

# neural conduction

## AND SYNAPTIC TRANSMISSION

### resting membrane POTENTIAL

- membrane potential recorded via microelectrodes: one on the extracellular side of the neuron, and measure the difference in membrane potential.
- Resting potential:  $-70\text{mV}$ .

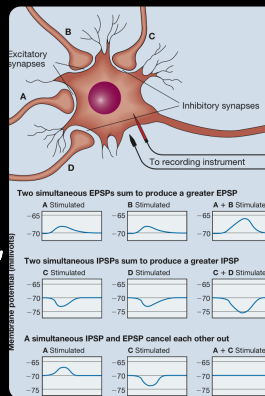
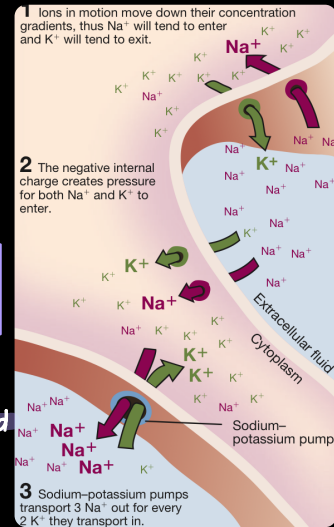
$\begin{matrix} \text{Na}^+ \\ \text{Na}^+ \\ \text{Na}^+ \end{matrix}$ 
 $\begin{matrix} \text{K}^+ \\ \text{K}^+ \end{matrix}$

$\text{K}^+$  always more intracellular than  $\text{Na}^+$   
 3 main forces which drive  $\text{Na}^+$  in:  
 - electrostatic pressure (-ve charge inside)  
 - concentration gradient (high  $\rightarrow$  low)  
 - random motion.

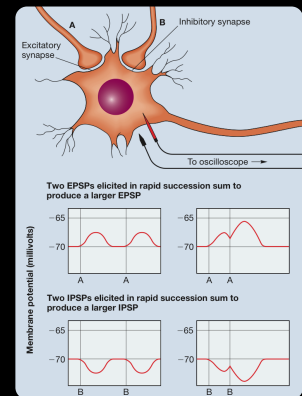
For every 3  $\text{Na}^+$  out of cell 2  $\text{K}^+$  come in, via the sodium potassium pump.

### conduction of ACTION POTENTIAL

- neuron membranes are either **hyperpolarized** (more negative), or **depolarized** (less negative), by postsynaptic signals.
  - postsynaptic signal = IPSP/EPSP
    - inhibitory  $\hookrightarrow$  excitatory.  $\hookrightarrow$  decrease w/ distance.
  - postsynaptic signal = passively transported, decremental, graded. **stronger stimuli = stronger potential initiated.**
- Action potential = generated through the summation of all IP/EPSP.
  - threshold for AP:  $-55\text{mV}$ ; AP = all or none response, non-decremental.
  - AP generated @ the initial axon segment.
- Integration of the post-synaptic signal:
  - spatial summation** (the addition of all the IPSP/EPSP) around the neuron, to determine if it reaches the threshold of excitation.
  - temporal summation** (addition of all the subsequent EP/IPSP) that occurs in succession; one potential can stay long enough until the next one adds onto it.

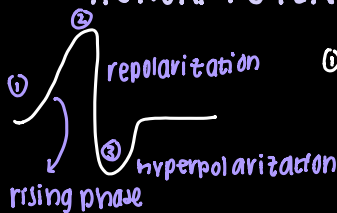


spatial summation.



