DIRECT ACTIVATION PATHWAY (PART OF THE UMN SYSTEM)
(a.k.a. pyramidal or direct motor system).

- DIRECTS SKILLED, DISCRETE VOLUNTARY (CONSCIOUSLY CONTROLLED) MOVEMENTS (SPEECH)

- FACILITATORY

- MAIN LAUNCHING AREA IS PRIMARY MOTOR CORTEX, PREMOTOR CORTEX, SUPPLEMENTARY MOTOR AREA.

- CORTICOBULBAR TRACTS (control cranial nerves)
- CORTICOSPINAL TRACTS (control spinal nerves)

- PATHWAYS ORIGINATE IN CORTEX OF ONE HEMISPHERE AND TERMINATE IN THE CR. AND SPINAL NUCLEI THAT INNERVATE SPEECH ON THE SIDE OF THE BODY OPPOSITE THE CEREBRAL CORTEX OF ORIGIN (EG. LEFT HEMISPHERE STROKE CAUSES RIGHT SIDED PARALYSIS). Also true for Lower face (VII) AND tongue (XII).

- OTHER CRANIAL NERVES RECEIVE BILATERAL INPUT FROM DAP AND IAP (I.E. SERVES PROTECTIVE FUNTION). THEREFORE, UNILATERAL UMN LESIONS HAVE MINOR EFFECTS ON V, IX, X, XII.

DAMAGE TO DIRECT ACTIVATION PATHWAY

- A LOSS OR REDUCTION IN IT ABILITY TO FACILITATE FINE, DISCRETE MOVEMENTS

- INITIALLY REDUCED TONE AND WEAKNESS, BUT EVENTUALLY LEADS TO INCREASED TONE AND SPASTICITY.

- POSITIVE BABINSKI SIGN

- PATHOLOGICAL ORAL REFLEXES… SUCK, SNOT.
The Direct Activation Pathway

Figure 2-16 The direct activation pathways.
Effects of Damage to IAP: Generally referred to as UMN damage

- Usually in combination with damage to the direct activation system (pyramidal tract).
- Lesions affect muscle tone and reflexes.
- Depending upon location of the lesion (how high in the central nervous system i.e., cerebrum or brainstem) can contribute to different types of problems.

Corticoreticular fibers (cortex to reticular formation)

- Lesions are different for flexors and extensor muscles
- Lesions above the level of the midbrain and red nucleus produce decorticate posturing. Increased extensor tone in legs and increased flexor tone in arms (i.e., legs tend to be extended and resist bending and arms tend to flex and resist extension). All descending pathways are uninhibited. (leads to spasticity – increased tone).

- Lesions at the level of the midbrain below the red nucleus remove arm flexor pattern and result in excitation of all extensor muscles… decerebrate posturing.

- Lesions below the médulla result in a loss of all descending input and produce generalized flaccidity in muscles supplied by spinal nerves.
The Indirect Activation Pathway a.k.a extrapyramidal or indirect motor system:

- multiple synapses from its origin in cerebral cortex and its destination in the FCP.

- The “milk” run (local route), whereas DAP (pyramidal tract) is the “express” run.

- Primary function is motor control to regulate reflexes and maintain posture and tone. Ensures that specific speech movements occur without constant or variable interference with their speed, range and direction.

- Effect seems to be inhibitory, whereas the direct activation system seems to be facilitory.

- In speech, the IAS probably inhibits interference with the movements of specific muscles so that appropriate speed, range, and direction of movement can be maintained.
Figure 2-18 The indirect activation pathway. Note that the tracts of this system are intermingled with those of the corticobulbar and corticospinal tracts (the direct activation pathway).
Control Circuits (Basal Ganglia and Cerebellum)

- Unlike direct and indirect activation pathways, control circuits do not have direct contact with LMN’s.

- They coordinate, integrate and help control the diverse activities of the many structures pathways involved in motor performance (i.e., integrate activities of direct and indirect pathways).

- Integration is completed by the basal ganglia control circuit and the cerebellar control circuit.
FUNCTION of the BASAL GANGLIA

- Function not completely understood, but seem important for maintaining normal posture and static muscle contraction upon which voluntary skilled movements are superimposed.

- Important to regulating the amplitude, velocity and possibly the initiation of movements.

- Help maintain a stable musculoskeletal environment in which discrete speech movements can occur.

