MECHANISM OF SKELETAL MUSCLE CONTRACTION

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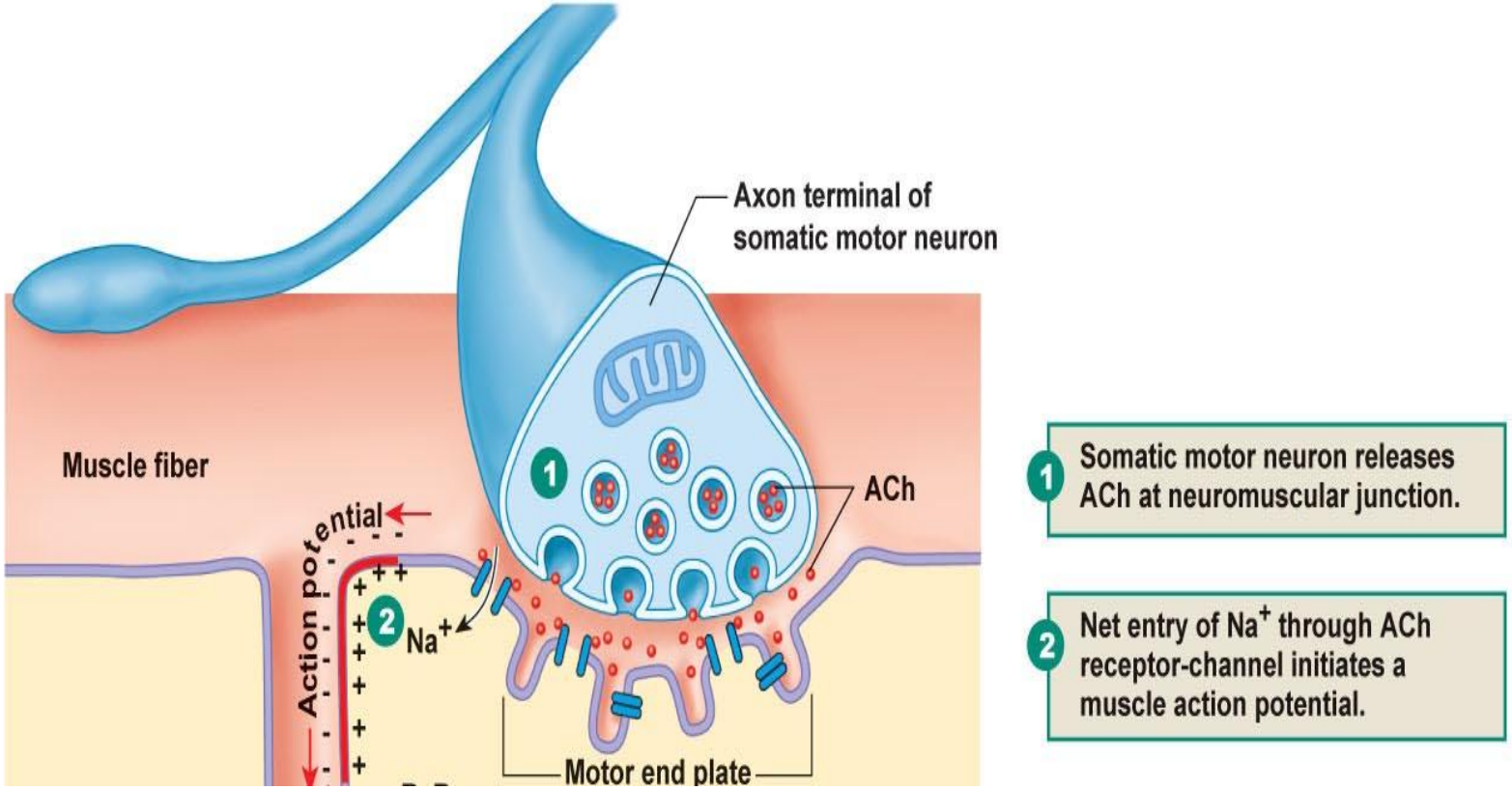
SKELETAL MUSCLE

