhythmic, oscillatory involuntary eye movement of one or both eyes that may occur spontaneously or be evoked by a specific direction of gaze. Nystagmus may be horizontal, vertical or rotatory, and typically has a fast and a slow component. By definition, the direction of nystagmus is the direction of the fast component. Nystagmus may be physiologic or pathologic, as noted in table 3.

**TABLE 3**  
**CAUSES OF NYSTAGMUS**

<table>
<thead>
<tr>
<th>PHYSIOLOGIC</th>
<th>PATHOLOGIC</th>
</tr>
</thead>
<tbody>
<tr>
<td>End-gaze</td>
<td>Vestibular lesions</td>
</tr>
<tr>
<td>Heights</td>
<td>Cerebellar lesions</td>
</tr>
<tr>
<td>Optokinetic</td>
<td>Brain stem lesions (INO)</td>
</tr>
<tr>
<td></td>
<td>Drugs</td>
</tr>
<tr>
<td></td>
<td>Congenital</td>
</tr>
</tbody>
</table>

**Gaze palsies** producing dysconjugate gaze can be due to lesions involving: the extraocular muscles as can be seen with hyperthyroidism; the neuromuscular junction as can be seen with myasthenia gravis; individual lesions affecting CNs III, IV or VI; or a lesion involving the MLF producing an internuclear ophthalmoplegia (INO). INOs are frequently seen with multiple sclerosis in younger individuals, and with small brain stem strokes in older individuals.

A **gaze preference** is a conjugate paresis of gaze that is frequently seen following lesions to the frontal eye fields following a large hemispheric stroke.

**Ptosis**

Ptosis refers to drooping of the eyelid. Two separate muscles, innervated by two different nerves, elevate the eyelid:

The **levator palpebrae muscle** is a skeletal muscle innervated by CN III and is responsible for opening the eyes. The **superior tarsal muscle** is a smooth muscle innervated by the sympathetic nervous system and also helps elevate the eyelid. A lesion to either of these nerves can produce ptosis, although the ptosis due to a lesion of CN III is more pronounced.

These two forms of ptosis are usually associated with anisocoria, and the proper diagnosis can be made by noting the pupillary size on the side of the ptotic eyelid. A sympathetic lesion causing ptosis has an accompanying small pupil (Horner's syndrome), while a CN III lesion causing ptosis has an accompanying large pupil.
Trigeminal Nerve

The trigeminal nerve has both sensory and motor function.

Trigeminal Sensory Function

The sensory component of the trigeminal nerve relays sensory information from the face via three divisions, the ophthalmic, maxillary and mandibular divisions. Testing of this component is accomplished by touching all three divisions of the face with cotton or a pin.

The Corneal Reflex

The corneal reflex is an excellent way to test the sensory component of the trigeminal nerve, as well as the facial nerve. To test this reflex, the cornea is touched with a wisp of cotton and the resultant direct and consensual eye blink is noted. The afferent information for this reflex is carried by the ophthalmic division of the trigeminal nerve, and the efferent information is carried by the facial nerve.

Trigeminal Motor Function

Trigeminal motor function includes the muscles of mastication: the temporalis, masseter, and lateral and medial pterygoid muscles. These are innervated by the mandibular division of the trigeminal nerve and can be tested by having the patient clench the jaw tightly or deviate the jaw from side to side against resistance.

Facial Nerve

Motor component

This nerve supplies motor innervation to the face and has numerous divisions. To test this nerve, facial symmetry is observed at rest. The patient is then asked to wrinkle the brow, close the eyes firmly, smile and frown. Facial weakness can be due to both lower motor neuron or upper motor neuron lesions. With a lower motor neuron lesion of the facial nerve, ipsilateral weakness of the entire half of the face results. Bell’s palsy is an example of lower motor neuron facial weakness.

Facial weakness can also be seen with an upper motor neuron lesion involving the motor cortex or the corticobulbar tract. In this case, the weakness is contralateral to the lesion and involves only the lower half of the face. This pattern of weakness is seen with upper motor neuron lesions because the upper face receives bilateral cortical innervation and is therefore unaffected in unilateral upper motor neuron lesions.

Taste component

The facial nerve supplies taste sensation to the anterior 2/3 of the tongue via the chorda tympani nerve. Taste can be checked by applying sugar or salt solutions to the anterior tongue with a cotton applicator.
Vestibulo-Cochlear Nerve

This nerve has two divisions, each of which carries vestibular or auditory information respectively.

**Cochlear Division**

This can be tested rather crudely by assessing the patient's ability to hear a ticking watch or rubbing fingers held a certain distance away from the ear. The examiner is the control. This form of testing evaluates global auditory function.

Hearing loss is frequently differentiated into **conductive hearing loss** and **sensori-neural hearing loss**. Conductive hearing loss implies a lesion to structures in the outer or middle ear that convert air conduction into bone conduction. Bone conduction is perceived as louder than air conduction in this form of hearing loss.

Sensori-neural hearing loss is due to a lesion involving the inner ear (cochlear apparatus) or the eighth cranial nerve. Both air and bone conduction are reduced in this form of hearing loss. Sensori-neural hearing loss is sometimes further subdivided into cochlear and retro-cochlear. **Cochlear hearing loss** results from destructive lesions involving the labyrinth, such as Meniere's disease, occupational noise, certain ototoxins and certain infections including syphilis. **Retro-cochlear hearing loss** is usually due to a tumor invading the eighth cranial nerve (acoustic Schwannoma).

The **Weber test** and **Rinne test** are two tests of hearing that help differentiate conductive from sensori-neural hearing loss.

In the **Rinne test**, the base of a vibrating tuning fork (512 Hz) is placed against the mastoid process until the sound is no longer heard. The tines of the tuning fork are then moved adjacent to the external ear where sound should still be appreciated in normal individuals, since air conduction is normally better than bone conduction. If the sound is no longer heard in this second position a conductive hearing loss is suspected.

In the **Weber test**, a vibrating tuning fork (512 Hz) is placed at the vertex of the skull and the patient is asked to localize the sound. Normally the sound should be heard equally in both ears. Lateralization of the sound to one ear is abnormal, with the sound localizing to the "bad ear" in a conductive hearing loss and to the "good ear" in a sensori-neural hearing loss.

