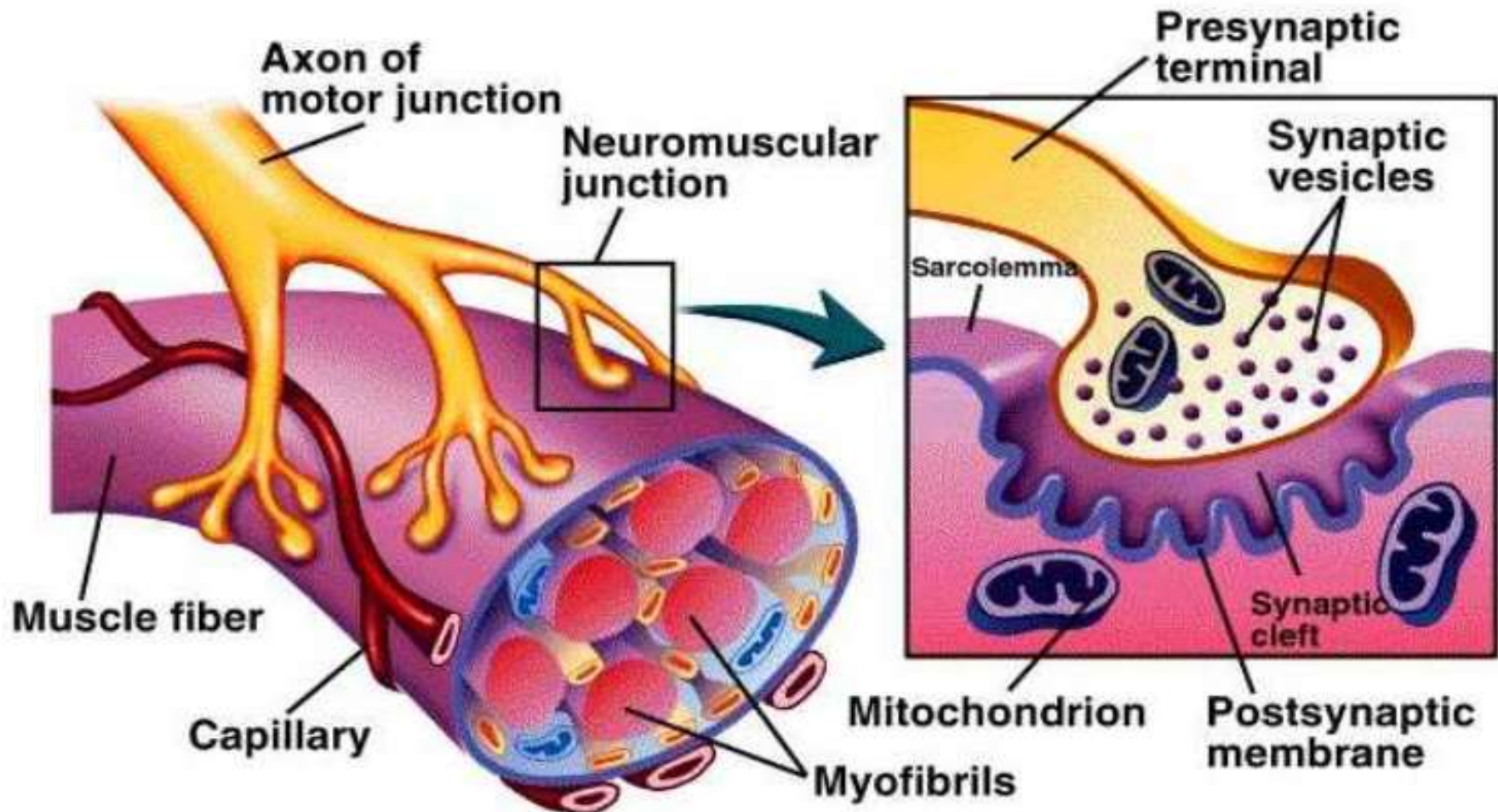
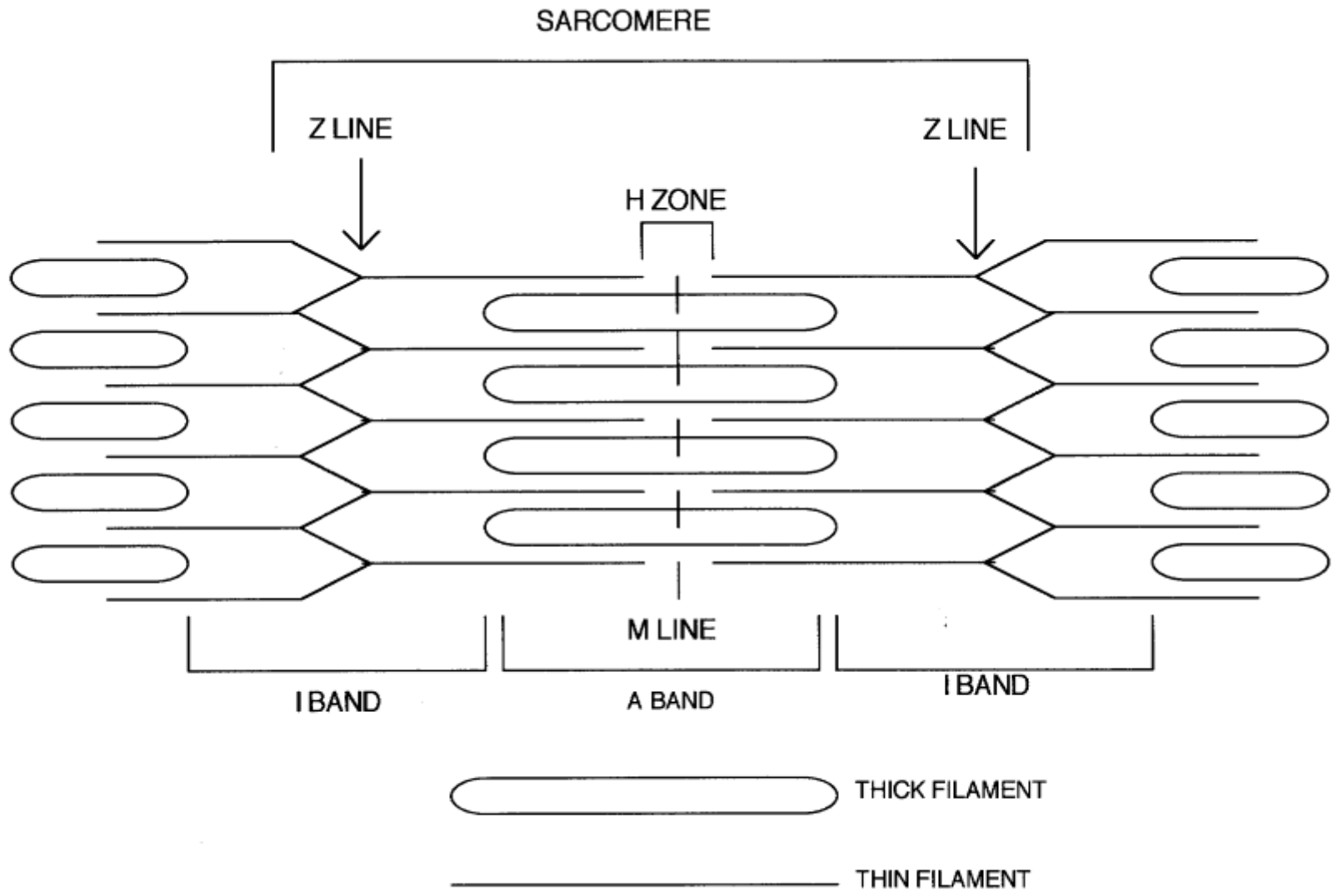


# Notes: Muscle Contraction

EQ: How do muscles contract?