- Seem to have a damping effect on cortical discharges. Cortex sends messages that are in excess of what is necessary to achieve the movement goals, the one role of the basal ganglia is to damp or modulate those impulses to an appropriate degree.
Effects of Damage to the Basal Ganglia control circuit

Damage to the BG produce more profound speech disturbances than do lesions of the cortex.

Two manifestations of damage to Basal Ganglia

- Hypokinesia (reduced mobility)
- Hyperkinesia (involuntary movements)

- Hypokinesia is often associated with disease of the substantia nigra, which results in a deficiency of the neurotransmitter “dopamine.” The effect is an increase in muscle tone, with subsequent increased resistance to passive movements = “rigidity”.

- Hyperkinesia can result from excessive activity of the dopaminergic nerve fibers, thereby reducing the circuit’s damping effect on cortical discharges.

- This results in involuntary movements, which vary in their locus, speed, and constancy.
The Basal Ganglia (Caudate Nucleus, Putamen, Globus pallidus) and its connections

FIGURE 9-18. The basal ganglia consist of the caudate nucleus, amygdala, globus pallidus, and putamen. The basal ganglia are shown in relation to the other subcortical structures, including the diencephalic thalamus and hypothalamus, as well as the red nucleus and substantia nigra.
Corpus Striatum:
(A) Caudate Nucleus
(B) Lentiform Nucleus:
 (1) Putamen
 (2) Globus Pallidus

Figure 12-1. Coronal section of the cerebrum anterior to the thalamus, stained by a method that differentiates gray matter (dark) and white matter (light), showing the components and relations of the corpus striatum. (× 75)

Horizontal Section

Figure 12-2. Horizontal section of the cerebrum, stained by a method that differentiates gray matter (dark) and white matter (light), showing the components and relations of the corpus striatum. (× 75)
Figure 2–19 The basal ganglia control circuit.
Summary of the UMN input into the Motor Nuclei of the Cranial Nerves involved in Speech

FACT 1: Cranial Nerves V, VII (upper face), IX, X, XI receive Bilateral UMN input. This means that each cranial nerve motor nucleus (in the brainstem) receives bilateral (two) messages from the cortex (launching pad). So the left cranial nerve motor nucleus would receive a message from both the right and left sides of the brain. And, the right cranial nerve motor nucleus would receive a message from both the right and left sides of the brain.

For example, let's consider the Trigeminal Nerve (V). In the diagram below, the Right motor nucleus of the Trigeminal nerve resides in the pons of the brainstem, and supplies the muscles of mastication (via the Trigeminal) on the Right side of the face (jaw). It is important to note, that the Right motor nucleus receives UMN input from both the left and right hemispheres of the brain. There is also a left motor nucleus for the Trigeminal N. that is the pons of the brainstem. The left motor nucleus and trigeminal nerve supply the muscles of mastication on the left side of the face. The left motor nucleus also receives bilateral input from the UMN as well. So both motor nuclei (i.e., the left and the right) receive two messages from the brain. There is redundancy built into the nervous system. This bilateral UMN input into the motor nuclei is true for the other cranial nerves (i.e., V, VII-upper face, IX, X, XI).

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note — right motor nucleus receives umn input from both left + right hemispheres (corticospinal tracts)