The significance of abnormalities in Weber and Rinne testing are listed in table 4.
TABLE 4
CLINICAL TESTS OF AIR CONDUCTION (AC) AND BONE CONDUCTION (BC)

<table>
<thead>
<tr>
<th>TEST</th>
<th>NORMAL RESPONSE</th>
<th>CONDUCTIVE HEARING LOSS</th>
<th>SENSORI-NEURAL HEARING LOSS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rinne</td>
<td>AC &gt; BC</td>
<td>BC &gt; AC</td>
<td>AC &gt; BC but both</td>
</tr>
<tr>
<td>Weber</td>
<td>No lateralization</td>
<td>Lateralized to defective ear</td>
<td>Lateralizes to normal ear</td>
</tr>
</tbody>
</table>

AC  Air conduction
BC  Bone conduction

Vestibular Division

This portion of CN VIII can be evaluated by observing for nystagmus at rest, as well as following labyrinthine stimulation. Labyrinthine stimulation can be performed by means of the Nylen-Barany (Dix-Hallpike) positioning maneuver. In this test, the patient is quickly moved from the sitting position to a supine position with the head positioned 45° below the plane of the table and turned to one side (figure 6).

![Figure 6](image)

The Nylen-Barany maneuver for positional vertigo and nystagmus. The patient is moved abruptly from a seated [A] to a prone [B] position, with his head hanging 45° below the horizontal and rotated 45° to one side. He is observed for the development of nystagmus and vertigo.

This position is maintained for about one minute, during which time the patient is observed for nystagmus. The test is then repeated with the head turned to the other side. If the patient reports vertigo during the maneuver, or if nystagmus develops, vestibular dysfunction may be present.
Caloric testing is an alternate method for stimulating the labyrinth. In this test, hot or cold water is introduced into the external auditory meatus and the patient is observed for the development of nystagmus. Both ears are irrigated sequentially and the degree of resultant nystagmus following irrigation of either ear is compared.

Nystagmus and vertigo can both be seen following a peripheral lesion involving the vestibular apparatus, or following a central lesion involving the vestibular nuclei in the brain stem. The vertigo and nystagmus that result from either a central or peripheral lesion have different characteristics, and these differences are useful in localizing lesions of the vestibular system. Table 5 lists the different responses seen with central or peripheral lesions for the Nylen-Barany maneuver.

<table>
<thead>
<tr>
<th></th>
<th>PERIPHERAL</th>
<th>CENTRAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Latency period before onset of nystagmus</td>
<td>2-20 sec.</td>
<td>None</td>
</tr>
<tr>
<td>Duration of nystagmus</td>
<td>&lt;1 min.</td>
<td>&gt;1 min.</td>
</tr>
<tr>
<td>Fatigability</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Direction of nystagmus in one head position</td>
<td>Unidirectional</td>
<td>May change direction</td>
</tr>
<tr>
<td>Vertigo intensity</td>
<td>Severe</td>
<td>Slight</td>
</tr>
<tr>
<td>Head positions eliciting vertigo</td>
<td>Single position</td>
<td>More than one position</td>
</tr>
<tr>
<td>Caloric and rotary tests</td>
<td>Vestibular paresis</td>
<td>Hyperactive responses, impaired fixation, suppression</td>
</tr>
</tbody>
</table>

Glossopharyngeal and Vagus Nerves

These nerves are usually tested together since they have overlapping functions. The glossopharyngeal nerve primarily carries sensation from the posterior pharynx and the larynx. The vagus nerve supplies motor innervation to the soft palate, pharyngeal muscles and the vocal cords. The vagus nerve is easily tested by asking the patient to phonate and observing for a symmetric rise in the palate and uvula.

Another way to test both of these nerves is to elicit the gag reflex. In this test, the examiner touches the posterior pharyngeal wall with a tongue blade and observes for a symmetric rise in the palate and uvula. The afferent arm of this reflex is carried by the glossopharyngeal nerve and the efferent arm by the vagus nerve.

Spinal Accessory Nerve

The primary function of this nerve is to supply motor innervation to the sternocleidomastoid (SCM) muscle and the upper third of the trapezius muscle. Sternocleidomastoid function is assessed by asking the patient to rotate the head against resistance. Recall that contraction of the right SCM muscle allows one to turn the head to the left. Trapezius function is assessed by shoulder shrug.
Hypoglossal Nerve

This nerve supplies motor innervation to the tongue, and is evaluated by observing the tongue at rest, and by asking the patient to protrude the tongue in the midline or to apply lateral pressure against each cheek. A lesion of the hypoglossal nerve will eventually cause atrophy of the ipsilateral half of the tongue. Recall that the tongue will also deviate towards the side of the lesioned nerve when protruded.

Cranial Nerve Reflexes

Practically the entire brain stem can be evaluated by means of five cranial nerve reflexes. This is extremely useful in evaluating the cause of coma in an unresponsive patient. These five reflexes are detailed in table 6.

<table>
<thead>
<tr>
<th>REFLEX</th>
<th>AFFERENT NERVE</th>
<th>EFFERENT NERVE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pupillary *</td>
<td>II</td>
<td>III</td>
</tr>
<tr>
<td>Jaw Jerk *</td>
<td>V</td>
<td>V</td>
</tr>
<tr>
<td>Corneal *</td>
<td>V</td>
<td>VII</td>
</tr>
<tr>
<td>Gag *</td>
<td>IX</td>
<td>X</td>
</tr>
<tr>
<td>Vestibulo-ocular</td>
<td>VIII</td>
<td>III, IV, VI (via MLF)</td>
</tr>
</tbody>
</table>

* has direct and consensual response
THE MOTOR EXAMINATION

When performing the motor examination on a patient presenting with weakness, it is important to remember that weakness could be a result of a lesion at any point in the neuraxis: cerebral hemispheres, brain stem, spinal cord, anterior horn cell, nerve root (myotome), peripheral nerve, neuromuscular junction, or muscle. Another important distinction in the evaluation of weakness is whether the weakness has a characteristic "upper motor neuron pattern" or "lower motor neuron pattern". These differences will be detailed below.

The motor examination consists of several parts: assessment of muscle bulk, evaluation of muscle tone, observation for spontaneous movements, and functional and formal muscle strength testing.

Muscle Bulk

In general, muscle bulk should be symmetric throughout the limbs, when comparing the right and left sides, and proximal and distal portions of the extremities. Loss of muscle bulk is known as atrophy, and is seen in two pathologic settings:

- **Denervation atrophy**: A profound form of muscle atrophy that is seen with lower motor neuron lesions.
- **Disuse atrophy**: A mild form of muscle atrophy that can be seen in a variety of clinical settings, including upper motor neuron disease, disuse, corticosteroid use, collagen-vascular disorders, and with musculoskeletal problems.

Spontaneous Movements

Several different types of abnormal spontaneous movements can be seen as follows:

- **Fasciculations**: Worm-like contractions of muscle due to random discharge of an entire motor unit. Although frequent fasciculations are seen with anterior horn cell disorders (i.e. amyotrophic lateral sclerosis), occasional fasciculations are commonly seen with simple muscle fatigue following exercise and are of no clinical consequence.

- **Myoclonus**: Sudden contractions of a muscle or group of muscles that move an entire limb across a joint. Myoclonus is frequently seen with metabolic or hereditary neurologic disorders.

