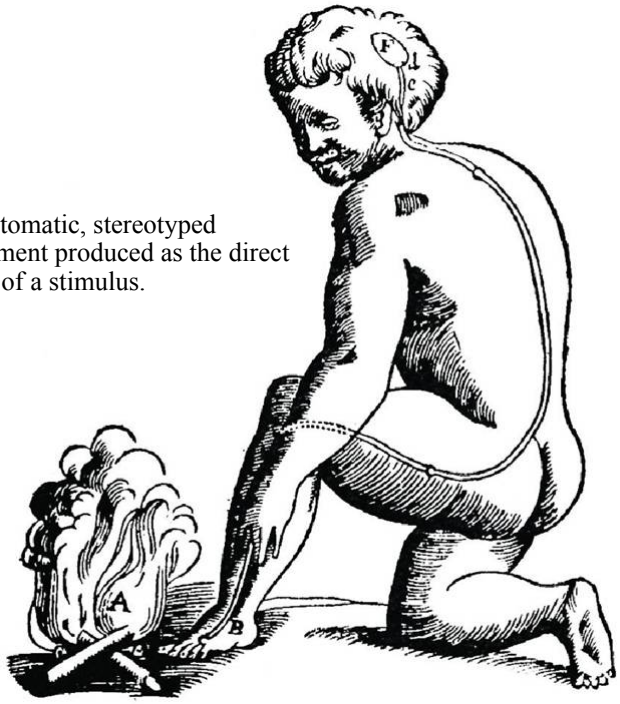
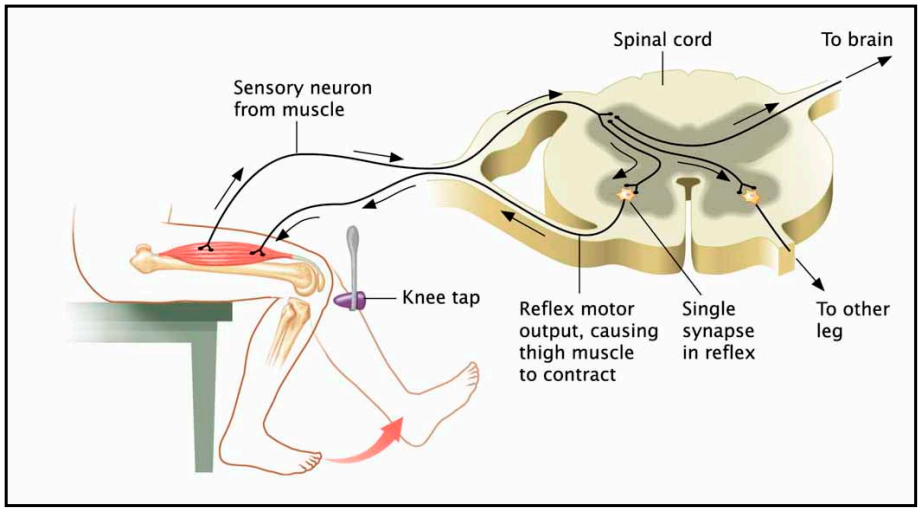
Reflex:

An automatic, stereotyped movement produced as the direct result of a stimulus.



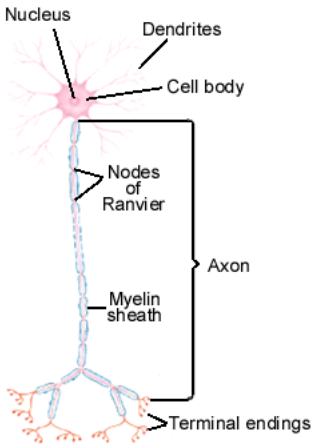
A reflex pathway as we know it now

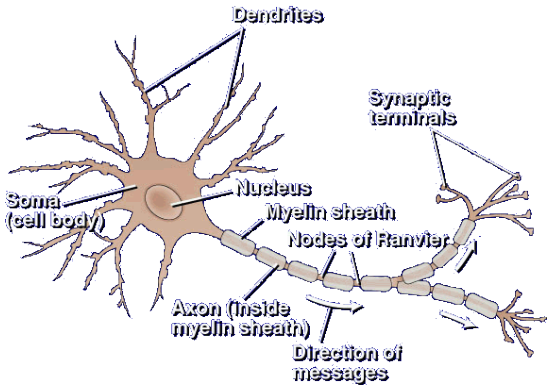


The Neuron

number: 10 billion to a trillion
(most popular estimate is 86 billion)
10,000 connections each

parts: dendrites
cell body (or "soma")
axon
terminal endings (or terminal buttons)





Questions…

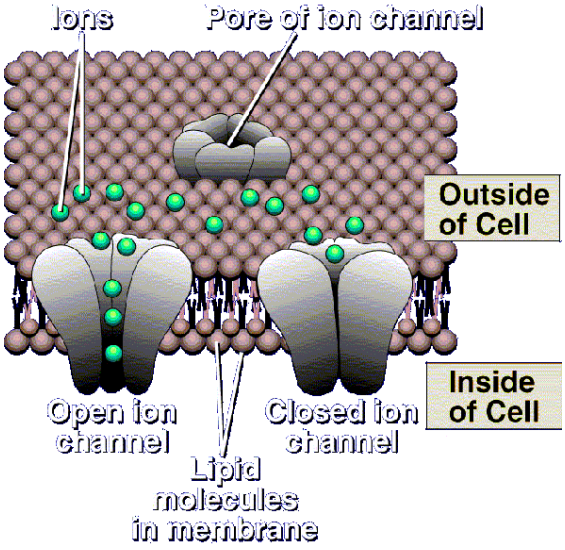
1) how does a neuron "fire"? (what is the nerve impulse?)

2) how does it cause the next neuron to fire? (how does it communicate?)

nerve impulse = ACTION POTENTIAL:

1) start with electrical RESTING POTENTIAL: inside of cell is 70 mV more negative than outside due to Cl- ions inside and Na + ions outside (so RESTING POTENTIAL is -70 mV).

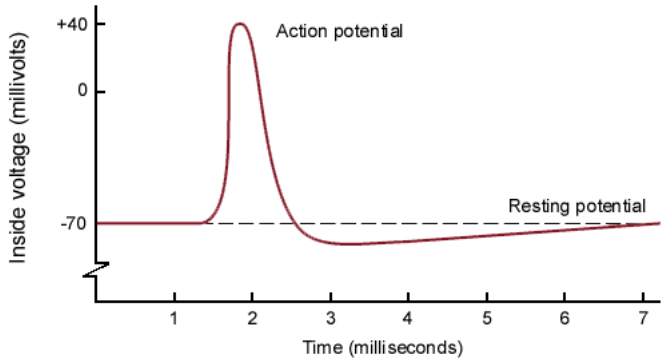
2) stimulation of neuron lets in Na+ ions, which makes the inside more positive: -70,-69,-68,-67...



