Upper and Lower Motoneurons
Objectives

• Explain the concept of lower motoneurons and upper motoneurons
• Describe the locations of lower motoneurons and upper motoneurons
• Describe the corticospinal tract and cortico-bulbo-spinal tract and their role in movement
• Describe the somatotopic distribution of motor cortex
• Describe the symptoms of lower motoneuron and upper motoneuron lesions.
• Explain spasticity and clonus.
• Describe the developmental aspects of the motor system including myelination and the appearance of primitive reflexes.

http://nba.uth.tmc.edu/neuroscience/s3/chapter02.html
http://nba.uth.tmc.edu/neuroscience/s3/chapter03.html (relevant portions)
Overview

1. The upper motoneuron/lower motoneuron system provides the signals to muscles for movement.

2. Commands for movement originate in motor cortical areas. They are conveyed to spinal cord motoneurons for movement of body muscles and CN motoneurons for movement of head muscles.
Overview

3. This is a 2-tier system – the cortical command neurons are called *upper motoneurons*; the actual motoneurons they control are *lower motoneurons*.

4. Damage to each of these components produces specific *deficits that are key* to their involvement. First and foremost, you should recognize that UMN/LMN damage causes *weakness or inability to move*. 
1. What do symptoms mean and what do they tell us about part of motor system damaged?

2. Which symptoms **shared** and which **different**?

3. What is location of lesion?

<table>
<thead>
<tr>
<th>A 30 year-old woman who was trimming tree branches, fell off her ladder and dropped 20 feet to the ground landing on her back. She was rushed to the hospital where an exam revealed the following:</th>
<th>A 24-year-old man was brought to the emergency department after he was shot during an argument outside a bar. Examination several days after the injury revealed the following symptoms:</th>
</tr>
</thead>
<tbody>
<tr>
<td>LEFT LOWER LIMB</td>
<td>LEFT LOWER LIMB</td>
</tr>
<tr>
<td>• She could not move it.</td>
<td>• He could not move it</td>
</tr>
<tr>
<td>• <strong>Decreased</strong> resistance to passive flexion and extension.</td>
<td>• <strong>Increased</strong> resistance to passive flexion and extension.</td>
</tr>
<tr>
<td>• Patella reflex was <strong>absent</strong>.</td>
<td>• Patella reflex was <strong>hyperactive</strong>.</td>
</tr>
<tr>
<td></td>
<td>• <strong>Upgoing</strong> toes (<strong>Babinski sign</strong>).</td>
</tr>
<tr>
<td>RIGHT LOWER LIMB</td>
<td>RIGHT LOWER LIMB</td>
</tr>
<tr>
<td>• Movement and reflexes were normal.</td>
<td>• Movements and reflexes were normal</td>
</tr>
<tr>
<td>• Similar findings were obtained on exam 2 weeks later. (<strong>Sensory deficits omitted</strong>)</td>
<td>• This condition remained unchanged several months later. (<strong>Sensory deficits omitted</strong>)</td>
</tr>
</tbody>
</table>
## Functional Distinctions

<table>
<thead>
<tr>
<th>FUNCTION</th>
<th>COMPONENT</th>
<th>DEFICITS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Start</td>
<td>Basal Ganglia</td>
<td>Spontaneous Movements</td>
</tr>
<tr>
<td>Move</td>
<td><strong>UMN/LMN</strong>&lt;br&gt;Cerebral Cortex&lt;br&gt;Brainstem, Spinal cord Roots/peripheral nerves</td>
<td>Weak/Paralyzed</td>
</tr>
<tr>
<td>Plan</td>
<td>Cerebellum</td>
<td>Ataxia</td>
</tr>
<tr>
<td>Adjust</td>
<td>Cerebellum</td>
<td>Ataxia</td>
</tr>
<tr>
<td>Balance/EyeMove</td>
<td>Cerebellum</td>
<td>Loss of balance&lt;br&gt;Nystagmus</td>
</tr>
</tbody>
</table>
Hierarchy of Control