- **Chorea and Athetosis**: Brief, irregular, asymmetric writhing movements of basal ganglia origin. Chorea is a quick, distal dance-like movement and athetosis is a more proximal slower movement.

- **Tremor**: A rhythmic, oscillatory movement of the trunk or limbs due to numerous causes, including lesions of the cerebellum, motor system, sensory system or the basal ganglia. Tremor is frequently differentiated into resting tremor and action tremor. Resting tremor is one of the hallmarks of Parkinson's disease. Action tremor
can be seen with lesions of the cerebellum or the sensory system, and may also be idiopathic (benign familial tremor or senile tremor).

**Muscle Tone**

Muscle tone is defined as the resistance of muscle to passive stretch, and is assessed by moving a relaxed limb passively through an entire range of motion. Tone may be increased or decreased in various pathologic states, and various forms of altered muscle tone are detailed in table 7.

**TABLE 7**
**MUSCLE TONE**

<table>
<thead>
<tr>
<th>TYPE</th>
<th>DESCRIPTION</th>
<th>PATHOLOGY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increased</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Spasticity</td>
<td>Has a catch which varies with position and is velocity dependent</td>
<td>UMN lesion</td>
</tr>
<tr>
<td>Rigidity</td>
<td>Steady resistance to movement at all speeds and positions. Superimposed tremor leads to &quot;cog-wheeling&quot;.</td>
<td>Basal ganglia lesion</td>
</tr>
<tr>
<td>Paratonia</td>
<td>Inability to relax the muscle</td>
<td>Bihemispheric lesions</td>
</tr>
<tr>
<td>Gegenhalten</td>
<td>Opposes examiner</td>
<td></td>
</tr>
<tr>
<td>Mitgehen</td>
<td>Assists examiner</td>
<td></td>
</tr>
<tr>
<td>Decreased</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Flaccidity</td>
<td>Limp</td>
<td>LMN lesion</td>
</tr>
<tr>
<td></td>
<td>Loss of check</td>
<td>Cerebellar lesion</td>
</tr>
</tbody>
</table>

**Muscle Strength**

Muscle strength is assessed by both functional testing and formal testing.

**Functional Testing**

Functional testing is a very reliable form of testing muscle strength that is easily reproducible and reflects the ability of the patient to perform certain tasks. Functional testing of the upper extremity includes the following: ability to touch the chin to the chest when lying supine; ability to raise the arms above one’s head; ability to blanch the knuckles when making a fist. Functional testing of the lower extremities includes the following: ability to arise from a chair without using one’s hands; ability to arise from a squat; ability to step up on a chair with one leg; ability to walk on one’s toes or heels.
The Pronator Drift

The pronator drift is another very important functional muscle test for the upper extremities. This test is performed by having the patient hold both hands outstretched with the palms up and the eyes closed. The examiner watches for subtle pronation of the arm, which sometimes is accompanied by abduction and internal rotation at the shoulder and flexion at the elbow. Pronation of the arm is a subtle sign that is strongly indicative of upper motor neuron dysfunction.

Formal Testing

Formal muscle strength testing involves grading muscle strength for individual muscle groups on a 0-5 scale by means of push/pull testing by the examiner. Typically, a group of muscles is tested together. Representative muscle groups that are often evaluated in a screening examination are listed in table 8.

<table>
<thead>
<tr>
<th>UPPER EXTREMITY MUSCLE GROUPS</th>
<th>LOWER EXTREMITY MUSCLE GROUPS</th>
</tr>
</thead>
<tbody>
<tr>
<td>shoulder abduction</td>
<td>hip flexion</td>
</tr>
<tr>
<td>elbow flexion</td>
<td>hip extension</td>
</tr>
<tr>
<td>elbow extension</td>
<td>hip abduction</td>
</tr>
<tr>
<td>wrist flexion</td>
<td>hip adduction</td>
</tr>
<tr>
<td>wrist extension</td>
<td>knee flexion</td>
</tr>
<tr>
<td>finger flexion</td>
<td>knee extension</td>
</tr>
<tr>
<td>finger abduction</td>
<td>ankle plantar flexion</td>
</tr>
<tr>
<td></td>
<td>ankle dorsiflexion</td>
</tr>
</tbody>
</table>

The 0-5 grading scale for muscle strength testing is based on the ability of the muscle group to oppose gravity, and was devised by the medical research council in Great Britain during the polio epidemic. Table 9 defines each of the grades.

<table>
<thead>
<tr>
<th>GRADE</th>
<th>DESCRIPTION</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No movement</td>
</tr>
<tr>
<td>1</td>
<td>Flicker of contraction</td>
</tr>
<tr>
<td>2</td>
<td>Full range of motion with gravity eliminated</td>
</tr>
<tr>
<td>3</td>
<td>Full range of motion against gravity</td>
</tr>
<tr>
<td>4</td>
<td>Full range of motion against gravity and offers some resistance</td>
</tr>
<tr>
<td>5</td>
<td>Full power</td>
</tr>
</tbody>
</table>
THE SENSORY SYSTEM

When evaluating a patient with sensory dysfunction, it is important to keep in mind all of the levels of the nervous system at which a lesion can produce sensory dysfunction: peripheral nerve, brachial or lumbo-sacral plexus, nerve root (dermatome), spinal cord, brain stem, thalamus, or sensory cortex.

The sensory examination is largely a subjective examination that requires an alert, cooperative patient who can give reliable subjective impressions of various stimuli. In general, sensory symptoms precede sensory signs, and the sensory examination may not be revealing early on in the course of an illness that produces sensory dysfunction.

When performing the sensory examination, one looks for asymmetries. In general, the examiner looks for a proximal-to-distal gradient, or for findings in the distribution of a specific nerve or nerve root.

The sensory examination is divided into three parts: primary modalities, cortico-sensory modalities, and functional testing (the Romberg test).

Primary Modalities

Protopathic sensation

Examples of protopathic sensation include poorly localized touch, pain and temperature perception. These modalities are carried by small, unmyelinated fibers, travel contralaterally in the lateral spinothalamic tracts in the spinal cord, and are ultimately processed in the brain stem reticular formation and in the thalamus.

- **Pain:** This is typically assessed with a pin. The "prickly" sensation may be reported by the patient as diminished, absent or heightened in the affected areas.

- **Temperature:** This can be assessed with a cool tuning fork, or with test tubes filled with cold or hot water.

Epicritic sensation

Examples of epicritic sensation include fine, discriminative touch, vibration, and proprioception (position sense). These modalities are generally subserved by encapsulated nerve endings and are carried by large, myelinated nerve fibers, ascending ipsilaterally in the dorsal columns in the spinal cord. This information then crosses in the medulla, projects to the thalamus and is ultimately processed in the primary sensory cortex.