ACTION POTENTIAL (continued)…

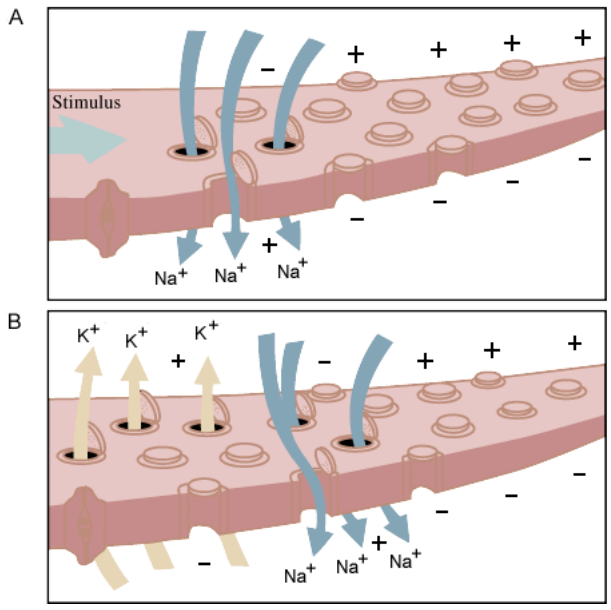
3) when enough Na+ ions get in for the potential to be reduced to -55 mV, suddenly the doors (ion gates) to the cell membrane are flung open allowing Na+ to rush in.

4) so much Na+ enters that the potential doesn't just go to 0 -- it shoots all the way up to +40 mV, so the inside is now positive relative to the outside (the ACTION POTENTIAL)



Action potential (conclusion)

5) ion pumps work to reduce potential back to -70 mV by pushing positive ions out (actually K+ because Na+ goes out slower; then ANOTHER pump takes Na+ back out and puts K+ back in)

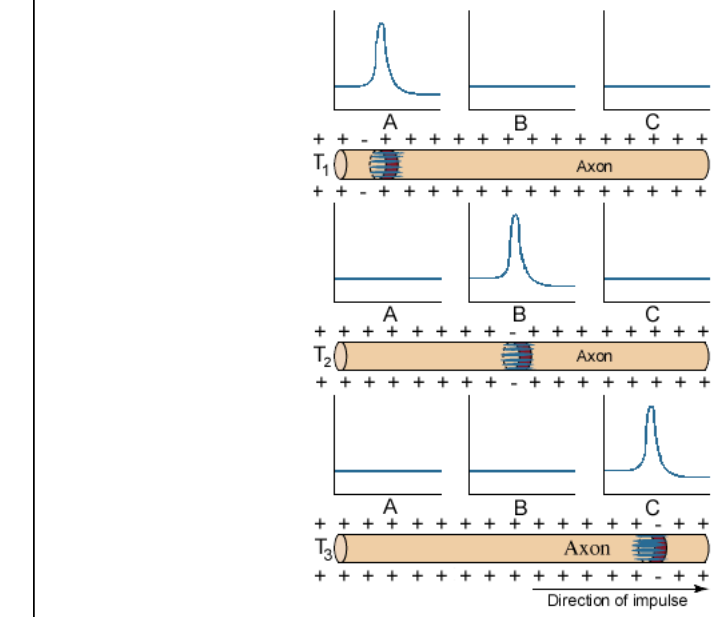


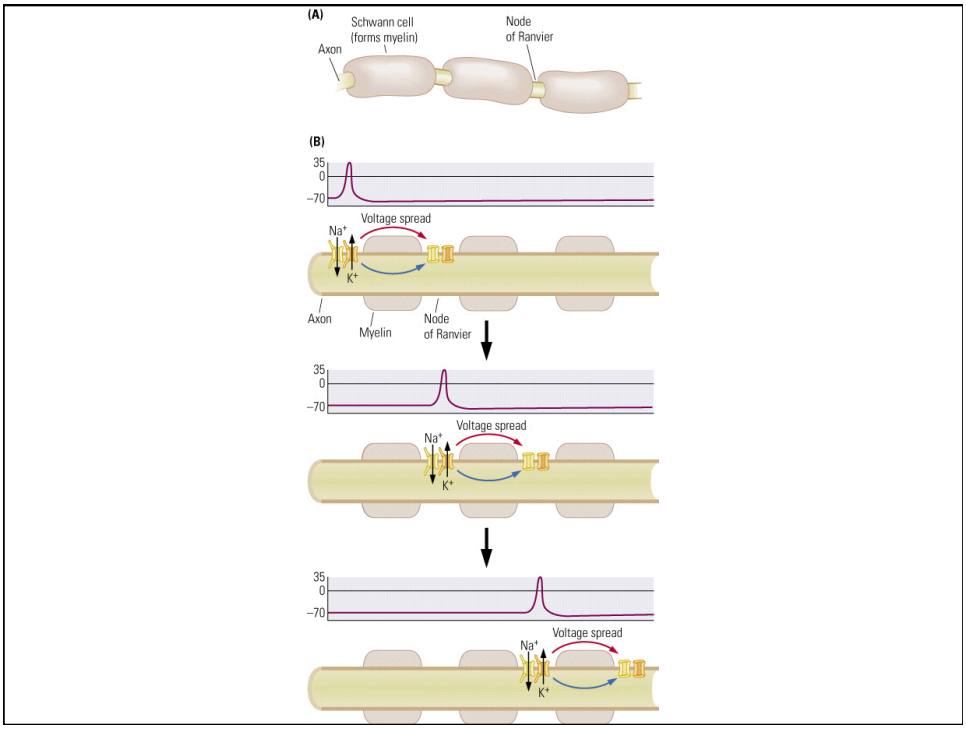
ACTION POTENTIAL (continued)...

