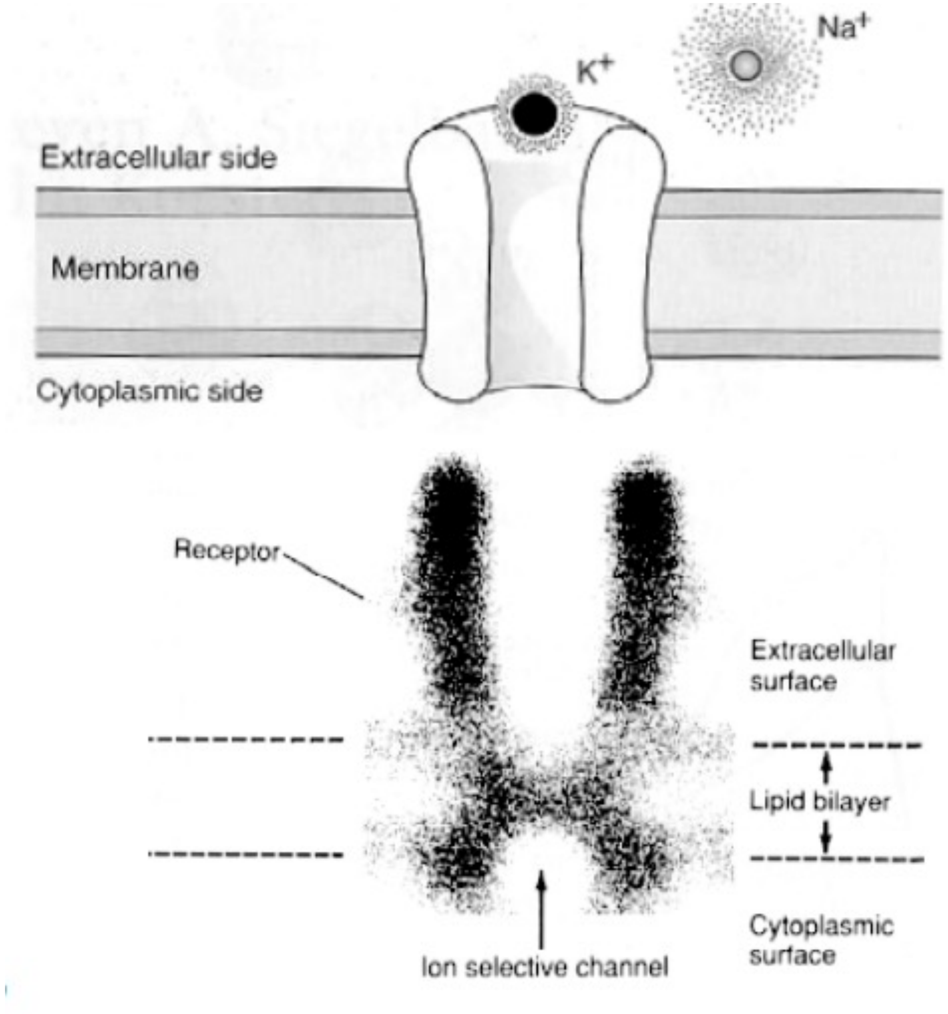
BCI-S2022

Basics of Neuroscience for BCI

Ionic Channels: The Gatekeepers

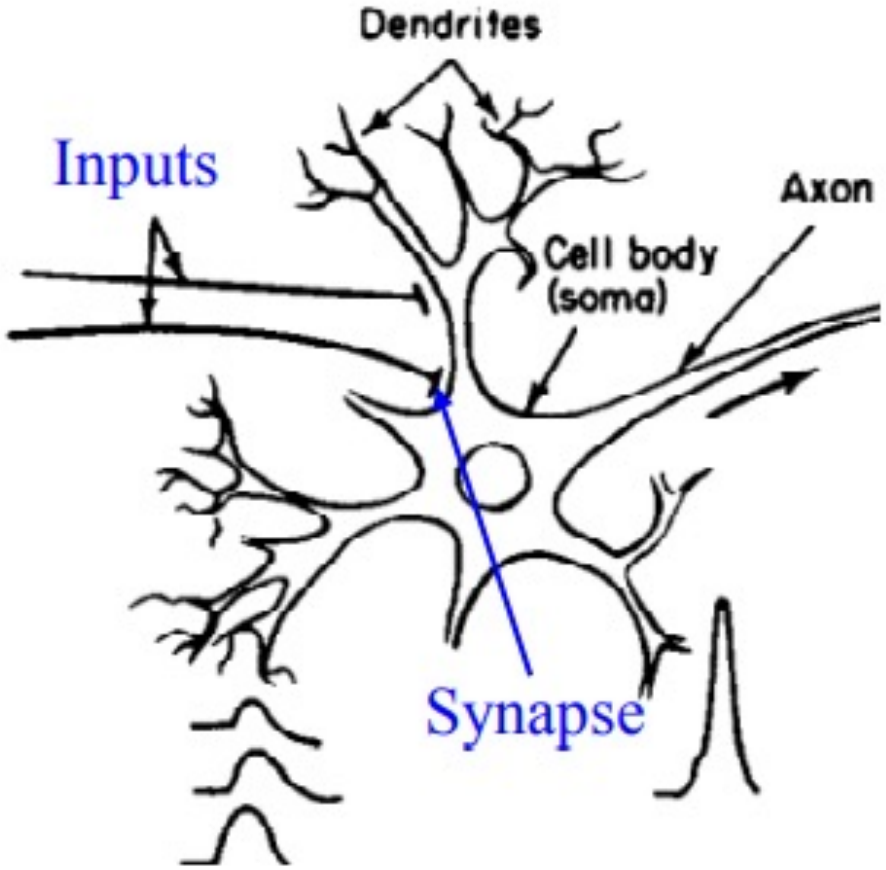
- Changes in membrane potential occur because neurons contain **gated ion channels** that open or close in response to stimuli
- Proteins in membranes act as channels that allow specific ions to pass through.
  - E.g.: Pass K+ but not Cl- or Na+
- These **IONIC CHANNELS** are gated
  - Voltage-gated: Probability of opening depends on membrane voltage
  - Chemically-gated: Binding to a chemical causes channel to open
  - Mechanically-gated: Sensitive to pressure or stretch



From Kandel, Schwartz, Jessel, Principles of Neural Science, 3rd edn., 1991, pgs. 68 & 137

