The Nervous System

Chapter 37 – Campbell Biology in Focus

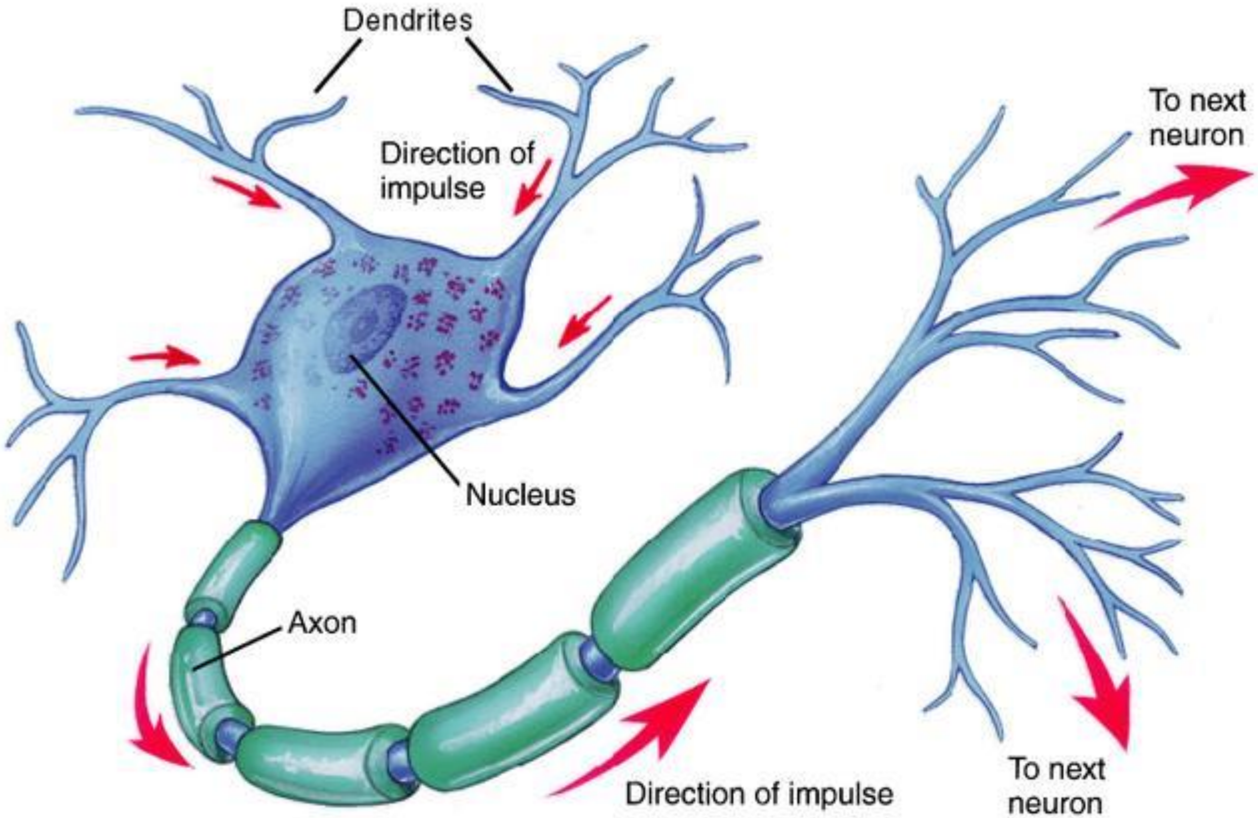
37.1 -Neuron structure and organization reflect function in information transfer

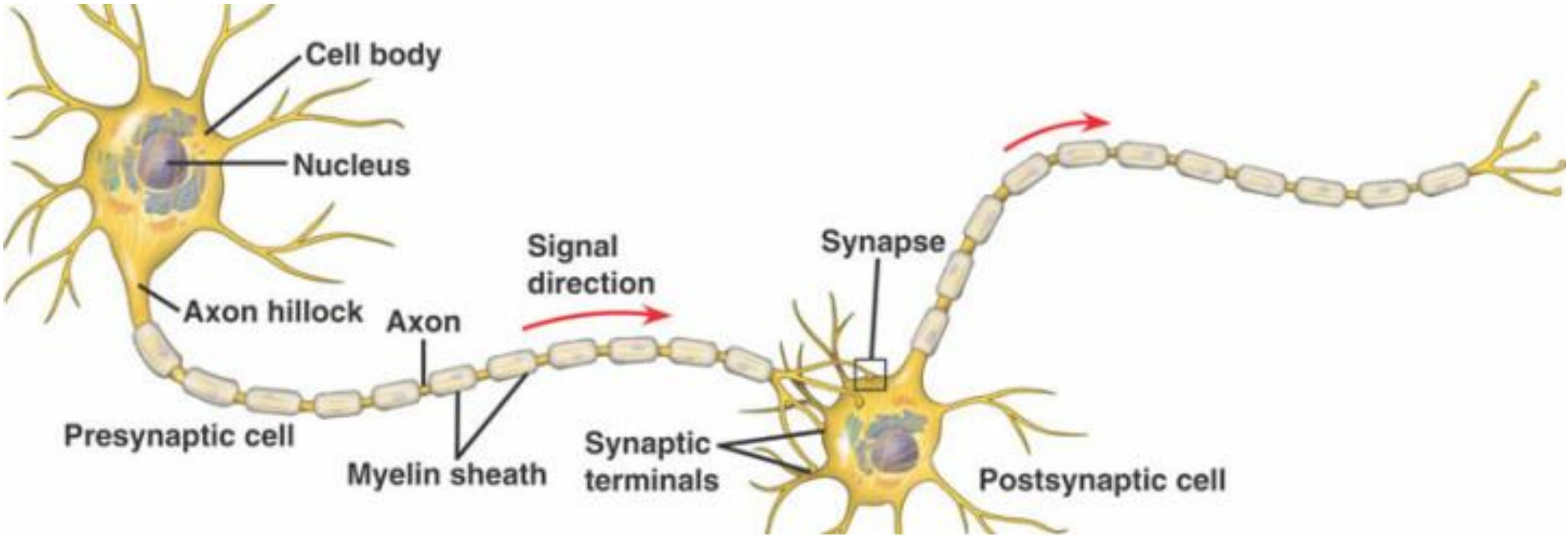
- The specialized cells of the nervous system are called ***neurons.***
  - *They are responsible for transferring information within the body*
- ***All neurons transmit electrical signals within the cell in an identical manner → Action Potentials***
- In more complex animals neurons are usually grouped into highly organized clusters called ***brains*** – or simpler structures called ***ganglia***.

Neuron Structure & Function

1. Cell body
   - Contains nucleus and organelles
   - Carries out normal cell functions
2. Dendrites
   - Extensions leading towards cell body
   - ***Receive signals*** from other neurons and direct them toward cell body
3. Axon
   - Extension leading away from cell body
   - Transmits signals (nerve impulses) away from cell body towards other neurons or effectors
   - Usually much longer than dendrites