PHYSIOLOGY OF CONTRACTION



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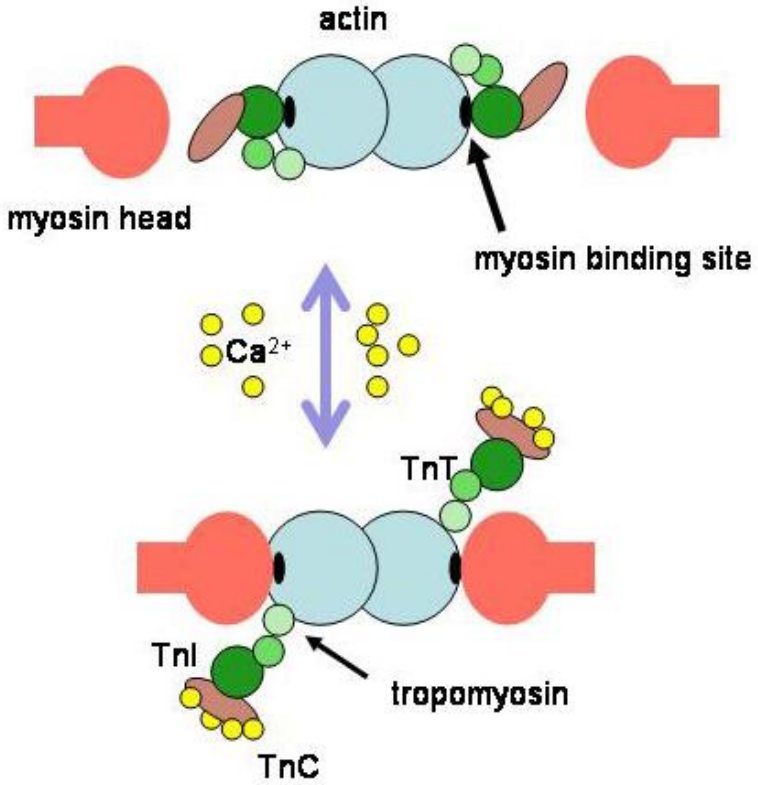
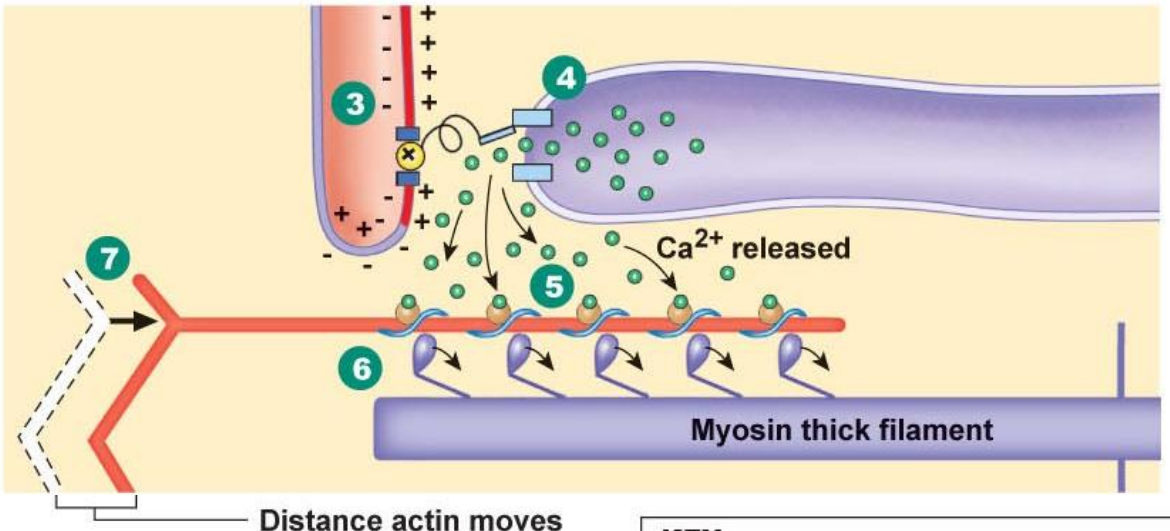




(b) Excitation-contraction coupling

3 Action potential in t-tubule alters conformation of DHP receptor.

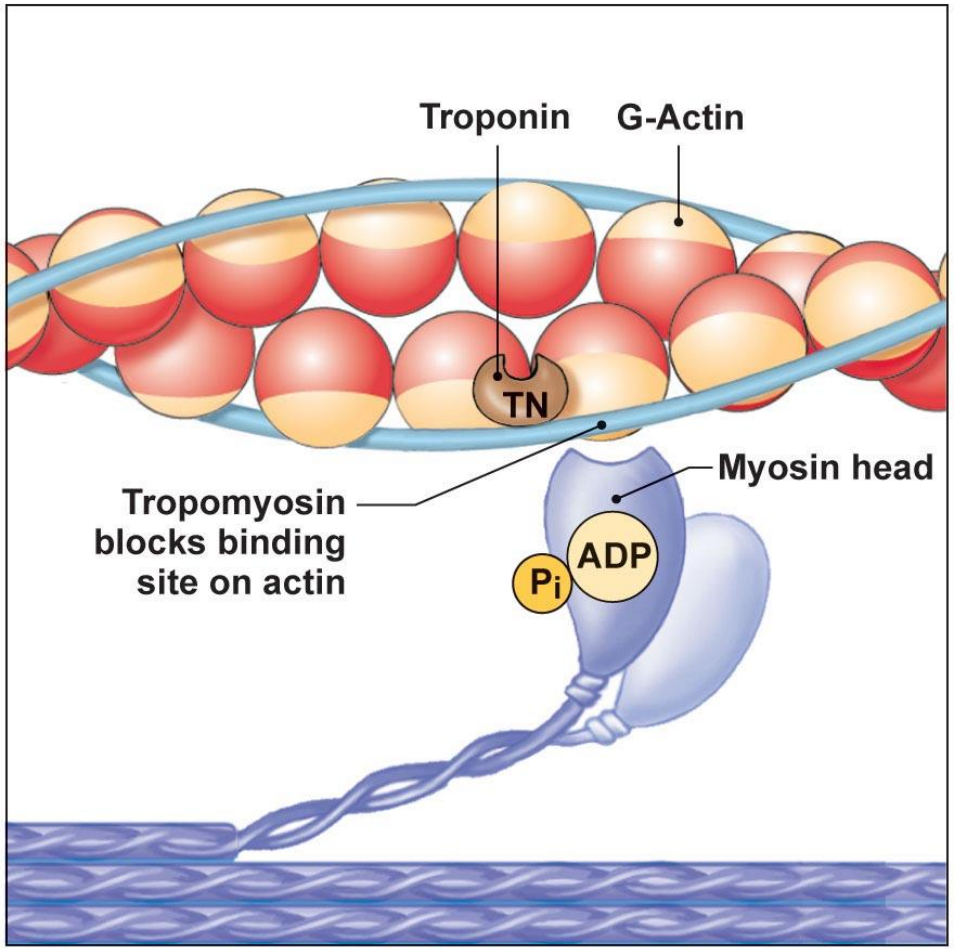
4 DHP receptor opens RyR Ca^{2+} release channels in sarcoplasmic reticulum and Ca^{2+} enters cytoplasm.

