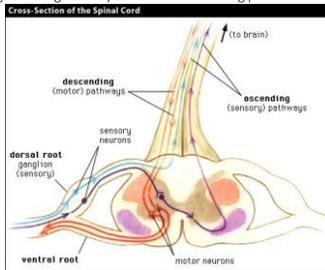


Now it is time to focus on the pathways running down the cord( on both sides of the cord even though the diagram only shows the descending paths on the left)



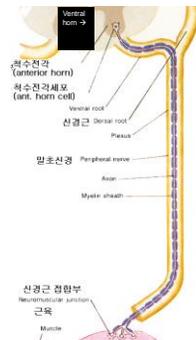
## •Descending Motor Pathways

- Pathways from brain carrying motor commands down to
  - "lower motor neurons"
- (review the motor neurons which synapse on muscle fibers at the neuromuscular junction, using acetylcholine as the messenger to trigger contraction)

### •"Lower Motor Neurons" (LMNs)

- Neurons whose axons synapse on skeletal muscle fibers (to stimulate contraction)
- aka "alpha motor neurons"
- aka "final common path" to all behavior
- majority are found in ventral horns of cord
- some are in the brainstem (cranial nerve motor neurons serving muscles of head)

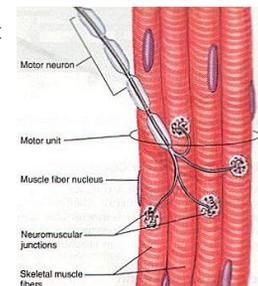
### •An Example of a LMN



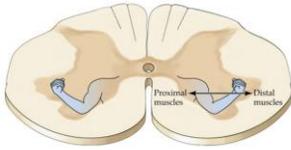
### •Motor Units

- Motor unit = a single LMN + the extrafusal muscle fibers it synapses on
- Some motor units are small (axon synapses on just a few fibers); some motor units are large (axon synapses on ~1000 fibers).
- All fibers of a motor unit would be stimulated by neurotransmitter (ACh) release at the same time.
- All movements depend on LMNs.

### •Motor Unit



**Location** of motoneurons in ventral horn

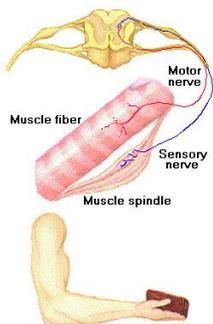
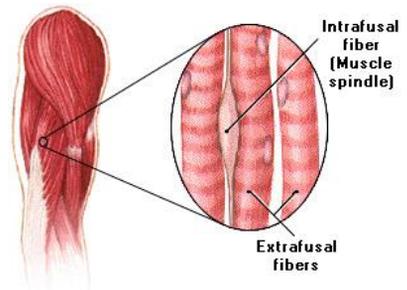


• Spinal Anatomy and Physiology  
Spinal Nerves

- Myotomes
  - Muscles innervated by each spinal nerve (not as easy to diagram as "dermatomes")
  - Examples of myotomes of different levels
    - Arm extension: C-5
    - Elbow extension: C-7
    - Small finger abduction: T-1
    - Knee extension: L-3
    - Ankle flexion: S-1

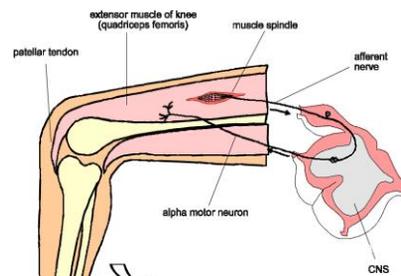
• Spinal Reflexes

- Spinal cord is not just a cable between brain and body. Segments of cord also have "local" functions relatively independent of the brain.
  - Monosynaptic stretch reflex
  - Polysynaptic withdrawal or flexor reflex
  - Polysynaptic withdrawal plus crossed extensor reflex