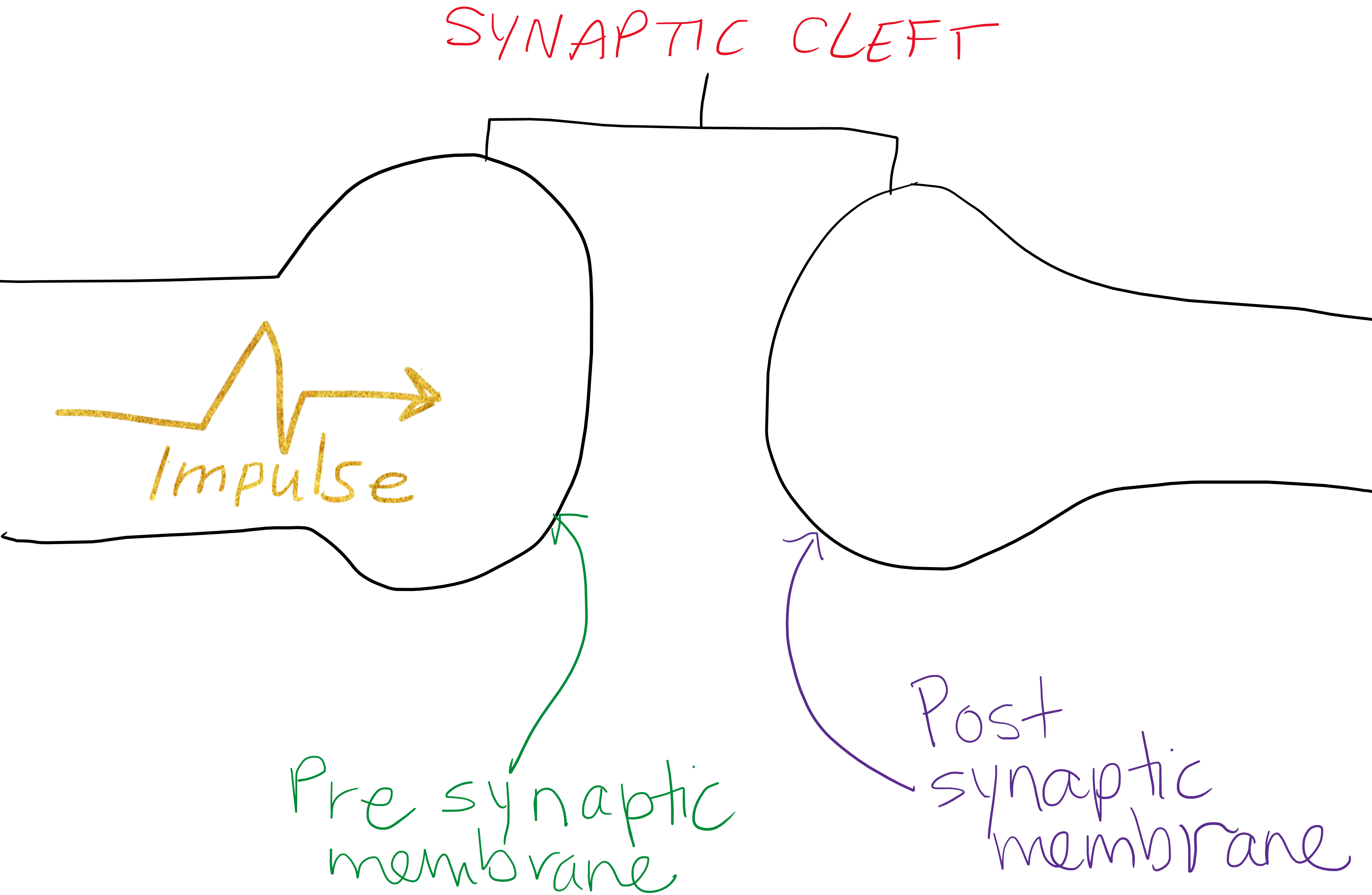
Remember: D→CB → A




Cell body
Nucleus
Axon hillock
Axon
Signal direction
Synapse
Presynaptic cell
Myelin sheath
Synaptic terminals
Postsynaptic cell

At the end of an axon…

- The axon hillock (attached to the cell body) is where most signals are generated
- At its other end, the axon usually divides into many branches
- Each branched end of an axon transmits impulses to other cells at junctions called ***synapses***
  - There can be thousands of synapses at the end of a single axon.
- Chemical messengers called ***neurotransmitters*** at the synapse pass the information from transmitting neuron to the receiving cell

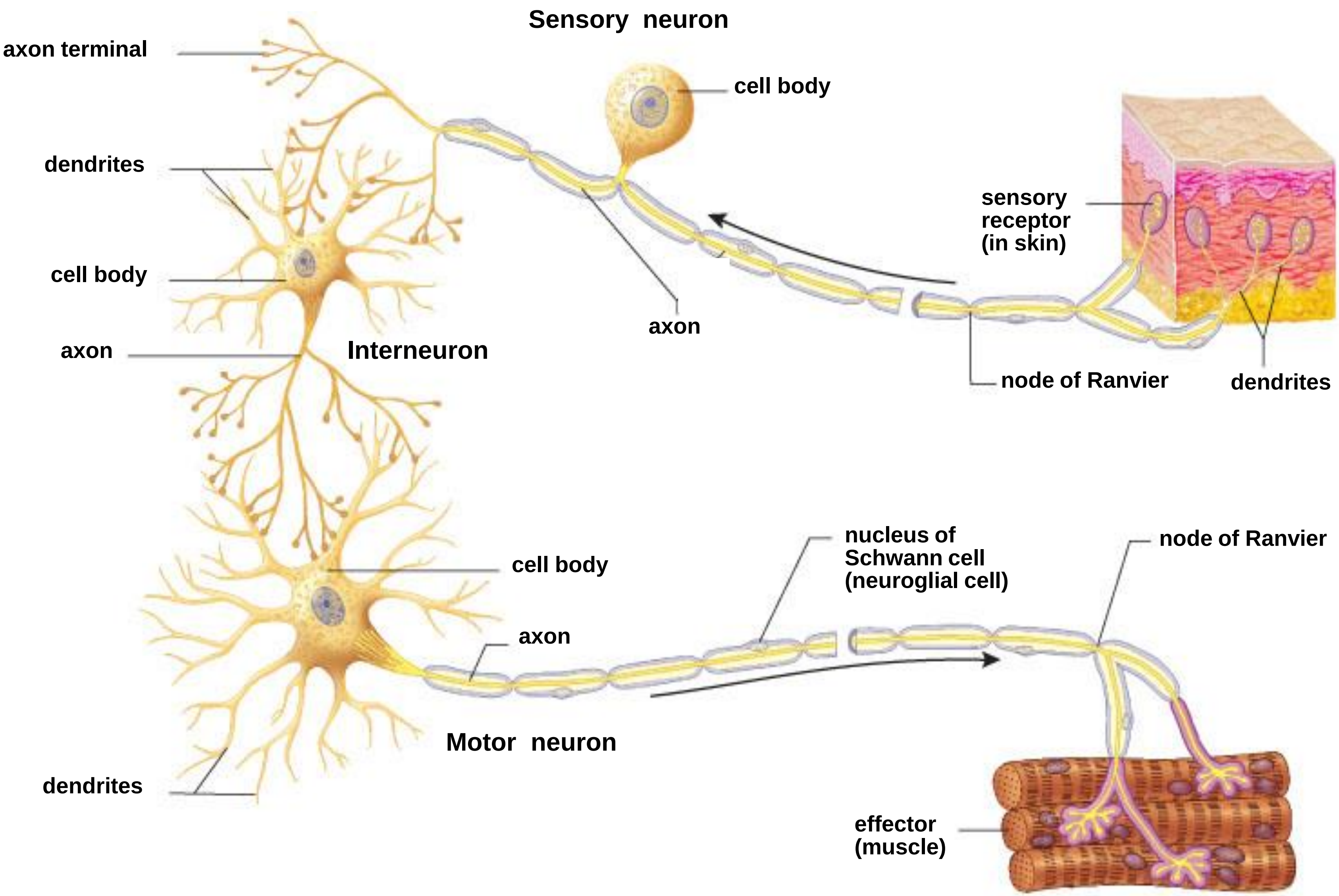

SYNAPTIC CLEFT
Impulse
Presynaptic membrane
Post synaptic membrane

2 Types Of Cells In The N.S.

NEURONS	**NEUROGLIA**
• Transmit impulses	• Support and nourish neurons
• SENSORY NEURON	• Maintain homeostasis
• INTERNEURON	• Form myelin
• MOTOR NEURON	• May aid in signal transmission

Introduction to Information Processing

- Sensory Neurons
  - Transmit info about external stimuli
  - Ex. Light, touch or smell or blood pressure and muscle tension
- Interneurons
  - Form local circuits connecting neurons of the brain
  - Integrate sensory input
- Motor Neurons
  - Transmit signals to effectors – glands or muscle cells