- note that -55mV is a threshold: below that voltage there is no action potential - firing is "all-or-none"
- more intense stimulation doesn't cause a more intense action potential -- just more frequent ones (up to 1000/sec!), and in more neurons

ACTION POTENTIAL (continued)…

- action potential travels down length of axon by depolarizing neighboring areas
- travels NOT at speed of electrical current in wire, but rather at about 50 to 100 m/sec


A
B
C
T1
Axon
A
B
C
T2
Axon
A
B
C
T3
Axon
Direction of impulse


(A)
Schwann cell (forms myelin)
Axon
Node of Ranvier
(B)
35
0
−70
Voltage spread
Na+
K+
Axon
Myelin
Node of Ranvier
35
0
−70
Voltage spread
Na+
K+
35
0
−70
Voltage spread
Na+
K+

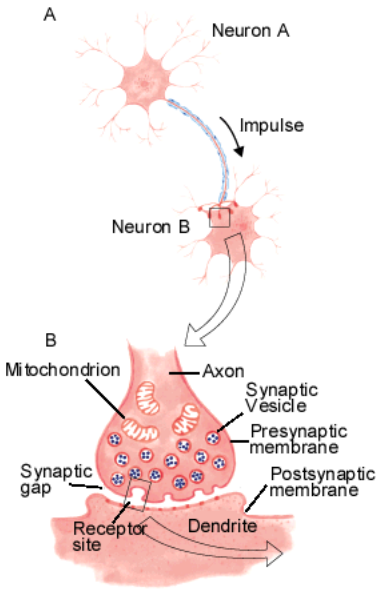
communication across the synapse: NEUROTRANSMITTERS

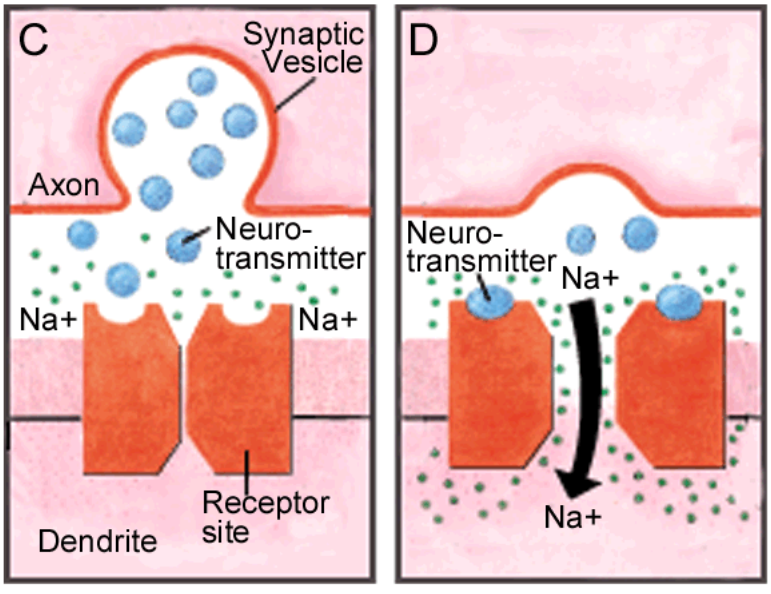
1) synapse is gap between two neurons (the presynaptic and the postsynaptic neurons); terminal endings of presynaptic neuron relay impulse to dendrites of postsynaptic neuron

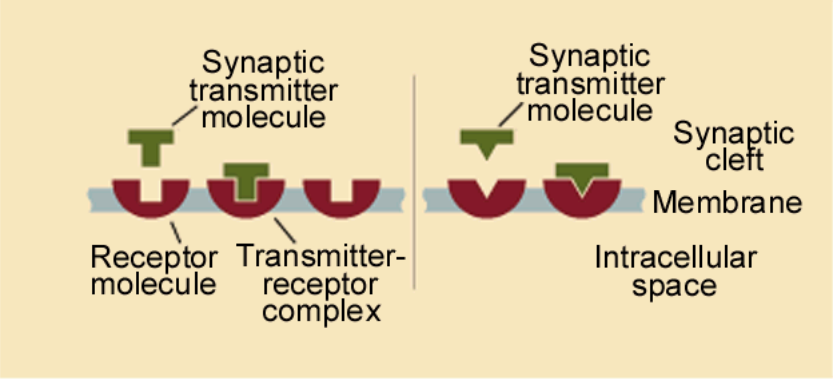
NEUROTRANSMITTERS (continued)

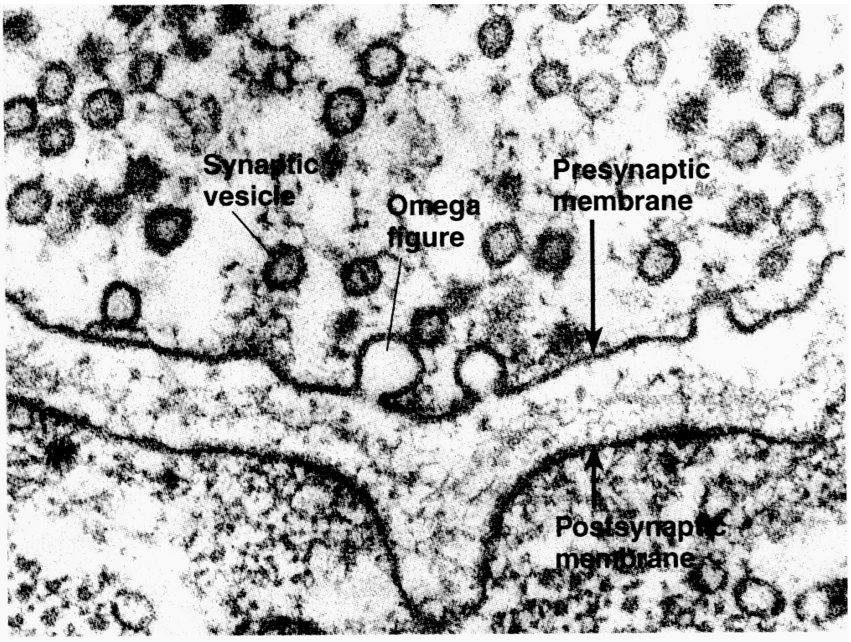
2) terminal buttons contain little sacs ("vesicles") of chemicals ("neurotransmitters"); at action potential, vesicles burst and release neurotransmitters into synapse

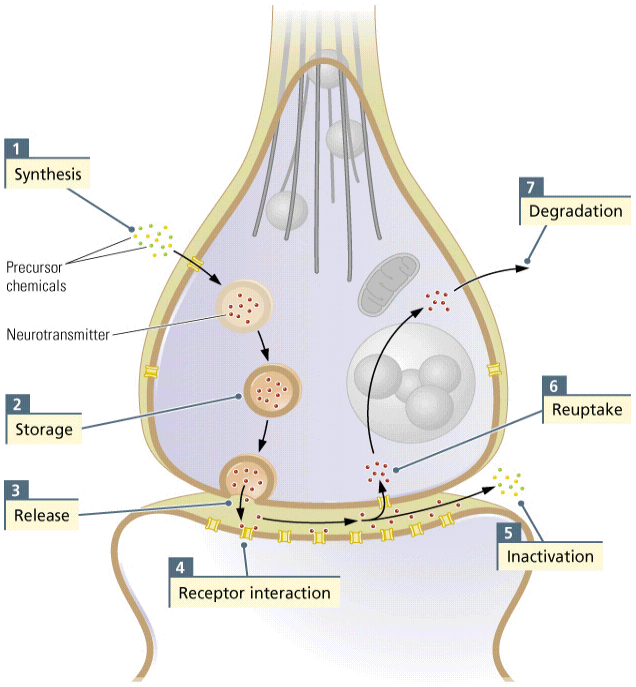
3) receptor molecules on membrane of dendrite are like little locks to be opened: neurotransmitters are the keys, and this is what opens ion gates to allow Na+ inside in the first place