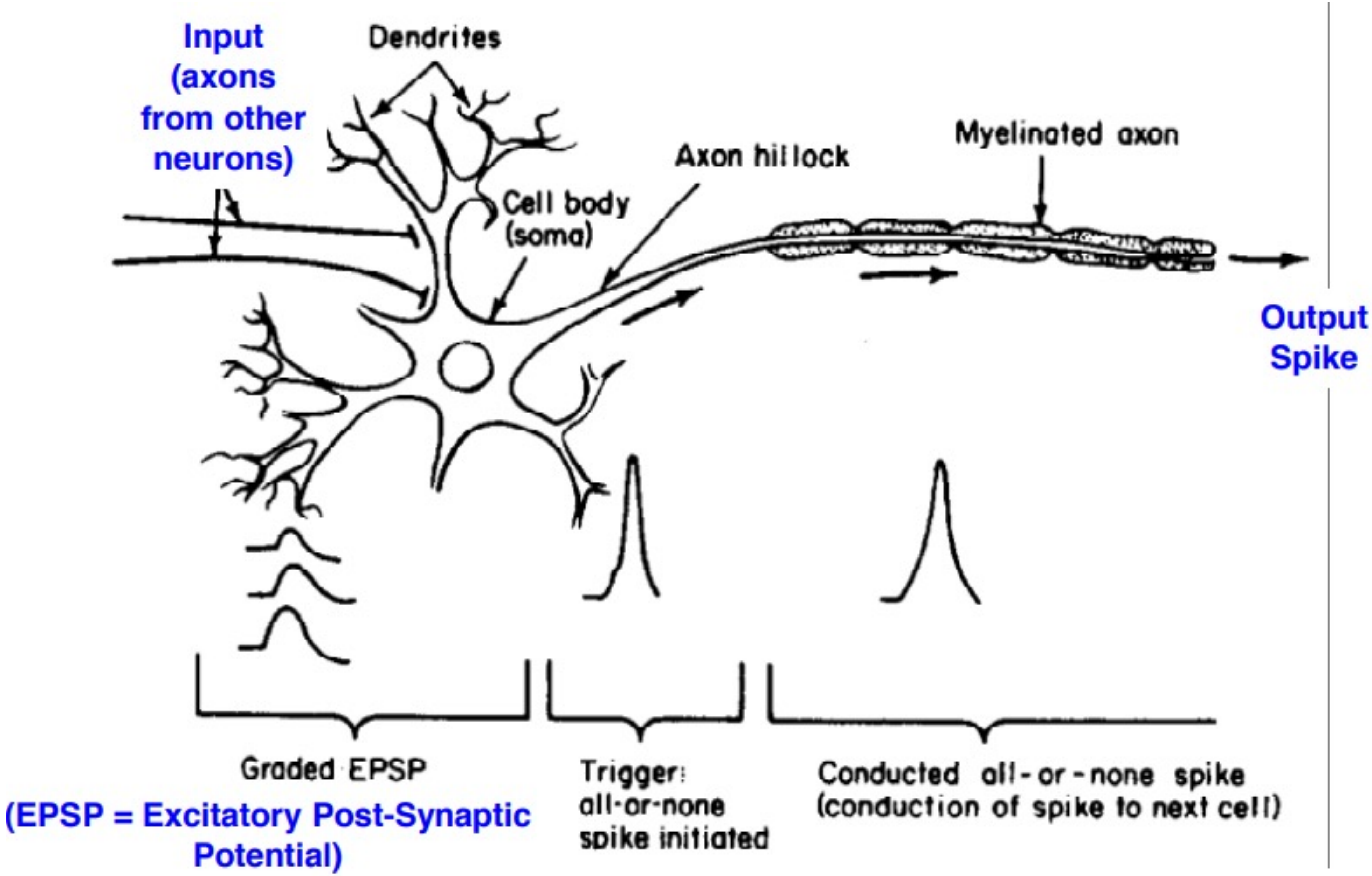
Gated Channels allow Neuronal Signaling

- Inputs from other neurons -> chemically-gated channels (at "synapses") -> Changes in local membrane potential
- This causes opening/closing of voltage-gated channels in dendrites, body, and axon, resulting in depolarization (positive change in voltage) or hyperpolarization (negative change)



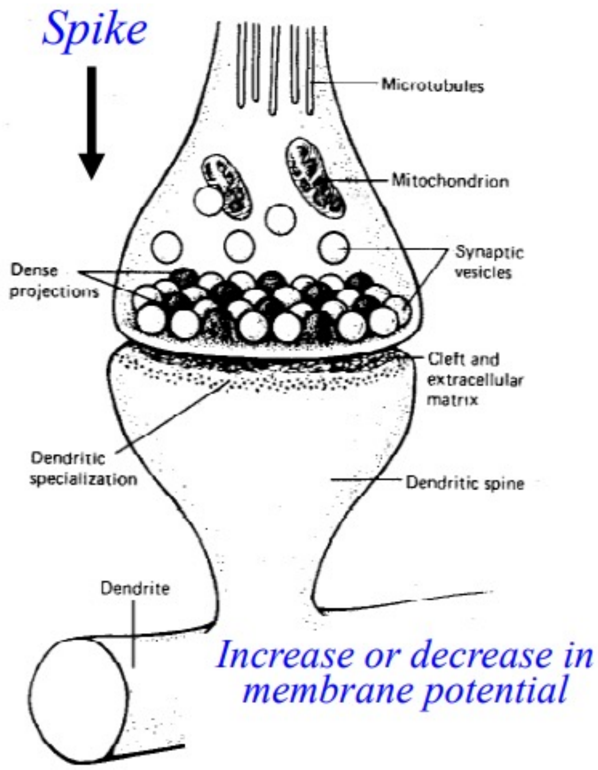
Regions of Neurons

- Neurons in different regions of the brain have different morphological structures
- The typical structure includes a cell body (called the soma) connected to a tree-like structure with branches called dendrites
- A single branch called the axon that emanates from the soma and conveys the output spike to other neurons.
- The spike is typically initiated near the junction of the soma and axon and propagates down the length of the axon.
- Many axons are covered by myelin, a white sheath that significantly boosts the speed of propagation of the spike over long distances.


Input
(axons
from other
neurons)
Dendrites
Cell body
(soma)
Axon hillock
Myelinated axon
Output
Spike
Graded EPSP
(EPSP = Excitatory Post-Synaptic
Potential)
Trigger:
all-or-none
spike initiated
Conducted all-or-none spike
(conduction of spike to next cell)

Synapse

- Neurons communicate with each other through connections known as synapses.
- Synapses can be electrical but are more typically chemical.
- A synapse is essentially a gap or cleft between the axon of one neuron (called the presynaptic neuron) and a dendrite (or soma) of another neuron (called the postsynaptic neuron)