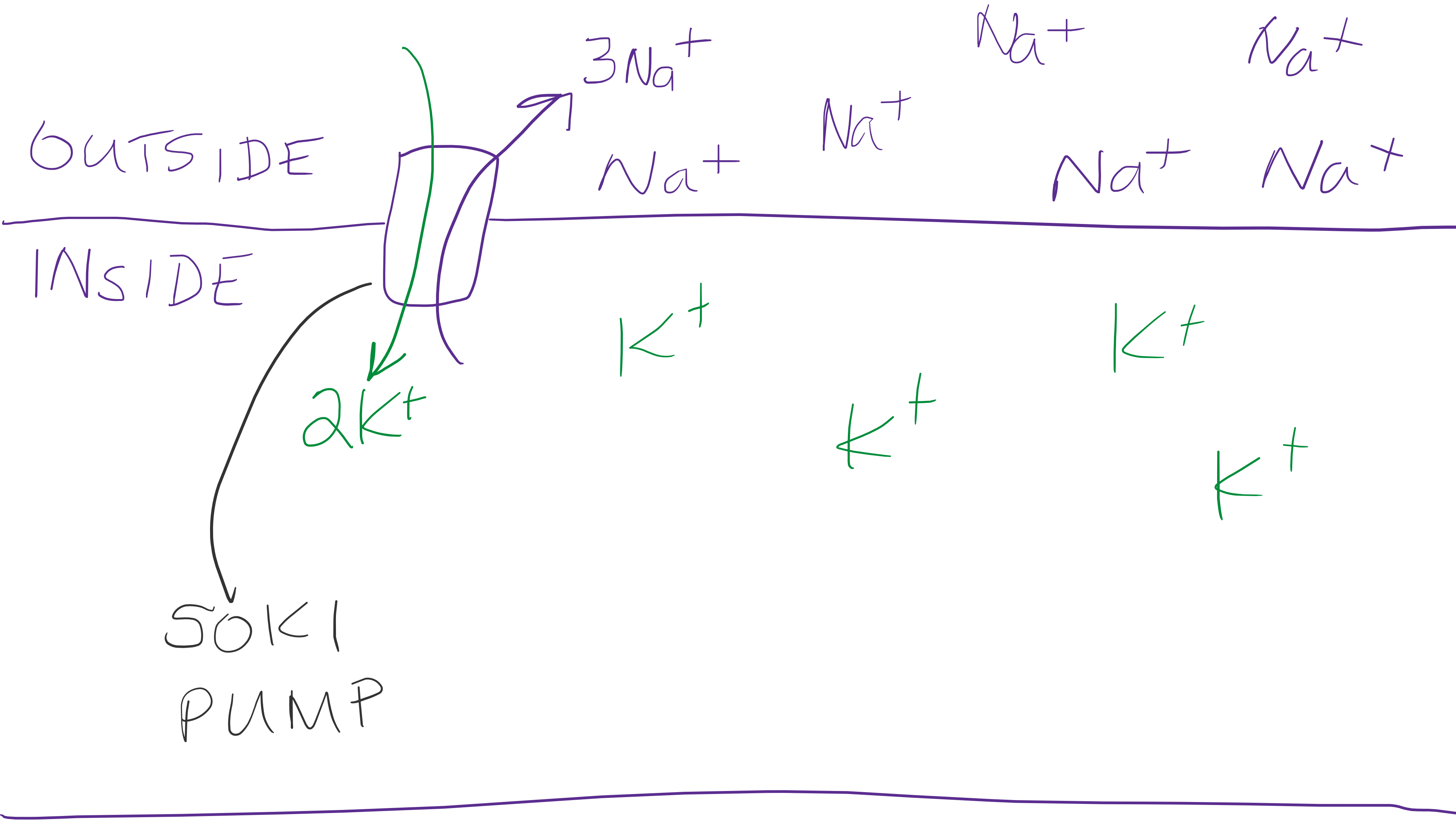

Sensory neuron
axon terminal
cell body
dendrites
sensory receptor (in skin)
cell body
axon
axon
Interneuron
node of Ranvier
dendrites
nucleus of Schwann cell (neuroglial cell)
node of Ranvier
cell body
axon
Motor neuron
dendrites
effector (muscle)

37.2 – Ion pumps and ion channels establish the resting potential of a neuron

- In neurons ions are unequally distributed between the interior and exterior of the cell
- This results in a charge difference
- This charge difference, or ***voltage***, across the membrane is called the **membrane potential**
- The membrane potential of a neuron at rest is called the RESTING POTENTIAL
  - Resting potential of a cell is between -60mV and -80mV
  - The inside of the cell is more negative than the outside…

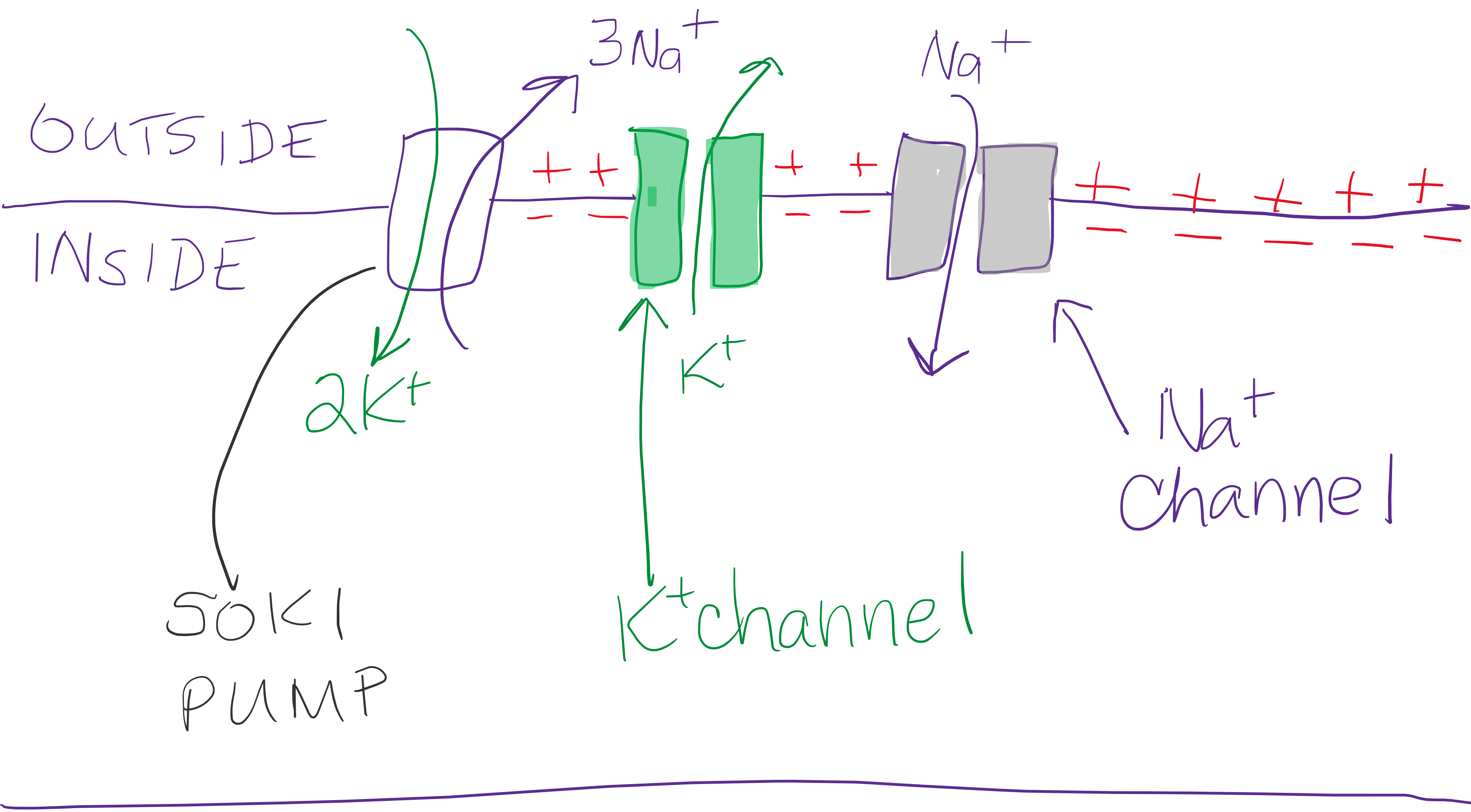
Formation of the Resting Potential

- Sodium and Potassium ions help establish the resting potential of a neuron
- The sodium-potassium (SOKI) pump pumps Na+ out (SO) and K+ in (KI)
  - The protein pump uses energy from ATP hydrolysis to create these gradients