A
Neuron A
Impulse
Neuron B
B
Mitochondrion
Axon
Synaptic Vesicle
Presynaptic membrane
Synaptic gap
Postsynaptic membrane
Receptor site
Dendrite


C
Synaptic Vesicle
Axon
Neuro-transmitter
Na+
Na+
Receptor site
Dendrite
D
Neuro-transmitter
Na+
Na+


Synaptic transmitter molecule
Synaptic transmitter molecule
Synaptic cleft
Membrane
Receptor molecule
Transmitter-receptor complex
Intracellular space

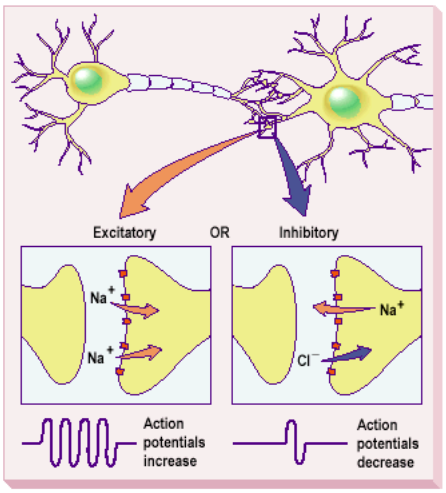

Synaptic vesicle
Omega figure
Presynaptic membrane
Postsynaptic membrane

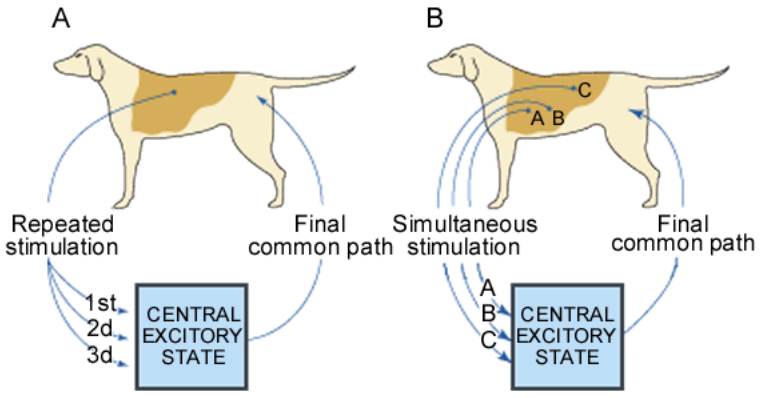


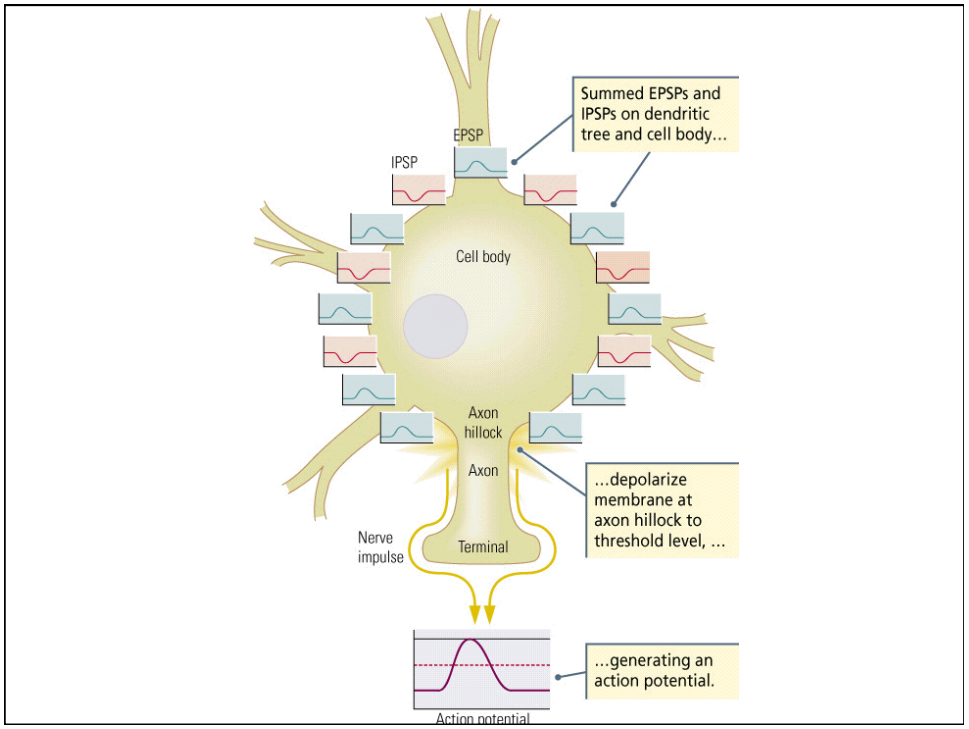
NEUROTRANSMITTERS (continued…)

4a) neurotransmitters may open a gate to let Na+ inside: excitatory (more likely to fire) because potential is getting smaller, toward -55

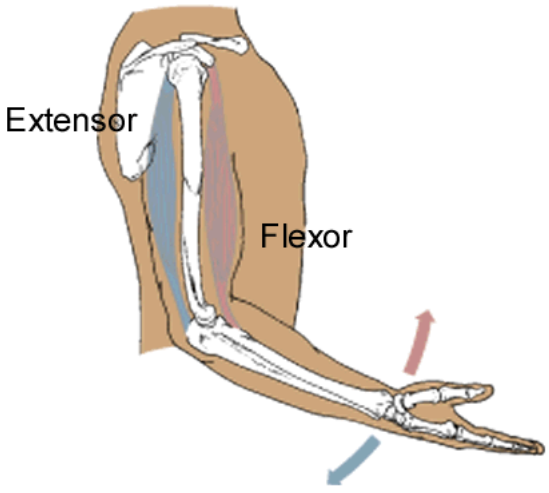
4b) or they may open a gate that pushes positive K + ions out: inhibitory (less likely to fire) because potential is getting larger (e.g., -70, -71, -72...)


