

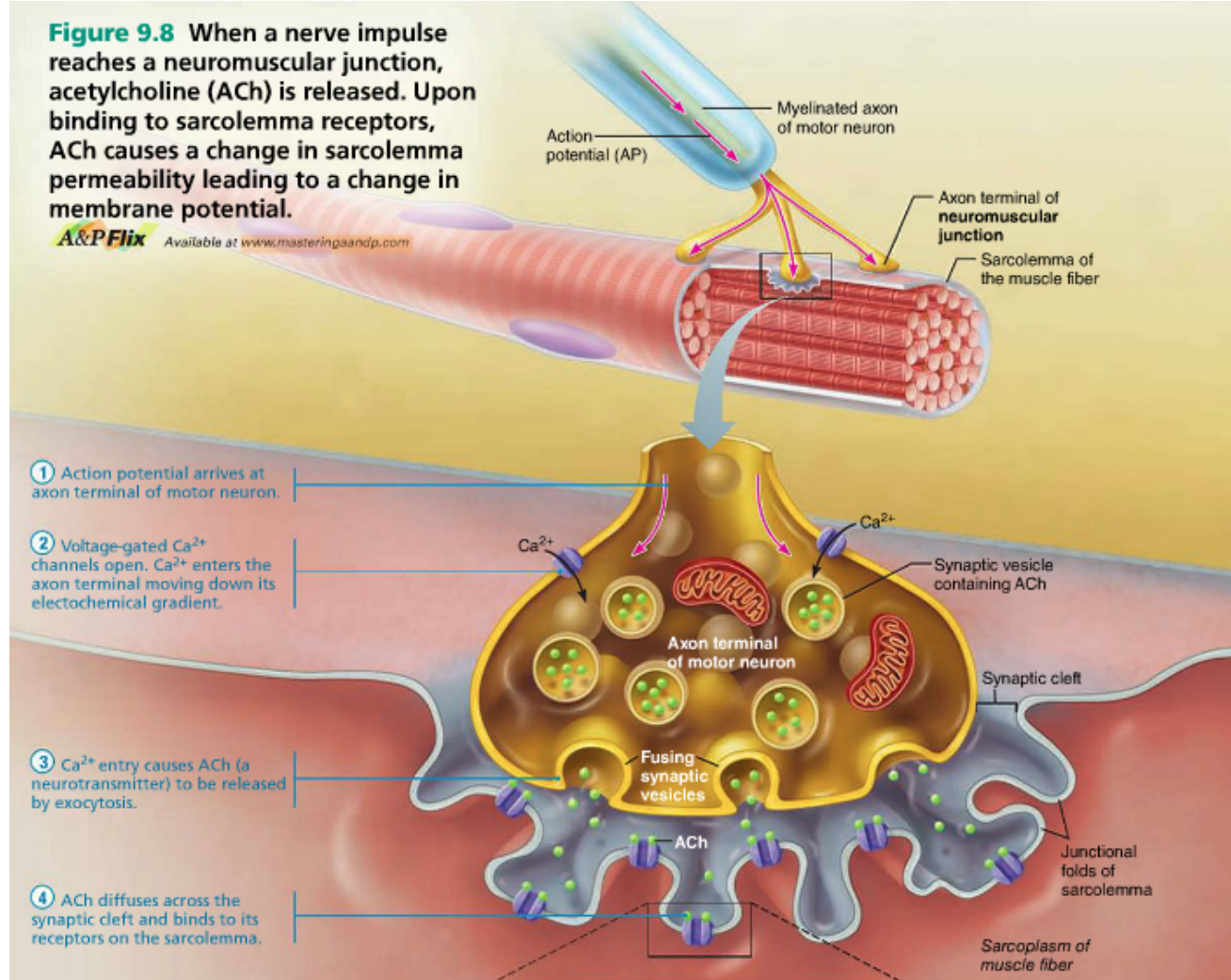
Neuromuscular Junction

Last time...

- Neuromuscular junction
 - Where a neuron meets a muscle fiber (cell)
- Synaptic Cleft, Synaptic Vesicles, Junctional Folds
- Neurotransmitter
 - Acetylcholine
- Degrading Enzyme
 - Acetylcholinesterase