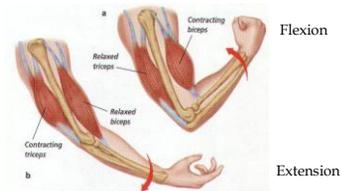
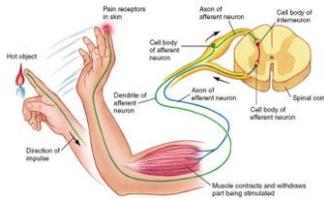
Muscle spindle receptor is stretched and begins to fire when muscle stretches or relaxes too much. This firing activates the LMN that will make that relaxed muscle fiber contract to re-establish muscle tone/tension. Notice reflex arc only involves 2 neurons (sensory neuron synapses on motor neuron) – a "monosynaptic reflex"

- <http://www.youtube.com/watch?v=TVawvNU5cI&feature=related> kneejerk
- <http://www.youtube.com/watch?v=v5OPtqnQjk> other stretch



Tapping tendon stretches muscle, activating stretch receptors. Reflex automatically maintains muscle tone

•The Flexor or Withdrawal Reflex is slightly more complicated because it involves an interneuron



Flexors and extensors in a limb are never active at the same time. It is wired into our nervous system that when one is stimulated the other is inhibited from contracting (so is relaxed).

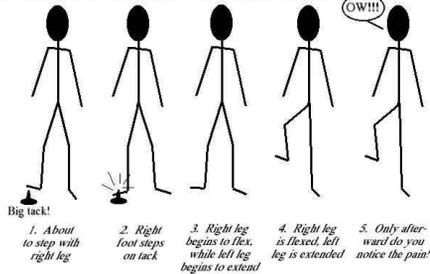
### Withdrawal of a Supporting Limb

If we step on a tack and the withdrawal reflex is triggered in that leg, an even more complicated spinal reflex occurs because your opposite leg must support your weight while the other leg is withdrawing.

### The Crossed Extensor Reflex



### Function of Crossed Extensor Reflex



•Muscle Fibers Depend on Their Innervation by a LMN

- Muscle fibers that lose their LMN show:
  - no reflexive contraction (so no muscle tone)
  - no voluntary contraction
  - (This is called "flaccid paralysis")
  - atrophy of the muscle fibers over time
  - (These are "symptoms of LMN damage")

• Example of Disease Causing Loss of LMNs :  
Poliomyelitis

- Viral infection causing a summer cold/flu-like illness in most, but which, in some, infects LMNs – most often those controlling the lower limbs.
- If those neurons die, the muscle fibers in their motor units will be paralyzed, show no reflexes and will atrophy.

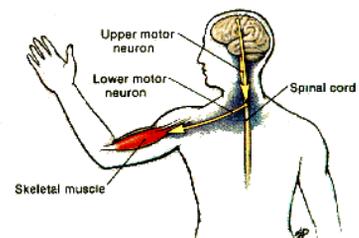
• Muscle Atrophy

- Caused by loss of LMNs due to polio



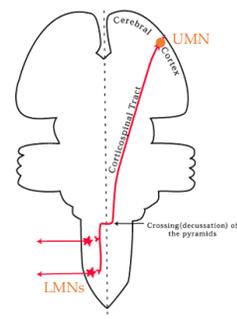
• “Upper Motor Neurons”(UMNs):

- The brain cells that send motor commands down to the LMNs.
- UMNs are the source of the “descending motor pathways”
- Damage to UMNs affects motor function/control because there is a loss of commands to the LMNs

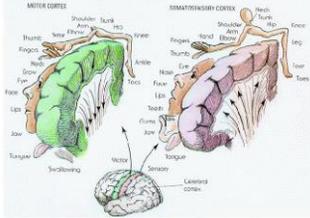


• The Corticospinal or Pyramidal Pathway

- Most direct route for UMNs to influence LMNs (Cortex to LMN)
- Evolutionarily recent, largest in man
- Most important for fine, precise, rapid, skilled voluntary movements of small motor units (e.g. hands, facial muscles, tongue)
- Big body muscles receive more input from other “extrapyramidal” motor pathways