- **Vibration:** This is assessed with a tuning fork (128 Hz). The vibrating tuning fork is applied over a distal joint, such as the DIP joint of the great toe or of the index finger. The examiner places his/her finger under the patient's joint and the patient is asked to indicate when the stimulus decays. Vibratory loss is present if the examiner still feels the stimulus. If vibratory perception is absent distally, more proximal joints are assessed in a similar fashion.
• **Proprioception**: This is evaluated by assessing position sense at the interphalangeal joints with slight degrees of motion of the joint. The examiner grasps the patient's joint laterally so as not to provide digital pressure cues to the patient.

**Cortico-sensory Modalities**

These are more complex forms of sensation that require significant cortical processing. Four different cortico-sensory modalities are typically evaluated: stereognosis, graphesthesia, two-point discrimination, and double simultaneous stimulation.

• **Stereognosis**: The ability to identify objects by touch alone. To evaluate this modality, objects such as a safety pin or coin are placed in the hand of a patient for identification.

• **Graphesthesia**: The ability to recognize numbers drawn on the palm of the hand.

• **Two Point Discrimination**: The ability to localize and discriminate between two points that are close together. This is typically tested on the tip of the index finger with a paper clip that is bent open. The examiner applies both ends of the clip, keeping them several millimeters apart and moving them closer and closer together. Normal subjects have a detection threshold of 2 mm at the tip of the index finger.

• **Double Simultaneous Stimulation**: A normal subject should be able to localize two stimuli that are applied simultaneously to different parts of the body. Patients with a parietal lobe lesion have a phenomenon known as extinction in which they consistently fail to identify a stimulus on the side of the body contralateral to a parietal lobe lesion, when it is presented simultaneously with a stimulus on the opposite side of the body. In a broad sense, extinction to double simultaneous stimuli is a type of agnosia known as sensory neglect. To test for extinction, the right and left sides of the body are touched at the same time and the patient is asked to localize both stimuli with the eyes closed.

**Functional Testing**

Functional sensory testing is assessed by means of the Romberg test. This test is performed by asking the patient to stand with his/her feet together and then close the eyes. The patient is then observed to see if balance can be maintained with the eyes closed. The Romberg test is reported as positive if the patient falls to one side.

Recall that three systems are routinely used to maintain balance, namely proprioception, the vestibular apparatus and vision. Only two of these systems are required at any one time. Eye closure removes visual cues for maintaining balance. If balance is maintained with the eyes closed, this implies integrity of both the vestibular apparatus and proprioception. Falling to one side implies dysfunction of one of these balance systems.

The Romberg test can only be performed if the patient is able to stand well with feet together and eyes open. If the patient cannot do this well, a lesion of the cerebellum is suspected; the Romberg test cannot be performed under these circumstances.
COORDINATION

Coordination is an integral function of the motor, sensory and cerebellar systems. Tests of coordination typically assess cerebellar function, but the contributions of the other systems, including the motor, sensory and vestibular systems, must be considered when interpreting these tests.

Coordination testing is usually divided into two parts: truncal stability and limb coordination. The ability to check movements and vestibular coordination are also assessed if the clinical situation warrants.

Truncal Stability

Truncal stability is assessed by observing the patient's balance when sitting or standing with feet together and eyes open. Truncal ataxia suggests a lesion involving the midline cerebellar vermis.

Limb Coordination

Limb coordination may be assessed in both the arms and legs:

- **Finger-to-Nose Test**: The patient is asked to touch his/her nose with the index finger, then the examiner's finger, and then his/her nose again. Speed, accuracy and any tremor are noted.

- **Heel-to-Shin Test**: The heel of one leg is run smoothly down the other shin, and speed, accuracy and any tremor are noted.

Rapid Alternating Movements (Diadochokinesia):

The patient is asked to alternately slap the thigh with the front and back of the hand, or to touch each finger to the thumb. Each side is tested separately and compared with the other. Foot tapping is a rapid alternating movement frequently evaluated in the lower extremity.

Ataxia and dysmetria are general terms used to describe unevenness in the performance of any of the above tests, and are frequently due to lesions involving the cerebellar hemispheres. Recall that cerebellar lesions produce ataxia on the side ipsilateral to the lesion. Upper motor neuron lesions or sensory lesions that result in altered proprioception can also result in ataxia and dysmetria.

Ability to Check Movements

Ability to check movements is evaluated by asking the patient to maintain flexion of his/her arm at the elbow against resistance provided by the examiner. The examiner then abruptly lets go of the patient's arm and observes the ability of the patient to "check" or break the flexion movement. An inability to check movements can be seen with lesions of the ipsilateral cerebellar hemisphere, as well as with severe sensory disturbances causing altered proprioception.
Vestibular Coordination

Past pointing and compass turning are two tests that evaluate the integrity of the vestibular system.

- **Past Pointing**: To perform this test, the patient is asked to repeatedly elevate his/her arm vertically and then return to the horizontal such that the index finger touches the examiner's finger that is held directly in front of the patient. This is performed with the eyes open initially, and closed later on. A drift of the patient's arm in one direction is strongly suggestive of a lesion involving the ipsilateral vestibular apparatus.

**Compass Turning**: The patient is asked to march in place with the eyes closed. Rotation of the body in one direction is suggestive of ipsilateral vestibular pathology.
REFLEXES

Evaluation of reflexes is perhaps the most objective way to examine the nervous system. Not only are reflexes helpful in evaluating awake individuals, but they also are invaluable in examining comatose patients.

The cranial nerve reflexes have already been summarized in table 6.

Reflexes can be divided into non-pathologic and pathologic. The non-pathologic reflexes include muscle stretch reflexes (deep tendon reflexes) and superficial (cutaneous) reflexes. The pathologic reflexes include the Babinski sign as well as the frontal release signs.

Muscle Stretch Reflexes

Muscle stretch reflexes are monosynaptic spinal cord reflexes that are elicited by striking the muscle tendon with a percussion hammer and evaluating the subsequent contraction of that muscle. Striking the muscle tendon stretches the muscle spindle and this afferent information is carried by the Ia afferent sensory nerve fibers through the dorsal root and dorsal horn of the spinal cord, eventually synapsing on a corresponding anterior horn cell in the ventral horn of the spinal cord. The efferent arm of this reflex originates in the anterior horn cell, exits the spinal cord in the ventral root, and is carried by the alpha motor neuron, eventually synapsing on the same muscle.

Five muscle stretch reflexes are usually elicited in a routine neurologic examination, and these are listed in table 10.

<table>
<thead>
<tr>
<th>MUSCLE STRETCH REFLEX</th>
<th>NERVE ROOTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Biceps</td>
<td>C5-6</td>
</tr>
<tr>
<td>Triceps</td>
<td>C7-8</td>
</tr>
<tr>
<td>Brachioradialis</td>
<td>C5-6</td>
</tr>
<tr>
<td>Knee Jerk</td>
<td>L3-4</td>
</tr>
<tr>
<td>Ankle Jerk</td>
<td>S1-2</td>
</tr>
</tbody>
</table>

Muscle stretch reflexes are usually graded on a 0-4 scale, as shown in table 11.