temporal summation

### conduction of ACTION POTENTIALS

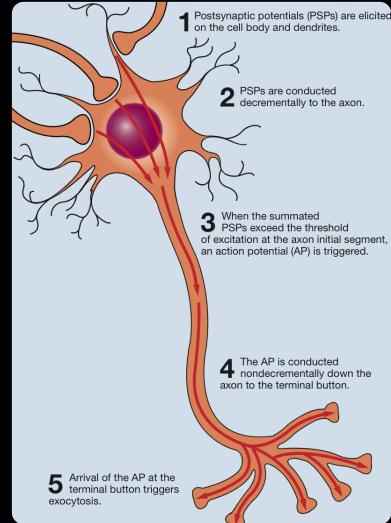


- Excitatory threshold reached,  $\text{Na}^+$  opens to allow the  $\text{Na}^+$  ions to go in.
  - the  $\text{Na}^+$  changes the charge to  $+50\text{mV}$ , & closes. (1ms after opening).
- Repolarization: the  $\text{K}^+$  channels open for the  $\text{K}^+$  to go out of the cell, to decrease the +ve charge.
- Hyperpolarization: too many  $\text{K}^+$  went out, so neuron temp. in hyperpolarized state.

- Absolute refractory period: no new AP can be generated.
- Relative refractory period: can generate new AP but needs more stimulation.  $\hookrightarrow$  ensure that the AP only goes one way.

Action potential in myelinated vs. unmyelinated cells:

- AP = active transport, and non decrementally.
- **unmyelinated:**
  - AP travels down the axon (@ each of the Na channels) and then reaches the terminal buttons causing exocytosis.
  - **orthodromic conduction:** soma → axon.
  - **antidromic conduction:** axon → soma.
- **myelinated**
  - jumps along **nodes of Ranvier**, the nodes of Ranvier have  $\text{Na}^+$  channels that are clumped up @ the nodes, and reconduct the signal when it reaches it.
  - **saltatory conduction.**
  - faster than non myelinated, small axons.

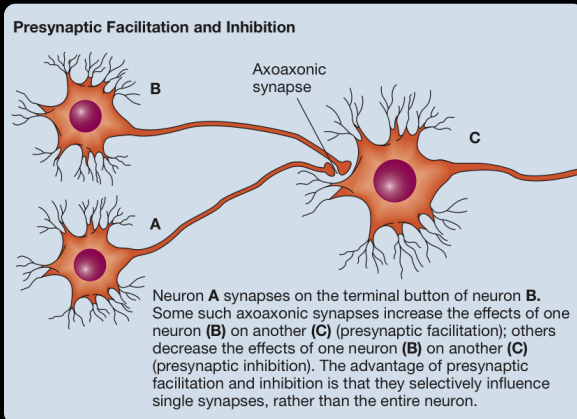


**Hodgkin-Huxley Model:** doesn't work when applied to mammals b/c they don't have the same qualities that a motor neuron has.

↳ they used motor neurons, but can't be applied to human neurons in CNS.

↳ AP = diff in motor neurons and CNS neurons, and many CNS neurons don't have AP (interneurons)

## Chemical transmission OF NEURONS



Different types of synapses:

- **dendritic spines** (nodules on dendrite surface).
- **axosomatic, axodendritic, dendrodendritic, axoaxonic.**
  - ↳ **most common**
  - ↳ **can alter another neuron indirectly.**
- **directed synapses:**
  - axon and dendrite in close proximity.

**Non-directed synapses:**

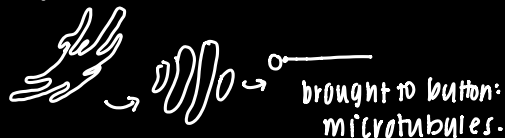
axon + dendrite not in close proximity, release via **varicosities** → bead on a string synapse.

### NT Release

- **Exocytosis:** when the AP reaches the neuron, the  $\text{Ca}^{2+}$  gates open.
- **Small molecule NT:** released in pulses (when the  $\text{Ca}^{2+}$  gates open)
- **neuropeptide:** released gradually, proportionate to the amount of  $\text{Ca}^{2+}$  in the cell.

