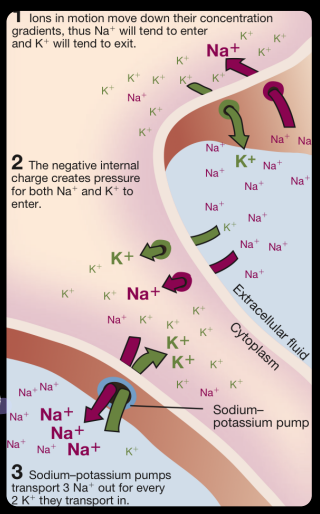
neural conduction
AND SYNAPTIC TRANSMISSION

resting membrane POTENTIAL

- membrane potential recorded via microelectrodes: one on the extra/intracellular side of the neuron, and measure the difference in membrane potential.
  → **resting potential**: -70mv.

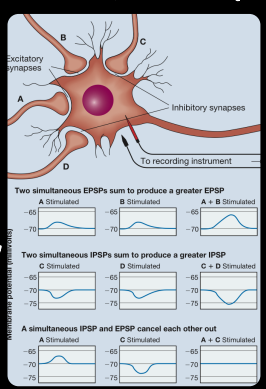
always more intracellular K than Na
3 main forces which drives Na in:
- electrostatic pressure (-ve charge inside).
- concentration gradient (high → low)
- random motion.



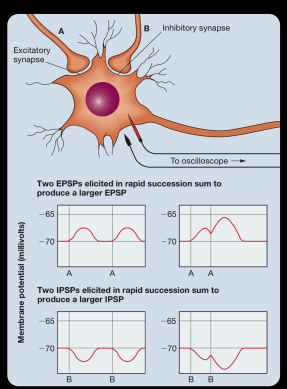
For every 3 Na^+_{out} of cell 2 K^+ come in, via the sodium potassium pump.

conduction of ACTION POTENTIAL

- neuron membranes are either **hyperpolarized** (more negative), or **depolarized** (less negative), by **postsynaptic signals**.
  - postsynaptic signals: IPSP / EPSP
    (inhibitory) (excitatory.)
  - postsynaptic signals: passively transported, decremental, graded. (decrease w distance.)
- Action potential: generated through the summation of all IP/EPSP. (stronger stimuli = stronger potential initiated.)
  - threshold for AP: -65mv; 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, the determine if it reaches the threshold of excitation.
  - **temporal summation** (addition of all the subsequent EPI/IPSP) that occurs in succession; one potential can stay long enough until the next one adds onto it.

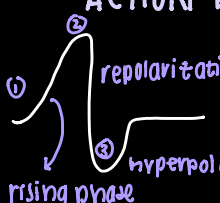


spatial summation.



temporal summation

conduction of ACTION POTENTIALS

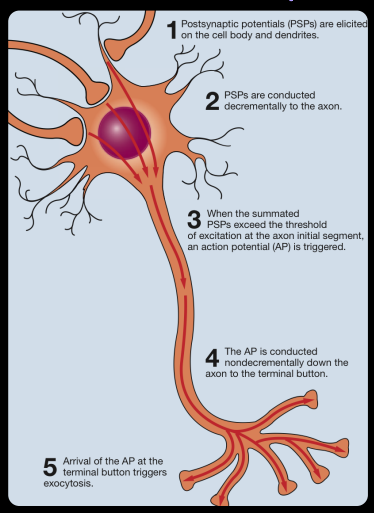


1. **Excitatory threshold** reached, Na^+ opens to allow the Na^+ ions to go in.
   - the Na changes the charge to +50mv, & closes. (1ms after opening).
2. **Repolarization**: the K^+ channels open for the K^+ to go out of the cell, to decrease the +ve charge.
3. **Hyperpolarization**: too many K went 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.

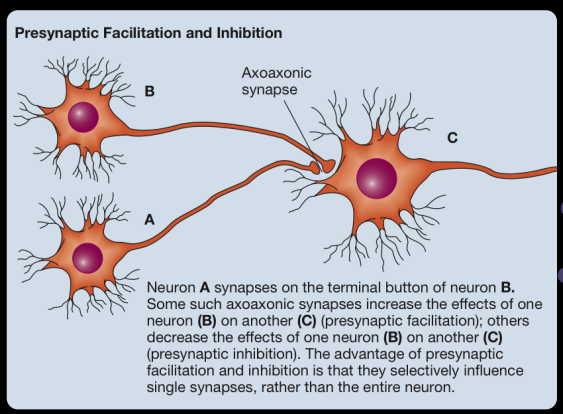
→ ensures that the AP only goes one way.

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 Na^+ channels that are clumped up @ the nodes, and reconducts 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, axoaonic.
  ↳ most common

Directed synapse: can alter another neuron indirectly.
axon and dendrite in close proximity.

Nondirected synapse:
axon + dendrite not in close proximity, release w/ a. varicosities → beads on a string synapse.

NT Release

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

NT synthesis & packaging

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

brought to button: microtubules.

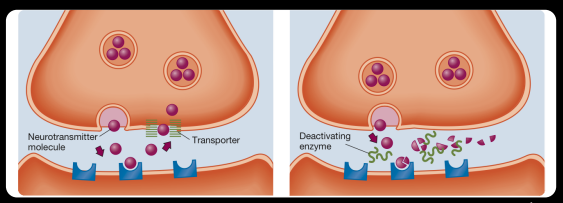
made: ER packaged: golgi

Coexistance: 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 pre/post-synaptic 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.

- ligand activated channels: NT binds, immediately initiating EPSP / IPSP via the ion channels opening / closing.
- metabotropic: more common, longer lasting effects.
- transmembrane region goes in/out 7x, 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° msnger 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.

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, serotonin.
  - conversion in between them need enzymes:

Tyrosine
↓
L-dopa → precursor to dopamine.
↓
norepi
↓
epi.
] catecholamines.

- Acetylcholine:
  - @ muscles, made w/ acetyl + choline.
- unconventional NT:
  - Soluble gases
    - CO_2, NO
    - retrograde transmission
    - 2° msnger activating.
    - short fcn; made by release.
  - endocannabinoids:
    - anadamides.
    - produced right before release.

Neuropeptide classes:
① gut-brain
② hypothalamic
③ misc
④ opiod
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 PAG = minimize pain.
  - enkaphalins include endorphin.

③ antischizophrenic drugs.