// bilateral input //
true (to v, vii-upper face, ix, x, xi)
```
FACT 2: Cranial Nerves VII-lower face and XII receive unilateral UMN input. For these two nerves, the input from the UMN into the motor nucleus is unilateral and comes from hemisphere opposite (contralateral) the motor nucleus. In the case of these nerves, the motor nucleus receives only one message and it originates from the opposite hemisphere.

Let’s consider the Right XIth Cr. Nerve, which supplies the muscles on the right-side of the tongue. The Right motor nucleus of the XIth Cr. N. receives the command to move only from a message originating from the Left hemisphere of the brain. In this case, the input from the UMN is said to be unilateral (and more specifically, contralateral). See the diagram below.

This unilateral UMN input is also true for the VIIth cranial nerve. However, the VIIth cr. nerve motor nucleus is organized into two parts—the upper and lower face. The motor nucleus that supplies the upper face receives bilateral UMN input (i.e., two messages), whereas the part of the motor nucleus that supplies the lower face receives only unilateral UMN input (i.e., only one message from the side of the brain opposite the motor nucleus).
So, what happens if we have UMN damage....

Let's deal with unilateral UMN damage. For instance, what if I have a stroke in the left hemisphere of the brain. Because each motor nucleus (for Cr. Nerves V, VII-upper face, IX, X, XI) receives bilateral messages (input) from the UMN, there will be no discernible effect on the function of those nerves (i.e., no functional effect). The reason is that the right hemisphere can still relay the message to the motor nucleus, and the movement can be accomplished.

Because of the redundancy built into the nervous system for these Cr. Nerves, one needs to experience bilateral UMN damage to have a negative functional effect on Cr. Nerves V, VII-upper face, IX, X, XI. Therefore, I would need to have bilateral strokes, or a tumor that somehow affected both the left and right UMN pathways, in order for me to have obvious effect on the muscles supplied by these nerves.
The EXCEPTION: Unilateral UMN damage is sufficient to cause functional weakness in the case of CR. N. VII (lower face) and XII (tongue). Why? Because these cranial nerves receive only unilateral input from the UMN (a single message that originates from the hemisphere opposite the motor nucleus). Therefore, the result of unilateral UMN damage is weakness (paresthesia) to the lower side of the face opposite the hemisphere of damage, or weakness of the ½ of the tongue contralateral to the side of UMN damage.

To be specific, left UMN (hemisphere) damage can cause right lower facial weakness, because of the unilateral input to the VIIth nerve motor nucleus (lower face). Also, left UMN damage can cause right tongue weakness, because of the unilateral input of the XIIth nerve motor nucleus (tongue).
Neuroanatomy and Speech and Language Production.

Speech Production is a complicated process. There is an average of 140,000 neuromuscular events every second when speaking at a rate of 4 sounds per sec.

Three processes leading to speech production.

1. **Cognitive-linguistic** (thoughts are organized and converted into verbal symbols that abide by the rules of our language). Damage at this level produces a language impairment known as **aphasia or dementia**.

2. **Motor speech programming** (intended verbal message must be organized into sensorimotor programs that will activate the speech muscles at appropriate times, durations, and intensities). Damage at this level produces something called **apraxia of speech**.

3. **Neuromuscular Execution** (CNS and PNS activity must execute speech motor programs by innervating the speech muscles i.e., respiratory, phonatory, resonatory, and articulatory muscles, in a manner that reflects the goals of the program). Damage at this level produces **dysarthria**. Different types of dysarthria reflect different sites of damage within the motor systems.

Neurological Damage at any point within this sequence will produce some kind of communication impairment. Where the damage occurs within the neurological system will often predict the kind of problem that surfaces. There is a relationship between where in the CNS the damage occurs and the final product i.e., aphasia, apraxia, dysarthria.

Therefore we need to know the major structures of the speech language system. Knowing the functional significance of these structures allows us to predict the effects of damage and/or infer where damage may be based upon speech and language features we observe.

**Major Speech and Language Circuitry**

Left hemisphere is dominant for Language expression and processing in 90-95% of the population. If right-handed you are left hemisphere dominant for language.

**Major Centers involved in Speech-Language production and processing.**

**Primary Auditory Cortex (Brodman Area 41)**—a.k.a. Heschl’s gyrus (located in lateral fissure, posterior two-thirds on upper surface of temporal lobe
Role is primary processing of auditory information.

**Supramarginal Gyrus (Brodman Area 40)**—fold of cortex that curves around end of lateral fissure. It responds to acoustic stimulation and is presumably involved in language processing.
Damage to this gyrus disturbs written language formulation....agraphia.