## Neuromuscular Junction

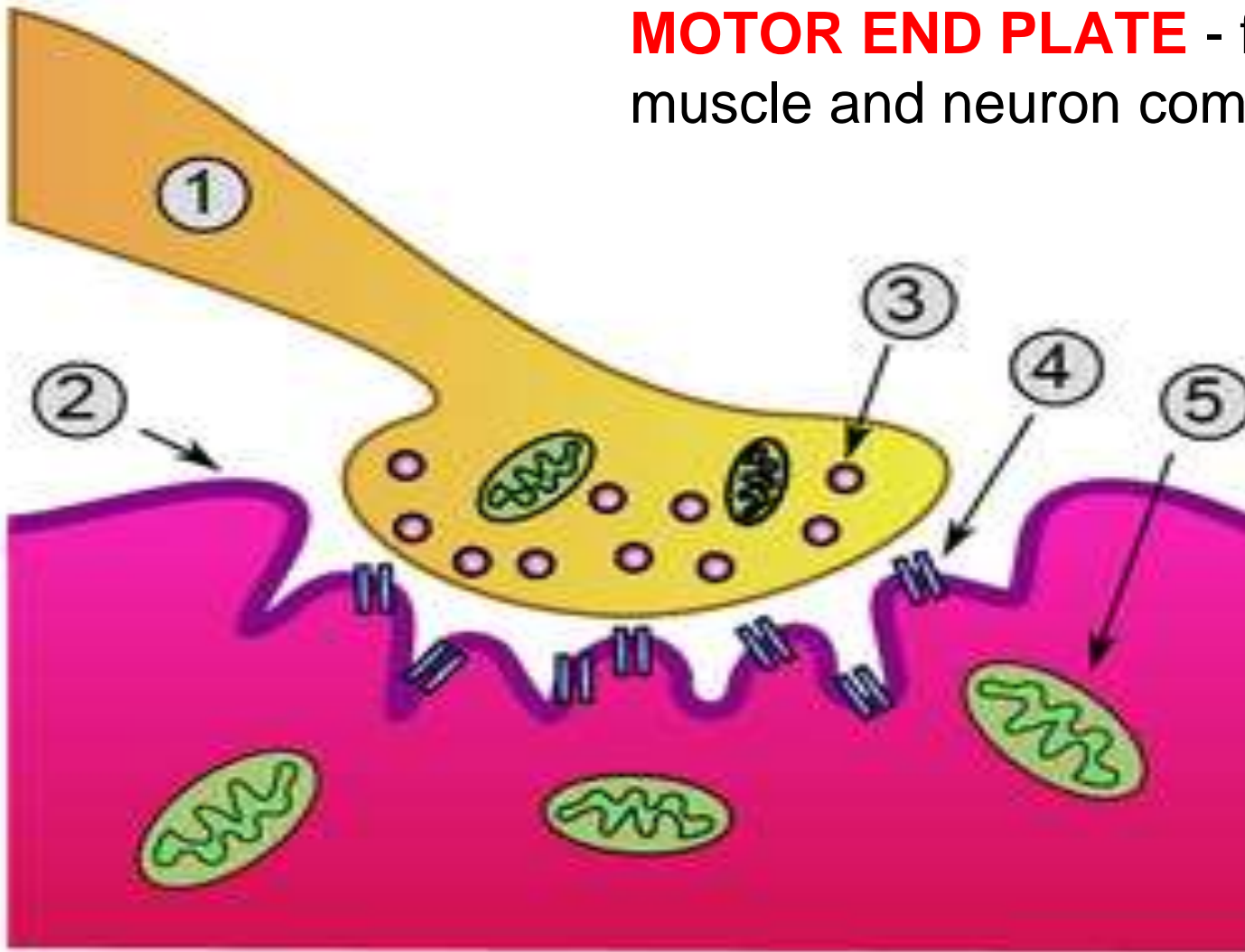




# Motor Unit or Neuromuscular Junction

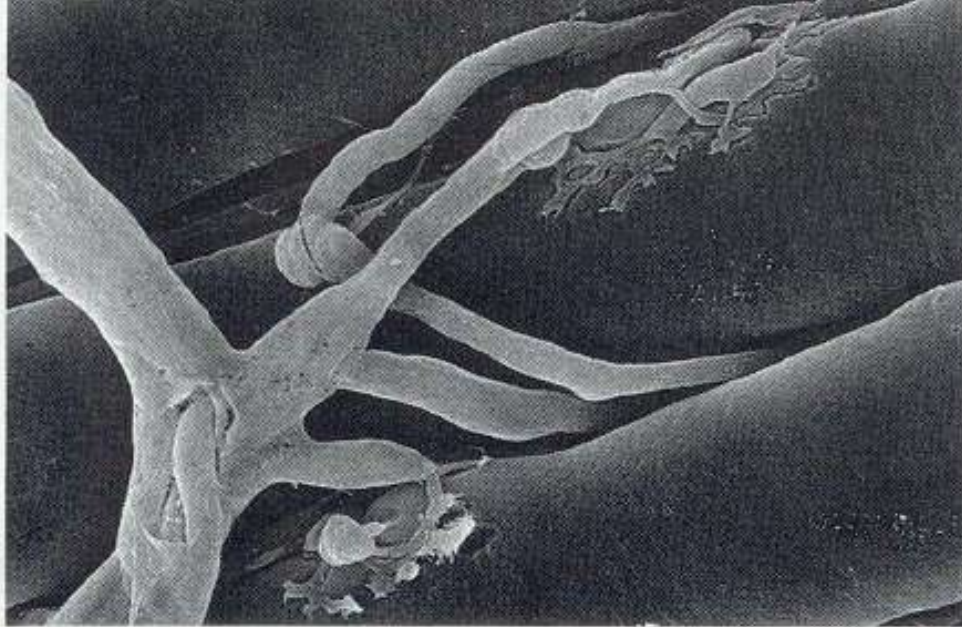
**NEUROMUSCULAR JUNCTION** - where a nerve and muscle fiber come together

**MOTOR END PLATE** - folded area where muscle and neuron communicate



1. Neuron
2. Sarcolemma (or motor end plate)
3. Vesicle
4. Synapse
5. Mitochondria





(a)

### Motor neuron

Myelin sheath of Schwann cell

Axon

(b)