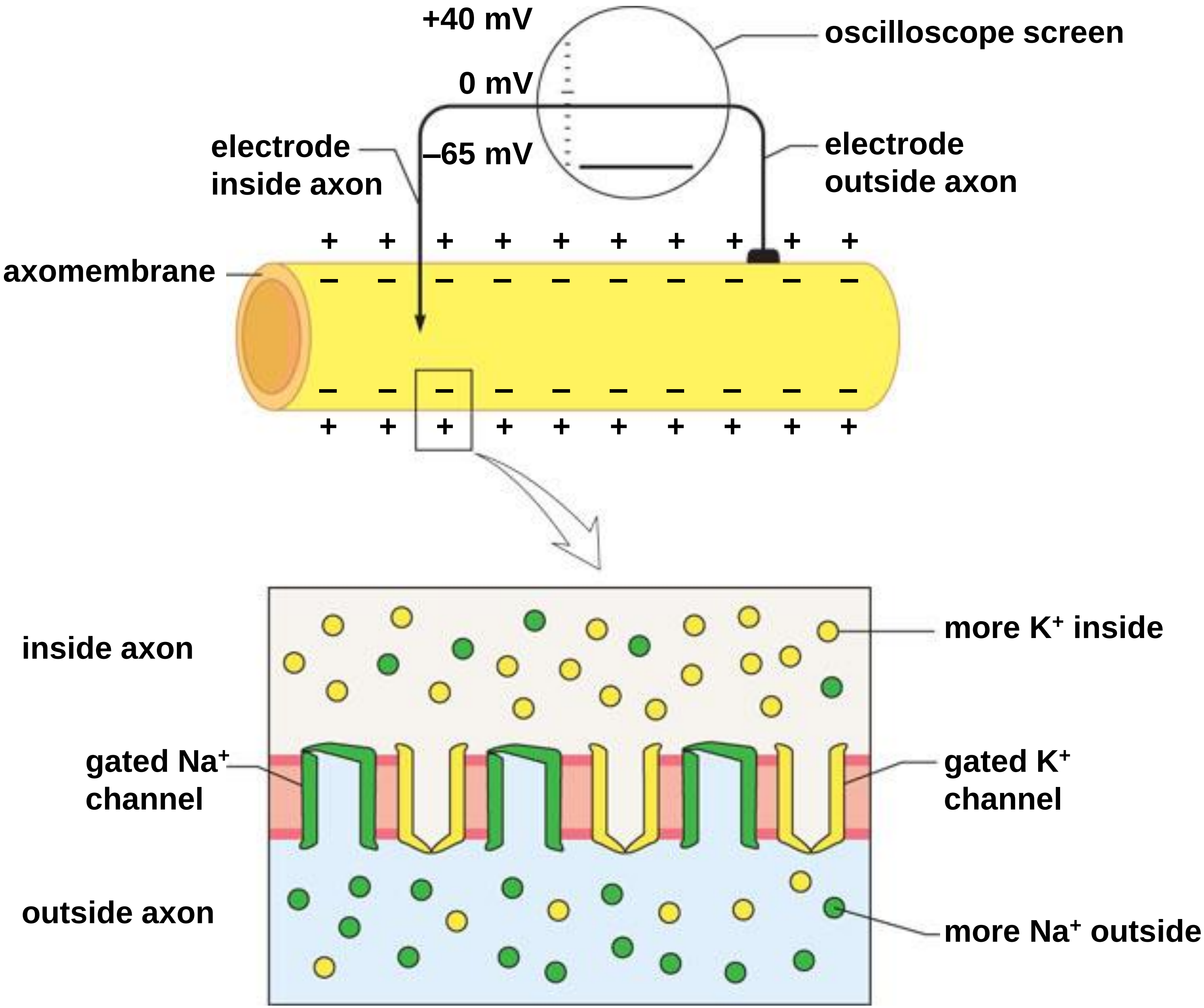


OUTSIDE
INSIDE
3Na+
Na+
Na+
Na+
Na+
Na+
Na+
2K+
K+
K+
K+
K+
SOKI
PUMP

The membrane is much more permeable to K+ so it flows out of the cell continuously. This contributes to a more negative charge in the axoplasm (the cytoplasm inside the neuron)



a. Resting potential



37.3 – Action Potentials are the signals conducted by axons

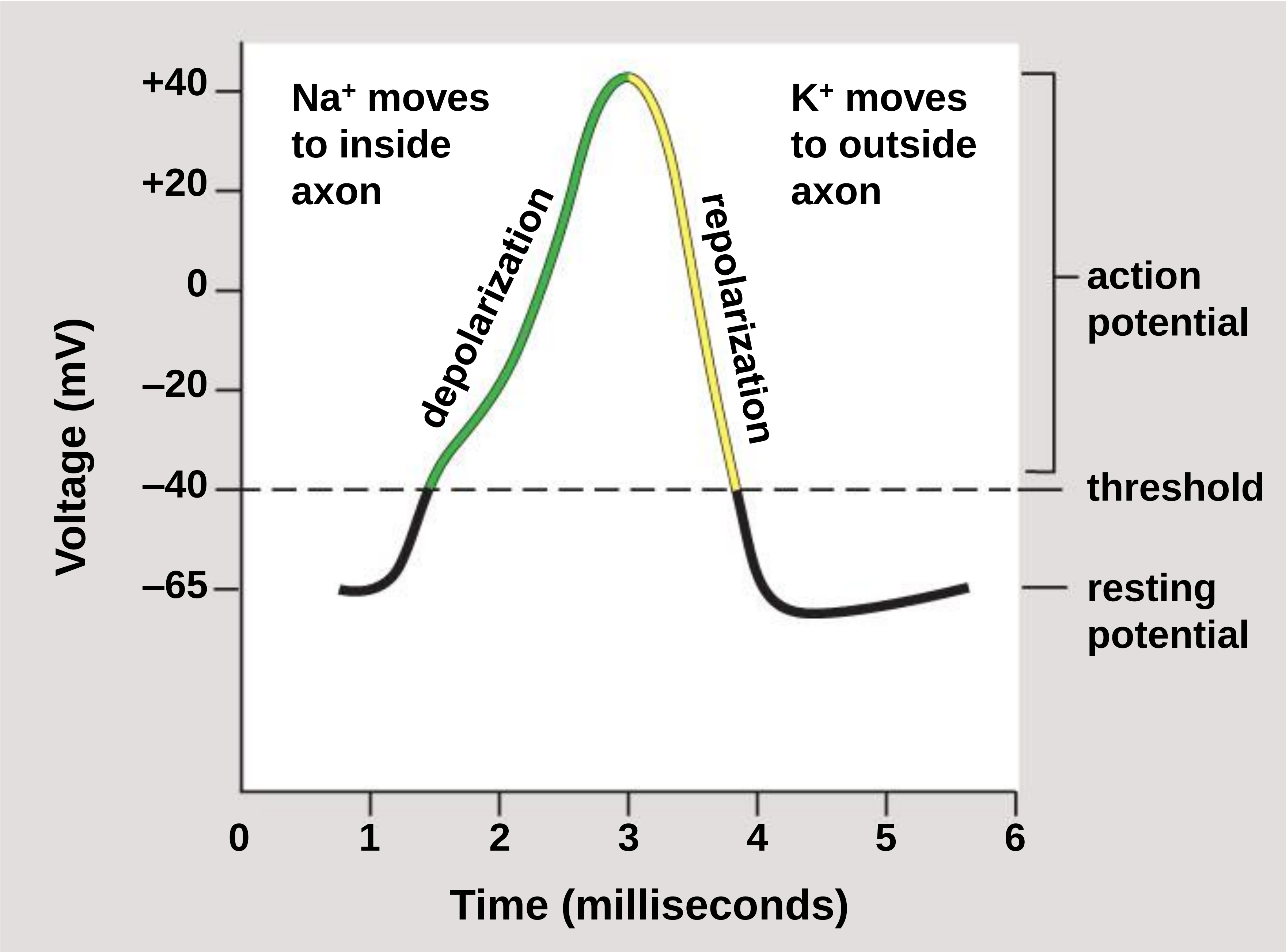
- When a neuron responds to a stimulus the membrane potential changes
- This is because some channels in the neuron's membrane are GATED ION CHANNELS that open in response to stimuli!
- When these channels open it allows ions to move through the membrane which in turns alters the membrane potential
- When there is a significant change in the membrane potential we call it an ACTION POTENTIAL
  - *There are 3 distinct phases to an action potential*

A closer look at Action Potentials

It all starts with a stimulus that opens a gated channel – this will change the membrane potential locally and can lead to other channels nearby opening too…

1. DEPOLARIZATION: *Rising Phase (Upswing)*
   - Na+ gated channels open and Na+ rushed into the cell
   - Membrane potential rises to around +45mV
2. REPOLARIZATION: *Falling Phase (Downswing)*
   - K+ gated channels open next and K+ rushes out of the cell
   - Membrane potential drops below resting potential (*Undershoot)*
3. REFRACTORY PERIOD: *"The Downtime"*
   - During this time the Na+ gated channels are closed and cannot reopen so if there's another stimulus another AP cannot be generated

d. Enlargement of action potential



The "All-or-none" Response

- An action potential (AP) is considered to be an **all-or-none** phenomena
- If a stimulus causes depolarization of the axonal membrane to a certain level (**threshold**) an AP occurs
  - Usually around -40mV is all it takes to start an AP
- That is, **the strength of an AP never changes**
  - *There is either an AP, or there isn't*
- The only variable that may change is frequency of APs
  - *The stronger a stimulus, the more frequent the APs*

An Overview: what's happening and when?