<table>
<thead>
<tr>
<th>GRADE</th>
<th>DESCRIPTION</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Absent</td>
</tr>
<tr>
<td>1</td>
<td>Hypoactive</td>
</tr>
<tr>
<td>2</td>
<td>Normal</td>
</tr>
<tr>
<td>3</td>
<td>Hyperactive with spread across a joint</td>
</tr>
<tr>
<td>4</td>
<td>Hyperactive with clonus</td>
</tr>
</tbody>
</table>

Clonus is a rhythmic series of involuntary muscle contractions induced by a sudden passive stretch to a muscle, and is indicative of hyperreflexia. Ankle clonus is easy to
obtain in a hyperreflexic patient, and can be elicited by sudden dorsiflexion of the foot. Patellar clonus can be obtained by a quick, downward motion on the patella, holding the knee slightly flexed.

Superficial (Cutaneous) Reflexes

Superficial (cutaneous) reflexes are polysynaptic, nociceptive reflexes that are elicited by stimulating the skin and observing for contraction of the corresponding muscle. Four superficial reflexes can be easily obtained: the abdominal reflexes, the anal wink reflex, the cremasteric reflex and the bulbocavernosus reflex.

- **Abdominal Reflexes** are obtained by stroking the skin lightly on the abdomen from the umbilicus towards any abdominal quadrant and observing for deviation of the umbilicus towards the quadrant that is stroked. The upper quadrants reflect T6-9 innervation and the lower quadrants T10-12 innervation. Abdominal reflexes may be diminished or absent in many circumstances, including a lesion to the corresponding nerve roots or with upper motor neuron lesions. In addition, obesity, previous pregnancy or abdominal surgery can result in a loss of abdominal reflexes.

- **The anal wink reflex** is elicited by stroking the perianal skin and observing for a contraction of the external anal sphincter on the stroked side. Presence of this reflex indicates integrity of the S3-5 nerve roots.

- **The cremasteric reflex** is elicited by stroking the skin on the inner thigh in a male and observing for elevation of the testicle on the stroked side. Presence of this reflex reflects integrity of the L1-2 nerve roots.

- **The bulbocavernosus reflex** is elicited by squeezing the glans penis and observing for contraction of the external anal sphincter. Presence of this reflex indicates integrity of the S3-4 nerve roots.

The Babinski Sign

The Babinski sign is perhaps the most important reflex in neurology and is obtained by stroking the lateral aspect of the sole of the foot with a noxious stimulus, starting at the heel and then crossing the ball of the foot towards the great toe. A normal response consists of flexion of the great toe. An abnormal (positive) response consists of dorsiflexion of the great toe, and occasionally fanning of the other toes. A positive Babinski response is indicative of an upper motor neuron lesion.

Frontal Release Signs

Frontal release signs are reflexes that are present in infancy, lost with maturation of the central nervous system, and regained with advanced age or with diffuse cortical or bihemispheric dysfunction, such as can be seen with Alzheimer's disease, Parkinson's disease or with bihemispheric strokes. Four frontal release signs are commonly tested: snout, palmomental, grasp and glabellar reflexes.
- **Snout Reflex:** This is elicited by repeatedly tapping the upper lip and observing for puckering of the lips. One way of eliciting this reflex is to place a tongue blade lightly over the upper lip and to tap the tongue blade with a percussion hammer.

- **Palmomental Reflex:** This is elicited by scratching the thenar eminence of the palm with a blunt object and observing for an ipsilateral contraction of the mentalis muscle on the chin.

- **Grasp Reflex:** This is obtained by having the examiner stroke the skin of the patient's palm with his/her fingers and observing for a resultant grasping of those fingers by the patient.

- **Glabellar Sign:** This is elicited by tapping the forehead repeatedly between the eyebrows over the glabella and observing for persistent blinking. It is important to note that a normal individual will blink once or twice only with this maneuver.
GAIT AND STATION

Gait

Examination of gait is most important in neurology, since it provides invaluable information concerning integrity of the motor system, sensory system, and cerebellum. Gait is evaluated by observing the patient walk briskly and turn corners. Particular attention is placed on any asymmetry involving a side or one limb, the distance the feet are kept apart (base), the length of stride and associated arm swing.

An important part of the gait examination is to observe a tandem gait, in which the patient is asked to walk heel-to-toe on a line. This narrows the base of the gait and will bring out subtle gait abnormalities that may not be otherwise evident. Inability to perform a tandem gait is frequently associated with altered proprioception or midline cerebellar lesions.

When evaluating gait, the examiner first notes if the gait is symmetric or asymmetric. If symmetric, the examiner then evaluates the base of the gait. Various abnormal gaits, including asymmetric gaits and wide-based and narrow-based symmetric gaits are detailed in table 12.

<table>
<thead>
<tr>
<th>TABLE 12</th>
<th>ABNORMAL GAITS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>GAIT</strong></td>
<td><strong>LESION</strong></td>
</tr>
<tr>
<td>ASYMMETRIC</td>
<td></td>
</tr>
<tr>
<td>Trendelenburg</td>
<td>Hip pathology</td>
</tr>
<tr>
<td>Hemiplegic</td>
<td>Upper motor neuron disorder</td>
</tr>
<tr>
<td>Steppage (foot drop)</td>
<td>Peroneal nerve palsy</td>
</tr>
<tr>
<td>Antalgic</td>
<td>Foot or leg pain</td>
</tr>
<tr>
<td>SYMMETRIC</td>
<td></td>
</tr>
<tr>
<td>Wide Based</td>
<td></td>
</tr>
<tr>
<td>Sensory ataxic (foot slap)</td>
<td>Posterior columns</td>
</tr>
<tr>
<td>Cerebellar ataxic</td>
<td>Cerebellum</td>
</tr>
<tr>
<td>Apractic (glue-footed)</td>
<td>Bihemispheric lesions</td>
</tr>
<tr>
<td>Narrow Based</td>
<td></td>
</tr>
<tr>
<td>Spastic (scissor)</td>
<td>Bilateral upper motor neuron lesions</td>
</tr>
<tr>
<td>Festinating</td>
<td>Basal ganglia (substantia nigra)</td>
</tr>
</tbody>
</table>
Station

Station is defined as the ability to maintain a stable sitting or standing posture. To evaluate for station, the patient is observed sitting without arm support and with eyes open. To evaluate standing posture, the feet are kept together, the hands are at the sides and the eyes are open. The examiner notes any tendency to lean or fall, as well as the most common direction of instability. Abnormalities in station typically occur with midline cerebellar lesions.