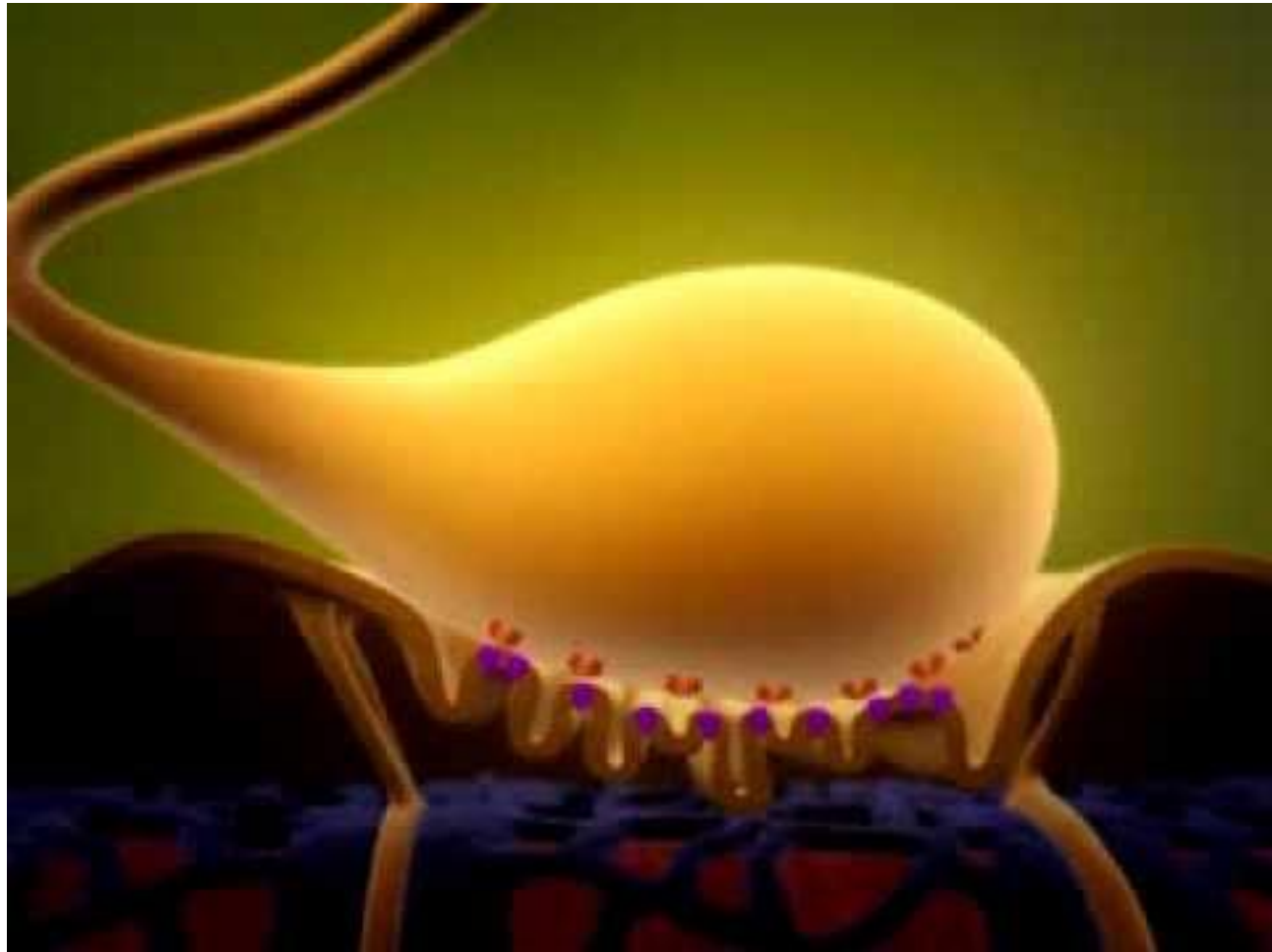
Figure 9.8 When a nerve impulse reaches a neuromuscular junction, acetylcholine (ACh) is released. Upon binding to sarcolemma receptors, ACh causes a change in sarcolemma permeability leading to a change in membrane potential.