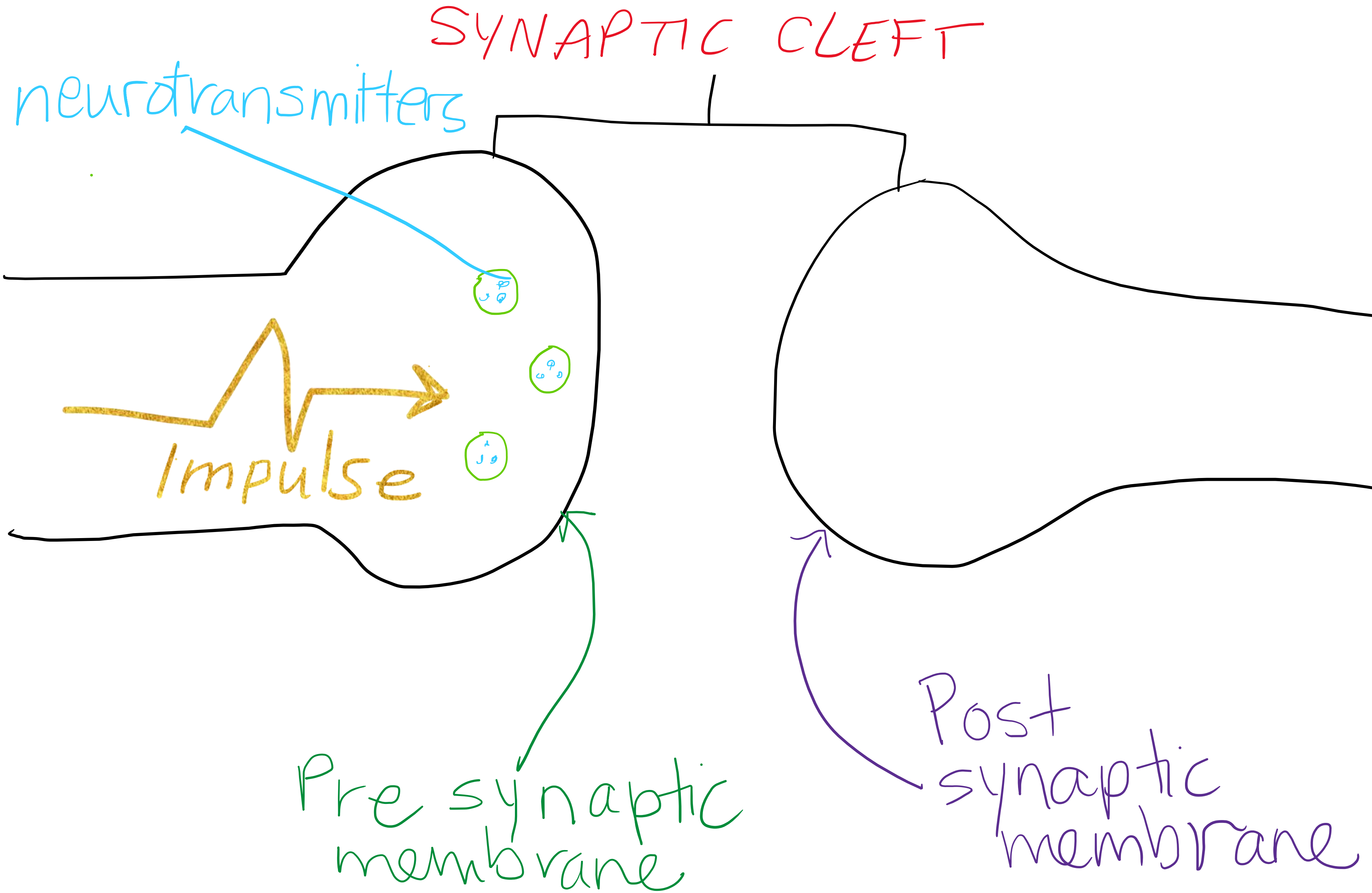
- *RESTING POTENTIAL*
  - *Na+/K+ pump at work*

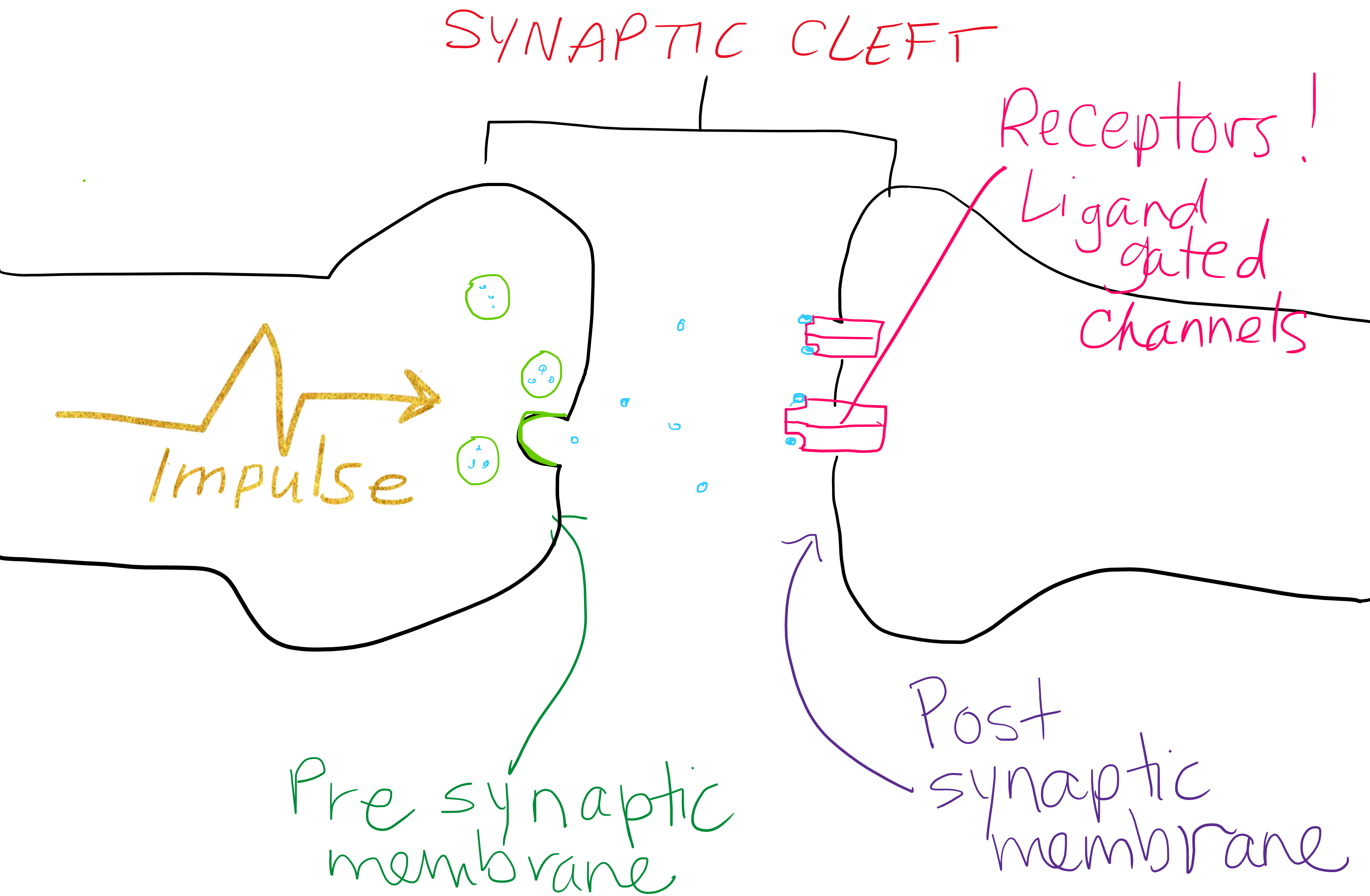
- *ACTION POTENTIAL*
  - *Depolarization: Na+ gates open*
  - *Repolarization: K+ gates open*
  - *Refractory Period: Na+ gates unable to open*

