

SNS COLLEGE OF ALLIED HEALTH SCIENCE
Affiliated to The Tamil Nadu Dr. M.G.R Medical University, Chennai



DEPARTMENT OF PHYSICIAN ASSISTANT

COURSE NAME : PHYSIOLOGY

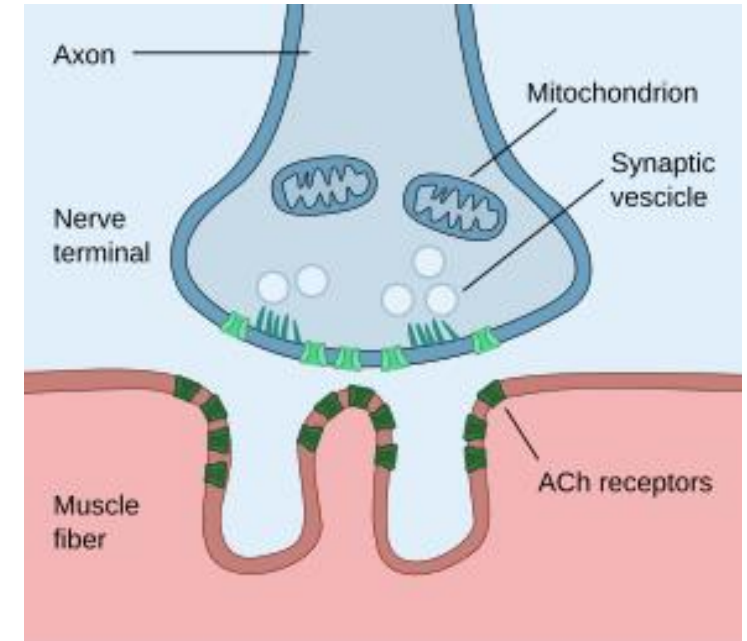