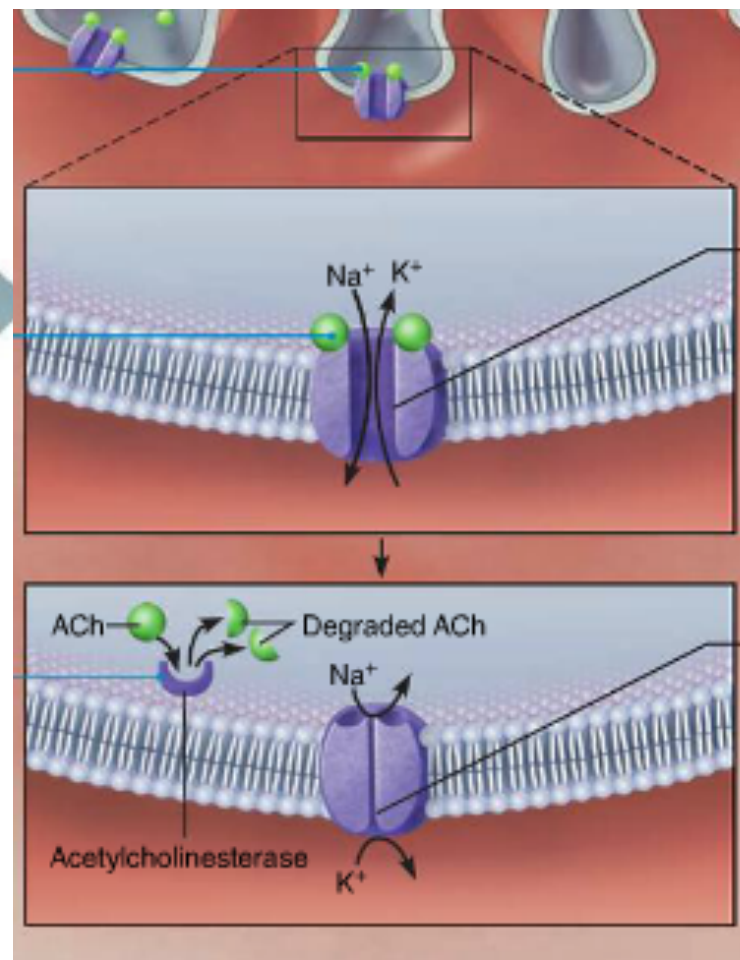
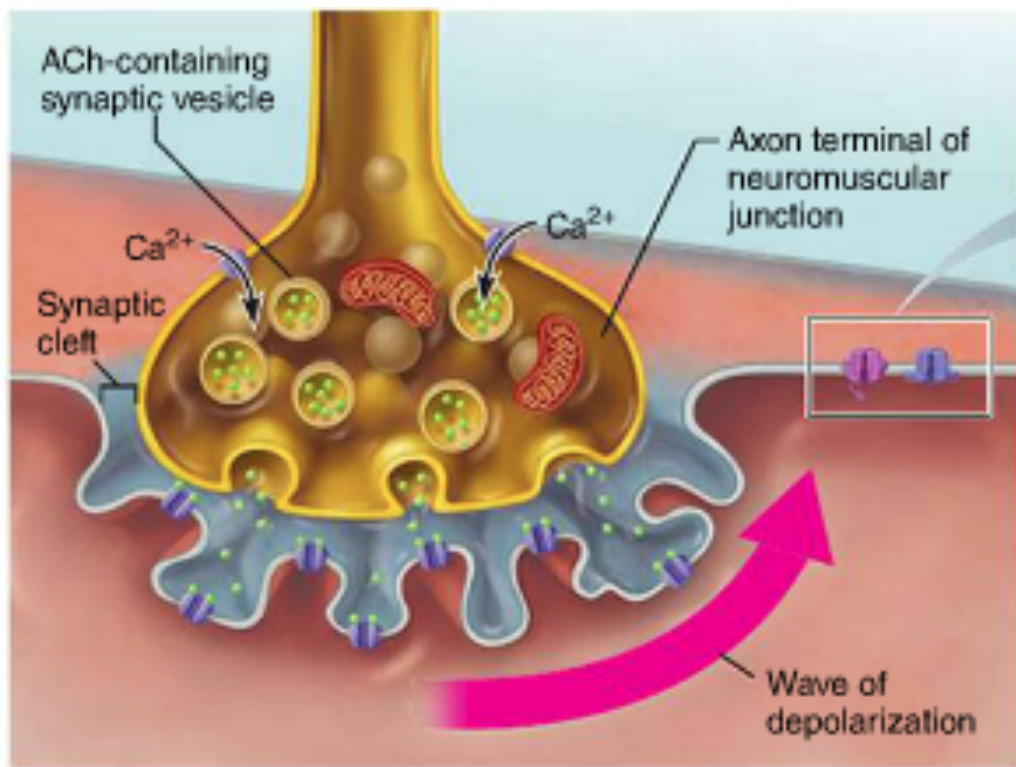
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Depolarization