- *RECOVERY PHASE (simultaneous w/ refractory period)*
  - *Na+/K+ pump at work to reestablish the ion distribution at rest*

37.4 – Neurons communicate with other cells at synapses

1. Action potential arrives at synaptic terminal depolarizing the presynaptic membrane
2. Voltage-gated Ca^{2+} channels open triggering an influx of Ca^{2+}
3. Elevated Ca^{2+} causes synaptic vesicles to fuse with presynaptic membrane releasing NTs into synaptic cleft
4. NTs bind to ligand-gated ion channels in the postsynaptic membrane causing them to open (Na+ and K+ diffuse into the receiving cell)


SYNAPTIC CLEFT
neurotransmitters
Impulse
Pre synaptic membrane
Post synaptic membrane


SYNAPTIC CLEFT
Receptors!
Ligand gated channels
Impulse
Pre synaptic membrane
Post synaptic membrane

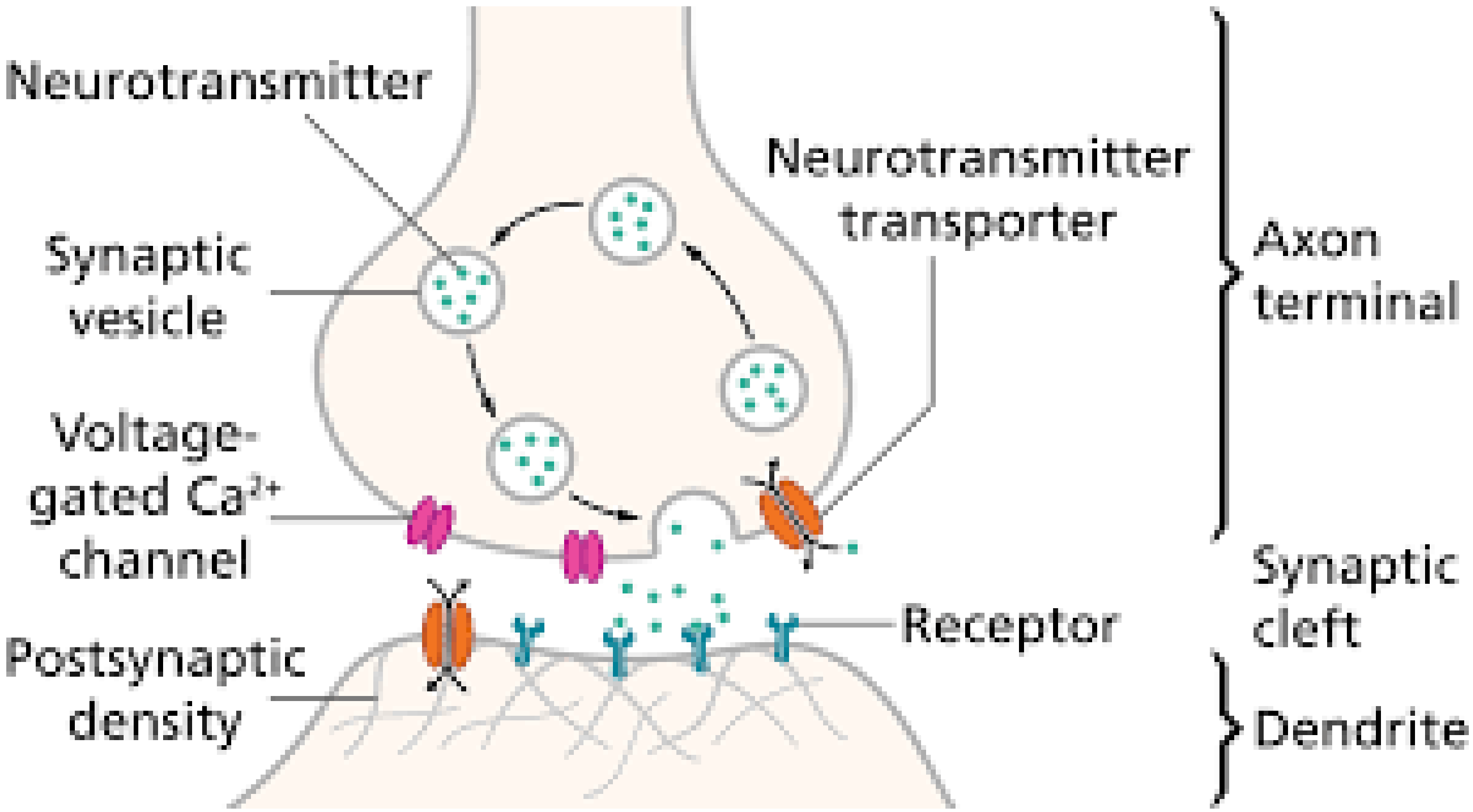
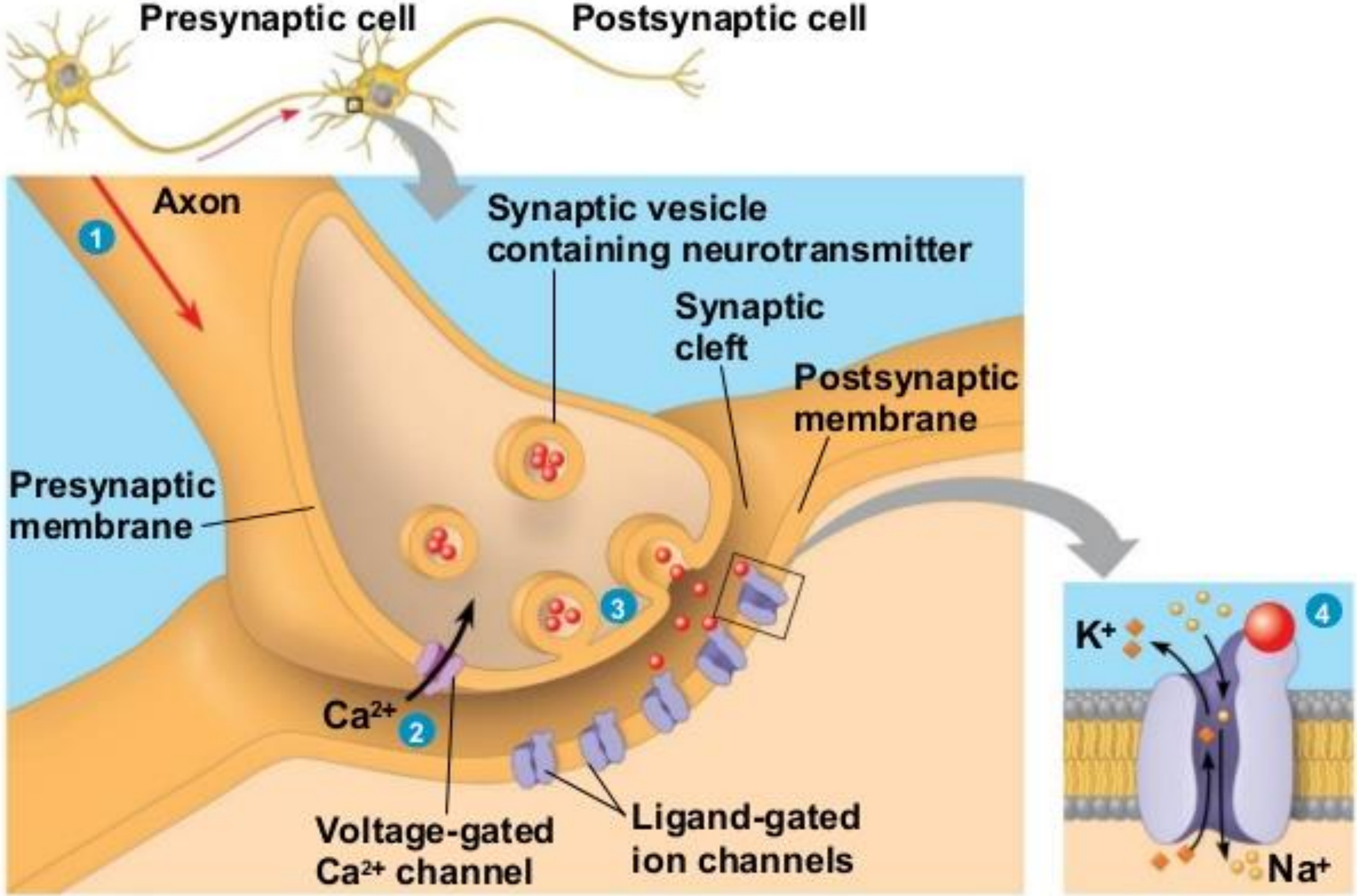
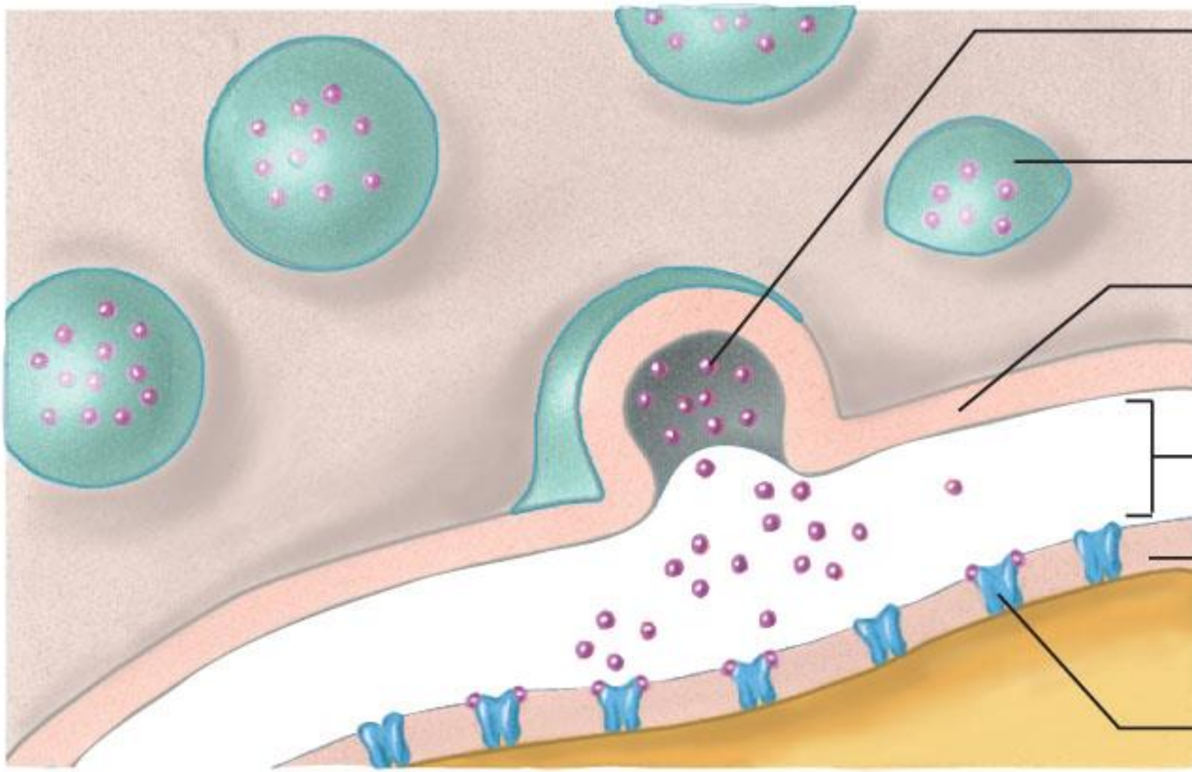