UNIT : INTRODUCTION TO PHYSIOLOGY

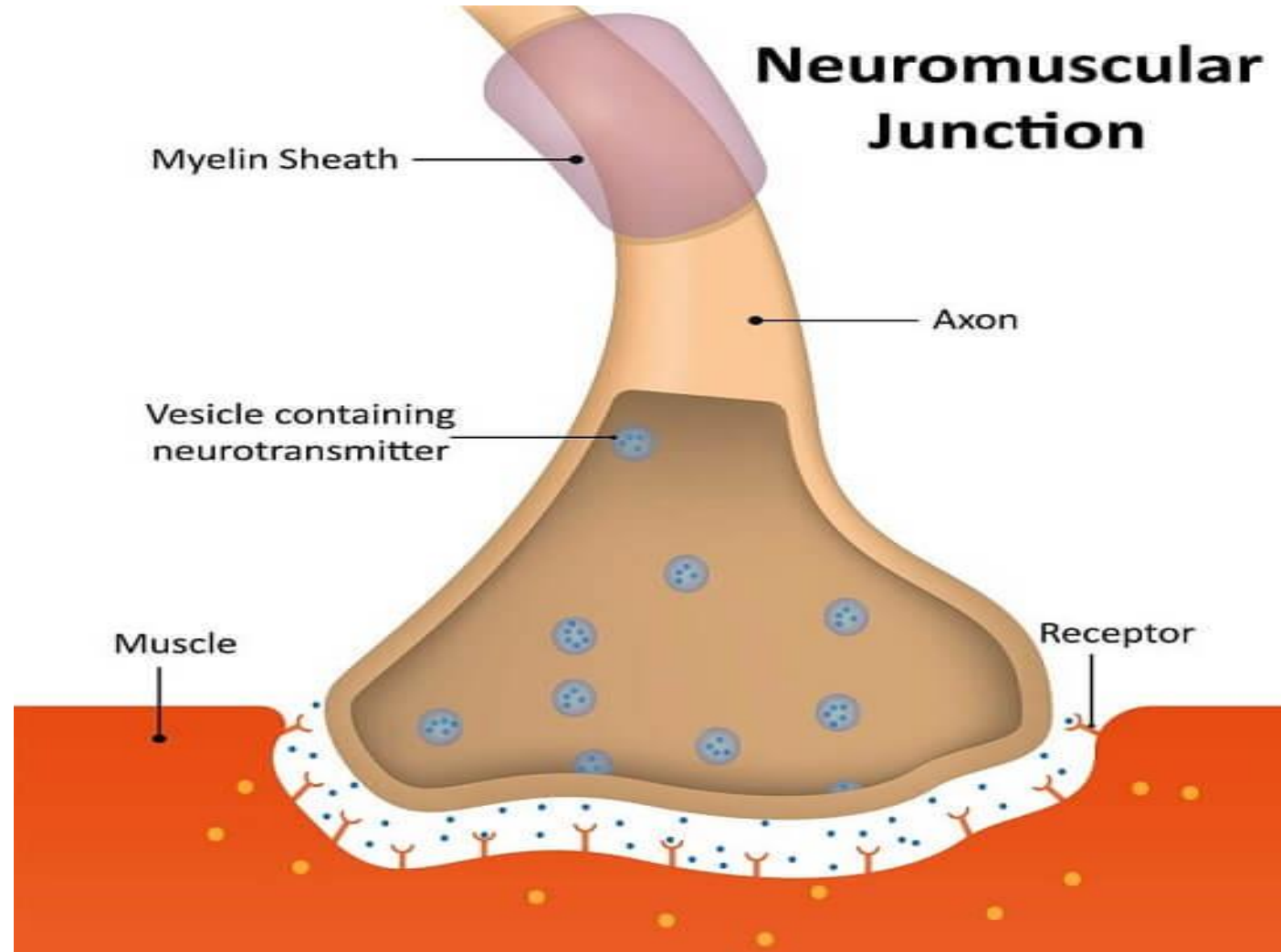
TOPICS : NEUROMUSCULAR JUNCTION AND MUSCLE CONTRACTION

FACULTY NAME : Ms. SINEKA M

INTRODUCTION (Define)