Corticospinal Tract begins in cortex – mostly in motor cortex but there are some contributing UMNS in other regions too

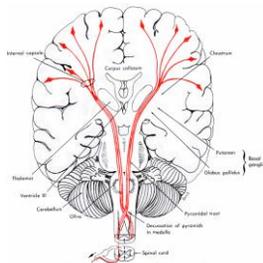


•Cortex → brainstem & spinal cord



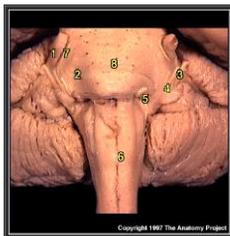
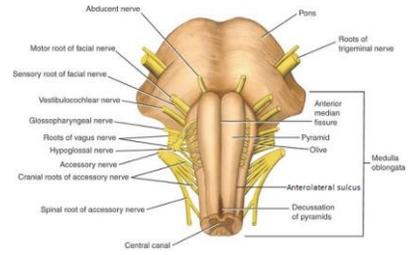
Some of the axons seen here heading towards cord are UMNS

Another name for the corticospinal tract is the pyramidal tract. This tract crosses to the opposite side (decussates) at the spinal cord/medulla junction.

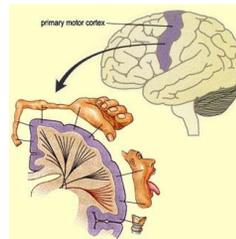


The crossed corticospinal (pyramidal) tracts. Axons that originate in the motor cortex cross to the opposite side of the brain in the medulla and descend through the spinal cord on the opposite side of the brain. This is called decussation. The decussation of pyramidal tracts occurs in the medulla. Each crossed corticospinal tract therefore carries motor impulses from one side of the brain to interneurons on either side of the spinal cord on the opposite side of the brain.

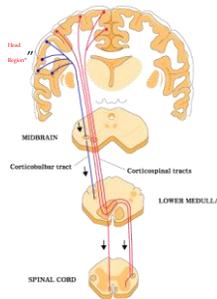
Ventral Surface of Brainstem Showing “Pyramids”



“Pyramids” (axons of corticospinal tract stained blue)

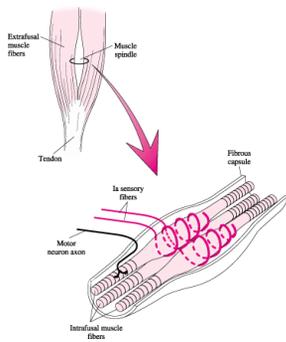


• LMNs for muscles of head are not in the spinal cord. Head region of motor cortex only sends its commands as far as the brainstem LMNs.

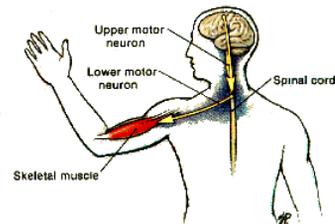


#### • Upper Motor Neurons Modulate Muscle Tone Via Gamma Motor Neurons

- "Gamma motor neurons" (the "other" motor neurons) do not go to the extrafusal fibers that move us.
- They synapse on "intrafusal muscle fibers" inside stretch receptors/muscle spindle receptors.
- Tensing these little fibers makes stretch receptors more sensitive.
- UMN also can **inhibit** stretch reflex to allow movement, so we are not stiff statues.



#### • UMN vs LMN



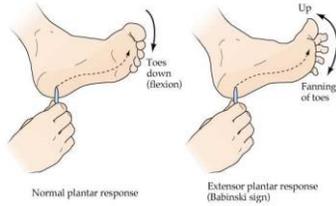
#### • Symptoms of UMN Damage

- Reflexes and muscle tone still present and, in fact, intensified → "hyperreflexia"
- **Voluntary** control impaired
- "Spastic paralysis" (excess muscle tone & loss of voluntary control of movement)
- No denervation induced atrophy (LMNs are okay)