### Muscle fiber

Branched terminus of axon

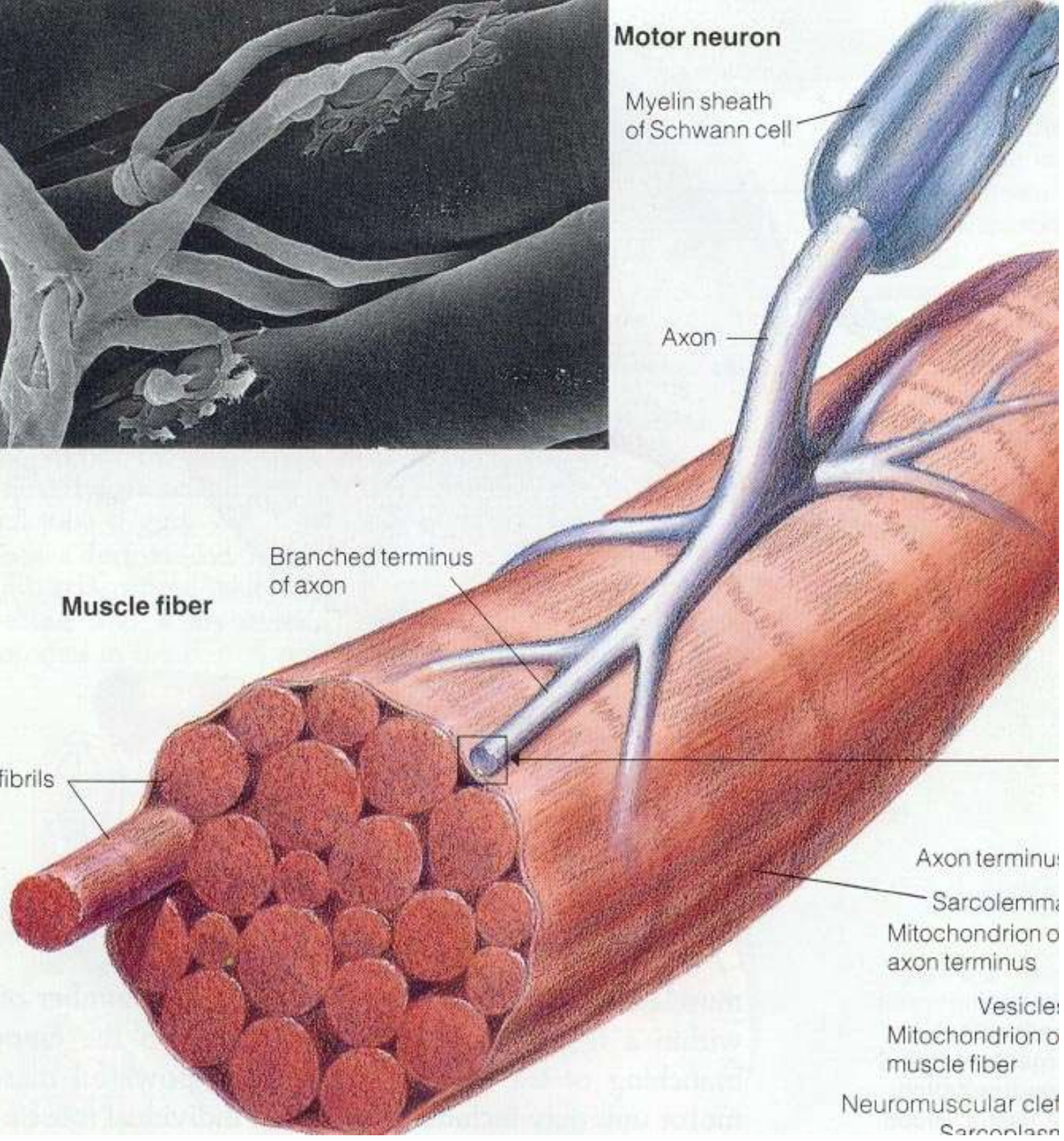
Myofibrils

Axon terminus

Sarcolemma  
Mitochondrion of axon terminus

Vesicles  
Mitochondrion of muscle fiber

Neuromuscular cleft  
Sarcolemma





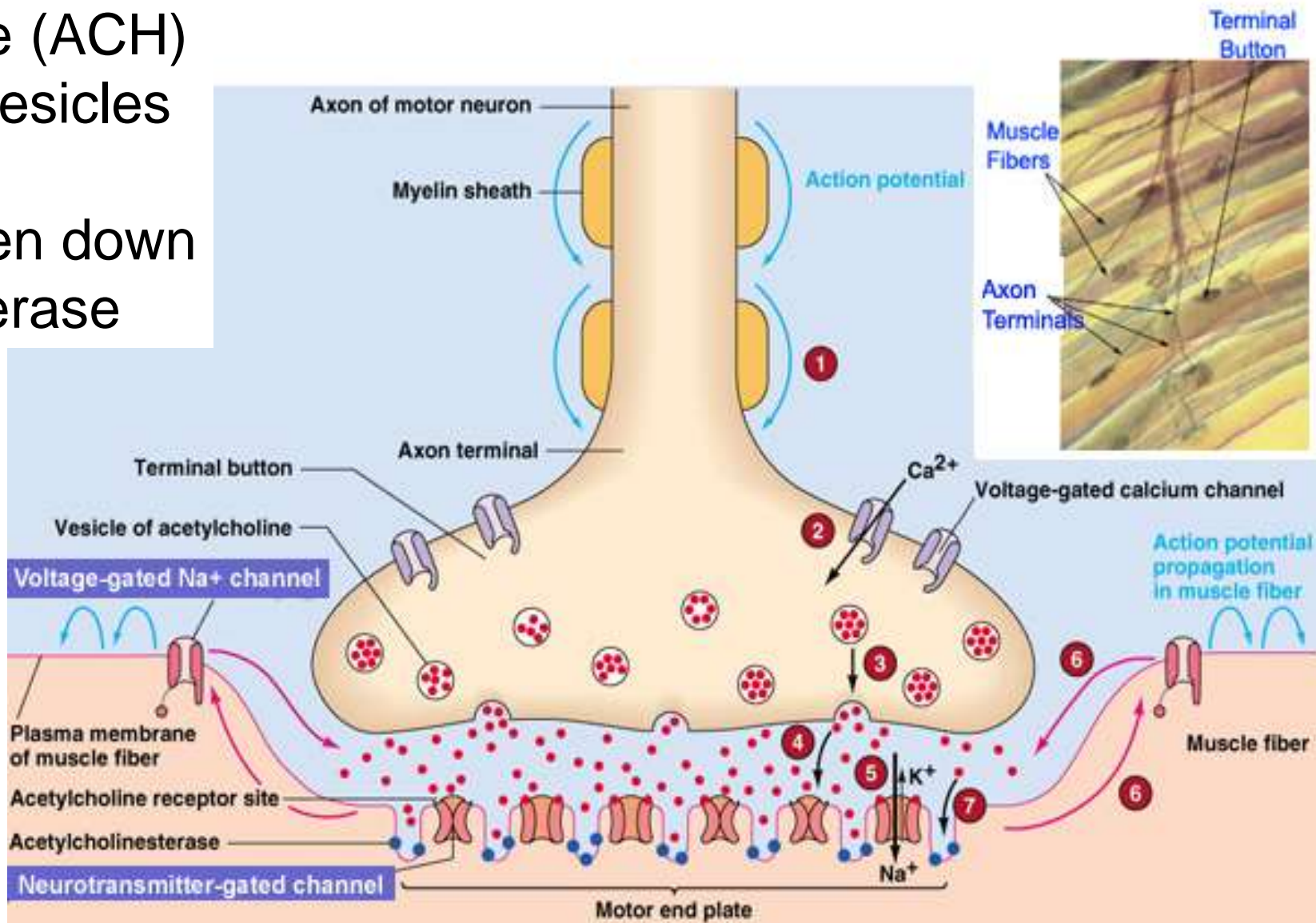
The neurotransmitter that crosses the gap is **ACETYLCHOLINE**.

This is what **activates** the muscle!!!!

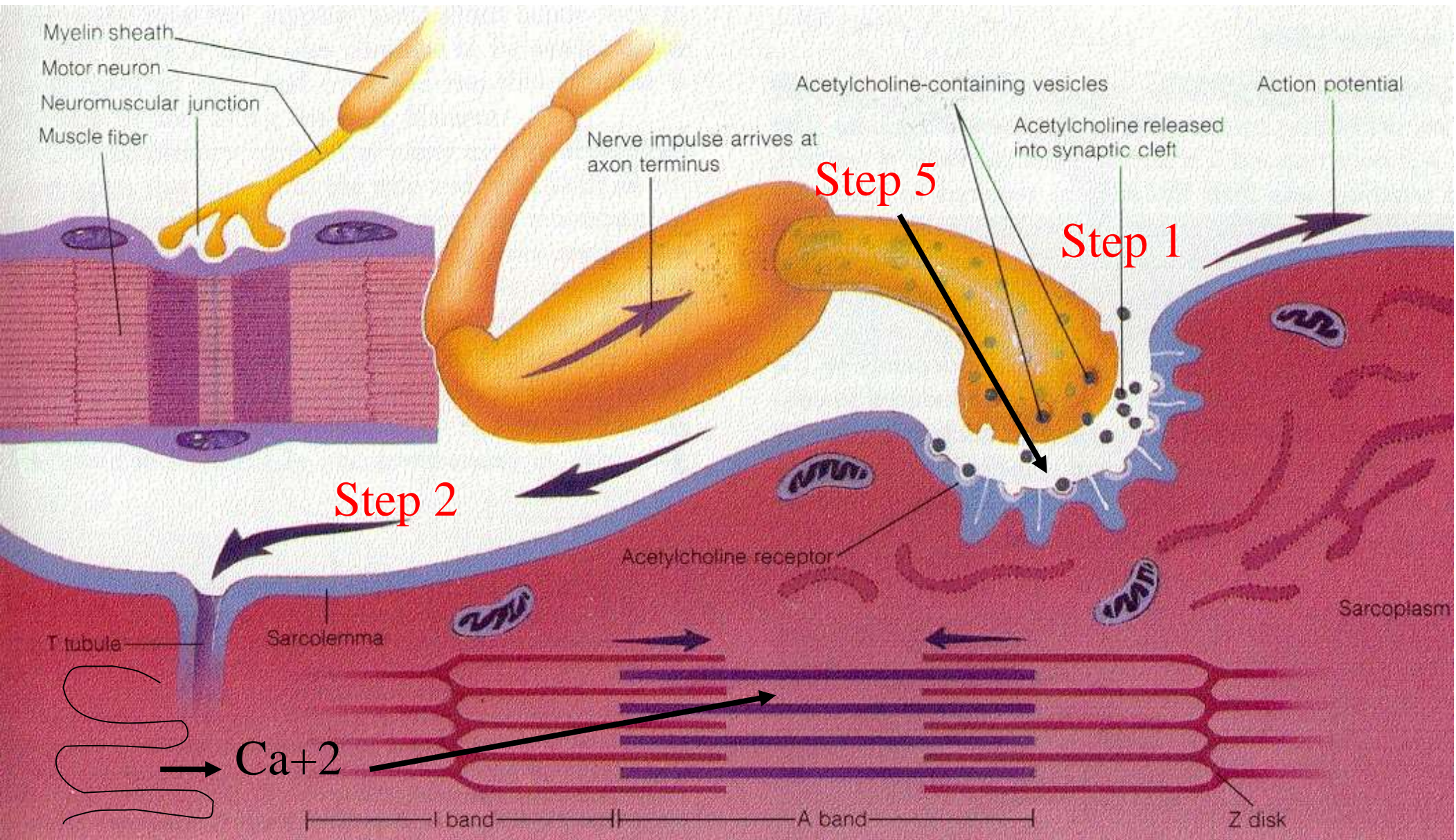
Acetylcholine (ACH)  
is stored in vesicles