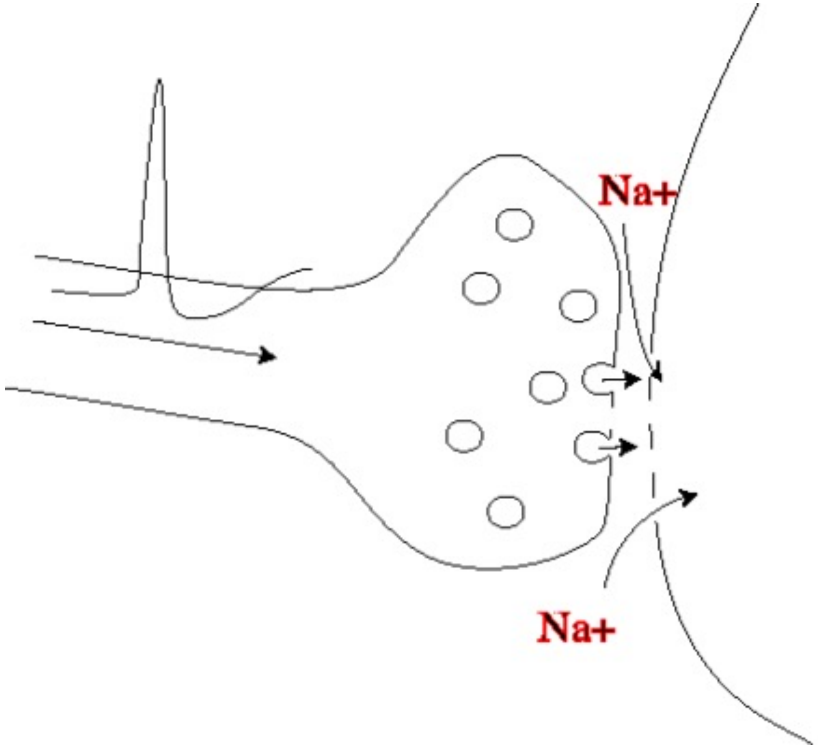
Synapse

- When an action potential arrives from a presynaptic neuron, it causes the release of chemicals known as neurotransmitters into the synaptic cleft.
- These chemicals in turn bind to the ionic channels (or receptors) on the postsynaptic neuron, causing these *<u>channels to open</u>*, thereby influencing the local membrane potential of the postsynaptic cell.

Synapse

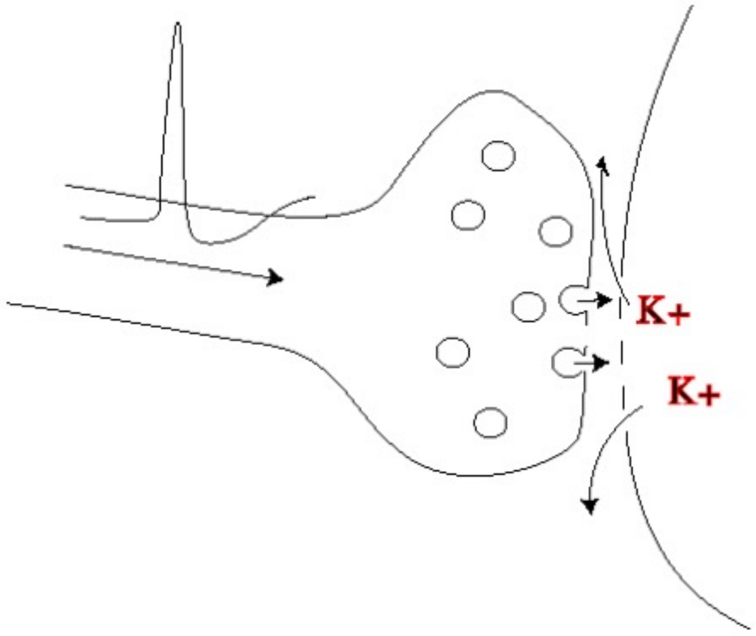
- Synapses can be excitatory or inhibitory.
- Excitatory synapses cause a momentary increase in the local membrane potential of the postsynaptic cell.
  - This increase is called an excitatory postsynaptic potential (EPSP).
  - EPSPs contribute to a higher probability of firing a spike by the postsynaptic cell.
- Inhibitory synapses do the opposite, temporarily decrease the local membrane potential of the postsynaptic cell
  - They cause inhibitory postsynaptic potentials (IPSPs)
- A neuron is called excitatory or inhibitory based on the kind of synapse it forms with *postsynaptic neurons*

An Excitatory Synapse



Input spike →
Neurotransmitter release →
Binds to Na channels (which open) →
Na+ influx →
Depolarization due to EPSP (excitatory postsynaptic potential)

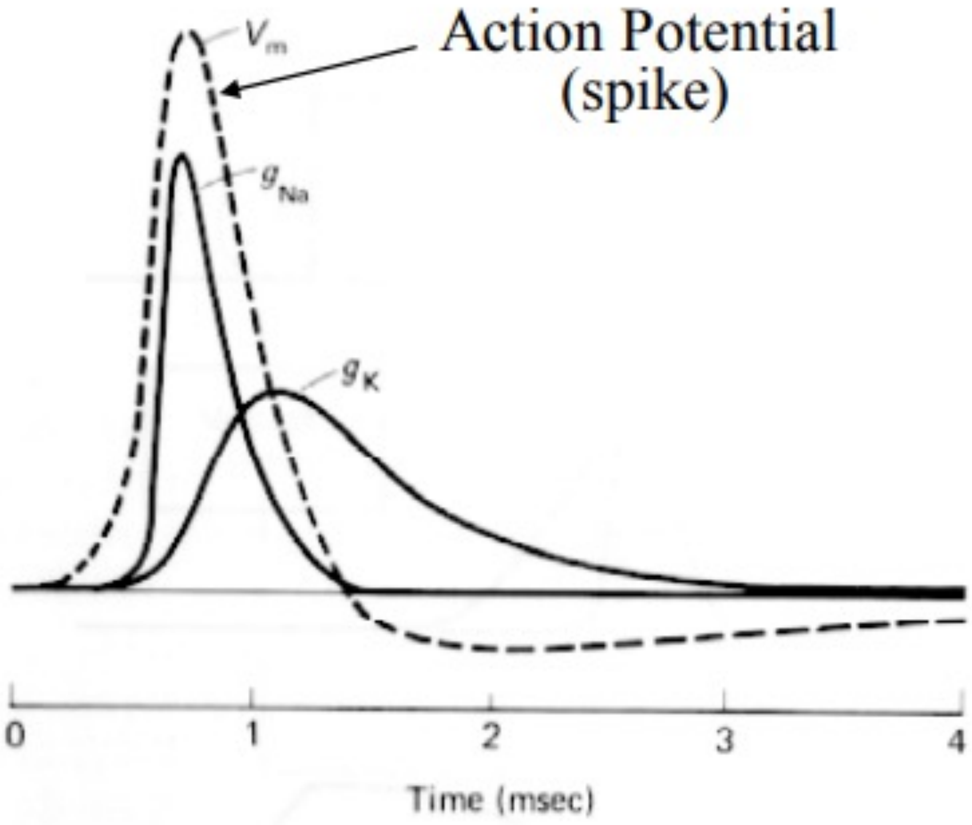
An Inhibitory Synapse



Input spike →
Neurotransmitter
release →
Binds to K
channels →
K+ leaves cell →
Hyperpolarization due
to IPSP (inhibitory
postsynaptic potential)

