Dental Neuroanatomy
10:00-12:00 noon, Thursday, February 18\textsuperscript{th}, 2011
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MOTOR PATHWAYS
Upper and Lower Motor Neurons

Objectives:
1. Diagram the corticospinal tract.
2. Recognize the names the corticospinal tract has in different regions of the brain stem, even though the axons are the same.
3. Name the different blood vessels that supply the corticospinal fibers at different levels of the CNS.
4. Locate nuclei with lower motor neurons in the brain stem. Explain how they are controlled.
5. Explain why a lesion in the internal capsule can be more devastating than a lesion of the same size in the cerebral cortex.
6. Compare and contrast the function and dysfunction of upper and lower motor neurons.
7. What happens to a patient when the corticospinal tract is damaged compared with a damaged ventral root?
8. Explain why a paraplegic can still have a patellar reflex.

http://library.med.utah.edu/kw/animations/hyperbrain/pathways/
http://library.med.utah.edu/kw/animations/hyperbrain/facial_muscles/facial_muscles.html
I. LOWER MOTOR NEURONS (MOTONEURON)

A. Skeletomotor neurons = Alpha motor neurons = anterior horn cell = ventral horn cell = lower motor neuron = LMN, the “final common pathway”

1. The cell body of a lower motor neuron is in the CNS … either the ventral horn gray matter of the spinal cord or cranial nerve motor nuclei.

2. The "Final Common Path". All processing and commands arising in the brain must be conveyed to a single target, the large, alpha motor neurons. The efferent limb of reflexes.

3. **Motor Unit.** A single lower motor neuron (alpha) and all of the striated muscle cells innervated by its axon. Variations in force, range and type of movement are determined by the differences in the number and size of the motor units as well as the frequency with which the motor neuron discharges.

4. The axon of the lower motor neuron terminates at the motor end plate = neuromuscular junction.
B. **Fusimotor Neuron - Gamma motor neurons** (fusi=spindle) referring to small muscle fibers located inside a sensory structure called a muscle spindle. The spindle is also a sense organ. Increased gamma motor neuron activity leads to increased contraction of muscle fibers in the spindle.

![Somatotopic Organization of muscle groups in ventral horn.](image)

C. **Somatotopic** organization of motor neurons

1. Spinal Cord
   
   a. The segmental innervation of muscle. Usually 2 (up to 4) cord segments supply a muscle depending on somites (myotomes) of origin.

   b. **Somatic motor cell columns**. The representation of axial (medial) vs. appendicular (lateral) muscles. Clustering of neurons innervating individual muscles are found in both the ventral horn of the spinal cord and in cranial nerve nuclei, e.g. facial nucleus, oculomotor nucleus, ..

2. Cranial nerve motor nuclei are composed of motor neurons too, and they innervate both somatic and branchial motor skeletal muscle under voluntary control (CNN III, IV, V-3, VI, VII, IX, X, XI and XII).
D. **Interneurons** of spinal cord. They are not motoneurons. Their axons remain in the CNS. Some are excitatory, others inhibitory. Most of the descending pathways terminate on dendrites of interneurons, which in turn end on motor neurons. **Only the corticospinal tract has direct access to motor neurons for fine, fast control** (particularly of flexor muscles of the hand).
E. Vascular supply of Spinal Cord: anterior and posterior spinal arteries.

1. **Anterior spinal artery supplies most of cord** except dorsal columns.
   a. **Anterior Spinal Artery**: An unpaired vessel originating from the vertebral artery joined by 4-10 cervical and thoracic arteries and one major lumbar artery, which enter through the intervertebral foramina.
   b. T4-T8 a vulnerable "watershed" region in terms of blood supply to upper and lower part of the cord. The anterior spinal artery receives its principal input from the vertebral - subclavian system in the upper portion and from the descending aorta in the lower portion. Cord can be damaged from hypotension during surgery or due to trauma.

2. **Posterior Spinal Arteries**
   a. Paired and form a plexus.
   b. Supply dorsal columns therefore, occlusion less devastating.