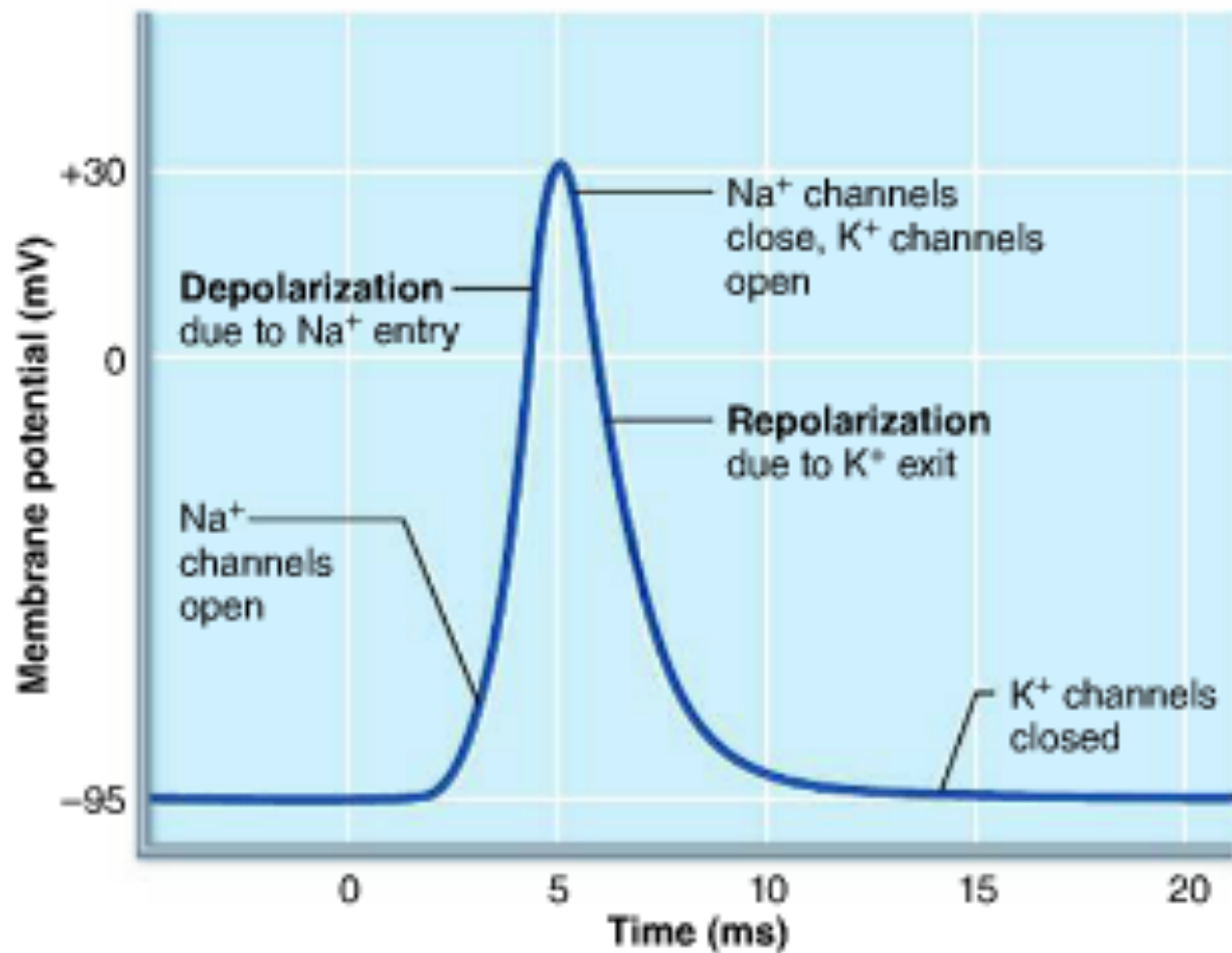
- ACh binds to a gated ion channel in the sarcolemma. ← KEY
- When ACh binds, it opens the channel allowing more Na^+ to flow inside the cell.
 - Na^+ enters faster than K^+ can leave.
 - Causes the interior of the sarcolemma to become less negative
 - depolarization
 - end plate potential





Repolarization

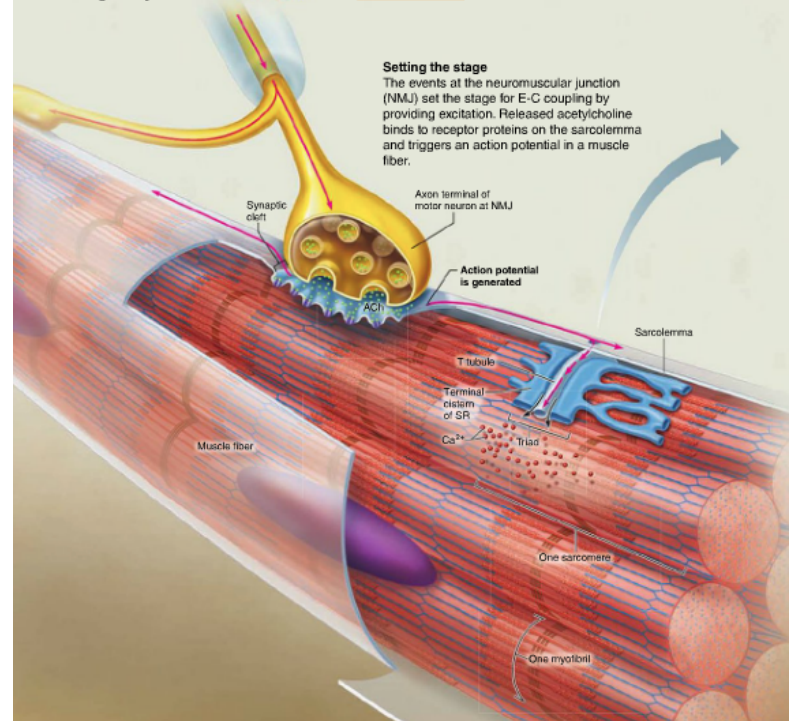
- AChE degrades ACh, closing the Na⁺ channels.
- As the Na⁺ channels close, K⁺ channels open.
 - K⁺ will rush out of the cell due to the high amount of positive charges inside the cell
- Restores the sarcolemma to its polarized state.