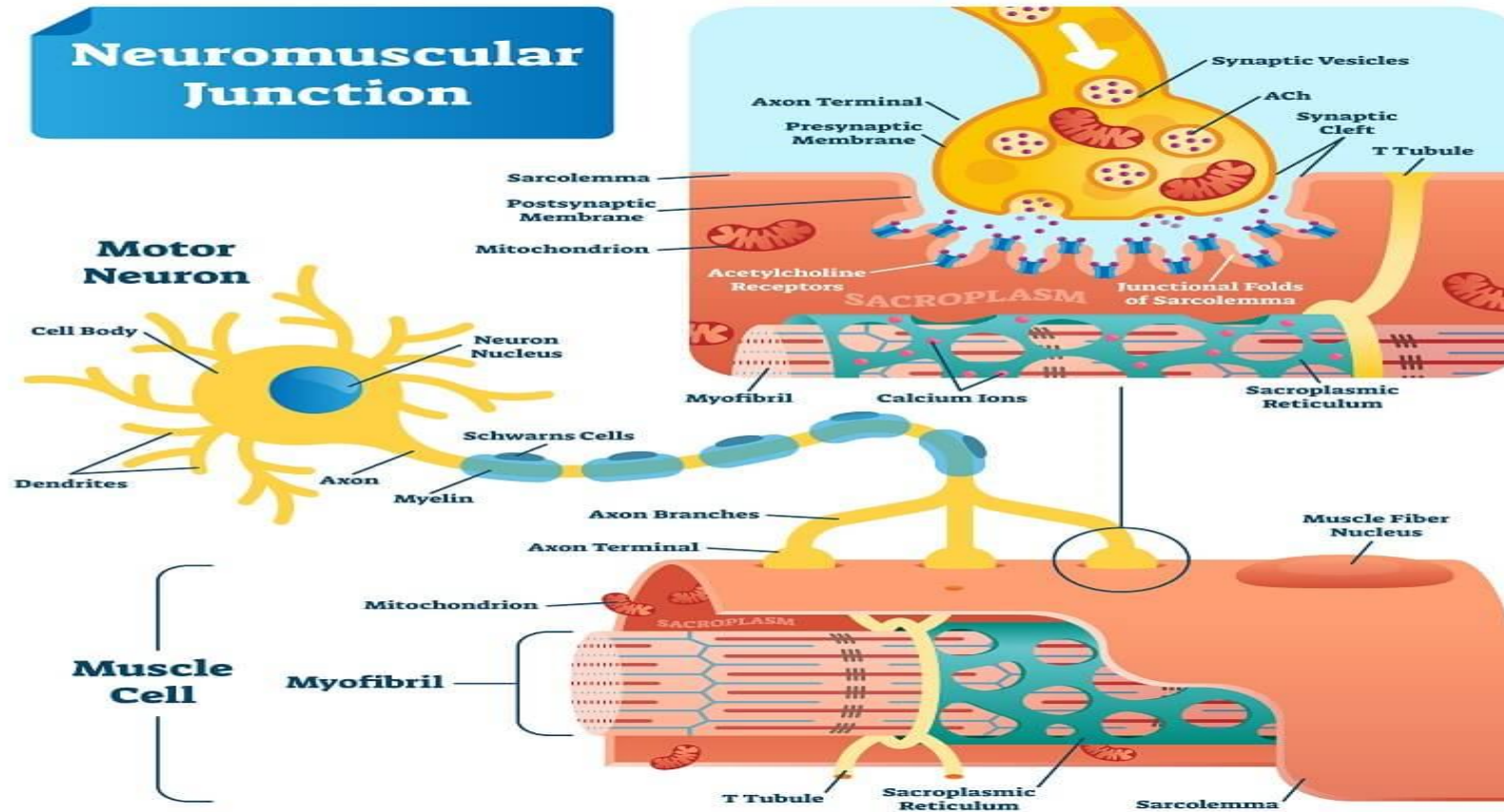
- The neuromuscular junction is the specialized **synapse** where a motor neuron communicates with a **skeletal muscle fiber** to initiate contraction.
- It's a chemical synapse that ensures precise, rapid transmission of signals from the nervous system to the muscle, enabling voluntary movements.





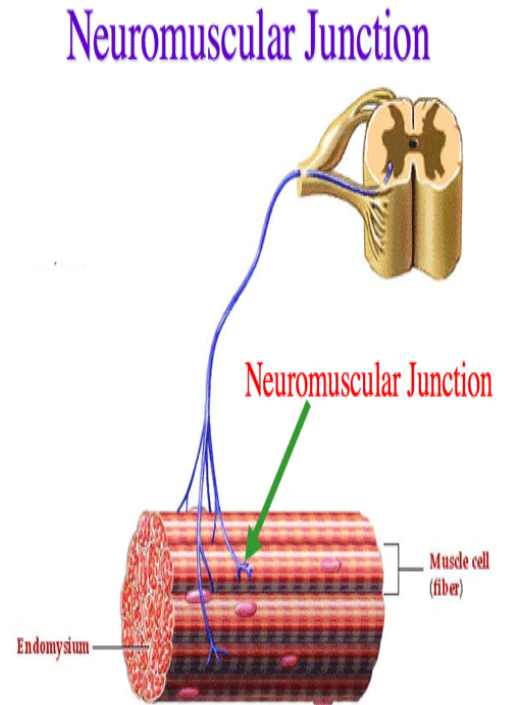
THREE MAIN COMPONENTS

- **Presynaptic terminal:** The end of the motor neuron axon, containing synaptic vesicles filled with the neurotransmitter acetylcholine (ACh).
- **Synaptic cleft:** A narrow gap ($\sim 20\text{-}50$ nm) between the neuron and muscle, filled with extracellular fluid.
- **Postsynaptic membrane:** The muscle cell's sarcolemma, featuring junctional folds that increase surface area and contain nicotinic acetylcholine receptors (nAChRs).