5 Ca^{2+} binds to troponin, allowing actin-myosin binding.

KEY
DHP = dihydropyridine L-type calcium channel RyR = ryanodine receptor-channel

Skeletal Muscle

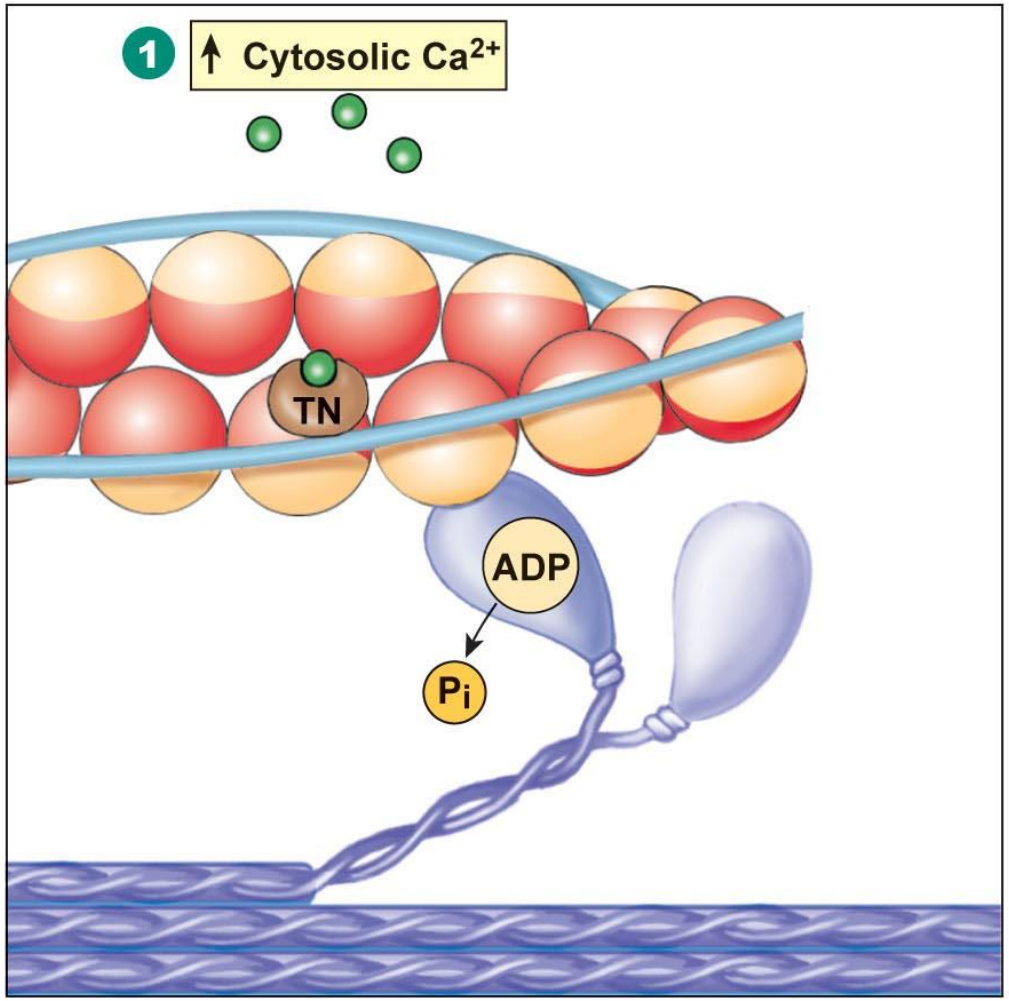
Physiology of Contraction



(a) Relaxed state. Myosin head cocked.