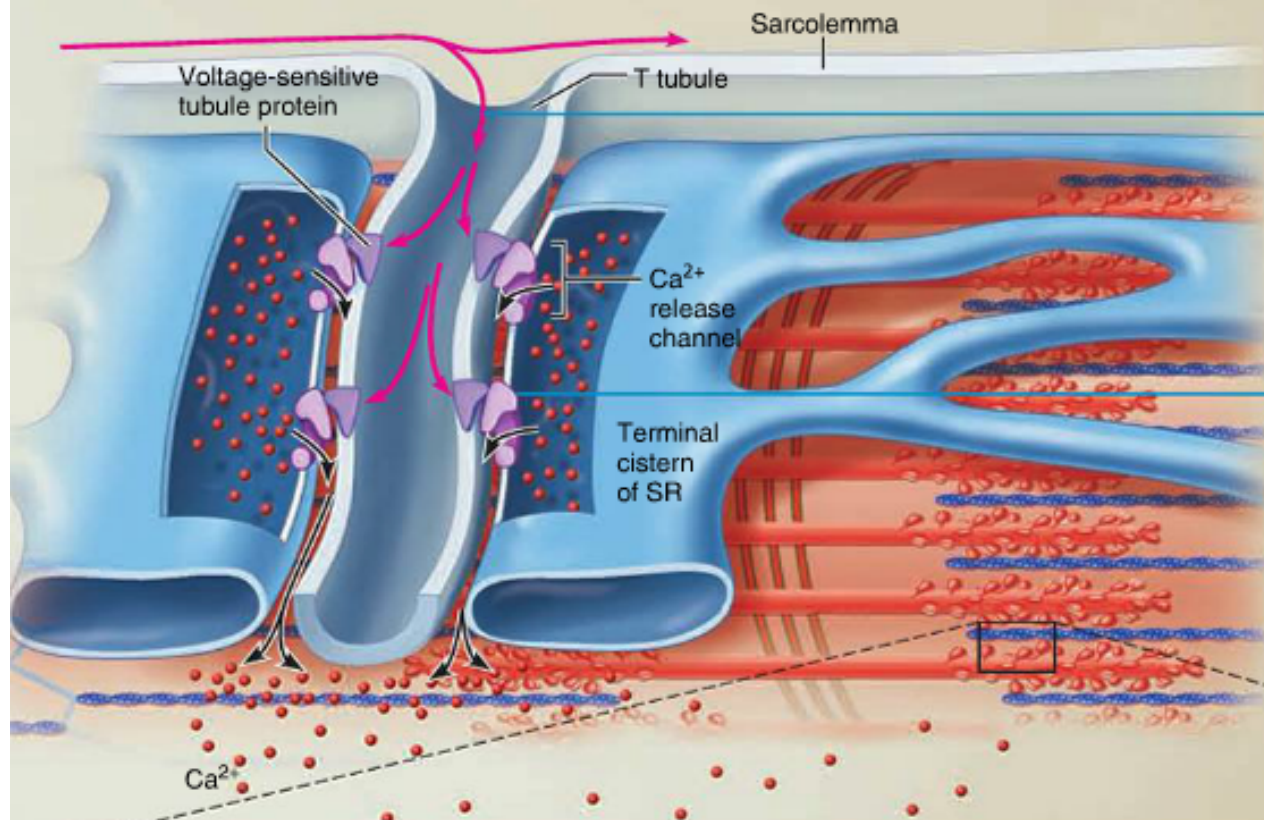
Excitation-Contraction Coupling

The sequence of events by which transmission of an action potential along the sarcolemma leads to myofilament sliding (contraction).

Figure 9.11 Excitation-contraction (E-C) coupling is the sequence of events by which transmission of an action potential along the sarcolemma leads to the sliding of myofilaments. *A&P11x* Available at www.masteringandp.com