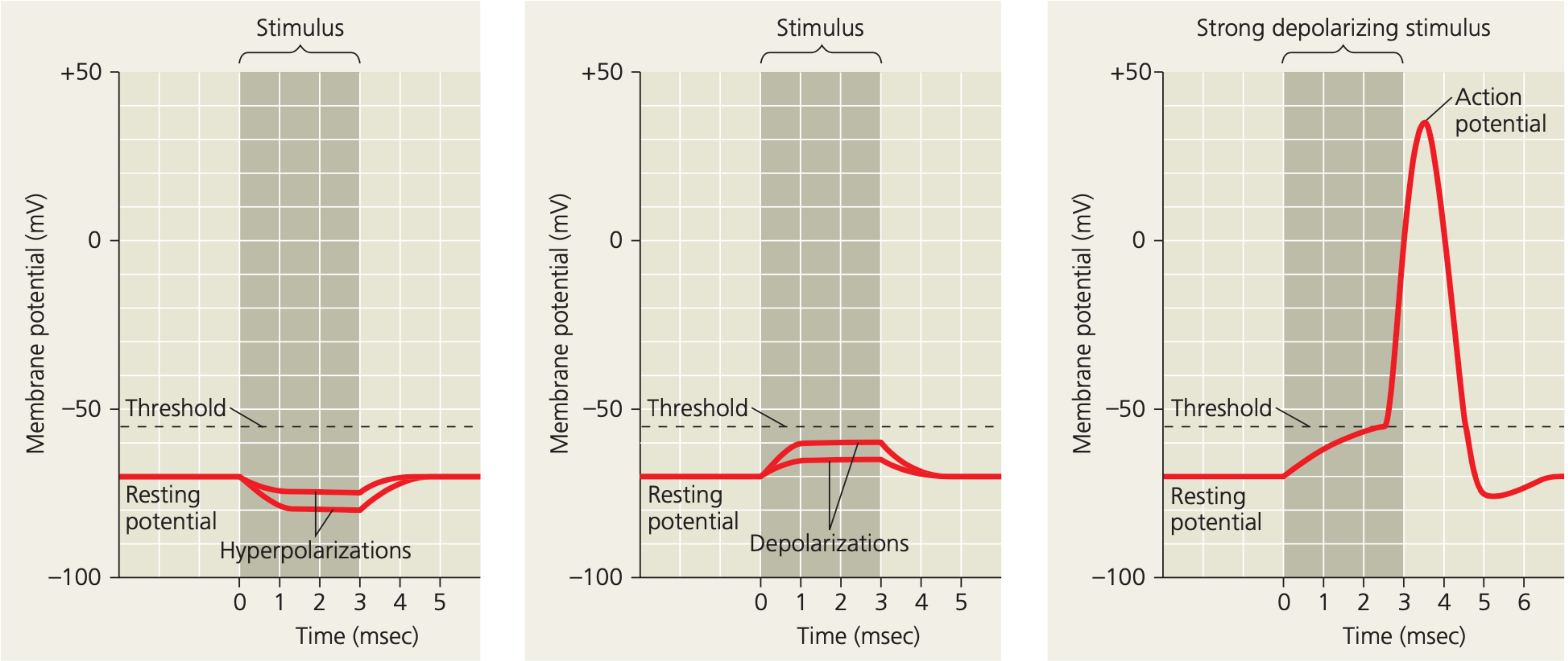
Action Potential or Spikes

- When the neuron receives sufficiently strong inputs from other neurons a cascade of events is triggered
- Rapid influx of Na+ ions into the cell
  - *Causing the membrane potential to rise rapidly.*
- The opening of K+ channels triggers the outflux of K+ ions
  - *Causing a drop in the membrane potential.*
- This rapid rise and fall of the membrane potential is called an ***action potential*** or **spike** and represents the dominant mode of communication between one neuron and another.



From Kandel, Schwartz, Jessel, Principles of Neural Science, 3rd edn., 1991, pg. 110

Graded potentials and an action potential in a neuron.



(a) Graded hyperpolarizations produced by two stimuli that increase membrane permeability to K^+. The larger stimulus produces a larger hyperpolarization.

(b) Graded depolarizations produced by two stimuli that increase membrane permeability to Na^+. The larger stimulus produces a larger depolarization.

(c) Action potential triggered by a depolarization that reaches the threshold.

Spike Generation

- The generation of a spike by a neuron involves a complex cascade of events involving sodium and potassium channels
- This process can be simplified into a simple threshold model of spike generation.
  - *When the neuron receives sufficiently strong inputs from its synapses for its membrane potential to cross a neuron-specific threshold, a spike is emitted.*

Synaptic plasticity: Adapting the connections

- **Long Term Potentiation (LTP):** Increase in synaptic strength of a synaptic connection between two neurons caused by correlated firing of the two neurons
  - lasts for several hours or more.
- Measured as an increase in the excitatory postsynaptic potential (EPSP) caused by presynaptic spikes
- LTP has been found in several brain areas including the hippocampus and the neocortex.
- Note: *LTP is regarded as a biological implementation of Donald Hebb's famous postulate (also called Hebbian learning or Hebbian plasticity) that if a neuron A is consistently involved in causing another neuron B to fire, then the strength of the connection from A to B should be increased.*

Synaptic plasticity: Adapting the connections

- **<u>Long-term depression or LTD:</u>** Decrease in the strength of a synaptic connection caused
  - by uncorrelated firing between the two neurons involved.
  - Reduction in synaptic strength that lasts for several hours or more
- LTD has been observed most prominently in the cerebellum, although it also coexists with LTP in the hippocampus, neocortex, and other brain areas