Angular Gyrus (Brodmann Area 39)—immediately posterior to the supramarginal gyrus. Damage to this area is frequently associated with reading difficulties (dyslexia).

Wernicke’s Area (Brodmann Areas 22, 41, 42- a.k.a. Auditory Association Area)—superior part of temporal lobe (behind primary auditory cortex). Involved in auditory comprehension of language. Assigns meaning to auditory input. Damage produces fluent incomprehensible speech and severe auditory comprehension deficits (i.e., receptive aphasia).

Broca’s Area (Brodmann Area 44)—inferior frontal gyrus. Expressive language area where programming of speech muscles for speech production occurs. Damage can produce expressive aphasia (difficulties with verbal expression with relatively spared comprehension). Damage can produce apraxia of speech (inability to program the positioning of speech muscles and sequencing of muscle movements for volitional speech production).

Arcuate fasciculus: connects frontal lobe to occipital and temporal lobes (i.e., connects expressive and receptive language areas). Damage to A.F. produces conduction aphasia. Preserved auditory comprehension, but patient has severe difficulties with repetition.

Supplementary and premotor areas (Brodmann Area 6). Along with Broca’s area is essential for speech output. It has important connection with the limbic system, and is involved with initiation of speech and maintenance of readiness to speak (involved in speech planning). Damage causes a disturbance in speech initiation and maintenance of normal fluency. May produce aphasia characterized by no initiation, but preserved repetition (i.e., transcortical motor).
The Basic Speech and Language Circuits of the Cerebral Cortex. (Arrows indicate primary flows of information among structures.)
DYSARTHRIA ...

a collective name for a group of speech disorders resulting from disturbances in muscular control over the speech mechanism due to damage of the central or peripheral nervous system. It designates problems in oral communication due to paralysis, weakness, or incoordination of the speech musculature (Darley, Aronson, & Brown, 1969, p.246).

(1) NEUROGENIC

(2) ABNORMAL NEUROMUSCULAR CONTROL MAY AFFECT SPEECH MOVEMENTS
   • SPEED
   • RANGE
   • STRENGTH
   • TIMING
   • ACCURACY

RESPIRATION, PHONATION, RESONANCE, ARTICULATION, & PROSODY

(3) CAN BE SUBDIVIDED INTO CATEGORIES, E.G.
   • FLACCID
   • SPASTIC
   • HYPOKINETIC
   • HYPERKINETIC
Apraxia of Speech

Is a motor speech disorder resulting from impairment of the capacity to program sensorimotor commands for the positioning and movements of the muscles for the volitional production of speech. It can occur without significant weakness or neuromuscular slowness, and in the absence of disturbances of thought or language.

- Deviant speech characteristics include abnormalities of articulation, rate, prosody, and fluency.

- Debate over whether it is a linguistic or motor disorder.

- Most distinctive characteristics include trial-and-error groping, dysprosody, difficulty initiating utterances, and articulatory inconsistency.

- Varies in severity. More severe forms have limited phonetic repertoire, little difference between voluntary and automatic speech utterances, and a highly consistent pattern of perceived speech errors.
Nonverbal Oral Apraxia.

- Inability to imitate or follow commands to perform volitional movements of the speech structures; the inability cannot be attributed to poor task comprehension or to sensory or neuromuscular deficits.
- No one-to-one correspondence between AOS and NVOA, although co-occurrence is quite high.
- NVOA much more frequent in patients with Broca’s aphasia than in those with Wernicke’s.
- Limb apraxia and NVOA also tend to co-occur.

Commonly used tasks for NVOA include...

- Cough
- Click the tongue
- Smack the lips
- Blow
- Whistle

Clinical Findings:

- Awkward, off-target responses, effortful groping correct movements, or inconsistent trial and error attempts.
- Sometimes they try to say the word and perform the act at the same time.

Patients usually are perplexed, frustrated, amused, or embarassed by these off-target responses and usually try to correct themselves.
Structural and Functional Aspects of Neurons

This unit will discuss: Neurons, Neuroglial cells, Excitation and Conduction, Synapses and Receptors

Neurons

The Structure of Neurons (overhead Zemiln page 244)

Neurons have...

- cell body--nucleus, variety of specialized bodies
  - many motor neurons have cell bodies with a pyramidal shape. will form pyramidal tract
- processes--axons & dendrites
  - Dendrites always conduct impulses TOWARD the cell body. They TAKE IN information.
  - Axons always conduct impulses AWAY from the cell body. They SEND OUT information.

Axons

- the bulk of the nervous system is axons
- axon hillock is junction of axon & cell body
- axons terminate in a number of branches called a telodendricle or end brush
- each ending is tipped with synaptic boutons (knobs) or button
- Telodendricle come close to, but do not touch the next neuron in the chain. They often approximate dendrites, but not always. - sometimes cell body
- Some axons are covered with fatty substance called myelin--from axon hillock to end brush.
  - myelin enables the axon to conduct the nervous message faster
  - neurons are rated as to amount of myelin using an alphanumeric system. A1, B3, etc.
  - Myelin is made of a type of a Neuroglial cell

Neuroglial Cells