Skeletal Muscle

Physiology of Contraction

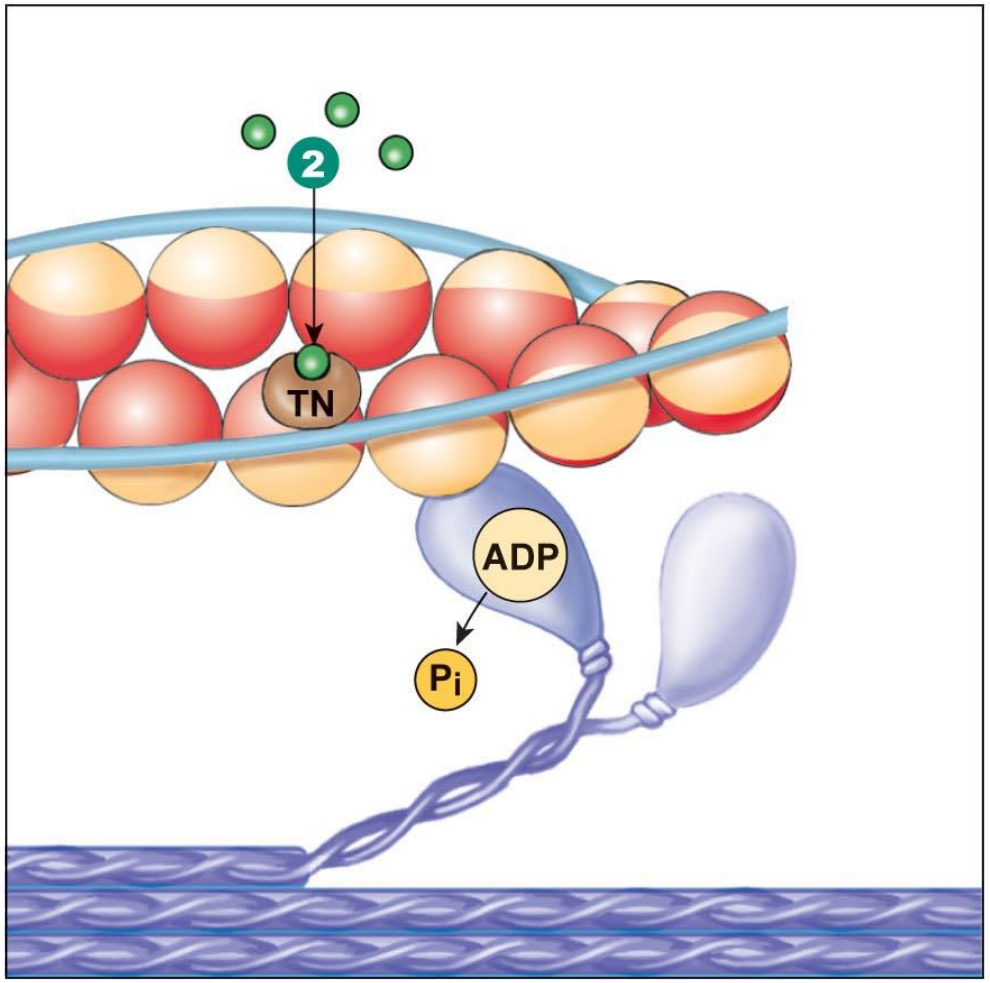


1 Ca^{2+} levels increase in cytosol.

(b) Initiation of contraction

Skeletal Muscle

Physiology of Contraction

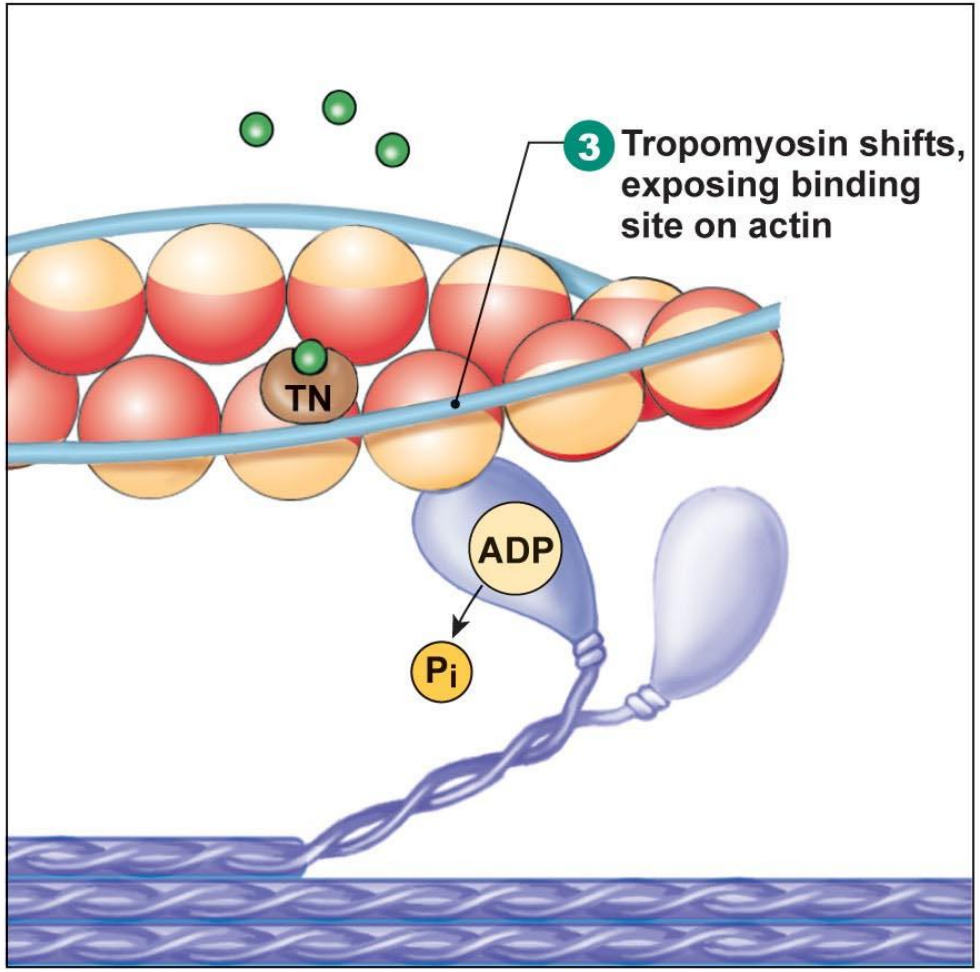


2 Ca^{2+} binds to troponin (TN).

(b) Initiation of contraction

Skeletal Muscle

Physiology of Contraction

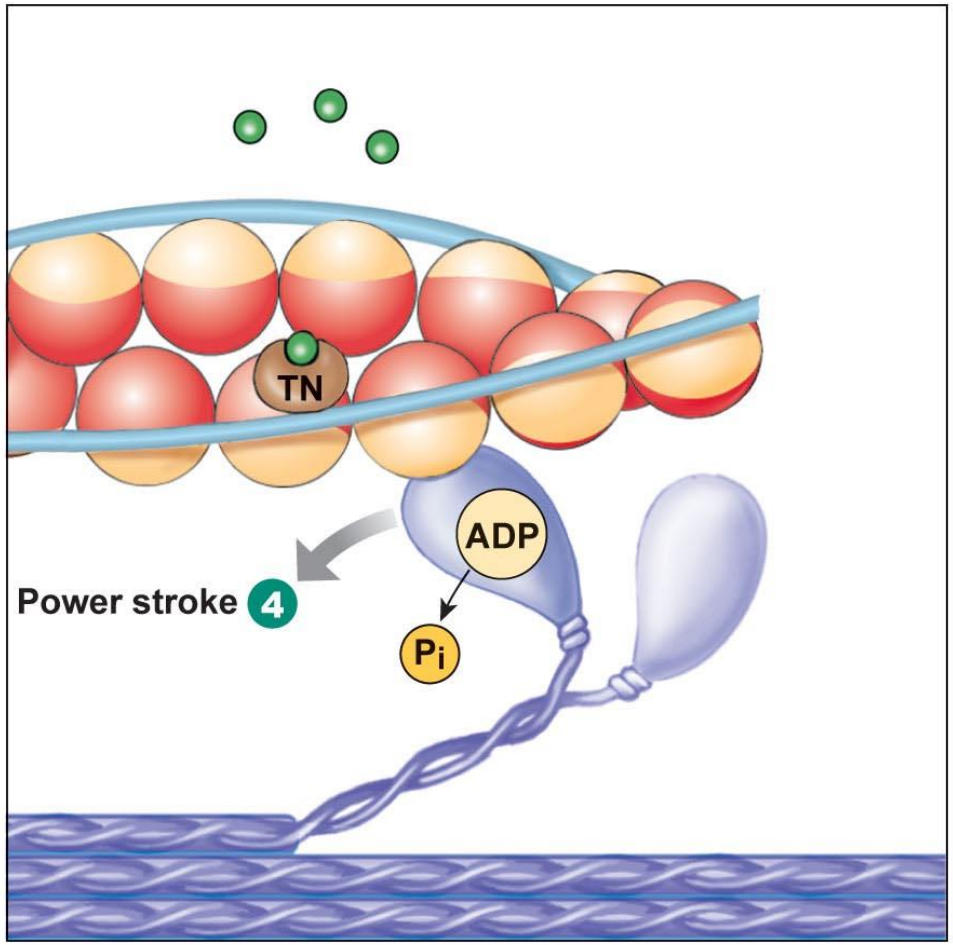