ACH is broken down  
by cholinesterase

## The Neuromuscular Junction



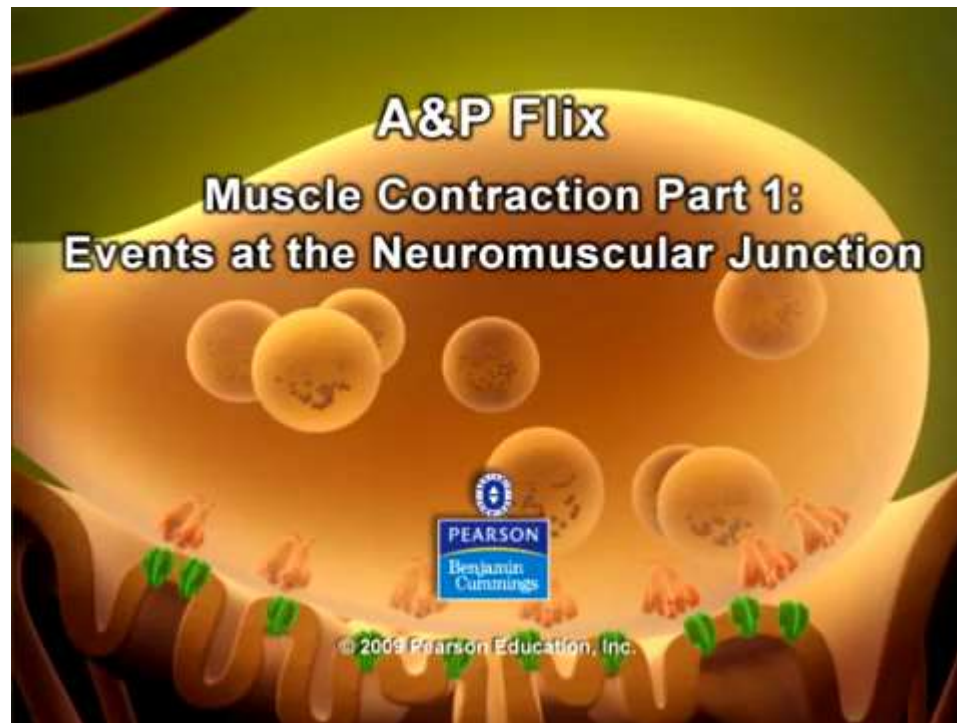




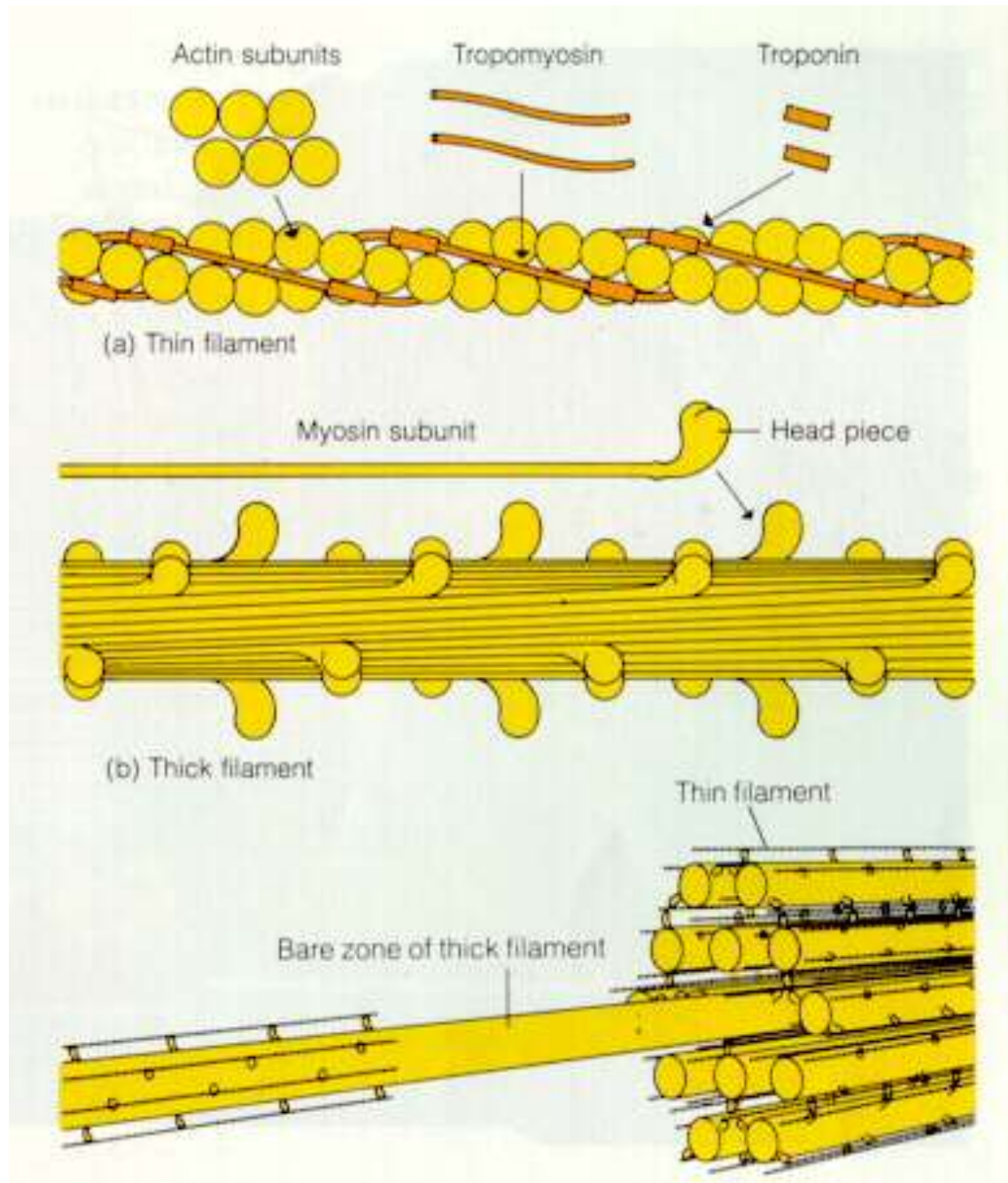
Step 3

Step 4 → CONTRACTION





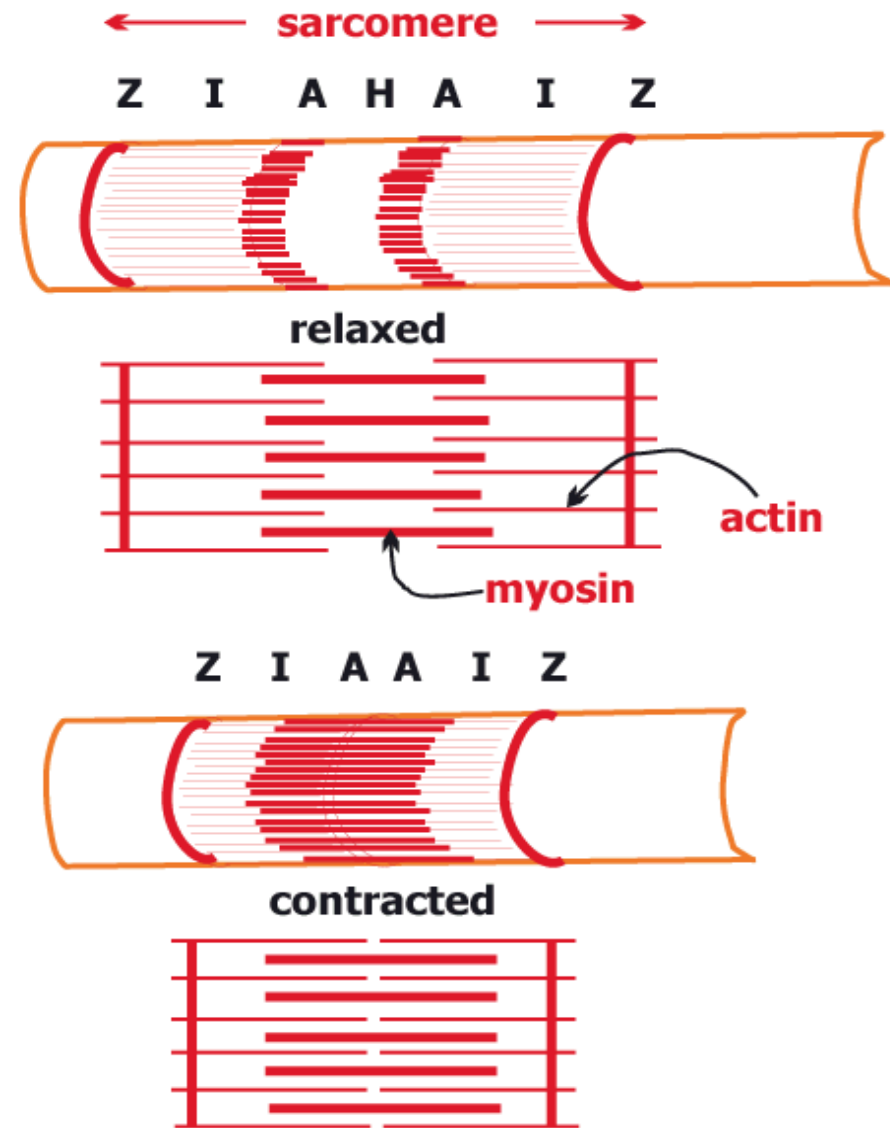
<https://www.youtube.com/watch?v=CLS84OoHJnQ>