Synaptic plasticity: Adapting the connections

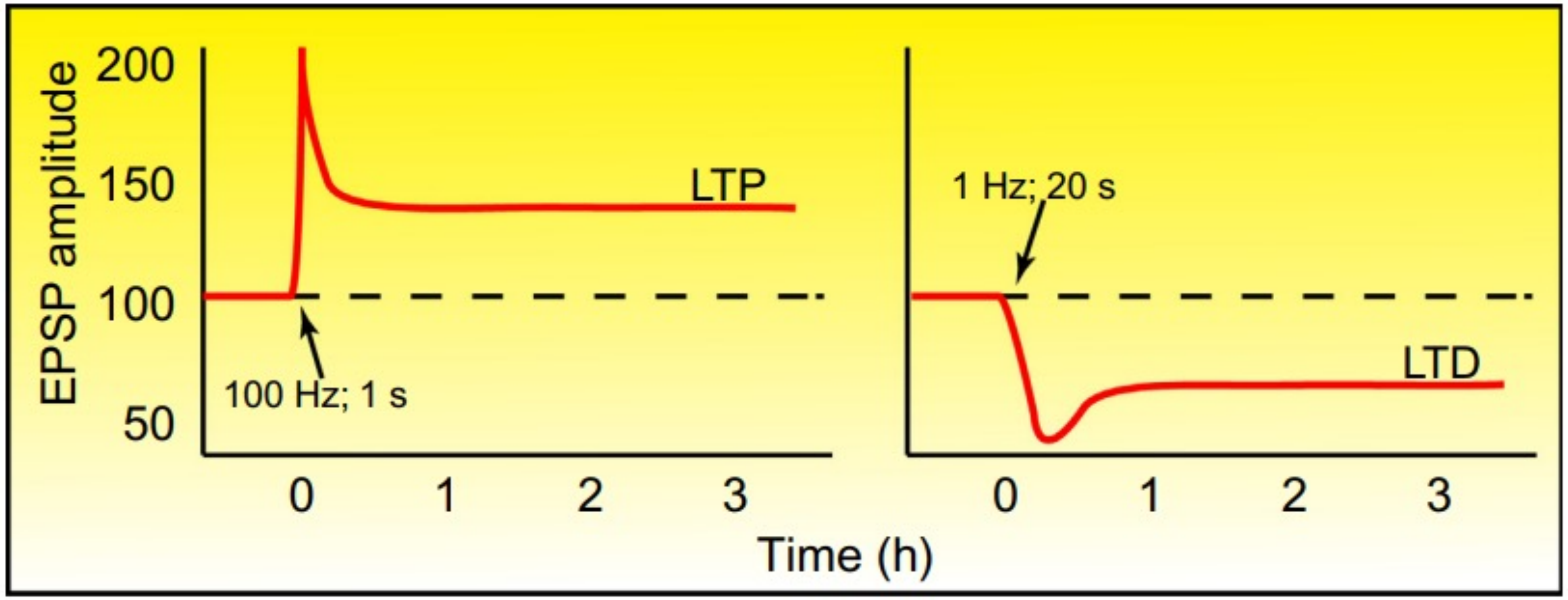
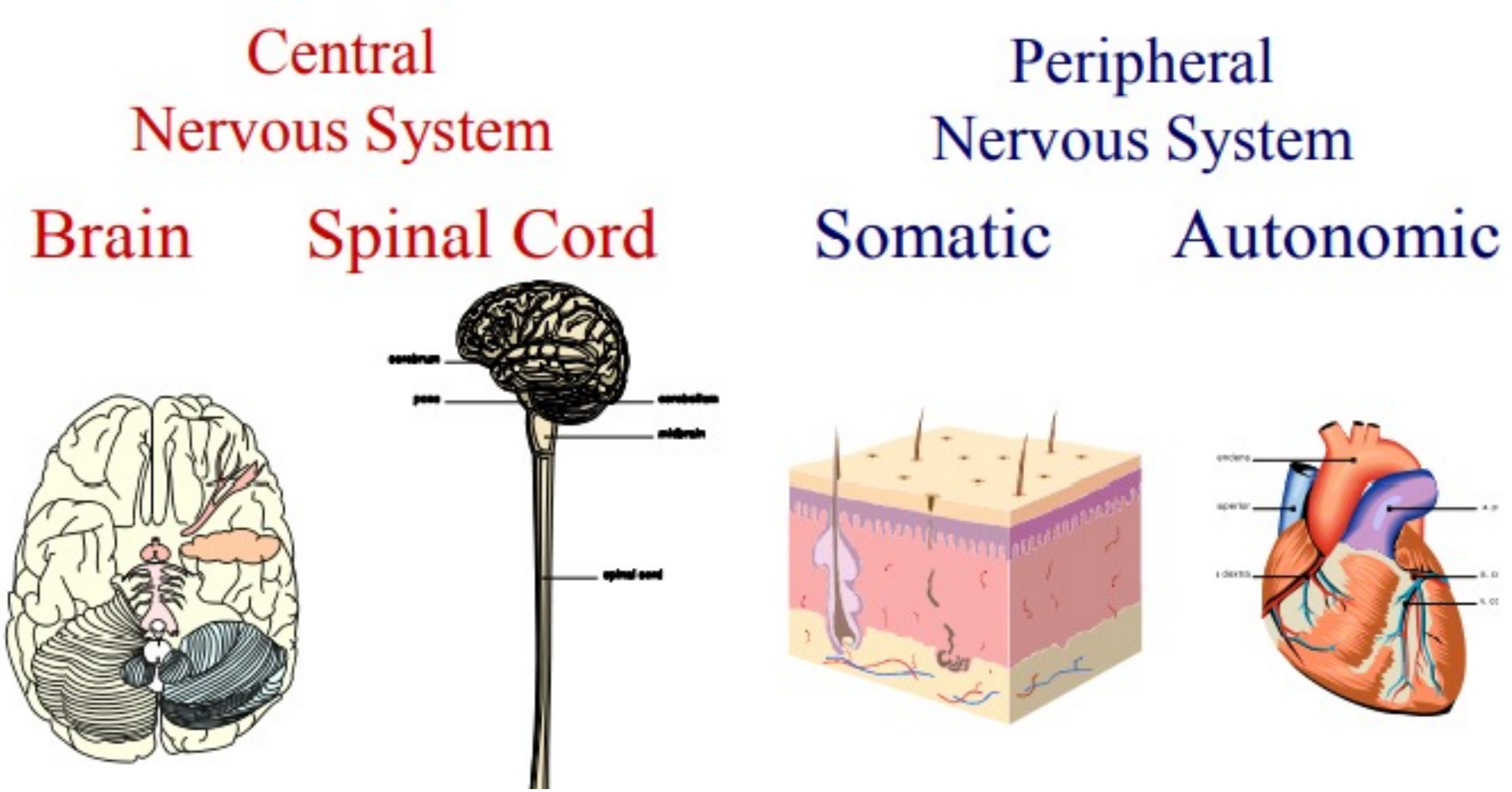


Figure: Long-term potentiation (LTP) and long-term depression (LTD).
Plots of excitatory postsynaptic potential amplitude at a hippocampal synapse over time during two different stimulus patterns. (Left panel) Following a long burst of high-frequency stimulation (100 Hz for 1 s), synapses strengthen, leading to a larger EPSP amplitude, and this is maintained for hours (LTP). The transient spike in strengthening that occurs immediately after the 100 Hz stimulus train results from post-tetanic potentiation. (Right panel) Following a low-frequency train of activity (1 Hz for 20 s), synapses weaken persistently, leading to a smaller EPSP amplitude (LTD).
Image from: *Meriney, Stephen D. (2019). Synaptic Transmission || Synaptic Plasticity. , (), 287–329.*