3 Troponin-Ca^{2+} complex pulls tropomyosin away from actin's myosin-binding site.

(b) Initiation of contraction

Skeletal Muscle

Physiology of Contraction

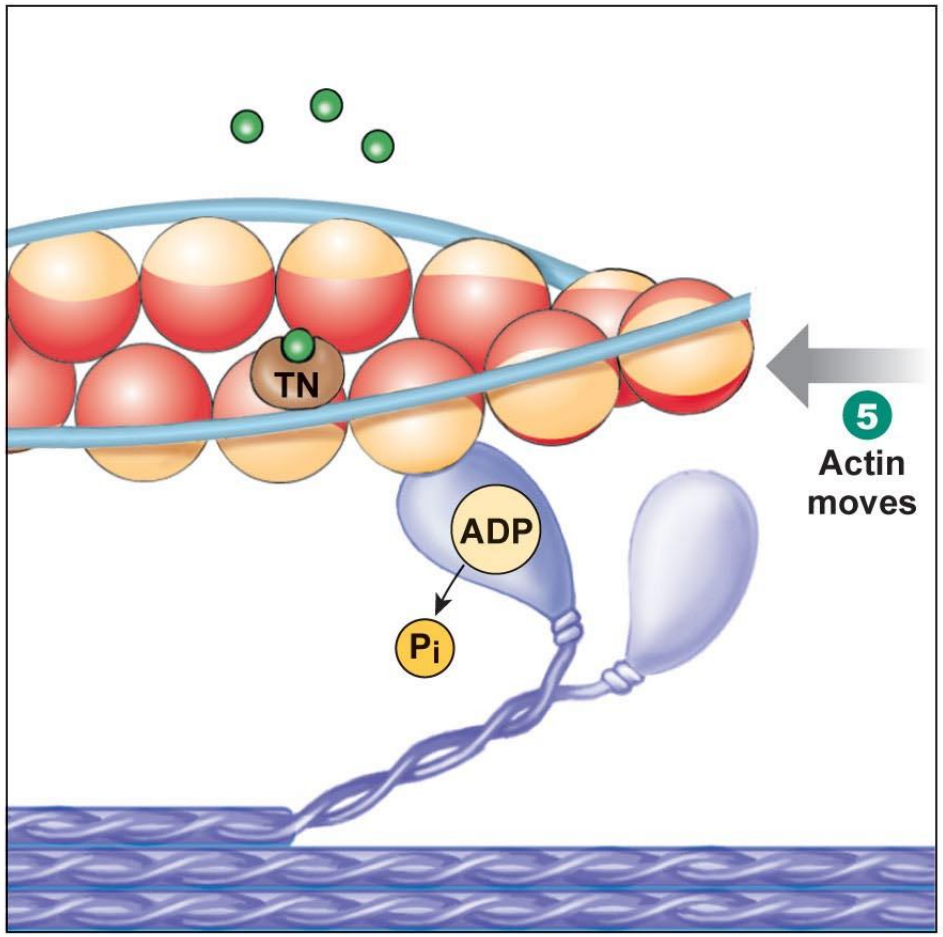


(b) Initiation of contraction

4 Myosin binds to actin and completes power stroke.

Skeletal Muscle

Physiology of Contraction

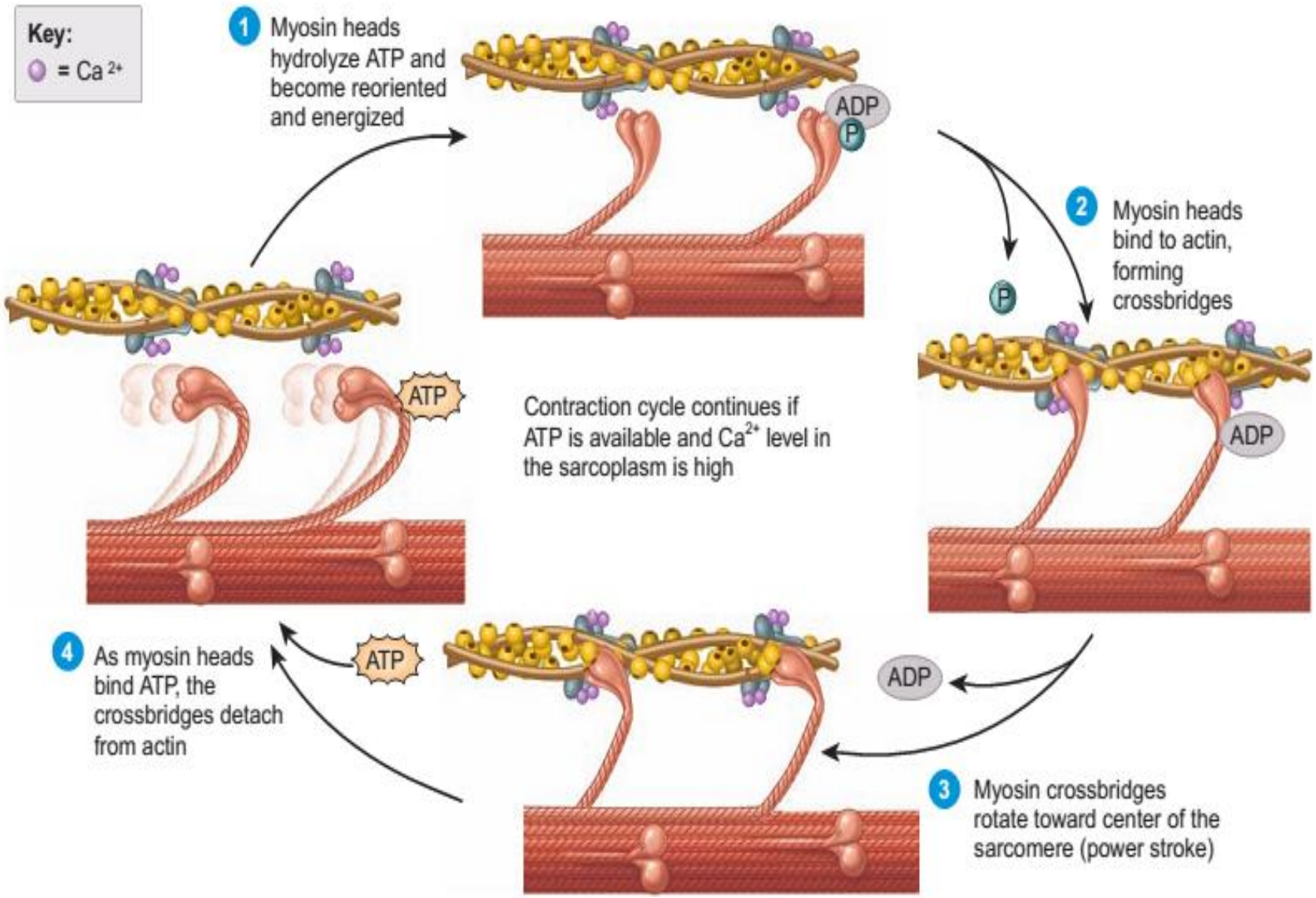


(b) Initiation of contraction