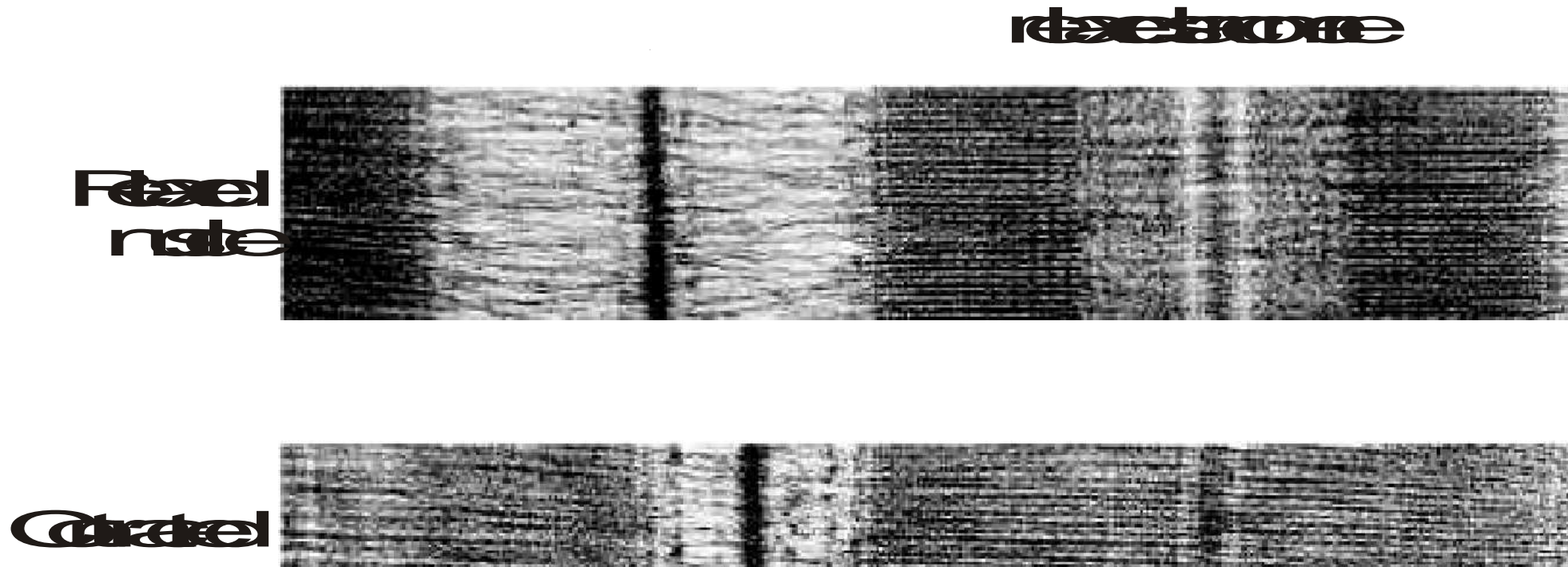


# Sarcomere shortens when muscle contracts

Shortening of the sarcomeres in a myofibril produces the shortening of the myofibril  
And, in turn, of the muscle fiber of which it is a part



## Mechanism of muscle contraction

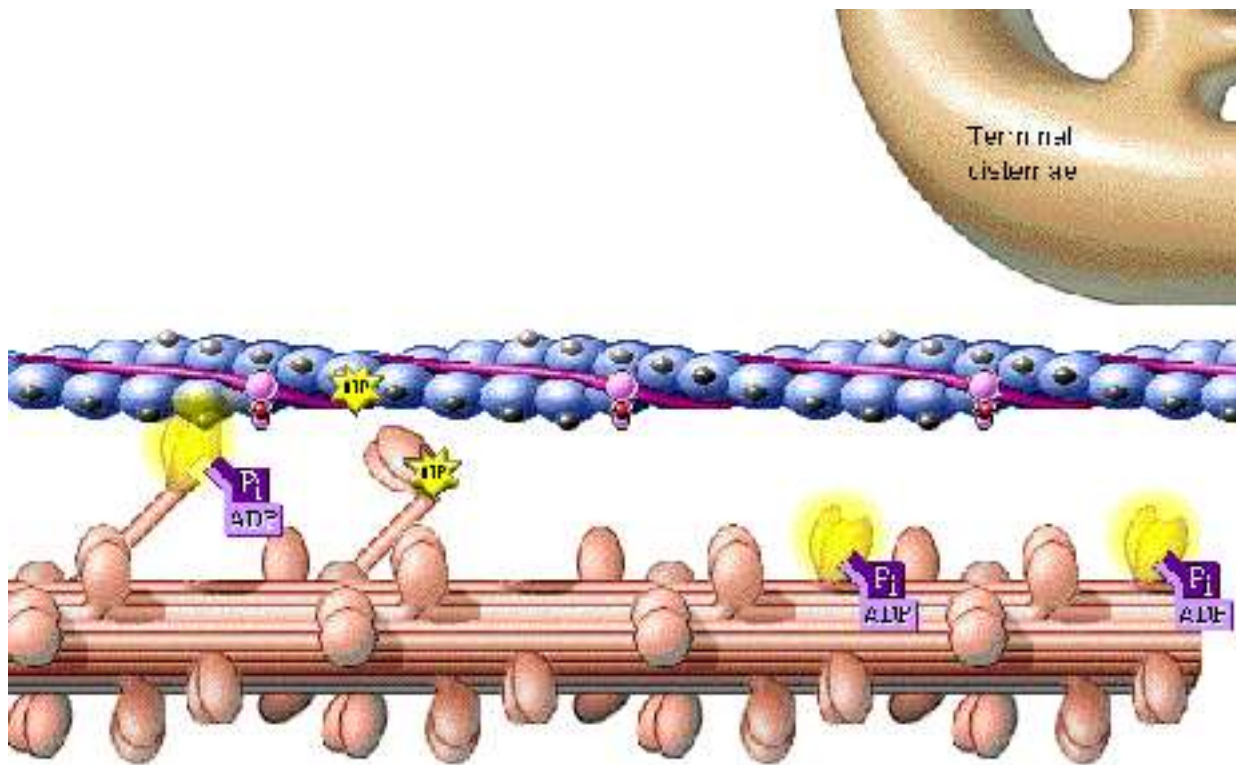


The above micrographs show that the sarcomere gets shorter when the muscle contracts  
The light (I) bands become shorter  
The dark bands (A) bands stay the same length



# SLIDING FILAMENT THEORY (MODEL)

The theory of how muscle CONTRACTS is the Sliding Filament Theory. The contraction of a muscle occurs as the thin filament slide past the thick filaments.