Excitatory
OR
Inhibitory
Na+
Na+
Na+
Cl−
Action potentials increase
Action potentials decrease


A
B
Repeated stimulation
Final common path
1st
2d
3d
CENTRAL EXCITORY STATE
Simultaneous stimulation
Final common path
C
A B
A
B
C
CENTRAL EXCITORY STATE



Reciprocal Inhibition

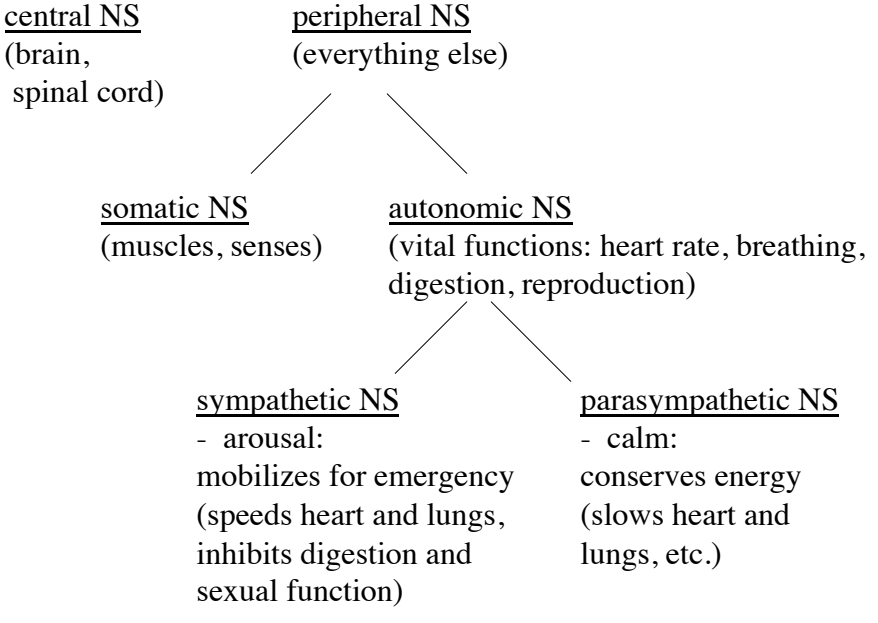


NERVOUS SYSTEM ("NS")

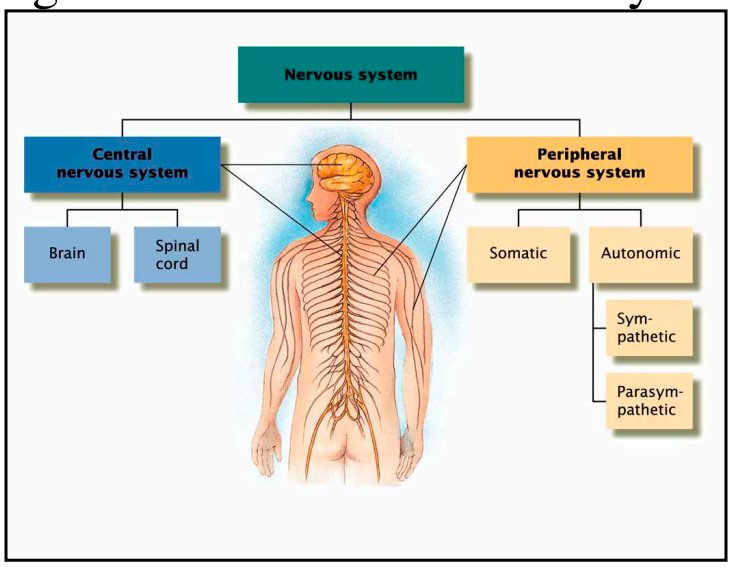
central - "center"
peripheral - "outside of center"

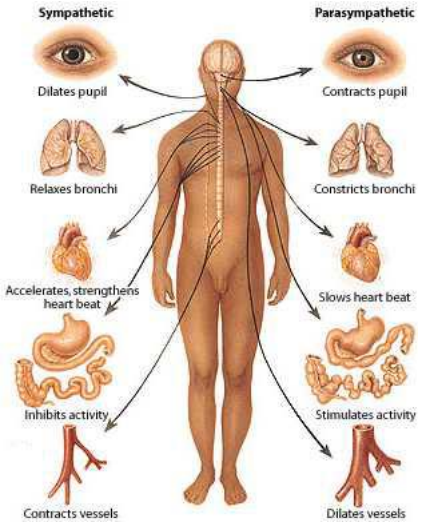
somatic - "body"
autonomic - "self rule"

sympathetic - excited states
parasympathetic - vegetative, calm states



Organization of the Nervous System





BRAIN: bottom to top (=inside to outside=old to new)

hindbrain:

medulla - breathing, heartbeat, blood circulation

pons - arousal and attention

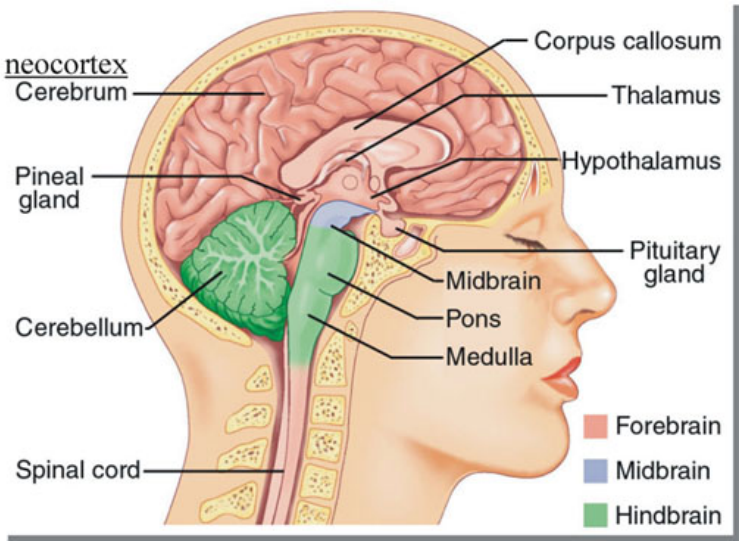
cerebellum - integration of muscles to perform fine movements, but no coordination / direction of these movements; balance

- cat transected above hindbrain: can move but not act

midbrain: forms movements into acts; controls whole body responses to visual and auditory stimuli

- cat transected above midbrain can act, but without regard to environment: without purpose

forebrain…



BRAIN (continued)

forebrain:

thalamus - sensory and motor relay center (to various cerebral lobes)

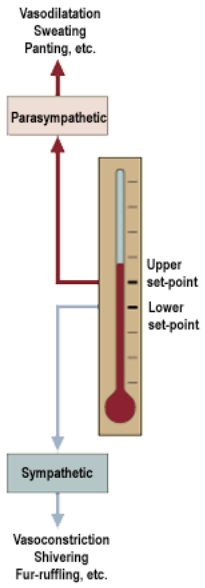
hypothalamus - controls responses to basic needs (food, temperature, sex)