5 Actin filament moves.

SLIDING FILAMENT THEORY

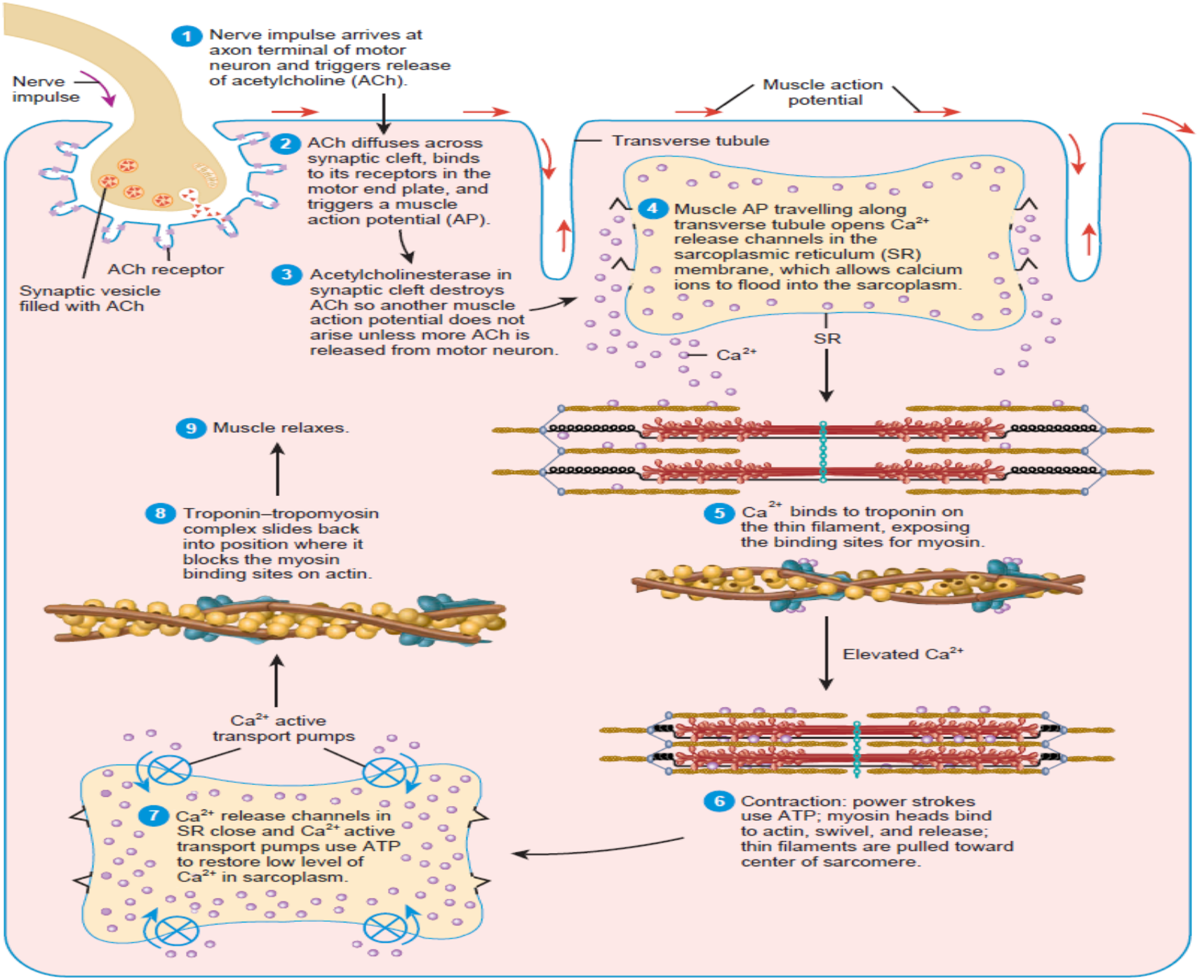
- Also called ***ratchet theory***
- Put forward by **A.F.Huxley** & **H.E.Huxley** in 1954.
- Explains *how actin filaments slide over myosin filaments* forming actin- myosin complex during muscle contraction.
- Also says that sliding brought about by repeated cross bridge cycles b/w myosin head & actin molecule.


Key:
= Ca 2+
1 Myosin heads hydrolyze ATP and become reoriented and energized
ADP
P
2 Myosin heads bind to actin, forming crossbridges
P
ADP
ATP
Contraction cycle continues if ATP is available and Ca2+ level in the sarcoplasm is high
4 As myosin heads bind ATP, the crossbridges detach from actin
ATP
ADP
3 Myosin crossbridges rotate toward center of the sarcomere (power stroke)