#### • Reflex Changes After UMN Damage

- hyperactive stretch reflex, particularly in anti-gravity muscles
  - too much muscle tone (hypertonia or spasticity)
  - clonus (rapid repetitive response to stretch)
- altered Babinski reflex after corticospinal damage

•Normal Adult vs. Positive Babinski



<https://www.youtube.com/watch?v=9hNh32VVAZQ>

•Clonus

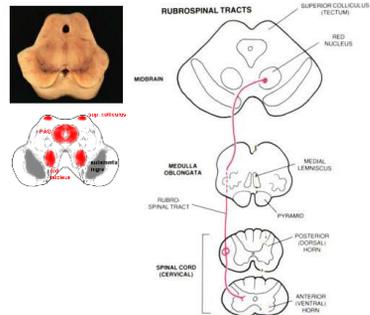
- Without descending modulation from brain, stretch reflex leads to repetitive contractions

<http://www.youtube.com/watch?v=kZweyLV95kA>

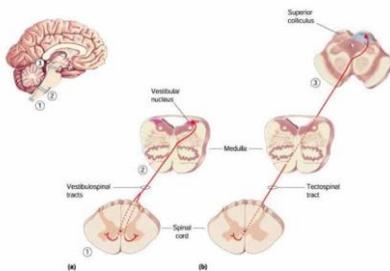
•Other Descending Tracts:

“Extrapyramidal Motor Pathways”

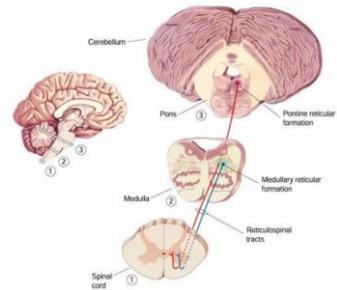
- Rubrospinal pathway to regulate tone of flexors in limbs for locomotion & to organize repetitive movements that involve the flexors (e.g. walking, running, crawling)
- Vestibulospinal pathway to stimulate extensors (antigravity) for standing, posture
- Tectospinal pathway for reflexive motor reactions to visual stimuli
- Reticulospinal pathway to regulate muscle tone by modulating the stretch reflex
- (don't need to memorize exact route of these)



Vestibulospinal and Tectospinal Pathways



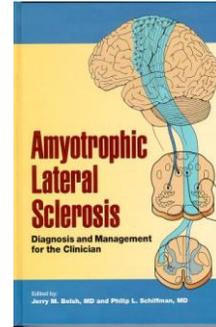
Reticulo-spinal tracts





### •Amyotrophic Lateral Sclerosis (ALS) or Lou Gehrig's Disease

- Fatal progressive loss of LMNs as well as corticospinal pathway (UMNs). Several genes involved.
- Onset most often in late 50's-early 60's; more men affected
- 70% will die within 5 years (eventually cannot swallow, breathe)

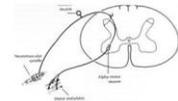


### •ALS – Symptoms

- First symptoms usually muscle cramping & twitching (fasciculations), with feelings of fatigue & weakness in a limb
- Loss of LMNs causes weakness, paralysis, loss of reflexes & atrophy in **affected** muscles. Loss of UMNs causes spasticity (muscle stiffness, cramping from too much tonus where LMNs have not been lost).
- Combination of UMN + LMN symptoms at multiple levels is fairly diagnostic

\*Listen to ALS link in syllabus

### •LMN



UPPER AND LOWER MOTOR NEURONS

- A lower motor neuron lesion interrupts the path from the alpha motoneuron to the muscle
- All excitation of the muscle is lost and the muscle becomes **paralyzed** (unable to move)
- the muscle becomes **flaccid** (muscle tone decreases).
- eventually the muscle will **atrophy**.
- An upper motor neuron lesion also causes **paralysis**.
- the muscle becomes **spastic** (muscle tone increases),
- the muscle does **not atrophy**.
- if the sole of the foot is stroked, the toe dorsiflexes (**Babinski response**)