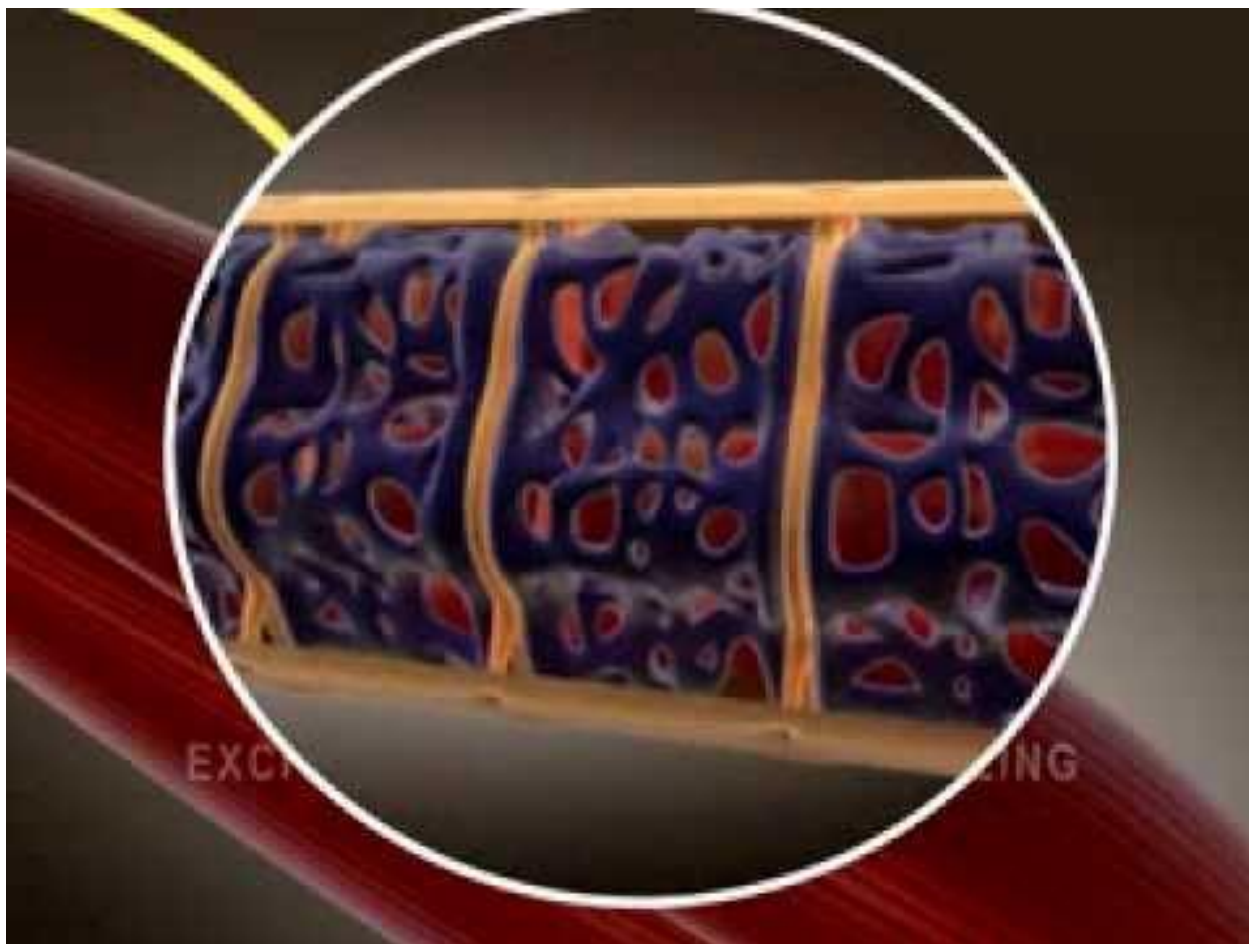


Steps in E-C Coupling:



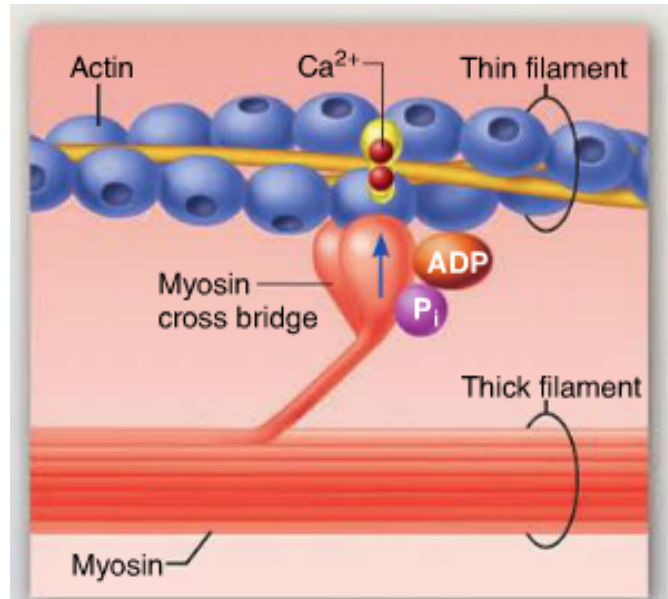
① The action potential (AP) propagates along the sarcolemma and down the T tubules.

② Calcium ions are released. Transmission of the AP along the T tubules of the triads causes the voltage-sensitive tubule proteins to change shape. This shape change opens the Ca²⁺ release channels in the terminal cisterns of the sarcoplasmic reticulum (SR), allowing Ca²⁺ to flow into the cytosol.