LESION LOCALIZATION

When a patient presents with motor weakness, it is often helpful to determine if the weakness could be due to an upper motor neuron lesion or to a lower motor neuron lesion. Table 13 lists several motor and reflex findings that can help one make such a determination.

<table>
<thead>
<tr>
<th></th>
<th>UMN LESION</th>
<th>LMN LESION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Muscle Bulk</td>
<td>Preserved</td>
<td>Atrophy</td>
</tr>
<tr>
<td>Muscle Tone</td>
<td>Spastic</td>
<td>Flaccid</td>
</tr>
<tr>
<td>Spontaneous Movements</td>
<td>None</td>
<td>Fasciculations</td>
</tr>
<tr>
<td>Reflexes</td>
<td>↑↑</td>
<td>↓↓</td>
</tr>
<tr>
<td>Babinski reflex</td>
<td>Present</td>
<td>Absent</td>
</tr>
<tr>
<td>Examples</td>
<td>Stroke, MS</td>
<td>Neuropathy</td>
</tr>
</tbody>
</table>
APPENDIX I

Outline of the Neurologic Examination
Ralph F. Józefowicz, MD

I. Mental Status
   A. Level of alertness
   B. Aphasia testing
      1. Fluency
      2. Naming
      3. Repetition
      4. Comprehension
      5. (Reading)
      6. (Writing)
   C. Apraxia and Agnosia
   D. Cognition
      1. Orientation
      2. Memory
      3. Intellect
      4. Abstraction
      5. Judgment
   E. Mood and Affect
   F. Thought content

II. Skull, Spine and Meninges
   A. (Skull palpation)
   B. Neck range-of-motion
   C. Back range-of-motion
   D. Meningismus
   E. Cranial and neck bruits

III. Cranial Nerves
   A. Olfactory
   B. Ophthalmic
      1. Visual acuity
      2. Visual fields
      3. Funduscopic examination
   C. Oculomotor, Trochlear, Abducens
      1. Pupils
      2. Ocular movements
      3. Ptosis
   D. Trigeminal
      1. Facial sensory
      2. Motor to muscles of mastication
   E. Facial
      1. Motor
      2. (Taste)
   F. Vestibulo-cochlear
      1. Hearing (256 or 512 Hz)
         a) (Rinne and Weber)
      2. (Balance testing)
   G. Glossopharyngeal, Vagus
      1. Palatal elevation
      2. Gag reflex
   H. Spinal accessory
      1. Sternocephalomastoid
      2. Trapezius
   I. Hypoglossal
      1. Tongue musculature
IV. Motor
   A. Muscle bulk and symmetry
   B. Spontaneous movements
   C. Motor tone
   D. Pronator drift
   E. Strength testing
      1. Functional
         a) Raise arms above head
         b) Arise from a squat
         c) Walk on toes/heels
      2. Formal

<table>
<thead>
<tr>
<th>UPPER EXTREMITY</th>
<th>LOWER EXTREMITY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Shoulder abduction</td>
<td>Hip flexion</td>
</tr>
<tr>
<td>Elbow flexion</td>
<td>Hip extension</td>
</tr>
<tr>
<td>Elbow extension</td>
<td>Hip abduction</td>
</tr>
<tr>
<td>Wrist flexion</td>
<td>Hip adduction</td>
</tr>
<tr>
<td>Wrist extension</td>
<td>Knee flexion</td>
</tr>
<tr>
<td>Finger flexion</td>
<td>Knee extension</td>
</tr>
<tr>
<td>Finger abduction</td>
<td>Ankle dorsiflexion</td>
</tr>
<tr>
<td></td>
<td>Ankle plantar flexion</td>
</tr>
</tbody>
</table>

V. Sensory
   A. Primary modalities
      1. Protopathic: pin, temperature
      2. Epicritic: vibration (128 Hz), position sense
   B. Cortico-sensory modalities
      1. Graphesthesia
      2. Stereognosis
      3. Two point discrimination
      4. Extinction to double simultaneous stimulation

VI. Coordination
   A. Truncal stability
   B. Finger-to-nose test
   C. Heel-to-shin test
   D. Rapid alternating movements (diadochokinesia)

VII. Reflexes
   A. Muscle stretch reflexes
      1. Biceps
      2. Triceps
      3. Brachioradialis
      4. Knee Jerk
      5. Ankle Jerk
   B. Plantar responses
   C. Frontal release signs
      1. Glabellar
      2. Snout
      3. Palmo-mental
      4. Grasp

VIII. Gait and Station
   A. Romberg test
   B. Normal gait
   C. Heel/toe walk
   D. Tandem gait
APPENDIX II
The Mini-Mental State Examination (MMSE)

Orientation

1. What is the
   Year? ______ 1
   Season? ______ 1
   Date? ______ 1
   Day? ______ 1
   Month? ______ 1

2. Where are we?
   State? ______ 1
   County? ______ 1
   Town/City? ______ 1
   Floor? ______ 1
   Address? ______ 1

Registration

3. Name three objects, taking one second to say each. Then ask the patient all three
   after you have said them. Repeat the answers until the patient learns all three. ______ 3

Attention and Calculation

4. Serial sevens. Give one point for each correct answer. Stop after five answers.
   Alternative: spell word backward. ______ 5

Recall

5. Ask for names of three objects learned in Question 3. Give one point for each correct
   answer. ______ 3

Language

6. Point to a pencil and a watch. Have the patient name them as you point. ______ 2

7. Have the patient repeat "No ifs, ands, or buts." ______ 1

8. Have the patient follow a three-stage command: "Take the paper in your right hand.
   Fold the paper in half. Put the paper on the floor." ______ 3

9. Have the patient read and obey the following: "Close your eyes." ______ 1

10. Have the patient write a sentence of his or her own choice. (The sentence should
    contain a subject and an object and should make sense. Ignore spelling errors when
    correcting.) ______ 1

11. Enlarge the design printed below to 1 to 5 cm per side and have the patient copy it.
    (Give one point if all the sides and angles are preserved and if the intersecting sides
    form a quadrangle.) ______ 1

Total ______ 30

Reprinted from Crum RM, Anthony JC, Bassett SS, Folstein MF. Population-based norms for the Mini-Mental State
Examination by age and education level. JAMA. 1993;269:2386-96
PERIPHERAL NEUROANATOMY
Ralph F. Józefowicz, MD

General Principles

When confronted with a patient who may have injury to the peripheral nervous system, one attempts to localize the lesion to a specific level in the PNS: nerve root, plexus or peripheral nerve. A lesion at any one of these levels typically results in both motor and sensory deficits that have a characteristic pattern, as follows:

- **Nerve root lesions** typically result in weakness in a *myotomal* pattern (all muscles innervated by a particular nerve root), and sensory loss in a *dermatomal* pattern (that portion of skin innervated by a particular nerve root).