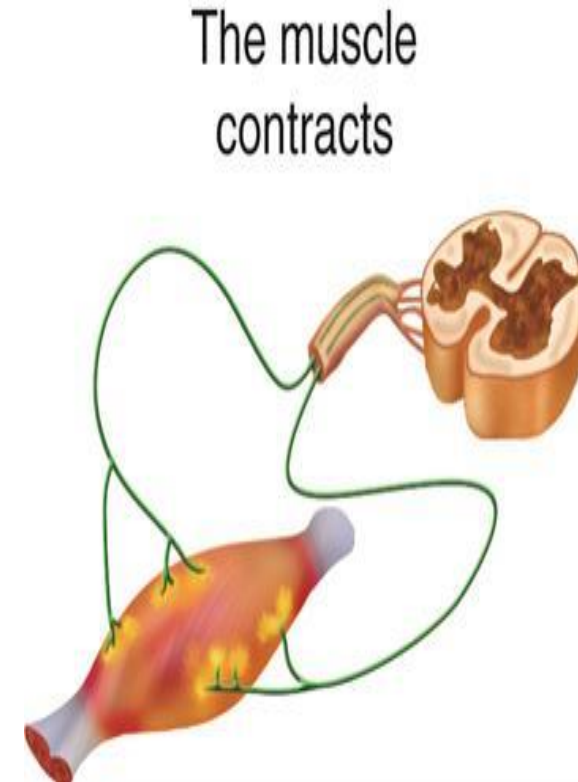


MECHANISM OF MUSCLE CONTRACTION

- **Action Potential Arrival:** An action potential (nerve impulse) travels down the motor neuron axon to the presynaptic terminal, depolarizing the membrane.
- **Calcium Influx and Vesicle Release:** Voltage-gated calcium channels open, allowing Ca^{2+} to enter the terminal. This triggers synaptic vesicles to fuse with the presynaptic membrane and release ACh into the synaptic cleft by exocytosis.

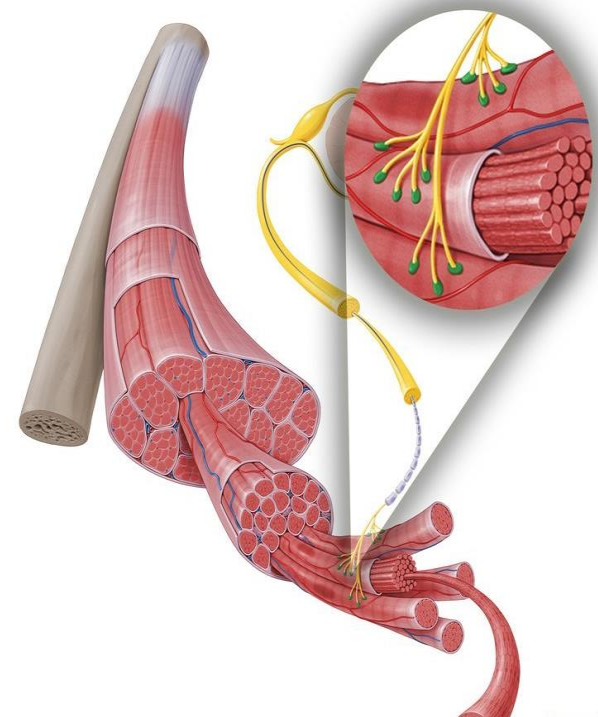


- **Neurotransmitter Binding:** ACh diffuses across the cleft and binds to nAChRs on the postsynaptic membrane. These ligand-gated ion channels open, permitting Na^+ influx, which depolarizes the membrane - this is the **End-plate potential**.
- **Muscle Action Potential Generation:** If the EPP reaches threshold ($\sim 15\text{-}20$ mV), it triggers voltage-gated sodium channels along the sarcolemma, generating a propagating action potential in the muscle fiber.