II. CLINICAL FINDINGS IN LOWER MOTOR NEURON DISEASE

Can involve cell body (Central Nervous System) or axon (Peripheral Nervous System) Includes branchial and somatic motor neurons in brain stem nuclei.

A. **Loss of reflexes**, areflexia, and voluntary contraction of the innervated muscle cells.

B. **Flaccid paralysis** -- loss of muscle tone; no resistance to passive movement (hypotonia, atonia, absence of deep tendon reflexes, DTRs).

C. **Muscle atrophy** due to denervation and absence of trophic factors.

D. **Fasciculation** -- fine rhythmic irregular twitching of small groups of denervated muscle fibers (motor units) visible through skin or to touch. Disappears with long-term atrophy.
III. UPPER MOTOR NEURONS (The Corticospinal Tract-Most important motor pathway).

A. Upper motor neurons are often used interchangeably with corticospinal or pyramidal tract. However, these cortical axons usually travel with other descending (reticulospinal) axons. Thus, lesions called upper motor neuron lesions often involve more than one group of axons. We will keep it simple: We will say they come from motor cortex = Corticospinal and Corticobulbar Tracts.

B. Origin. Giant pyramidal cells (30K) in the precentral gyrus (cerebral cortex).

C. Axon termination. Directly on skeletomotor (alpha) and fusimotor (gamma) motor neurons (55% in cervical region, 25% lumbosacral region).

D. Functional Significance
   1. Important in individual finger flexor movements = fractionation of movements.
   2. Important in movements that require speed, agility, adaptability.
   3. Terminate contralaterally on lower motor neurons to distal or appendicular muscles, especially flexors of upper limb.

From The Digital Anatomist Interactive Brain Syllabus. John Sundsten and Kate Mulligan, Univ.Washington School of Medicine. 1998 ©
E. The course of the axon, which forms the tract, is as follows:

1. **Pre-central gyrus** (site of the upper motor neuron cell body)
2. **Internal capsule** (posterior limb, see below)
3. **Cerebral peduncle** (crus cerebri) middle 3/5s
4. **Pons proper or basal pons**
5. **Pyramid** in the medulla
6. **Pyramidal decussation** (how the left brain controls the right body)
7. **Spinal cord**
   (a) The **lateral corticospinal tract**, crossed. -90%
   (b) The ventral or anterior corticospinal tract, uncrossed 10% to axial muscles.

F. Axons terminate on Ventral Horn Cells = Lower Motor Neurons.

a. Crossed (decussation) axons terminate contralaterally on motor neurons to appendicular muscles
(b. Uncrossed axons terminate ipsilaterally on axial muscles, they cross before terminating)
CORTICOSPINAL TRACT

Identify the sections of the CNS and outline the corticospinal tract.
Contrast the Corticospinal and Corticobulbar tracts
IV. THE CORTICOBULBAR TRACT

A. Definitions

1. **Bulb**: the medulla + pons + mesencephalon

2. The corticobulbar fibers are similar to corticospinal fibers except instead of terminating in the ventral horn of the cord, **they end in cranial nerve motor nuclei** (EXCEPT THE EXTRAOCULAR NUCLEI III, IV, and VI to be discussed later). The terms upper motoneuron and pyramidal tract are often used collectively as a term for both corticospinal and corticobulbar axons. Hence we have upper motor neurons that end on cranial motor neurons.

B. Origin -- Similar to that of the corticospinal axons except face region of cortex.

C. Course

1. Internal capsule -- posterior limb (some books say genu)

2. Cerebral peduncle -- the axons either leave the corticospinal fibers at this point or at a slightly more caudal level and make their way to the appropriate cranial nerve nuclei. Others travel more diffusely in the tegmentum. There is no visible corticobulbar tract as there is for the corticospinal tract.

D. Termination: Examples.

1. Most muscles act together such as the pharynx, larynx. They get input from both hemispheres.

5. Termination on the hypoglossal nuclei motoneurons is mostly crossed and can be useful in localizing lesions in the acute state. Signs may disappear after a few days.