**What is needed:**

ATP

Calcium

Myosin & Actin

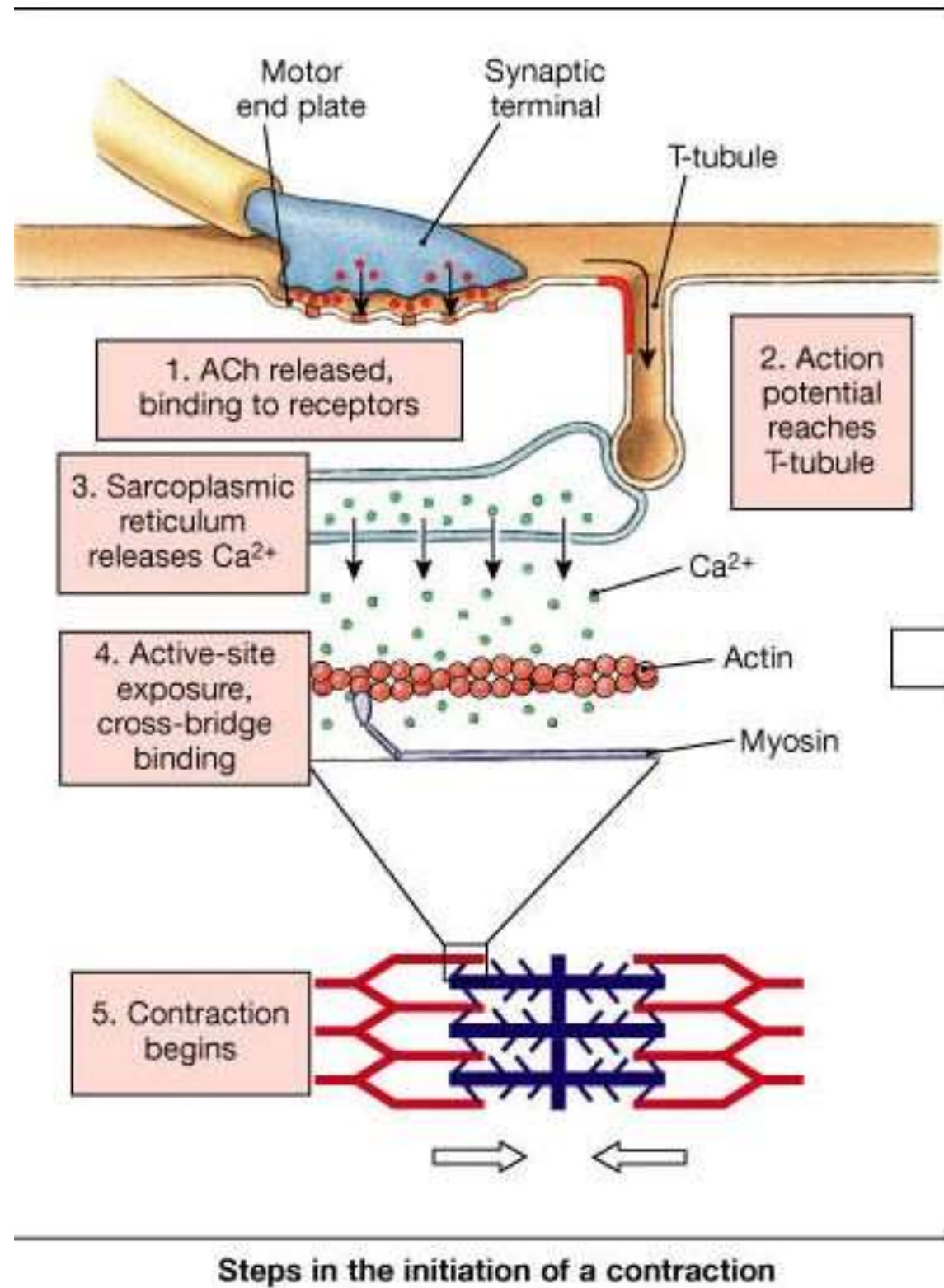
Acetylcholine

Cholinesterase

Step 3:  $\text{Ca}^{2+}$  binds with the troponin molecules and causes tropomyosin molecules on the thin fibers to move exposing the actin molecules.

Step 4: This allows the myosin head of the thick fibers to form a **cross bridge**.

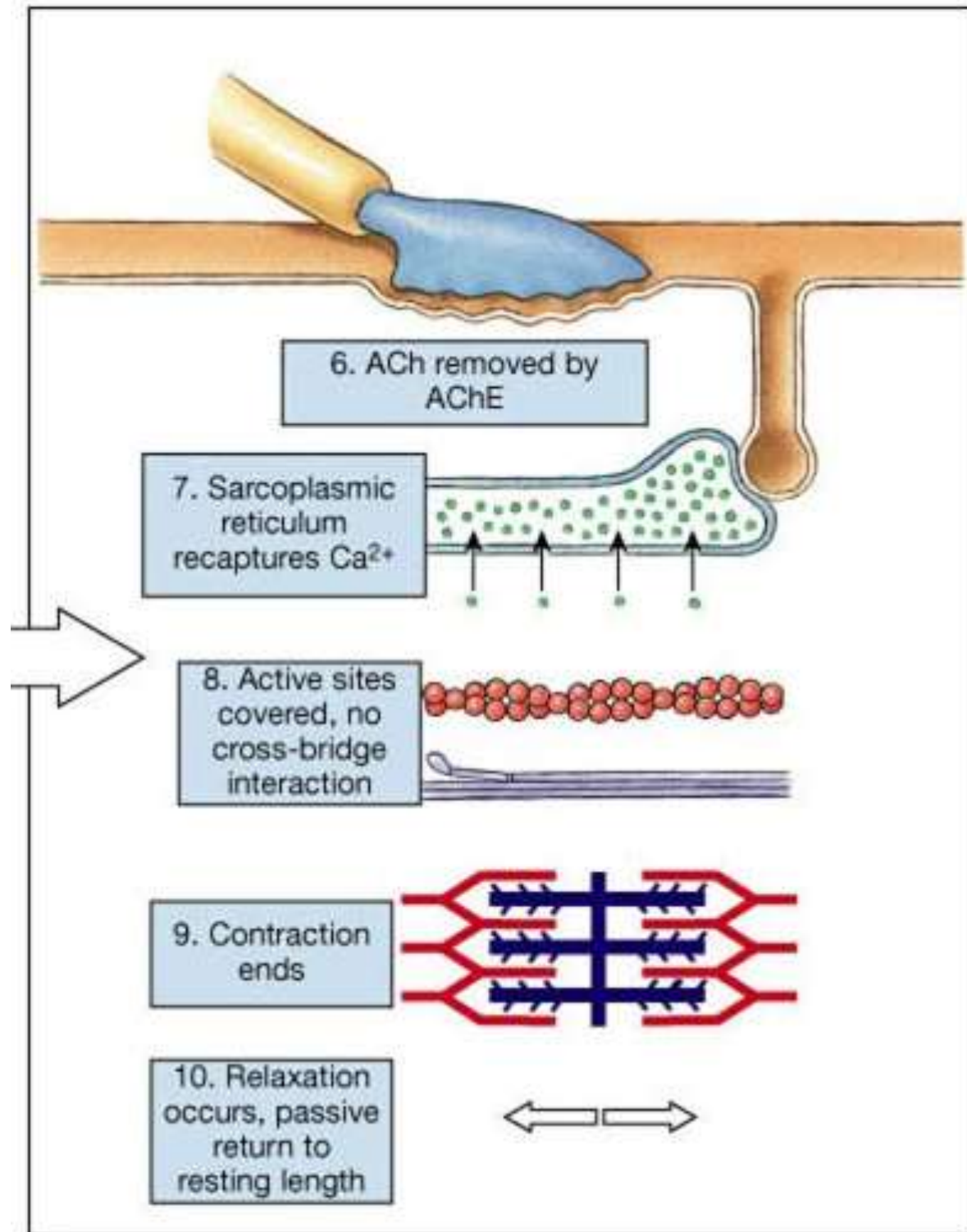
But this requires Adenosine triphosphate or ATP