Neurotransmitter
Synaptic vesicle
Voltage-gated Ca2+ channel
Postsynaptic density
Neurotransmitter transporter
Receptor
Axon terminal
Synaptic cleft
Dendrite

Figure 37.15



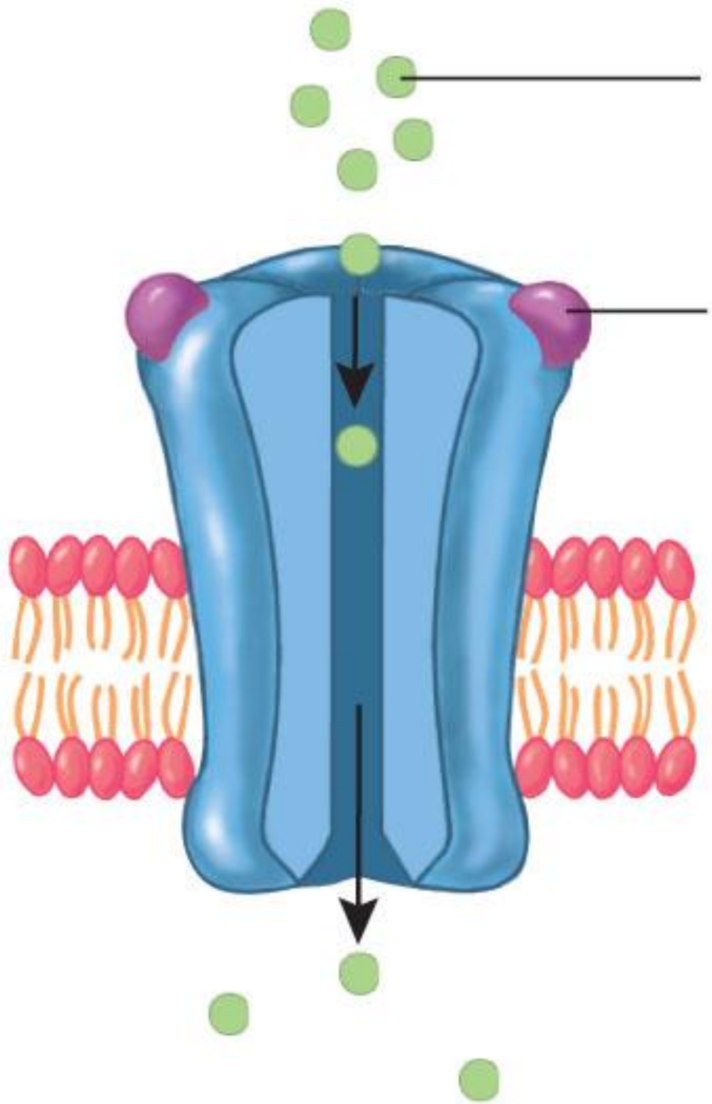
Presynaptic cell
Postsynaptic cell
Axon
1
Synaptic vesicle containing neurotransmitter
Synaptic cleft
Postsynaptic membrane
Presynaptic membrane
3
Ca2+
2
Voltage-gated Ca2+ channel
Ligand-gated ion channels
4
K+
Na+



Neurotransmitter molecules are released and bind to receptors on the postsynaptic membrane.

Neurotransmitters:

- Can be single amino acids, chains of a.a.'s, or protein derivatives
- Excitatory neurotransmitters *cause an AP to occur at the next neuron*
  - *Eg. Norepinephrine (NE), adrenalin, acetylcholine (Ach)*
- Inhibitory neurotransmitters *prevent an AP from occurring at the next neuron*
  - *Eg. Serotonin, GABA*



When a stimulatory neurotransmitter binds to a receptor, Na^+ diffuses into the postsynaptic neuron.