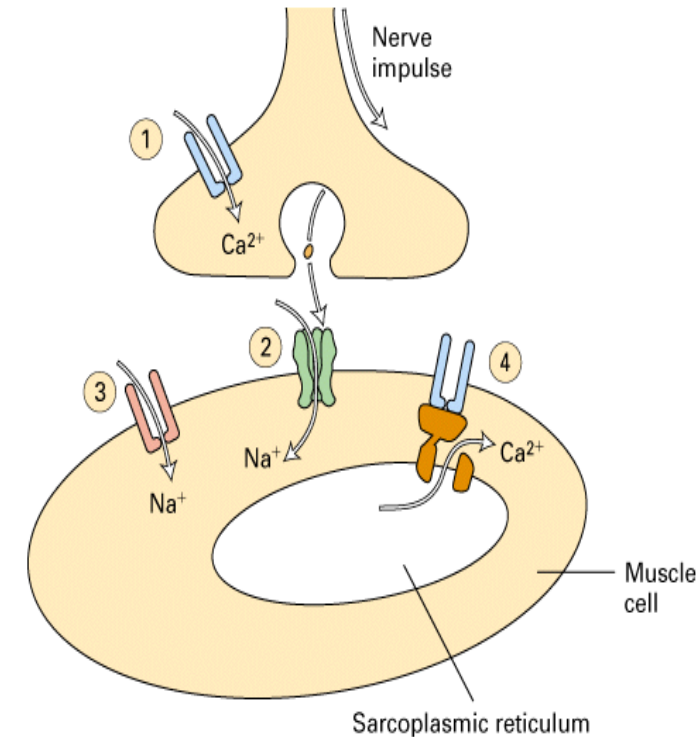
Excitation-Contraction Coupling:

- The action potential spreads via T-tubules (invaginations of the sarcolemma) into the muscle fiber.
- This activates dihydropyridine receptors (DHPRs), which mechanically open ryanodine receptors (RyRs) on the sarcoplasmic reticulum (SR), releasing stored Ca^{2+} into the cytosol (a process called **calcium-induced calcium release**).



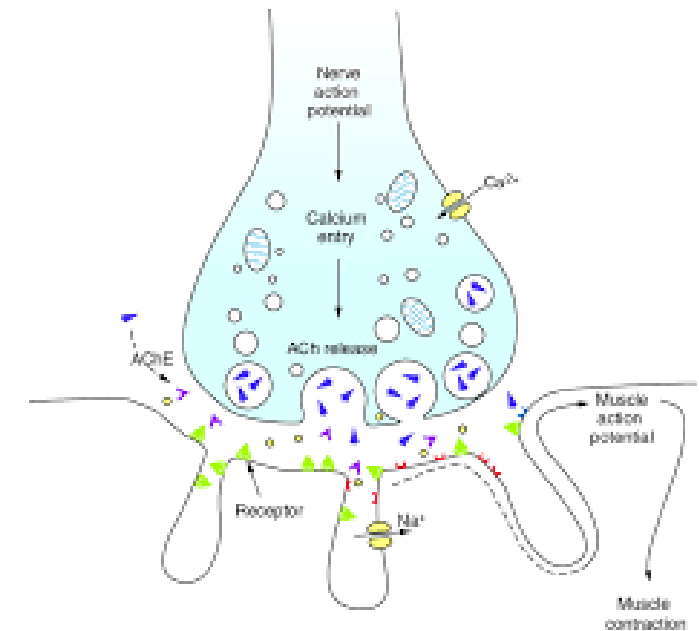
Cross-Bridge Cycling (Sliding Filaments):

- Ca^{2+} binds to troponin on thin filaments (actin), shifting tropomyosin to expose myosin-binding sites.
- Myosin heads bind actin, forming cross-bridges. Powered by ATP hydrolysis, myosin pulls actin filaments toward the sarcomere center, shortening the muscle (contraction).
- Cycle repeats as long as Ca^{2+} and ATP are available.