“upper motoneurons”
- cortex/brainstem
- axons stay in CNS
- synapse on LMN

“lower motoneurons”
- cell body in brainstem/spinal cord
- axons leave CNS
- synapse on muscle

Concept of 2 components:
- recognizes their different functions
- recognizes different symptoms of lesions
Lower motoneurons that control body movements
Body Movement - LMNs

Motoneurons for **limb** muscles
Motoneurons for **axial** muscles

• Motoneurons in **Ventral Horn** (Lamina IX):
  - $\alpha$ motoneurons innervate extrafusal muscle fibers
  - $\gamma$ motoneurons innervate intrafusal muscle fibers
Spinal Reflexes - LMNs

**Stretch Reflex**
- response to muscle stretch
- **basal activity sets resting muscle tension (tone)**
- basal activity is *modulated by brain* via UMN

Integrative feature of motor system. Muscle tone and DTR amplitude are clinical indicators of **entire** motor system.
Spinal Reflexes - LMNs

• What would be the effect of decreasing basal activity to a muscle?
• What about increasing basal activity to a muscle?
• How could you test it?
Clinical reflexes

Reflexes count up in order (main nerve root bolded):
- Achilles reflex = S1, S2 ("buckle my shoe")
- Patellar reflex = L3, L4 ("kick the door")
- Biceps and brachioradialis reflexes = C5, C6 ("pick up sticks")
- Triceps reflex = C7, C8 ("lay them straight")

Additional reflexes:
- Cremasteric reflex = L1, L2 ("testicles move")
- Anal wink reflex = S3, S4 ("winks galore")
Symptoms of LMN Lesions

• Paresis/paralysis – *Flaccid, ipsilateral* to injury
• Decreased tendon reflexes
• Decreased muscle tone

• Severe muscle atrophy (wasting)

• Fasciculations – twitching muscle fibers due to abnormal transmitter release at neuromuscular junction
A 30 year-old woman who was trimming tree branches, fell off her ladder and dropped 20 feet to the ground landing on her back. She was rushed to the hospital where an exam revealed the following:

<table>
<thead>
<tr>
<th>LEFT LOWER LIMB</th>
</tr>
</thead>
<tbody>
<tr>
<td>• She could not move it.</td>
</tr>
<tr>
<td>• <strong>Decreased</strong> resistance to passive flexion and extension.</td>
</tr>
<tr>
<td>• Patella reflex was <strong>absent</strong>.</td>
</tr>
<tr>
<td>• <strong>Down-going</strong> toes</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>RIGHT LOWER LIMB</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Movement and reflexes were normal.</td>
</tr>
</tbody>
</table>

• Similar findings were obtained on exam 2 weeks later. (Sensory deficits omitted)
Upper motoneurons that control body movements
UMNs for Body Movement

(ideation, planning, execution)

2 pathways travel together from cortex through brainstem into spinal cord to control movement:

- **Corticospinal tract** controls body movement especially fine movement
- **Cortico-bulbo-spinal tracts** control muscle tone and posture

- we recognize the different functions of these 2 pathways, BUT
- we treat them as a unit as they travel together through brainstem/spinal cord
- LESIONS affect BOTH pathways/functions
UMNs – Corticospinal Tract

What: corticospinal tract
Why: controls body movement especially fine movements
How: cell bodies in cortical motor areas send axons through brainstem to spinal cord LMNs
Lesions: cause weakness/paralysis
Corticospinal Tract

- **Lateral Corticospinal T (90%)** controls **limb** motoneurons to limb muscles.