6. **Muscles of facial expression** – see animation
E. Corticobulbar tract to the facial nucleus lower motor neurons.

1. The lower motor neurons that innervate muscles of the lower face receive only crossed corticobulbar axons from the cortex of the opposite side.

2. The motor neurons that innervate muscle of the upper face receive both crossed and uncrossed corticobulbar axons. That is that both hemispheres send cortical fibers to the nuclei on both sides of the brainstem.
V. THE INTERNAL CAPSULE

A. The **anterior** limb (to and from frontal lobe and basal ganglia is less important).

B. The **genu**- Corticobulbar

C. The **posterior limb** contains
   1. Some corticobulbar fibers or tract
   2. Corticospinal fibers or tract.
   3. Sensory radiations – somatosensory, auditory, visual
VI. BLOOD SUPPLY TO CORTICOSPINAL and CORTICOBULBAR SYSTEM

A. Motor cortex -- middle & anterior cerebral arteries.

B. Internal Capsule very variable
   1. Anterior limb -- lenticulostriate arteries from middle cerebral artery.
   2. Posterior limb -- middle cerebral artery, anterior choroidal artery and rarely branches from posterior cerebral artery.

C. Blood Supply of Cerebral Peduncle -- posterior cerebral artery (variable).

D. Blood Supply of Pons -- Basilar artery.

E. Blood Supply of Pyramid and lateral cord -- Anterior spinal artery.

VII. CLINICAL FINDINGS IN UPPER MOTOR NEURON DISEASE

Most corticospinal lesions are in the internal capsule or cerebral cortex in the distribution of the middle cerebral artery and result in classic signs. When the tract is lesioned in the brain stem these signs are associated with cranial nerve signs

A. Babinski sign (extensor plantar or dorsiflexion response or upgoing toe and fanning of the other toes) is abnormal and indicates damage to corticospinal tract. The big toe normally goes down. Hoffmann’s sign is a similar phenomenon for the hand.

B. Exaggerated tendon reflexes (hyperreflexia or increased DTRs). This includes clonus, crossed adductor, jaw jerk. Early on flaccidity may be due to "spinal" shock.

C. Spasticity: increased resistance to passive stretch. In upper extremity greater in flexors; in lower extremity greater in extensors. Clinical sign is the clasped-knife response.

D. Re-emergence of primitive reflexes, so-called Frontal (lobe) release signs: snout, grasp, suck, root, palmomental and glabellar
VIII. CONTRASTING UPPER MOTOR NEURON AND LOWER MOTOR NEURON DISEASE

Lesions of the upper motor neuron system (Corticospinal-Corticobulbar System) produce a different constellation of signs than do lesions of the lower motor neuron system (anterior horn cells).

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<thead>
<tr>
<th>UPPER MOTOR NEURON</th>
<th>LOWER MOTOR NEURON</th>
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<tbody>
<tr>
<td>Initial <strong>weakness</strong> or <strong>paralysis</strong> of muscles of entire limb or side of body and reduced reflexes</td>
<td><strong>Weakness or paralysis</strong> muscles in discrete area</td>
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<tr>
<td><strong>Spasticity</strong> of affected muscles, <strong>clased knife</strong></td>
<td><strong>Flaccidity</strong> of affected muscles</td>
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<tr>
<td><strong>Hyperactivity</strong> of <strong>deep tendon reflexes</strong>, <strong>clonus</strong></td>
<td><strong>Hypoactive</strong> or absent <strong>deep tendon reflexes</strong></td>
</tr>
<tr>
<td>No muscle atrophy or very slight from disuse</td>
<td>Prominent <strong>muscle atrophy</strong></td>
</tr>
<tr>
<td>No muscle fasciculations</td>
<td><strong>Fasciculations</strong> present</td>
</tr>
<tr>
<td><strong>Pathologic reflexes</strong>, Babinski</td>
<td><strong>No pathologic reflexes</strong> present</td>
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