Brain Organization, Anatomy, and Function

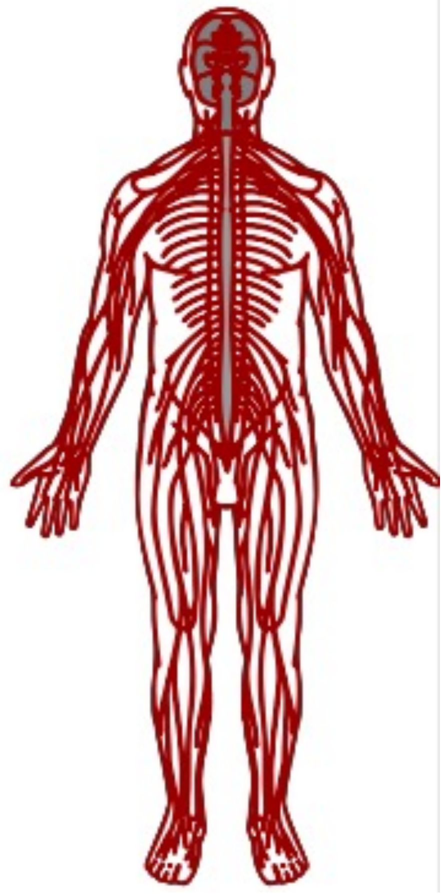
- The design of a brain-computer interface typically involves choices regarding which brain areas to record from and, in some cases, which brain areas to stimulate.
- The human nervous system can be broadly divided into
  - The central nervous system (CNS).
    - The CNS consists of the brain and the spinal cord.
  - The peripheral nervous system (PNS).
    - The PNS consists of the somatic nervous system (neurons connected to skeletal muscles, skin, and sense organs) and the autonomic nervous system (neurons that control visceral functions such as the pumping of the heart, breathing, etc.).

Brain Organization, Anatomy, and Function



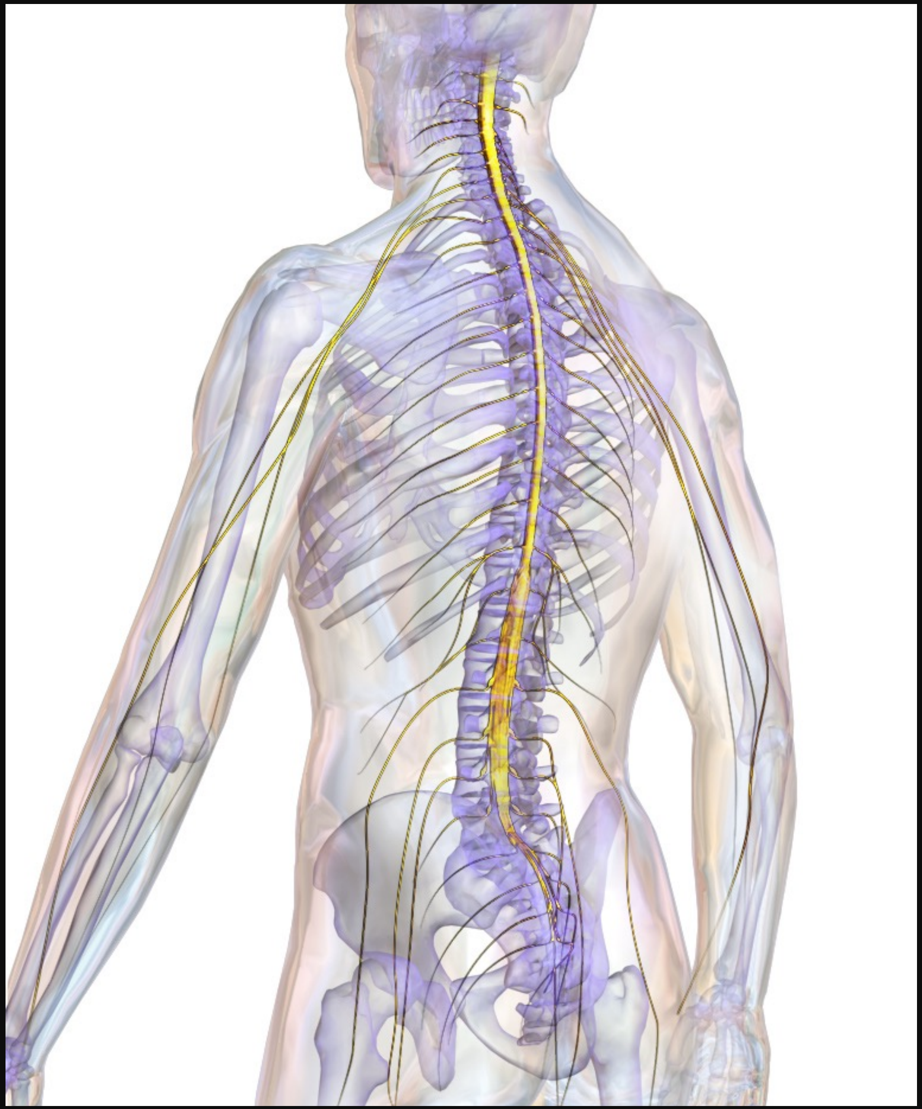
Skeletal/Somatic Nervous System

- Nerves that connect to voluntary skeletal muscles and to sensory receptors
- Afferent Nerve Fibers (incoming)
  - Axons that carry info away from the periphery to the CNS
- Efferent Nerve Fibers (outgoing)
  - Axons that carry info from the CNS outward to the periphery