- **Ventral Corticospinal T (10%)** crosses later in spinal cord to control axial motoneurons to axial muscles.
Origin/Path of Corticospinal Tract
The corticospinal tract is also known as the *pyramidal tract* because its fibers form the pyramids of the medulla.
somatotopic organization

Sensory pathways

The corticospinal fibers and 3rd order sensory fibers are in different locations in the posterior limb of internal capsule. Therefore, a small lesion could affect one area and not the other (eg a small lesion can affect corticospinal fibers but not sensory fibers causing purely motor symptoms).
Origin of Corticospinal Tract

Cortical motor areas:
- motor cortex
- premotor cortex
- supplementary motor area
(also a component from somatosensory areas)

How Do We Know:
1. Stimulation of precentral gyrus in animals and neurosurgery caused contraction of a few muscles contralaterally.
2. Stimulation revealed somatotopic distribution.
3. Amount of cortex correlated with functional importance for making fine movements.
anterior part of paracentral lobule
Cortical Motor Areas

Highly *interconnected* areas differing in structure and function:

1. **Motor Cortex** - 1 UMN activates several muscles - encodes *purposeful movement*

2. **Premotor Cortex** - movements involving *sensory guidance* (visual/tactile).

3. **Supplementary Motor Area** - planned, self-initiated movements; bimanual

Damage to

- **MC** - weakness/paralysis
- **PMC**
- **SMA**

**Apraxia** - inability to perform learned movement without paralysis or sensory impairment
Cortico-bulbo-spinal Pathway

**What:** *corticobulbar + bulbospinal* pathway

**Why:** dedicated pathway modulates *muscle tone, stretch reflex, and posture* during movement

**How:** cell bodies in cortical motor areas go to brainstem to synapse on 2 nuclei. Nuclei send axons to spinal LMNs.

**Lesions:** cause increased muscle tone and stretch reflexes and altered posture.
Corticobulbar Tract

• Cell bodies in motor cortical areas send axons to synapse on 2 brainstem nuclei:
  • red nucleus
  • reticular formation

• Travels with corticospinal axons
Bulbospinal Tracts

• Cell bodies in **red nucleus and reticular formation** send axons to LMNs in spinal cord.

• Although these tracts are dispersed in spinal cord, they are typically affected together by most structural/vascular lesions. Thus we consider them a unit with the corticospinal tract.
Bulbospinal Tracts

Tracts originate in

**Red Nucleus**
rubrospinal tract

**Reticular Formation**
reticulospinal tract

- Rubrospinal and reticulospinal tracts influence LMNs on opposite side consistent with effect of the corticospinal tract.
How Bulbospinal Tracts Affect Muscle Tone/DTRs

Inhibitory Effects

• The reticulospinal tract tonically inhibits LMNs.
• This inhibition decreases muscle tone and the amplitude of the stretch reflex, resulting in normal muscle tone.

Differential Effects on Limbs

• Reticulospinal inhibition influences upper and lower limb muscles differently:
  • inhibits upper limb flexors
  • inhibits lower limb extensors
How Bulbospinal Tracts Affect Muscle Tone/DTRs

- UMN lesions remove reticulospinal inhibition and thereby result in *increased* muscle tone and DTRs.
  - *upper limb flexors*
  - *lower limb extensors*

- The increased muscle tone from UMN lesions causes postural changes

Upper limb is adducted at the shoulder and *flexed* at elbow, wrist, fingers

Lower limb is *extended* at knee, ankle, toes
Increased muscle tone caused by UMN lesions is called **spasticity** and has specific characteristics.

- Spasticity is evaluated by passive movement of upper and lower limbs.
- Testing the stretch reflex causes its repeated activation (**clonus**) due to DTR hyperactivity.

### Spasticity Characteristics

1. **Limb and direction dependent**
   - upper limb – resistance to passive extension (due to increased tone in flexors)
   - lower limb – resistance to passive flexion (due to increased tone in extensors)

2. **Varies over range** — resistance greatest at start, diminishes at end of passive movement

3. **Velocity dependent** — resistance to passive movement is greater when the passive movement is faster
Lesions of the Spinal Cord

• The corticospinal and corticobulbospinal pathways cross before entering the spinal cord. **Lesions in the spinal cord** cause ipsilateral motor deficits.