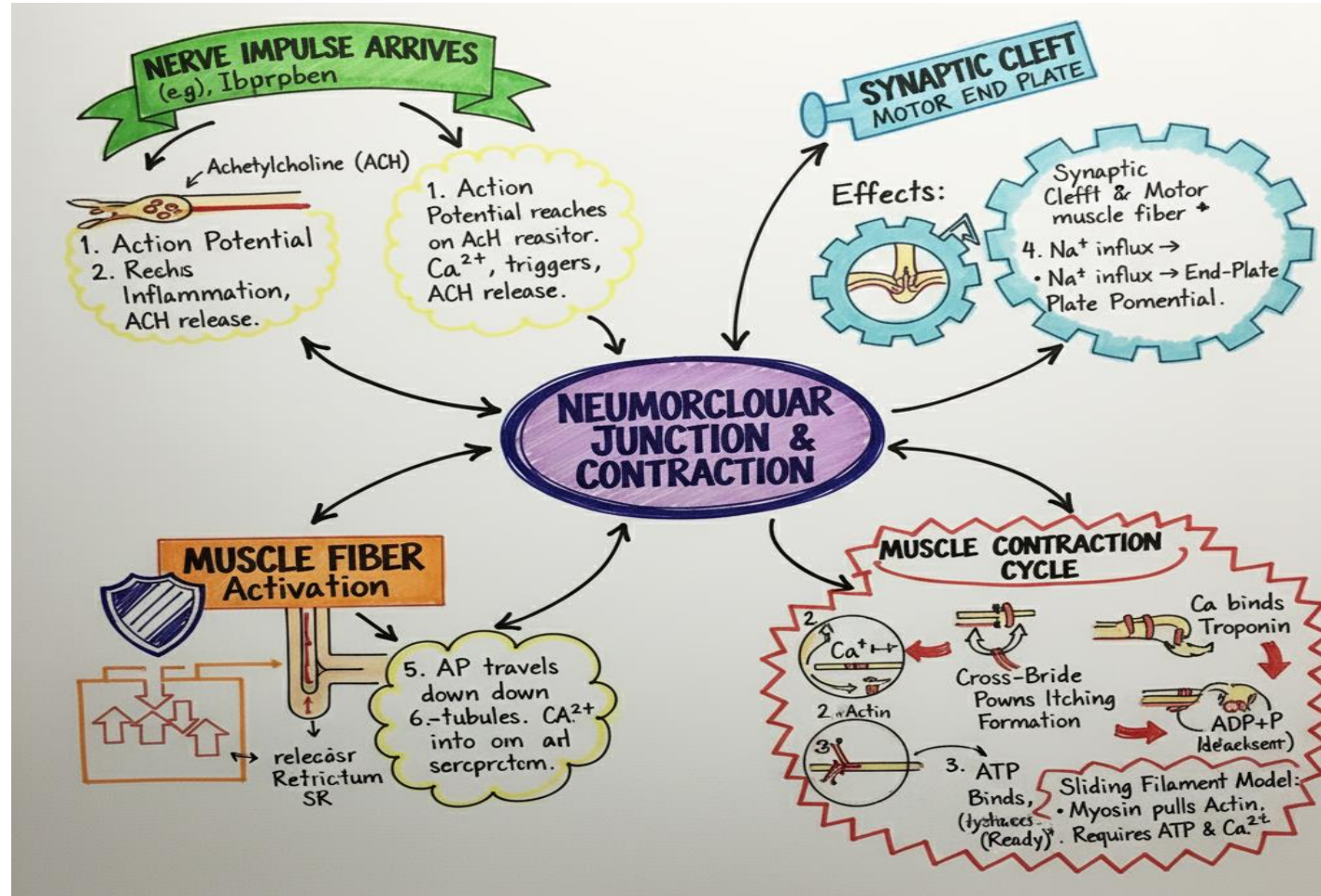


Relaxation:

- ACh is rapidly degraded by acetylcholinesterase (AChE) in the cleft, ending the EPP.
- Ca^{2+} is pumped back into the SR by SERCA pumps.
- Tropomyosin blocks binding sites again, allowing filaments to slide back (passive relaxation aided by elastic elements like titin).



SUMMARY



References

- <https://open.oregonstate.education/anatomy2e/chapter/muscle-fiber-excitation/>
- <https://www.sciencedirect.com/topics/neuroscience/neuromuscular-junction>
- <https://www.ncbi.nlm.nih.gov/books/NBK470413/>