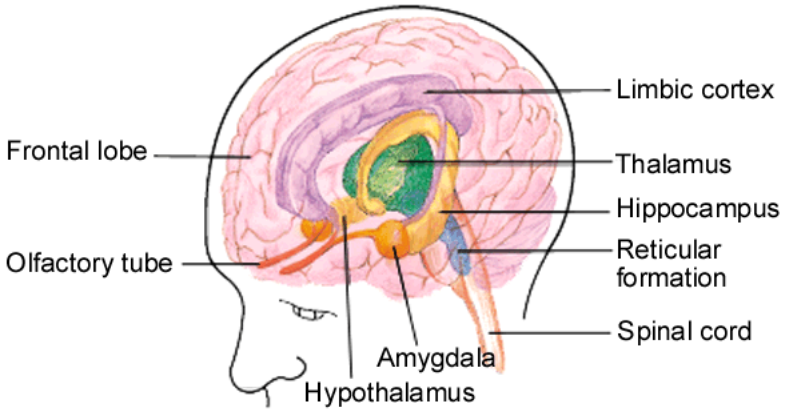
basal ganglia - regulates muscle contractions for smooth movements

limbic system - memory (hippocampus) and emotion (amygdala)

cerebral cortex (or "neocortex") - four lobes (frontal, parietal, occipital, temporal); seat of "higher" intellectual functions

- cat transected above limbic system: acts normal, with purpose -- but clumsy



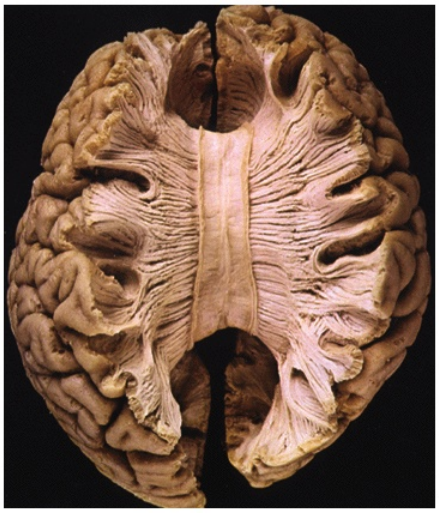


CEREBRAL HEMISPHERES (or CEREBRUM):

corpus callosum: connects hemispheres
- each hemisphere controls OPPOSITE SIDE of body

cerebral cortex (= skin or bark):
- 1 to 3 mm thick; 2 or 3 ft square if flattened out
- higher motor, sensory, and intellectual functions

Corpus Callosum



- large band of neural fibers
- largest "commissure" (or pathway between hemispheres) of the brain
- but not the ONLY one!

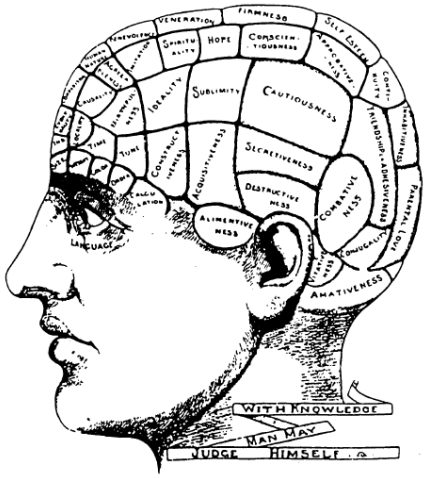
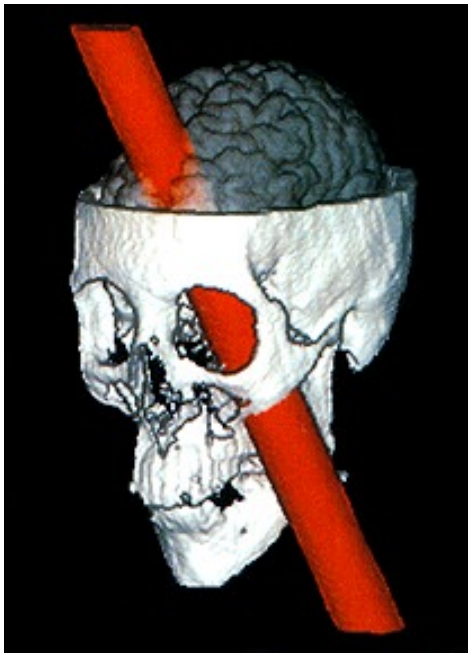


FIGURE 14-2. Originally Gall's system had 27 faculties. As phrenology (Spurzheim's name for the theory) expanded, the number of faculties increased. The figure shows the location of faculties according to Spurzheim. Language, indicated in the front of the brain (below the eye), actually derived from a case study of Gall's: a soldier had received a knife wound that penetrated the frontal lobe of his left hemisphere through the eye. The soldier became aphasic; he was thus the first comprehensive report of aphasia following left frontal damage.



localization of cortical function

- Phineas Gage (1848) – personality in brain
  - explosion sent iron rod through skull removing part of frontal lobe (probably pre-frontal area)
  - resulting personality change from responsible and gentle to argumentative, impulsive and vile
  - but this description is probably exaggerated
- Paul Broca (1865) – identified region in patient's brain responsible for speech
  - Carl Wernicke (1874) – identified separate region responsible for comprehension

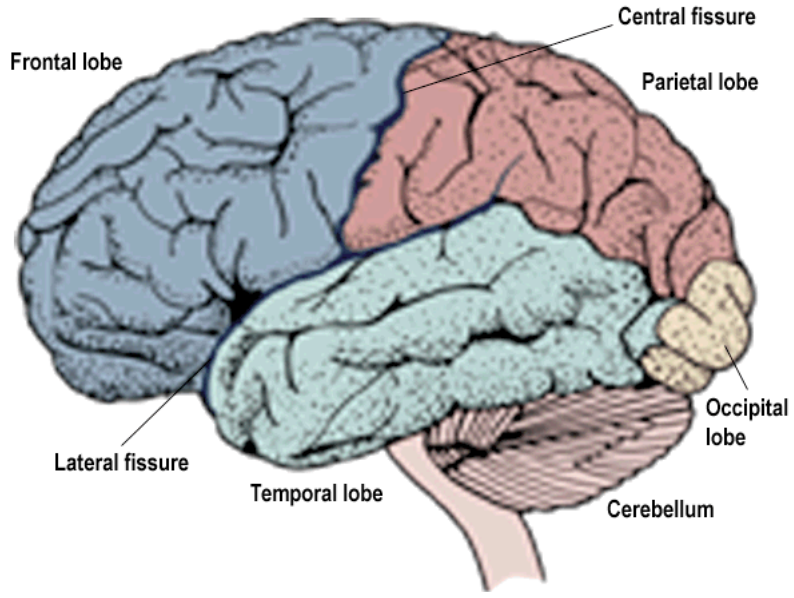
FOUR LOBES of cortex:

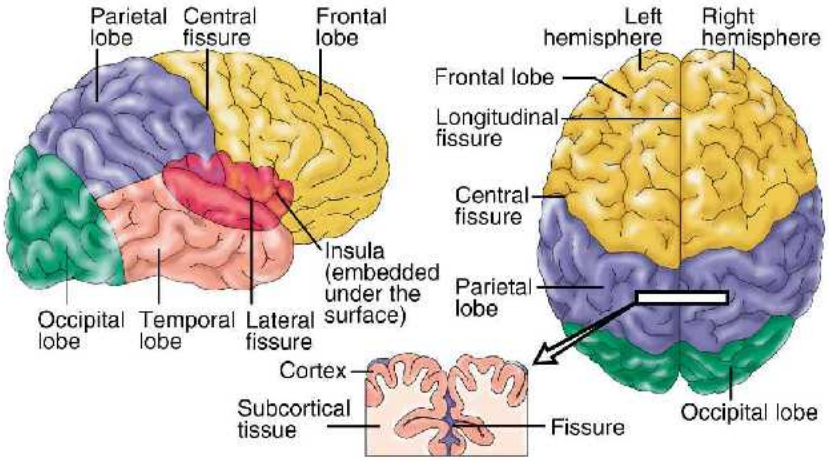
frontal lobe: planning; social behavior; motor control
- front of brain

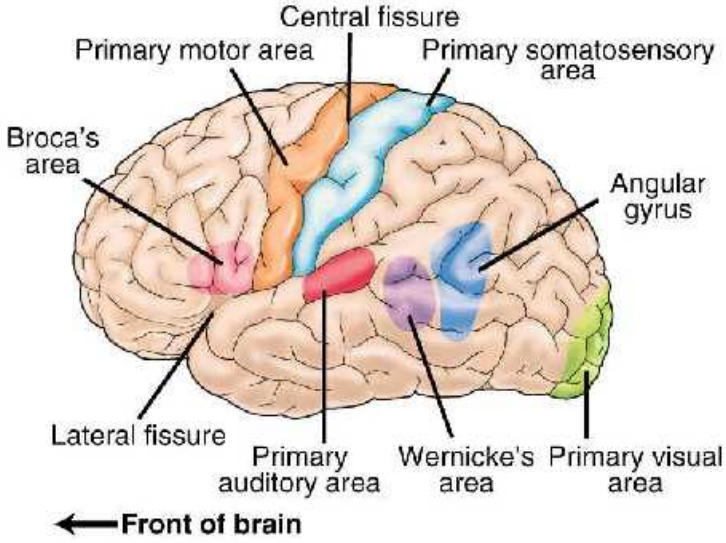
parietal lobe: somatosensory (sense of touch)
- on top and toward back of brain

occipital lobe: vision
- back of brain

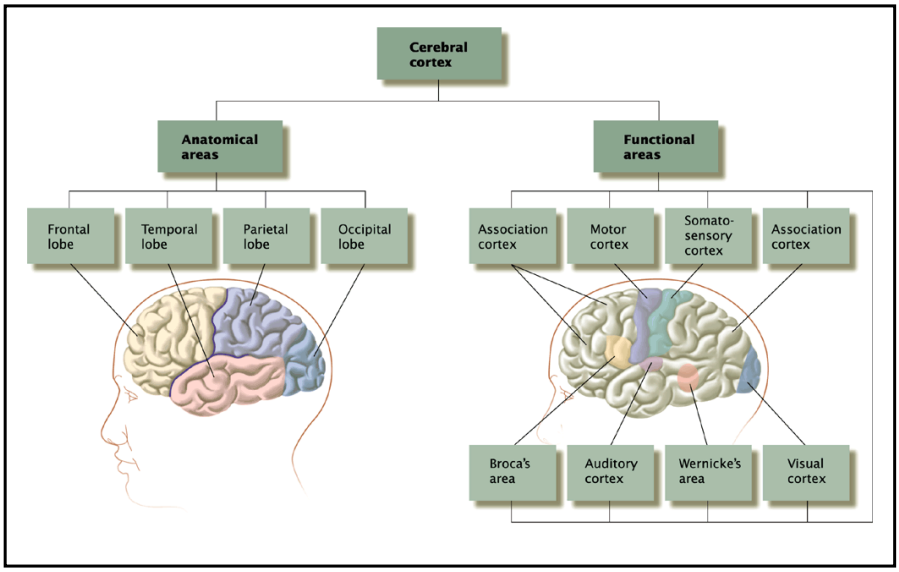
temporal lobe: hearing; memory
- side of brain

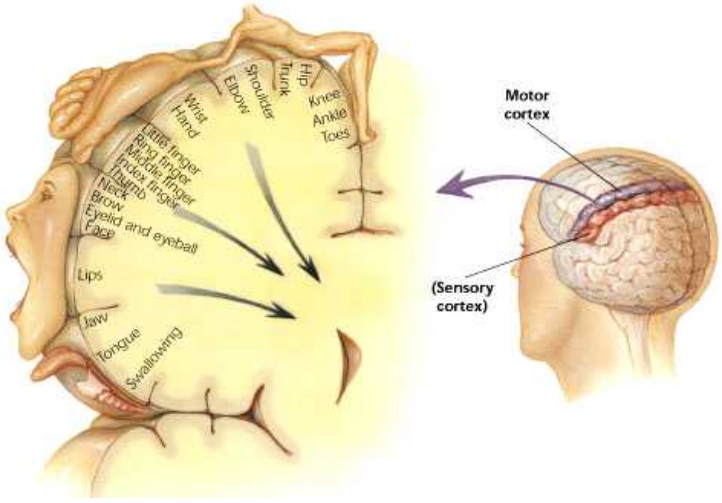



Parietal lobe
Central fissure
Frontal lobe
Insula (embedded under the surface)
Occipital lobe
Temporal lobe
Lateral fissure
Left hemisphere
Right hemisphere
Frontal lobe
Longitudinal fissure
Central fissure
Parietal lobe
Occipital lobe
Cortex
Subcortical tissue
Fissure

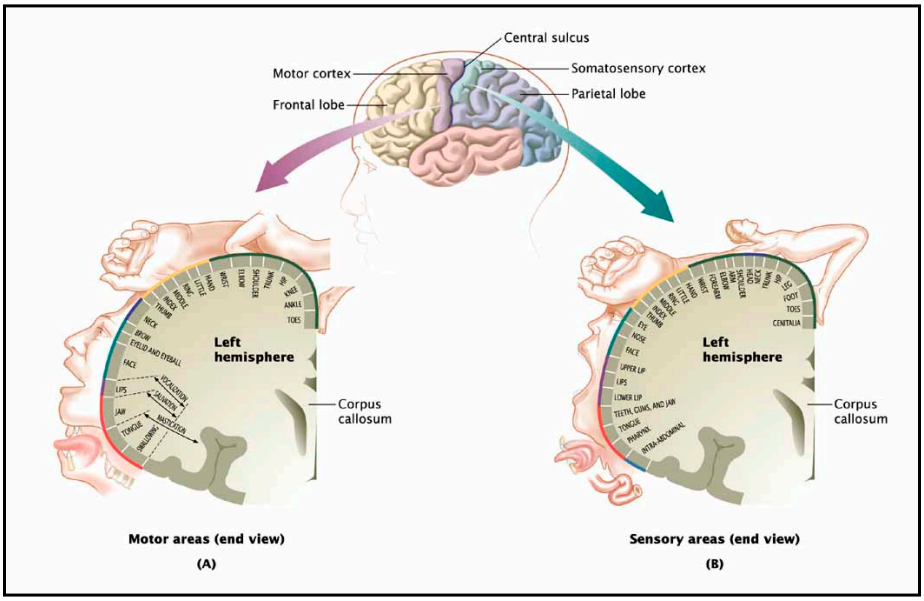

Central fissure
Primary motor area
Primary somatosensory area
Broca's area
Angular gyrus
Lateral fissure
Primary auditory area
Wernicke's area
Primary visual area
Front of brain