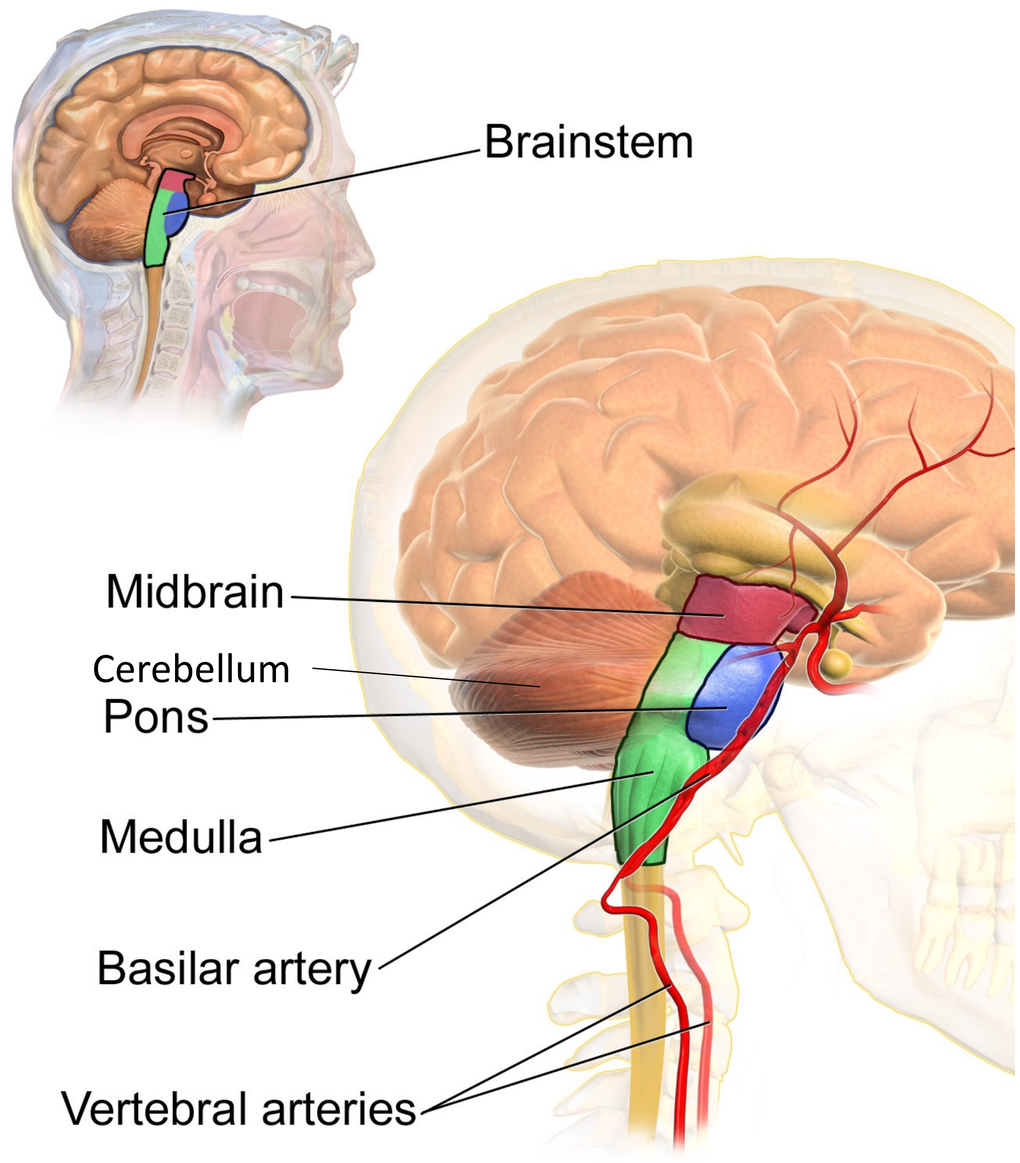
Autonomic and Central Nervous System

- Autonomic: Nerves that connect to the heart, blood vessels, smooth muscles, and glands
- CNS = Brain + Spinal Cord
- Spinal Cord
    - Local feedback loops control reflexes
    - Descending motor control signals from the brain activate spinal motor neurons
    - Ascending sensory axons transmit sensory feedback information from muscles and skin back to brain



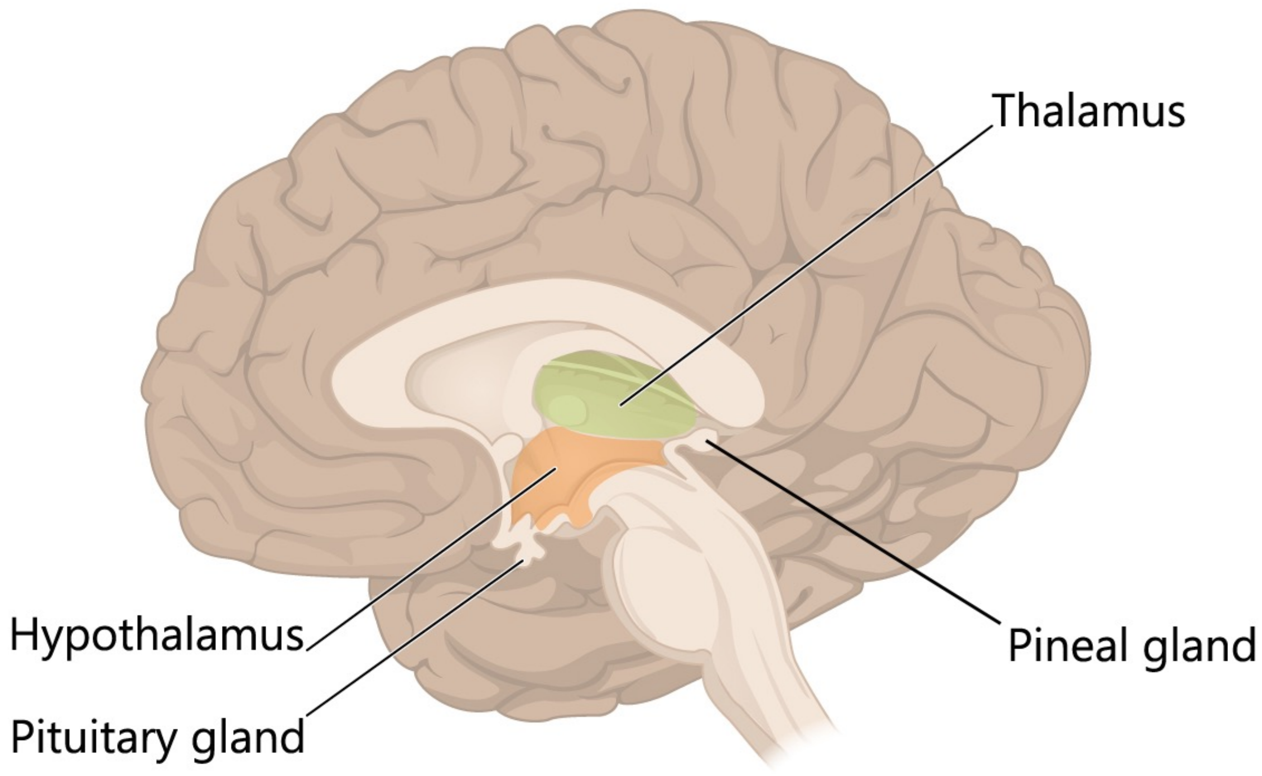
Major Brain Regions: Brain Stem

- Medulla: Breathing, muscle tone and blood pressure
- Pons: Connects brainstem with cerebellum & involved in sleep and arousal
- Cerebellum: Coordination of voluntary movements and sense of equilibrium
- Midbrain: Eye movements, visual and auditory reflexes