### NT synthesis & packaging

- **neuropeptides** = short aa (chains) of NT
- **small molecule NT, or neuropeptide (large).**



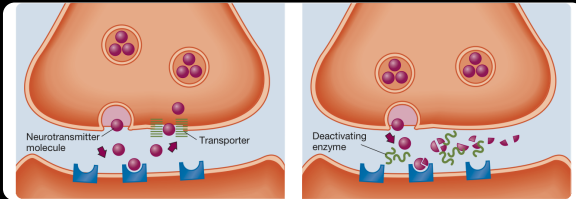
made: ER      packaged: golgi

**coexistence:** presence of both the small + large NT in the same cell.

### NT Receptors

- 2 main types: **ionotropic** and **metabotropic** receptors.
- **ionotropic:**

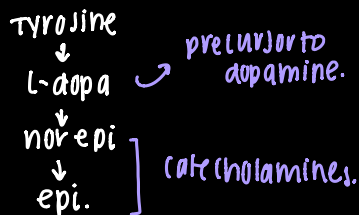
- special kind: **autoreceptor**
  - on the **presynaptic membrane** of a neuron
  - used to regulate how much enzymes are in the synapse of the **presynaptic neuron**.
  - involved in the **reuptake/degradation**.



- **reuptake**
  - most NT which are released = drawn back immediately.
  - transporter mechanism.
- **enzyme degradation**
  - enzyme released, breaks down the NT.
  - parts brought back for degradation + reuse.
  - **acetylcholinesterase**.

### Neurotransmitter Types

- amino acid, monoamine, ach (small NT)
- neuropeptides: only one class.
- **AA NT:**
  - GABA, glu, Tyr, Ala, gly.
  - ↳ made by glu parts.
  - glu = excitatory, GABA = inhibitory.
- **monoamine NT:**
  - epi, norepi, dopamine, serotonin.
  - catecholamines vs. indolamines.
  - ↳ epi, norepi      ↳ dopamine,
  - conversion in between serotonin.
  - them need enzymes:



- **Acetylcholine:**
  - e muscles, made w/ acetyl + choline.
- **unconventional NT:**
  - Soluble gases
    - CO<sub>2</sub>, NO
    - retrograde transmission
    - 2° messenger activating.
    - short fcn; made by release.
  - **endocannabinoids:**
    - anandamides.
    - produced right before release.

- **ligand activated channels:** NT binds, immediately initiating EPSP/IPSP via the ion (channels opening) closing.
- **metabotropic:** more common, longer lasting effects.
  - transmembrane region goes into tx, associated w/ G-coupled protein receptors.
  - attachment of NT = triggers G-coupled receptor, can do 2 things:
    - ① initiate IP/EPSP via ion channels.
    - ② initiate 2° messenger to postsynaptic neuron (chemical).
  - epigenetic (long effects).

### Glial cells + Gap Junction

- **gap junction:** electrical communication between the neurons.
- organization: neuron - neuron  
neuron - astrocyte  
astrocyte - astrocyte.
- **astrocytes** = arranged to make sure that the neurons are firing in sync.
- astrocyte only have gap junction @ the ends where they connect to each other and neurons.

### Neuropeptide classes:

- ① gut-brain
- ② hypothalamic
- ③ misc
- ④ opioid
- ⑤ pituitary

## Pharmacology of BEHAVIOUR

- How drugs influence transmission of the neurons:
  - binding to:
    - presynaptic / postsynaptic
  - or acting as:
    - agonist / antagonist
    - breakdown / prevent breakdown in the synapse.
    - prevent reuptake.

How drugs were used to influence neurotransmission:

① Atropine and Botox:

- muscarinic and nicotinic receptors of ACh are found in the CNS/PNS; they can bind ACh.

• muscarinic = metabotropic, binds atropine.

• nicotinic = ionotropic, binds botox.

↳ both are ACh antagonist.

② Pain Prevention

- injection of enkephalins (endogenous opioids) @ the PAA = minimize pain.
  - enkephalins include endorphin.

③ antischizophrenic drugs.