- **Plexus lesions** typically result in weakness in multiple myotomes, and sensory loss in multiple dermatomes, depending upon which divisions, trunks or cords of the plexus are involved.

- **Peripheral nerve lesions** typically result in weakness in a *peripheral nerve pattern* (all muscles innervated by a particular nerve), and similarly, sensory loss in a *peripheral nerve pattern* (that portion of skin innervated by a particular nerve).

### MUSCLE STRETCH REFLEXES

<table>
<thead>
<tr>
<th>MUSCLE STRETCH REFLEX</th>
<th>NERVE ROOTS</th>
</tr>
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<tbody>
<tr>
<td>Biceps</td>
<td>C5-6</td>
</tr>
<tr>
<td>Triceps</td>
<td>C7-8</td>
</tr>
<tr>
<td>Brachioradialis</td>
<td>C5-6</td>
</tr>
<tr>
<td>Knee Jerk</td>
<td>L3-4</td>
</tr>
<tr>
<td>Ankle Jerk</td>
<td>S1-2</td>
</tr>
</tbody>
</table>

### UPPER EXTREMITY MUSCLE GROUPS

<table>
<thead>
<tr>
<th>Movement</th>
<th>Muscle</th>
<th>Nerve</th>
<th>Roots</th>
</tr>
</thead>
<tbody>
<tr>
<td>Shoulder abduction</td>
<td>Deltoids</td>
<td>Axillary</td>
<td>C5, 6</td>
</tr>
<tr>
<td>Elbow flexion</td>
<td>Biceps</td>
<td>Musculocutaneous</td>
<td>C5, 6</td>
</tr>
<tr>
<td></td>
<td>Brachioradialis</td>
<td>Radial</td>
<td>C6</td>
</tr>
<tr>
<td>Elbow extension</td>
<td>Triceps</td>
<td>Radial</td>
<td>C7, 8</td>
</tr>
<tr>
<td>Wrist flexion</td>
<td>Flexor carpi radialis</td>
<td>Median</td>
<td>C6, 7</td>
</tr>
<tr>
<td></td>
<td>Flexor carpi ulnaris</td>
<td>Ulnar</td>
<td>C7, 8</td>
</tr>
<tr>
<td>Wrist extension</td>
<td>Extensor carpi radialis</td>
<td>Radial</td>
<td>C6, 7</td>
</tr>
<tr>
<td></td>
<td>Extensor carpi ulnaris</td>
<td>Radial</td>
<td>C7, 8</td>
</tr>
<tr>
<td>Finger flexion</td>
<td>Flexor digitorum profundus</td>
<td>Median (digits 1-3)</td>
<td>C8</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ulnar (digits 4-5)</td>
<td>C8</td>
</tr>
<tr>
<td>Finger abduction</td>
<td>Interossei</td>
<td>Ulnar</td>
<td>C8</td>
</tr>
</tbody>
</table>
LOWER EXTREMITY MUSCLE GROUPS

<table>
<thead>
<tr>
<th>Movement</th>
<th>Muscle</th>
<th>Nerve</th>
<th>Roots</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hip flexion</td>
<td>Iliopsoas</td>
<td>Femoral</td>
<td>L1-3</td>
</tr>
<tr>
<td>Hip extension</td>
<td>Gluteus maximus</td>
<td>Sciatic</td>
<td>S1, 2</td>
</tr>
<tr>
<td>Hip abduction</td>
<td>Gluteus medius and minimus</td>
<td>Sciatic</td>
<td>L4, 5</td>
</tr>
<tr>
<td>Hip adduction</td>
<td>Hip adductors</td>
<td>Obturator</td>
<td>L2-4</td>
</tr>
<tr>
<td>Knee flexion</td>
<td>Hamstrings</td>
<td>Sciatic</td>
<td>L5, S1</td>
</tr>
<tr>
<td>Knee extension</td>
<td>Quadriceps femoris</td>
<td>Femoral</td>
<td>L2-4</td>
</tr>
<tr>
<td>Ankle plantar flexion</td>
<td>Gastrocnemius</td>
<td>Tibial</td>
<td>S1, 2</td>
</tr>
<tr>
<td></td>
<td>Soleus</td>
<td>Tibial</td>
<td>S1, 2</td>
</tr>
<tr>
<td>Ankle dorsiflexion</td>
<td>Anterior tibialis</td>
<td>Deep peroneal</td>
<td>L4, 5</td>
</tr>
<tr>
<td>Ankle eversion</td>
<td>Peronei</td>
<td>Superficial peroneal</td>
<td>L5, S1</td>
</tr>
<tr>
<td>Ankle inversion</td>
<td>Posterior tibialis</td>
<td>Tibial</td>
<td>L4, 5</td>
</tr>
</tbody>
</table>

Upper Extremity

Cervical Radiculopathies

Herniated nucleus pulposus: Ruptured cervical discs have the potential for compressing the spinal cord as well as cervical nerve roots, and the signs and symptoms produced by a ruptured cervical disc are dependent upon this fact.

The most common location of a herniated cervical disc is C6-7, with compression of the C7 nerve root. The next most common location is C5-6, with compression of the C6 nerve root. Compression of the C5 and C8 nerve roots is the least common situation.

<table>
<thead>
<tr>
<th>Disc</th>
<th>Root</th>
<th>Pain</th>
<th>Sensory loss</th>
<th>Motor loss</th>
<th>Reflex loss</th>
</tr>
</thead>
<tbody>
<tr>
<td>C4-5</td>
<td>C5</td>
<td>Neck, shoulder, Upper arm</td>
<td>Shoulder</td>
<td>Deltoid, biceps</td>
<td>Biceps</td>
</tr>
<tr>
<td>C5-6</td>
<td>C6</td>
<td>Neck, shoulder, Lateral arm and forearm, Digits 1,2</td>
<td>Lateral arm and forearm, Digits 1,2</td>
<td>Deltoid, biceps</td>
<td>Biceps</td>
</tr>
<tr>
<td>C6-7</td>
<td>C7</td>
<td>Neck, lateral arm and forearm, Digits 2,3</td>
<td>Digit 3</td>
<td>Triceps, radial flexors and extensors</td>
<td>Triceps</td>
</tr>
<tr>
<td>C7-T1</td>
<td>C8</td>
<td>Neck, medial arm and forearm, Digits 4,5</td>
<td>Medial forearm, Digits 4,5</td>
<td>Intrinsic hand muscles</td>
<td>Triceps</td>
</tr>
</tbody>
</table>
Brachial Plexopathy

Upper plexus lesion:
- C5, C6 (Erb’s palsy)
- “Waiter’s tip” posture

Lower plexus lesion:
- C8, T1 (Klumpke’s palsy)
- Birth injury

Idiopathic brachial neuritis (Parsonage-Tuner syndrome)
- Superior trunk (C5, 6, 7)
- Pain in shoulder, axilla
- Weakness and atrophy in shoulder girdle, hands
- Etiology: vaccinations
- Treatment: steroids, physical therapy