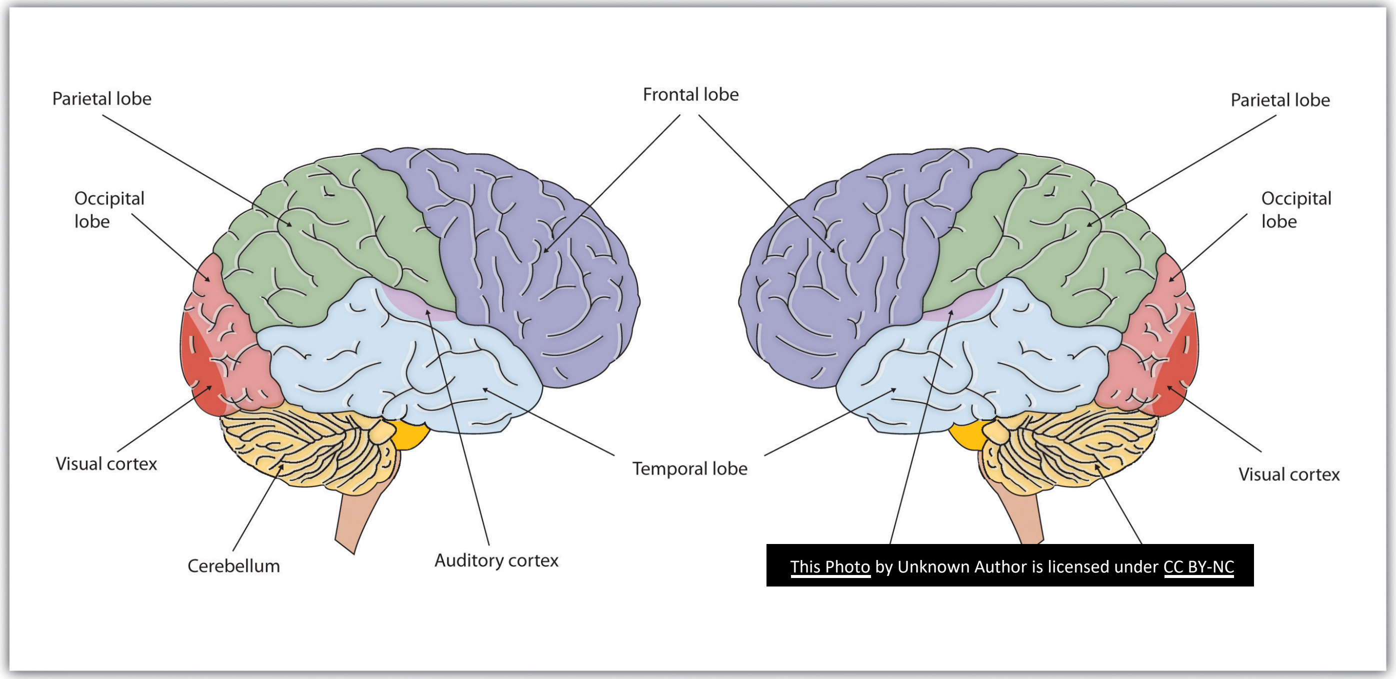
Major Brain Regions: Diencephalon

- Thalamus: Relay station for all sensory info (except smell) to the cortex
- Hypothalamus Regulates basic needs fighting, fleeing, feeding, and mating



Major Brain Regions: Cerebral Hemispheres

- Consists of Cerebral cortex, basal ganglia, hippocampus, and amygdala
- Involved in perception and motor control, cognitive functions, emotion, memory, and learning



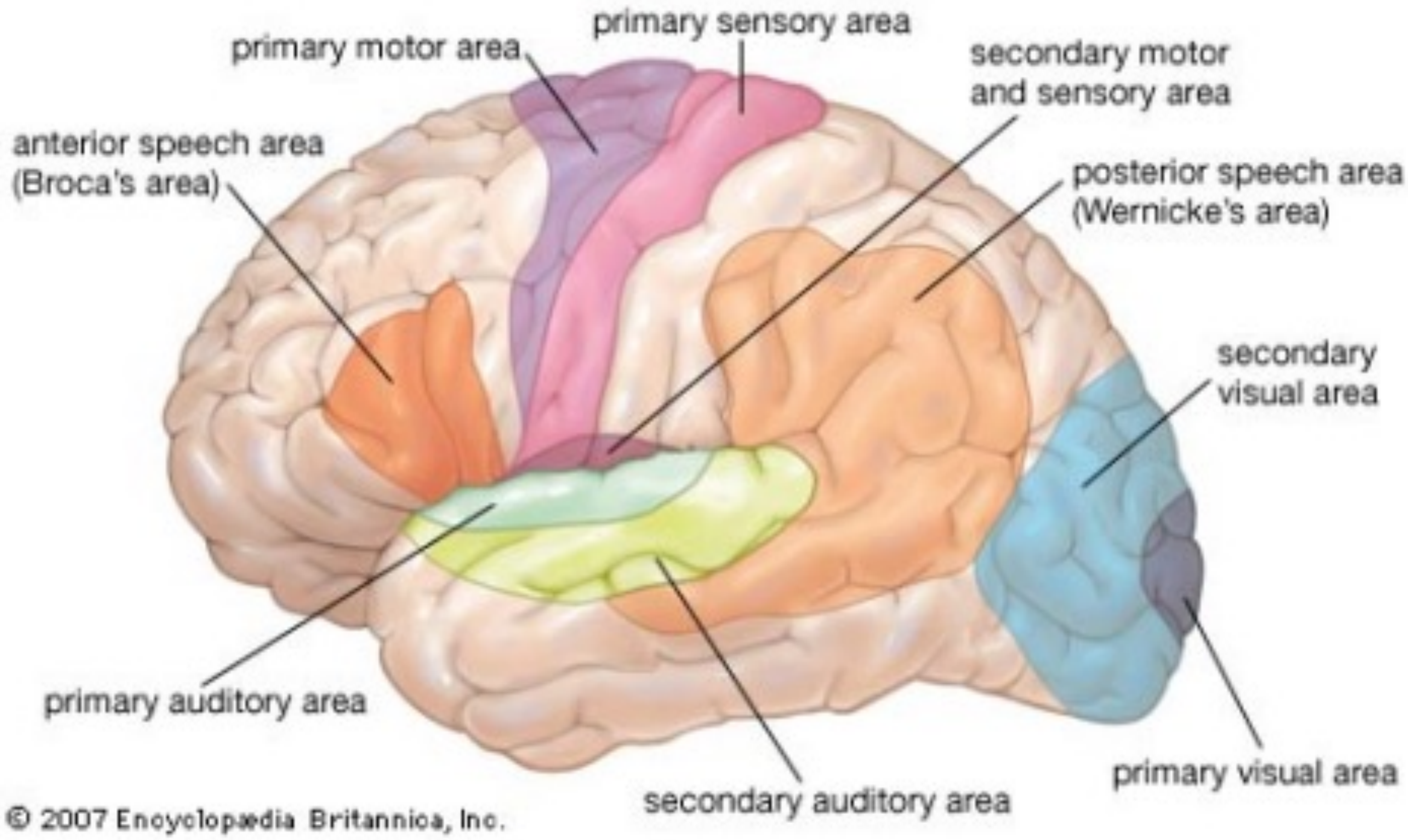


Figure 2.2: Regions of the cerebral cortex [Goldberg, 2002]

Cortical area	Function
Auditory association area	Complex processing of auditory information
Auditory cortex	Detection of sound quality (loudness, tone)
Broca's area (speech center)	Speech production and articulation
Prefrontal cortex	Problem solving, emotion, complex thought
Premotor cortex	Coordination of complex movement
Primary Motor cortex	Initiation of voluntary movement
Primary somatosensory cortex	Receives tactile information from the body
Sensory association area	Processing of multisensory information
Gustatory area	Processing of taste information
Wernicke's area	Language comprehension
Primary Visual Cortex	Complex processing of visual information