• Sensory and autonomic pathways are also affected
  • loss of tactile, pain, temp sensation (ascending pathways)
  • loss of bowel and bladder control
  • loss of thermo-regulation
  • **Respiratory** difficulty for lesions at/above C3-C5 (phrenic nucleus)

Lose **autonomic control** - via bulbospinal and corticospinal pathways.
Symptoms of UMN Lesions

1. Paresis/paralysis
2. Increased muscle tone- *Spasticity*
3. Increased tendon reflexes
4. Babinski sign — a reflex induced by stroking the plantar surface of the foot
Normal Plantar Reflex
A reflex induced by stroking the plantar surface of the foot.
# UMN Lesion Example

A 24-year-old man was brought to the emergency department after he was shot during an argument outside a bar. Examination several days after the injury revealed the following symptoms:

<table>
<thead>
<tr>
<th>LEFT LOWER LIMB</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>• He could not move it</td>
<td></td>
</tr>
<tr>
<td>• <strong>Increased</strong> resistance to passive flexion and extension.</td>
<td></td>
</tr>
<tr>
<td>• Patella reflex was <strong>hyperactive</strong>.</td>
<td></td>
</tr>
<tr>
<td>• <strong>Upgoing</strong> toes (<em>Babinski sign</em>).</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>RIGHT LOWER LIMB</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>• Movements and reflexes were normal</td>
<td></td>
</tr>
<tr>
<td>• This condition remained unchanged several months later. (Sensory deficits omitted)</td>
<td></td>
</tr>
</tbody>
</table>

**UMN injury**

- spinal cord - *Left* Side involving LCST
**SUMMARY**

**LMN Lesions**
1. Paresis/paralysis - *Flaccid, ipsilateral*
2. Decreased tendon reflexes
3. Decreased muscle tone
4. Severe muscle atrophy (wasting)
5. Fasciculations

Involving spinal motoneurons

**UMN Lesions**
1. Paresis/paralysis - *Spasticity*
2. Increased tendon reflexes
3. Increased muscle tone
4. Babinski sign

Involving corticospinal and cortico-bulbo-spinal tracts
UMN Syndromes

Brown-Sequard Syndrome (revisited)

- damage to one half of spinal cord causes loss of tactile sensation on same side of body, loss of pain on opposite side, **AND** weakness/paralysis (including increased tone and DTRs and Babinski) on same side as lesion.
Developmental Aspects

- Motor system provides important opportunity to assess development.
- Corticospinal tract does not complete myelination until end of year 1.
  - capacity for voluntary movement limited
  - Infant movement dominated by reflexes – survival benefit
- Reflexes appear and then disappear on a specific time schedule associated with CST myelination.
  - presence and timing of reflexes indicate CNS maturation.

Primitive reflexes can reappear in adults with CNS damage particularly with frontal lobe lesions.
## Primitive Reflexes

(from First Aid)

<table>
<thead>
<tr>
<th>Reflex</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Moro Reflex</td>
<td>“Hang on for life” reflex—abduct/extend arms when startled, and then draw together</td>
</tr>
<tr>
<td>Rooting Reflex</td>
<td>Movement of head toward one side if cheek or mouth is stroked (nipple seeking)</td>
</tr>
<tr>
<td>Sucking Reflex</td>
<td>Sucking response when roof of mouth is touched</td>
</tr>
<tr>
<td>Palmar Reflex</td>
<td>Curling of fingers if palm is stroked</td>
</tr>
<tr>
<td>Plantar Reflex</td>
<td>Dorsiflexion of large toe and fanning of other toes with plantar stimulation</td>
</tr>
<tr>
<td>Babinski Sign</td>
<td>Presence of this reflex in an adult, which may signify a UMN lesion</td>
</tr>
<tr>
<td>Galant Reflex</td>
<td>Stroking along one side of the spine while newborn is in ventral suspension (face down) causes lateral flexion of lower body toward stimulated side</td>
</tr>
</tbody>
</table>