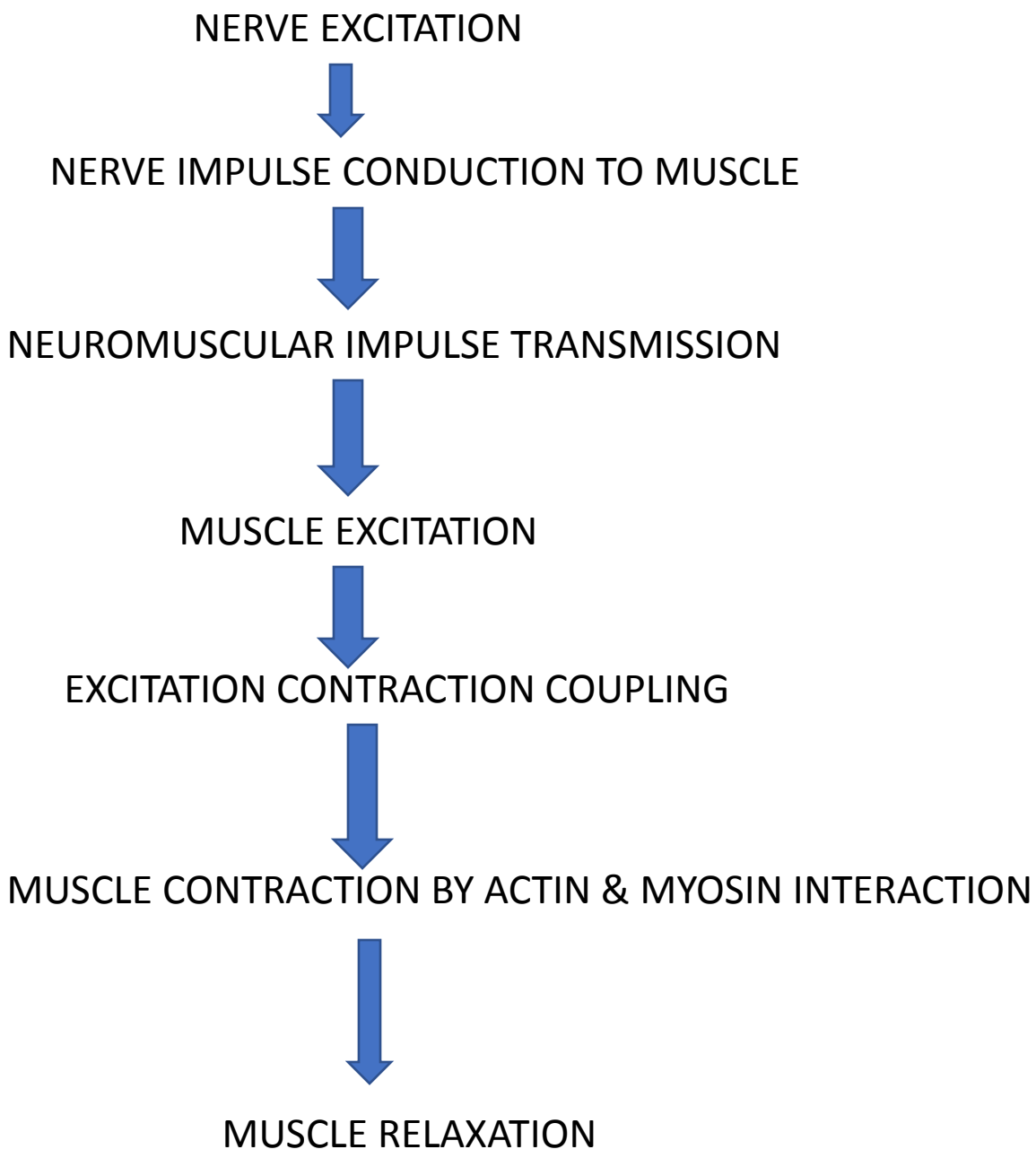
Applied aspect

RIGOR MORTIS :-

- Shortening & rigidity of all body muscles some hours after death.
- Rigidity occurs due to fixation of cross bridges due to ATP loss.
- Forensic importance:-Helps in fixing time of death.


1 Nerve impulse arrives at axon terminal of motor neuron and triggers release of acetylcholine (ACh).
Nerve impulse
2 ACh diffuses across synaptic cleft, binds to its receptors in the motor end plate, and triggers a muscle action potential (AP).
ACh receptor
Synaptic vesicle filled with ACh
3 Acetylcholinesterase in synaptic cleft destroys ACh so another muscle action potential does not arise unless more ACh is released from motor neuron.
Muscle action potential
Transverse tubule
4 Muscle AP travelling along transverse tubule opens Ca^{2+} release channels in the sarcoplasmic reticulum (SR) membrane, which allows calcium ions to flood into the sarcoplasm.
SR
Ca^{2+}
5 Ca^{2+} binds to troponin on the thin filament, exposing the binding sites for myosin.
Elevated Ca^{2+}
6 Contraction: power strokes use ATP; myosin heads bind to actin, swivel, and release; thin filaments are pulled toward center of sarcomere.
7 Ca^{2+} release channels in SR close and Ca^{2+} active transport pumps use ATP to restore low level of Ca^{2+} in sarcoplasm.
Ca^{2+} active transport pumps
8 Troponin–tropomyosin complex slides back into position where it blocks the myosin binding sites on actin.
9 Muscle relaxes.

SUMMARY


NERVE EXCITATION
NERVE IMPULSE CONDUCTION TO MUSCLE
NEUROMUSCULAR IMPULSE TRANSMISSION
MUSCLE EXCITATION
EXCITATION CONTRACTION COUPLING
MUSCLE CONTRACTION BY ACTIN & MYOSIN INTERACTION
MUSCLE RELAXATION