- About 10 times more numerous than nerves.
- Account for 1/2 the bulk of CNS
- Function to support the neurons and their processes.
- Electrically insulate neurons from each other, and help maintain balance on the fluid environments of the neurons.
- Schwann cells also called a neurilemma are one type of neuroglial cells. found in the PNS.
- Similar cells called oligodendrocytes serve the same function in the CNS
- Schwann cells form an insulating layer of myelin around many nerve processes.
- Schwann cells extend along the length of the axons of most motor neurons.
- Schwann cells are important in healing damaged nerves. Are found to some extent on all axons--even unmyelinated ones.

- How Neuroglial cells and nerve cells are related
  - One neurilemma may "wrap" about several un-myelinated axons.
  - These are considered unmyelinated axons.
  - If neurilemma wrap around one axon like a jelly role is considered myelinated axon.
  - Each neuroglial cell is about one mm of the axon about 1 mm in width.
  - Junctions between cells called the Nodes of Ranvier.
Neuron Excitation Conduction

- The whole environment of the neuron includes
  - intracellular fluid --inside the neuron's cell body, axon and dendrites
  - the cell membrane of the neuron -- (wall housing)
  - extracellular fluid--outside the cell membrane
- Both fluids contain electrolytes, salts (sodium chloride; potassium chloride)
- The concentration of positive ions OUTSIDE the cell is higher than inside.
- There are slightly more negative ions inside the cell. Initially the inside of the nerve fiber has -70 mV voltage. We say the inside of the neuron is negatively charged. We call this electrical charge the resting membrane potential.
- When stimulated to the critical firing level (we'll talk about this in a minute) the membrane becomes permeable to sodium ions. Na+. A bunch of positive sodium ions pass thru the membrane to the inside of the cell. The inside of the cell then becomes positively charged +30 mV.
- This point of excitation called **action potential**, i.e. depolarization.
- Immediately following, the membrane goes into a recovery phase that lasts several milliseconds. This is called **absolute refractory period**. The neuron cannot be further excited--No matter what.
- Followed by a period called **relative refractory period**--meaning a greater than average stimulus is required to reach the critical firing level.

All or none principal. Regardless of strength of initial stimulus, the spike of action potential is same. It either fires completely or not at all. (Example toilet flushing.)

- The action potential begins at axon hillock and travels the length of the axon. It travels faster in myelinated nerves as it jumps from one Node of Ranvier to the next.
- Important concept: A "stronger nervous impulse doesn't come from stronger stimulation on one axon but from many more axons being stimulated."

The Neural Synapse.

Information is relayed by one neuron exciting an impulse in an adjacent neuron.

First discuss the relationship of two neurons....

- Junction between neurons called **synapse**.
- There is a **pre-synaptic** and a **post-synaptic neuron**.
- The place of almost contact is called the **receptor site**. Is generally on dendrite but may be in other places.
- On average each neuron has about 1000 **boutons**. May connect with up to 100,000 other neurons.
- **Synaptic vesicles** located in the bouton have **chemical transmitter substances**.
- These are released into the **synaptic cleft**.
- The vesicles then open to release the **neurotransmitter substance** from the vesicle into the cleft.
- These neurotransmitter substances must act quickly -- within microseconds or they become ineffective at causing a reaction in the adjacent neuron.

**Excitation**

- To excite or cause a nervous impulse to be sent, a special neurotransmitter substance is released. It is called an **excitatory substance**.
- If it is effective it causes the receptor site of the post synaptic neuron to become positively charged. This slight positive charge is called the **excitatory post synaptic potential**--EPSP.
- By itself the EPSP of one neuron may not be enough to cause the next neuron to fire. If several accumulate within microseconds and become enough to stimulate the cell, an action potential...
appears at the axon hillock of the cell body and the impulse is on its way. Remember a neuron won't fire half way. Its all or none.

Inhibition

- Another neurotransmitter substance which may be released into the cleft is an inhibitor.
- It results in an inhibitory post synaptic potential IPSP.
- Reduces the ability of the next neuron to receive a positive electrical charge which is to say it inhibits the next neuron from firing.
- All neurons have many synapse, that is end buttons near them. At any moment it may receive XX IPSPs and XXX EPSPs. Whether or not it fires is the mathematical summation of all the synaptic information. If it reaches the critical level, it fires, if not it doesn't.

Receptors

Sensory nerves end with specialized receptors for pain, touch, pressure, cold, heat, taste, movement, smell, etc.

Neural Kiss
- An easy way to visualize and remember the functions of a synapse is to think of it as a 'neural kiss'.
  - Like a kiss, the synapse...
  - It communicates information.
  - Does not make a permanent commitment.
  - It may be excitatory or inhibitory.