Peripheral Nerve Lesions

Axillary nerve
- Motor: Deltoid muscle
- Sensory: Lateral upper arm

Musculocutaneous nerve
- Motor: Biceps muscle
- Sensory: Lateral forearm

Median nerve (Thumb and thenar eminence)

Carpal tunnel syndrome – (Compression at the carpal tunnel at the wrist)
- Clinical features
  - Motor: weakness of the LOAF muscles (lumbricals, opponens, abductor pollicis brevis, flexors of the first three digits)
  - Sensory: digits 1, 2, 3, lateral half of digit 4
  - Pain: wrist, median hand, forearm, shoulder
  - Symptoms worse at night
  - Tinel and Phalen signs
- Etiology: edema, pregnancy, rheumatoid arthritis, tenosynovitis, gout, myxedema, acromegaly, diabetes, amyloid
- Treatment: splints, steroids, surgery
Ulnar nerve (Little finger and hypothenar eminence)

"Tardy" ulnar palsy – Compression at the cubital tunnel at the elbow
• Clinical features:
  • Motor: weakness of the hypothenar and interossei muscles, flexor carpi ulnaris
  • Sensory: digit 5, medial half of digit 4
  • Claw hand deformity
• Etiology: chronic illness, pressure
• Treatment: remove pressure, surgery

Radial nerve (Extensors of the upper limb)

"Saturday night palsy" – Compression at the spiral groove in the forearm
• Clinical features:
  • Motor: wrist drop
  • Sensory: dorsum of hand over the first web space
  • Paradoxically weak grip
• Etiology: pressure
• Treatment: "cock-up" splint, surgery

Lower Extremity

Lumbar Radiculopathies

Herniated nucleus pulposus: Compression of a nerve root by a herniated intervertebral disc (nucleus pulposus) is the most common cause of a lumbar radiculopathy. Middle-aged adults are most commonly affected. Although most ruptured discs occur in the setting of significant back trauma, many cases do not and may follow a trivial activity, such as bending or lifting a light object.

The most common location for a ruptured lumbar intervertebral disc is L5-S1; L4-5 is the next most common location, followed by L3-4. Since the lateral portion of the disc is the most likely segment to rupture, the nerve root exiting below the disc is the one most likely to be compressed, (e.g. a ruptured L5-S1 disc will most likely compress the S1 nerve root).

<table>
<thead>
<tr>
<th>Disc</th>
<th>Root</th>
<th>Pain</th>
<th>Sensory loss</th>
<th>Motor loss</th>
<th>Reflex loss</th>
</tr>
</thead>
<tbody>
<tr>
<td>L3-4</td>
<td>L4</td>
<td>Anterior thigh, Medial calf</td>
<td>Anterior thigh, Medial calf</td>
<td>Quadriceps</td>
<td>Knee jerk</td>
</tr>
<tr>
<td>L4-5</td>
<td>L5</td>
<td>Posterior thigh, Lateral calf, Dorsum of foot</td>
<td>Lateral calf, Great toe</td>
<td>Dorsiflexors and evertors, EHL</td>
<td>None</td>
</tr>
<tr>
<td>L5-S1</td>
<td>S1</td>
<td>Posterior thigh and calf, Sole of foot</td>
<td>Posterior calf, Lateral foot</td>
<td>Plantar flexors and invertors</td>
<td>Ankle jerk</td>
</tr>
</tbody>
</table>
Lumbosacral Plexus

Diabetic amyotrophy
- Clinical features:
  - Seen in patients with diabetes mellitus
  - Pain in the thigh
  - Weakness in thigh and leg muscles
  - Numbness in the thigh and leg
  - Absent knee jerk reflex
- Etiology: infarction of the vasa nervorum supplying the lumbosacral plexus
- Treatment: time

Peripheral Nerve Lesions

Lateral femoral cutaneous nerve

Meralgia paresthetica
- Clinical features:
  - Sensory only – anterolateral thigh
  - Symptoms: pain and paresthesias
- Etiology: constriction at inguinal ligament, obesity, tight belts
- Treatment: remove constriction, amitriptyline

Femoral nerve
- Clinical features:
  - Motor: psoas, quadriceps femoris
  - Sensory: anteromedial thigh; anteromedial leg (saphenous nerve)
- Etiology of injury: hematoma, abscess, tumor, trauma, lymph nodes
- Differential diagnosis: L2, 3, 4 radiculopathy

Obturator nerve
- Clinical features:
  - Motor: hip adductors
  - Sensory: medial thigh
- Etiology of injury: prolonged labor

Sciatic nerve
- Clinical features:
  - Divides into the peroneal and tibial nerves just above the knee
  - Motor: hamstring muscles, all muscles of the leg and foot
  - Sensory: posterolateral leg and the entire foot
- Sciatica: Lower limb pain that radiates in the distribution of the sciatic nerve and usually due to an L4-S2 radiculopathy
- Peroneal compartment usually more involved with injuries
- Rarely injured, due to its deep location in the buttocks and posterior thigh
- Etiology on injury: intramuscular injections, tumors

**Peroneal nerve**

<table>
<thead>
<tr>
<th>Branch</th>
<th>Motor</th>
<th>Sensory</th>
</tr>
</thead>
<tbody>
<tr>
<td>Deep</td>
<td>Ankle dorsiflexors</td>
<td>1st web space</td>
</tr>
<tr>
<td>Superficial</td>
<td>Ankle Evertors</td>
<td>Lateral leg</td>
</tr>
</tbody>
</table>

**Peroneal nerve palsy – (Compression at the fibular head)**
- Clinical features:
  - Motor: foot drop – weakness of ankle dorsiflexion and eversion
  - Sensory: Lateral leg (superficial branch) and 1st web space of the foot (deep branch)
- Etiology: leg crossing, extreme weight loss
- Differential diagnosis: L5 radiculopathy

**Tibial nerve**

<table>
<thead>
<tr>
<th>Branch</th>
<th>Motor</th>
<th>Sensory</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lateral</td>
<td>Abductor digiti quinti</td>
<td>Lateral sole</td>
</tr>
<tr>
<td>Medial</td>
<td>Abductor hallucis</td>
<td>Medial sole</td>
</tr>
<tr>
<td>Calcaneal</td>
<td>(None)</td>
<td>Heel</td>
</tr>
</tbody>
</table>

**Tarsal tunnel syndrome**
- Clinical features:
  - Motor: intrinsic foot muscles
  - Sensory: sole of the foot
  - Pain with standing, worse at night
- Etiology: trauma, tenosynovitis, ganglion
- Treatment: surgery

**Reference**