NT Degradation

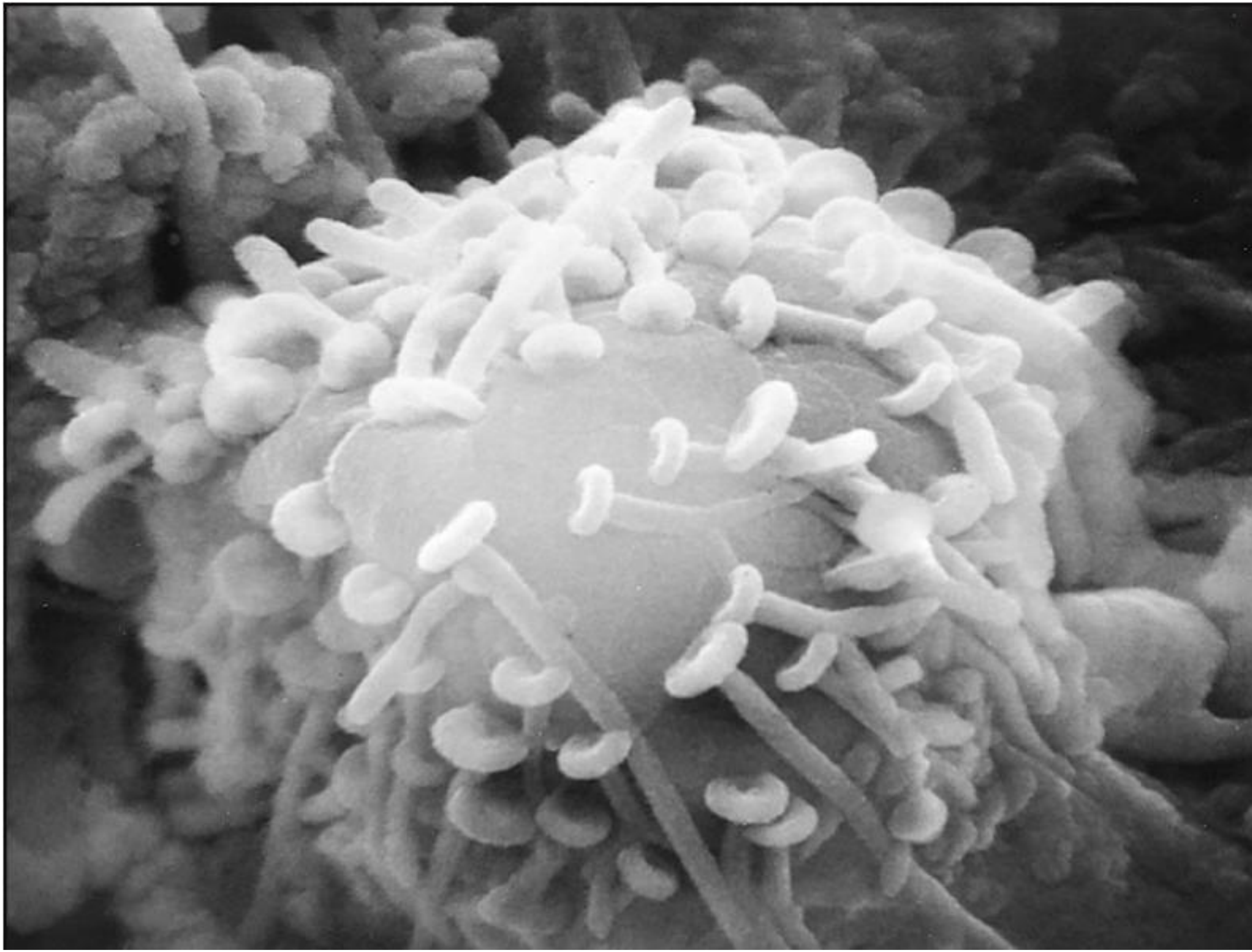
- NTs are quickly ***degraded by enzymes*** on the post-synaptic membrane or ***reabsorbed*** into the pre-synaptic axon terminal

- This prevents continual binding at the post-synaptic receptors
  - *Which would lead to continual stimulation or inhibition of the next neuron*

- **Ach** is degraded by <u>**acetylcholinesterase**</u>
- **NE** is degraded by <u>**monoamine oxidase**</u>
- **Serotonin** is reabsorbed by reuptake carrier proteins

Summation Of Signals…

- A single neuron may receive info from **thousands** of neighboring neurons
  - That is, there may be thousands of synapses around a neuron
- A neuron will **sum up** the excitatory inhibitory signals it receives
  - If a neuron receives significantly more excitatory signals than inhibitory ones, it will **"fire"**



Drugs Action At A Synapse:

At a synapse drugs can:

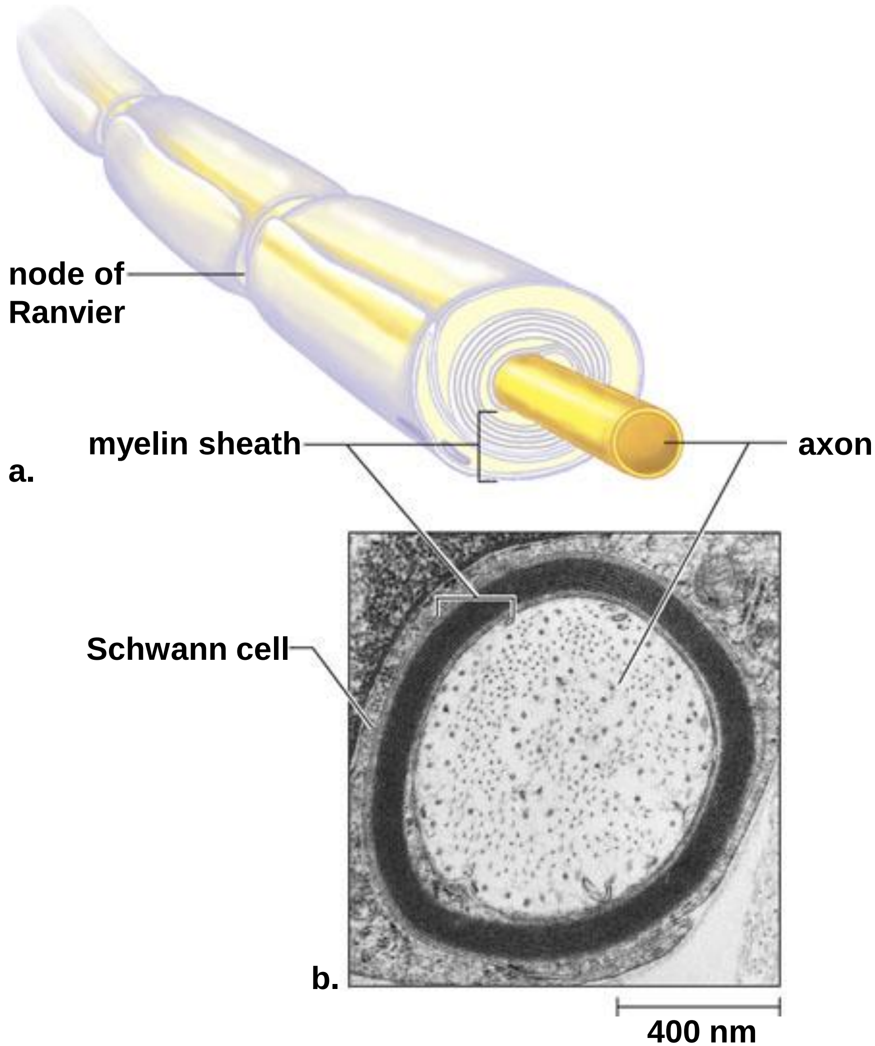
1. Cause NTs to leak out of a synaptic vesicle into the axon terminal
2. Prevent release of NTs into the synaptic cleft
3. Promote release of NTs into the synaptic cleft
4. Prevent reuptake of NTs by the presynaptic membrane
5. Block the enzyme that causes breakdown of the NT
6. Bind to a receptor, mimicking the action of an NT

Consideration: Myelin Sheath

- Some axons are covered with a protective lipid layer called myelin

- *Myelin sheath* is formed by types of neuroglia called **Schwann cells** (PNS) and **oligodendroglial cells** (CNS)

- Schwann cells wrap themselves up to 100 times around an axon, laying down multiple layers of plasma membrane
  - Each cell myelinates only a small portion of an axon (~1mm) so there are gaps between each segment
  - These gaps are called the **Nodes of Ranvier**
  - Speeds up AP transmission as signal can "jump" from node to node


node of
Ranvier
myelin sheath
axon
a.
Schwann cell
b.
400 nm

Why Myelin?

- Myelin serves as an **insulator**
  - Nerve impulses travel faster in myelinated cells:
    - Ie. Non-myelinated = 5 m/s
      Myelinated = 100-200 m/s
    - *Saltatory conduction:*
- Can **protect** nerve cells in the PNS to help them to regenerate if they are damaged