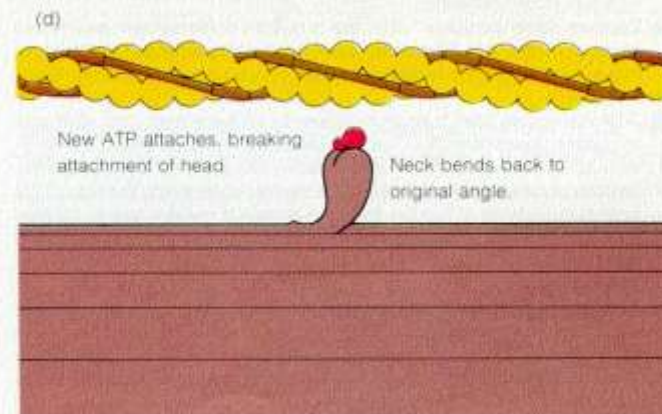
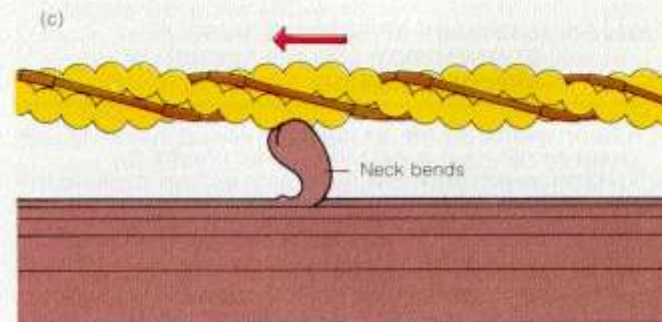
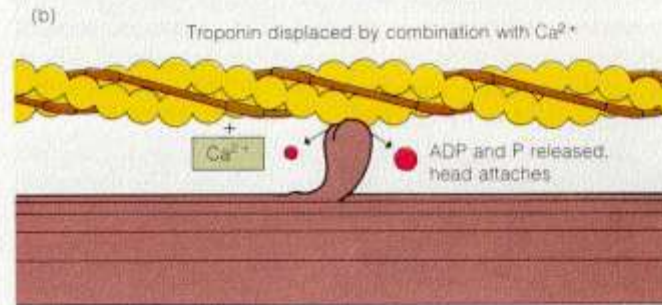
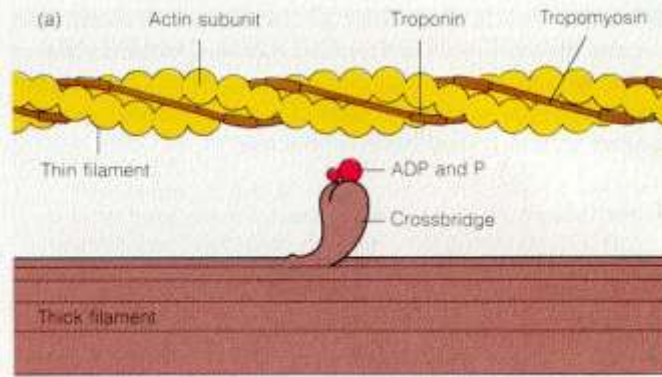


Step 8: A new molecule of ATP shows up and binds with the myosine head causing the head to release from actin – the cross bridge breaks down.

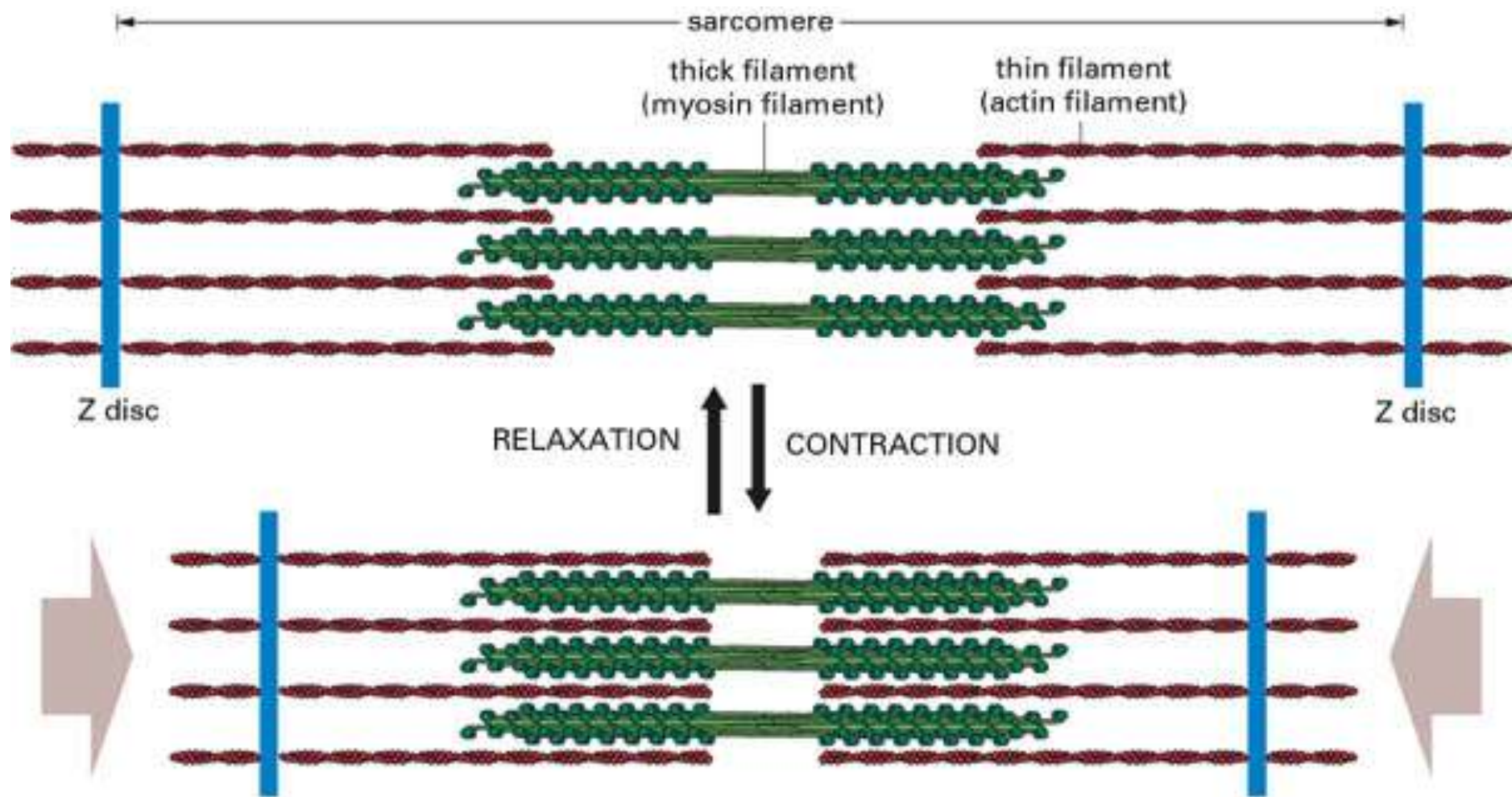
Step 7:  $\text{Ca}^{+2}$  is pumped (active transport) back to the SR and the muscle is “relaxed”.



Steps that end the contraction

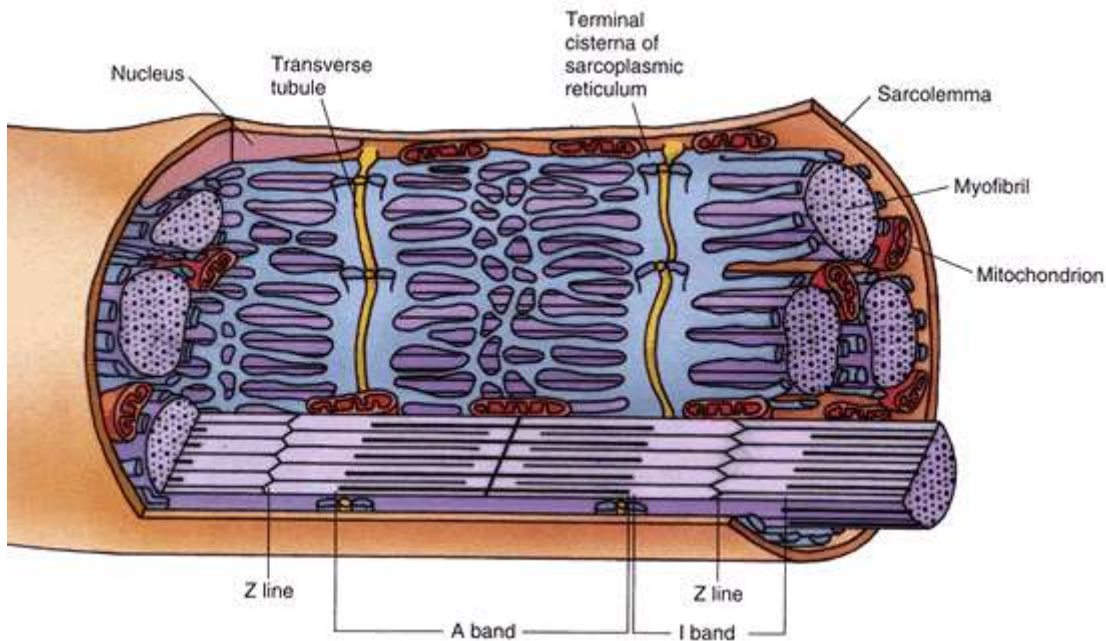






# Energy Source

-ATP is produced by CELLULAR RESPIRATION which occurs in the mitochondria



\* Only 25% of energy produced during cellular respiration is used in metabolic processes - the rest is in the form of HEAT.