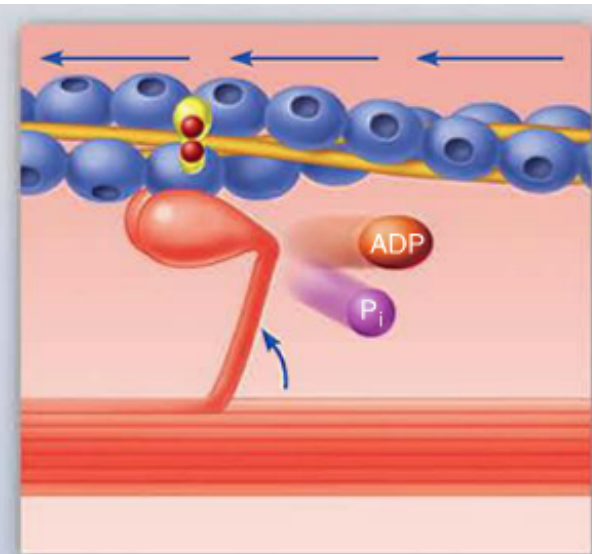


Cross-Bridge Cycling

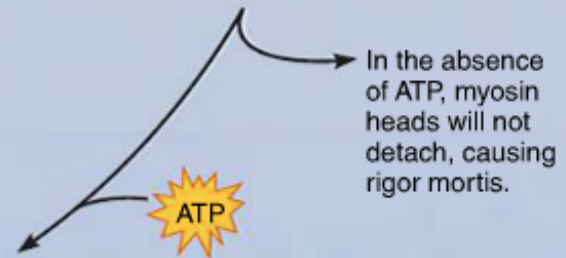
- Relaxed Muscle
 - Low Ca^{+2} levels
 - Troponin has locked tropomyosin into position so that it blocks the G Actin active spots
- Contraction
 - Ca^{+2} levels increase
 - Ca^{+2} binds to troponin, shifting the tropomyosin
 - Active sites exposed
 - Cross-bridging can now occur

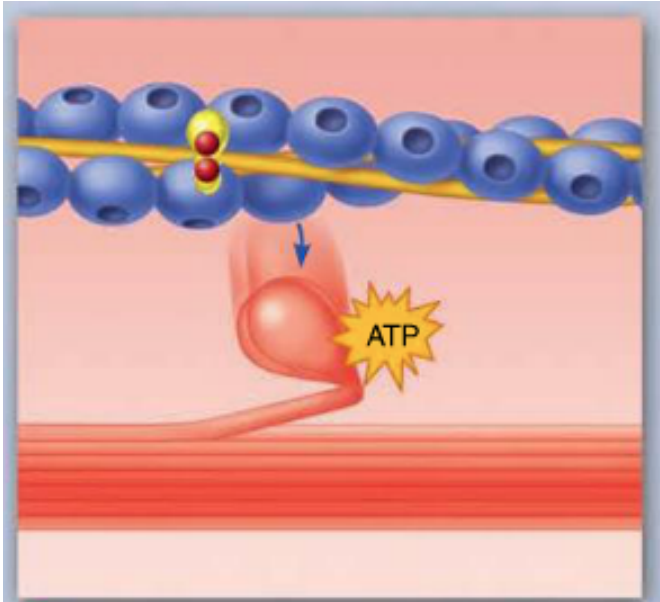


① **Cross bridge formation.** Energized myosin head attaches to an actin myofilament, forming a cross bridge.

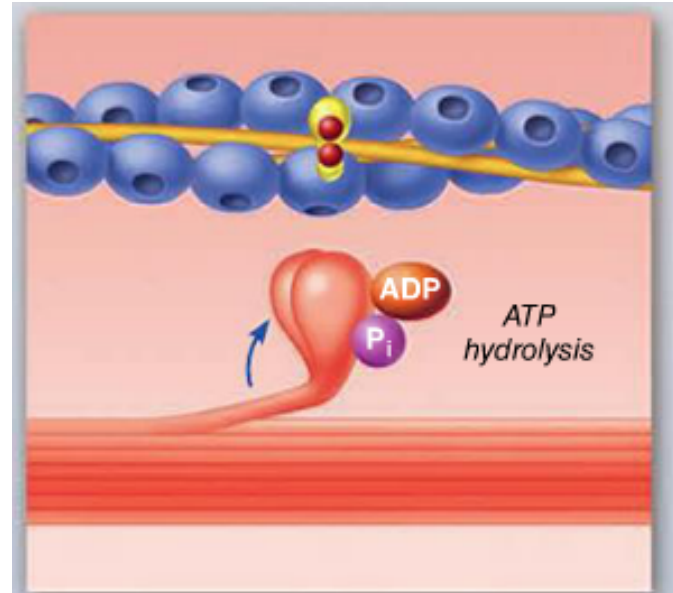


② **The power (working) stroke.** ADP and P_i are released and the myosin head pivots and bends, changing to its bent low-energy state. As a result it pulls the actin filament toward the M line.