The Cerebral Cortex





Motor and Somatosensory Cortex





TWO GENERAL RULES of cortical function:

1. **Left** Hemisphere: language
   Right Hemisphere: spatial abilities

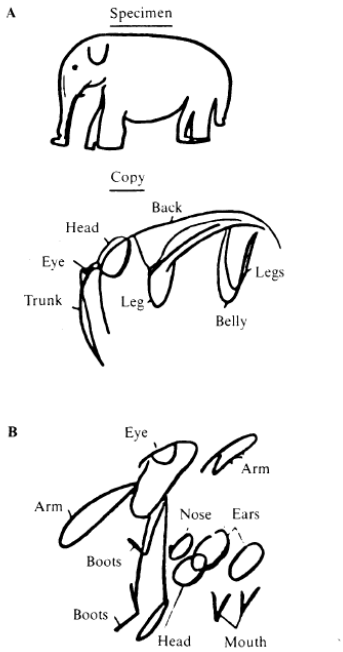
2. **Front**: expression / actions / plans
   Back: reception / perceptions / interpretations

DAMAGE TO NON-PRIMARY ("ASSOCIATION") CORTEX:

pre-frontal lesions: loss of planning, moral reasoning, sensitivity to social context
- or... loss of initiation of action, deliberation

apraxia ("no doing"): failure in sequencing components of actions; inability to organize movements
- FRONTAL - lesions just forward of motor cortex
- NOT paralysis, as from motor cortex lesion

agnosia ("no knowing"): - deficit in interpreting, categorizing, labeling, knowing
- OCCIPITAL (visual) or TEMPORAL (auditory) lesions
- sensory systems themselves (e.g., eyes) are okay

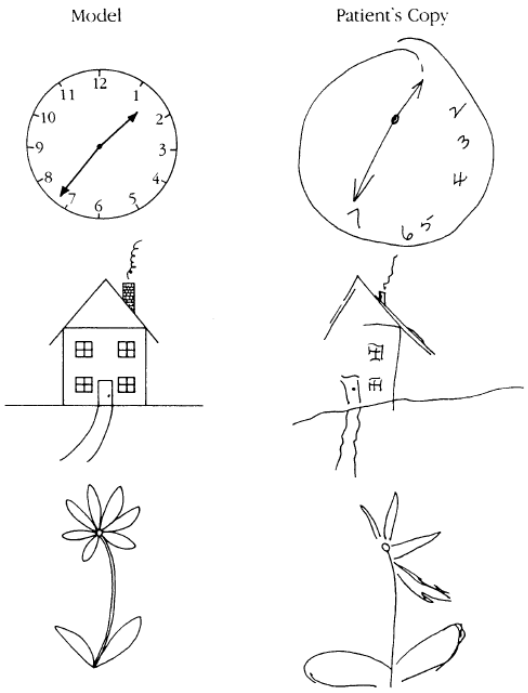


2.33 Drawings by a patient with visual agnosia

DAMAGE TO NON-PRIMARY ("ASSOCIATION") CORTEX (continued):

neglect: RIGHT hemisphere (PARIETAL) damage causes inattention to whole left side

aphasia: LEFT hemisphere (FRONTAL or TEMPORAL) damage causes deficits in language function...



APHASIA: disorder of language

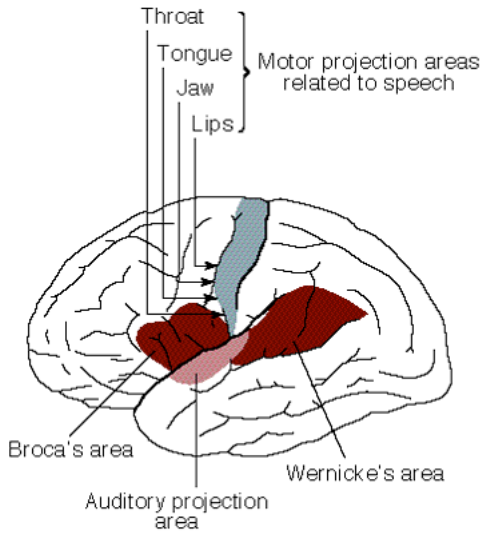
- **left hemisphere** brain lesions
- essentially *apraxia* (from frontal lesion) or *agnosia* (from back lesion) of language

expressive aphasia: cannot produce speech

- lesion to BROCA'S AREA (frontal assoc. area)

receptive aphasia: cannot understand speech - and consequently cannot produce speech!

- lesion to WERNICKE'S AREA (temporal assoc. area)



SPLIT BRAIN STUDIES

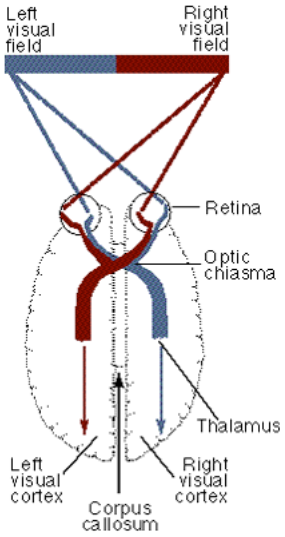
- sever corpus callosum to reduce severity of seizures
- leaves patient mostly normal, but with left and right brain independent in subtle ways

note visual pathways:

left side of each eye sends info to left hem.
right side of each eye sends info to right hem.

- result:

left visual field goes to right hem.
right visual field goes to left hem.



SPLIT BRAIN STUDIES (continued)

experiment on split brain patient:

- patient looks straight ahead; picture flashed quicker than eyes can move; ask "what did you see?"
- picture of cup on right: LH says "cup"
  picture of spoon to left: LH says"nothing"
- BUT when told to reach for that object with the left hand, RH grabs spoon
- ask "what is it?" and LH guesses "pencil" (and RH may frown at that)

Apparatus for Studying Split-Brain Patients