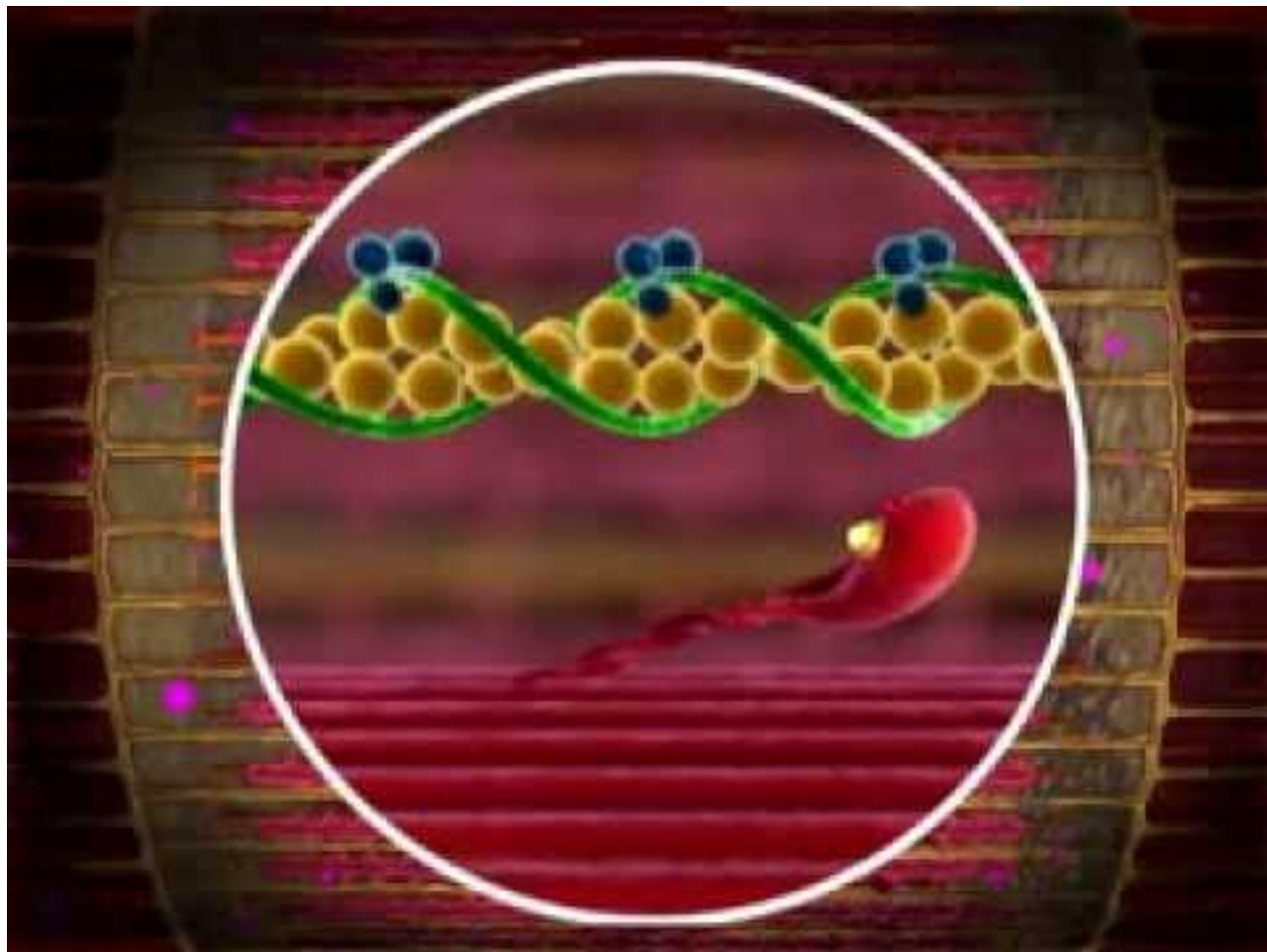




③ **Cross bridge detachment.** After ATP attaches to myosin, the link between myosin and actin weakens, and the myosin head detaches (the cross bridge "breaks").



④ **Cocking of the myosin head.** As ATP is hydrolyzed to ADP and P_i , the myosin head returns to its prestroke high-energy, or "cocked," position.*



Today's Assignment

Create a flow chart of muscle contraction:

- Begin at the neuromuscular junction and end when the sarcomere has returned to its relaxed state.
 - You need to be sure if there something happening that you include how it happened (stimulus, neurotransmitter, attachment of a molecule, transmission, etc.)