- maintains body temperature.

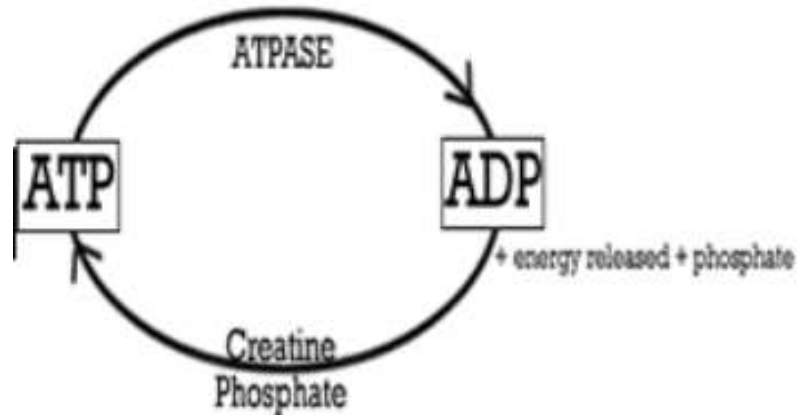
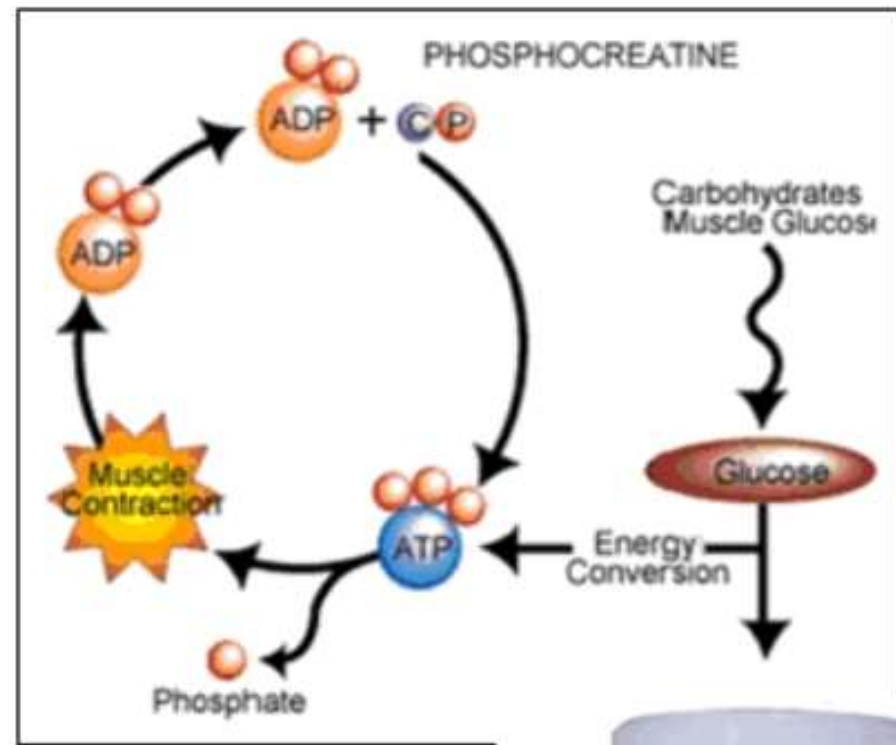


# Why might products like pro-creatine claim to increase energy?

-Creatine phosphate increases regeneration of ATP

ATP = adenosine triphosphate

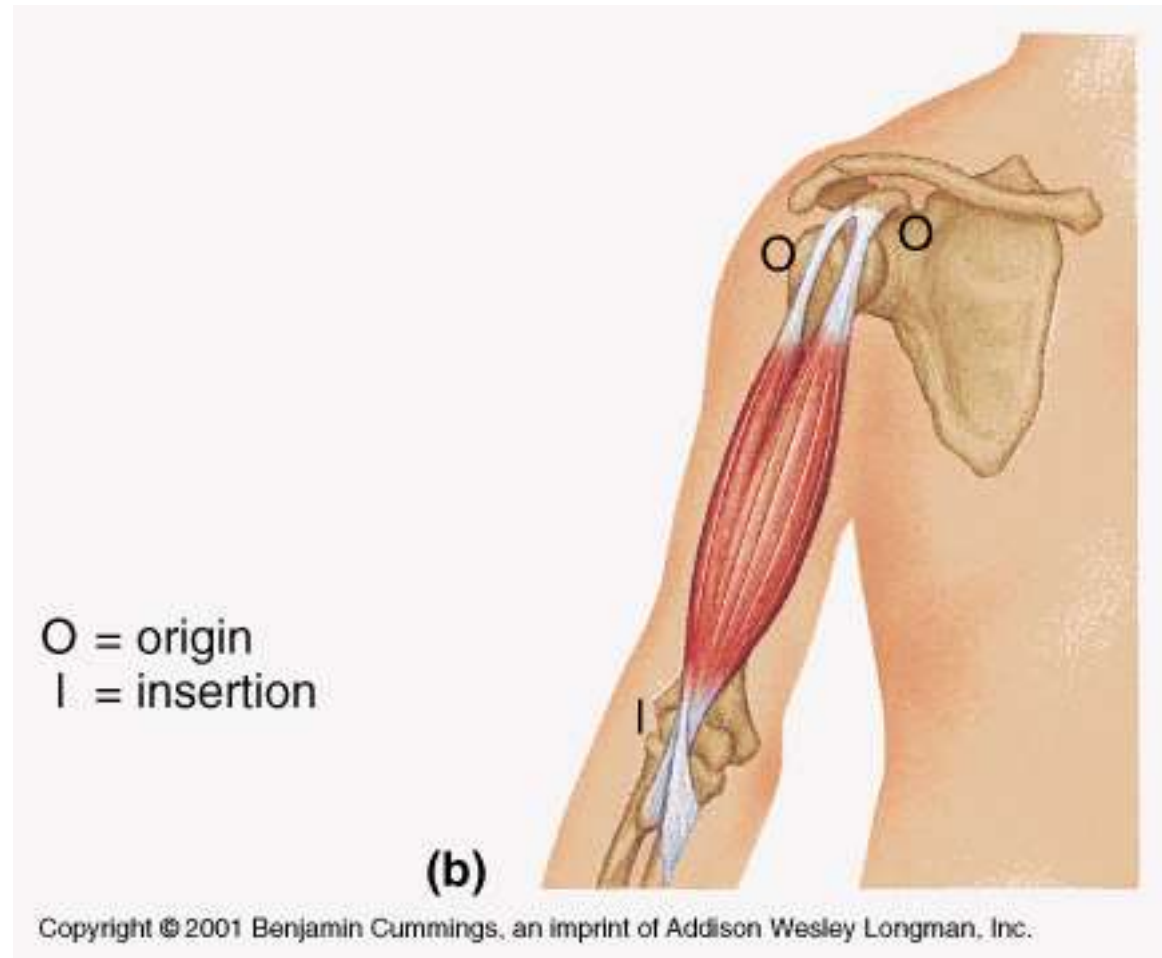
ADP = adenosine diphosphate



# Origin and Insertion

**Origin** = the immovable end of the muscle

**Insertion** = the movable end of the muscle



The biceps brachii has two origins (or two heads).



# Slow- & Fast-Twitch Fibers

- Two types of fibers based on speed of contraction
  1. Slow-twitch or **type I fibers**
  2. Fast-twitch or **type II fibers**
- The eye lids use type II, a muscle in your calf (soleus) would use mostly type I

# Characteristics of Type I & II

- Type I have a lot of blood vessels and many mitochondria, with a lot of **myoglobin**.
  - Myoglobin is similar to the blood protein hemoglobin and brings plenty of oxygen to the muscle.
  - These fibers are also called red fibers (dark meat).
- Type II have fewer of the above and can run off glycogen w/o a lot of O<sub>2</sub> present.
  - These fibers are also called white fibers (white meat)

# What is rigor mortis?



A muscle becomes rigid after death because the tissue loses its ability to produce ATP. RM ends after about 48 hours as the muscle proteins that make the cross bridges start to rot. RM is important in forensic medicine for determining the time of death.



# What is tetanus?

Tetanus causes cholinesterase to not break down the acetylcholine in the synapse.

This results in a person's muscles contracting and NOT relaxing!!



A tetanus shot must be administered shortly after exposure to the bacteria.

Once you develop tetanus, there is NO cure.

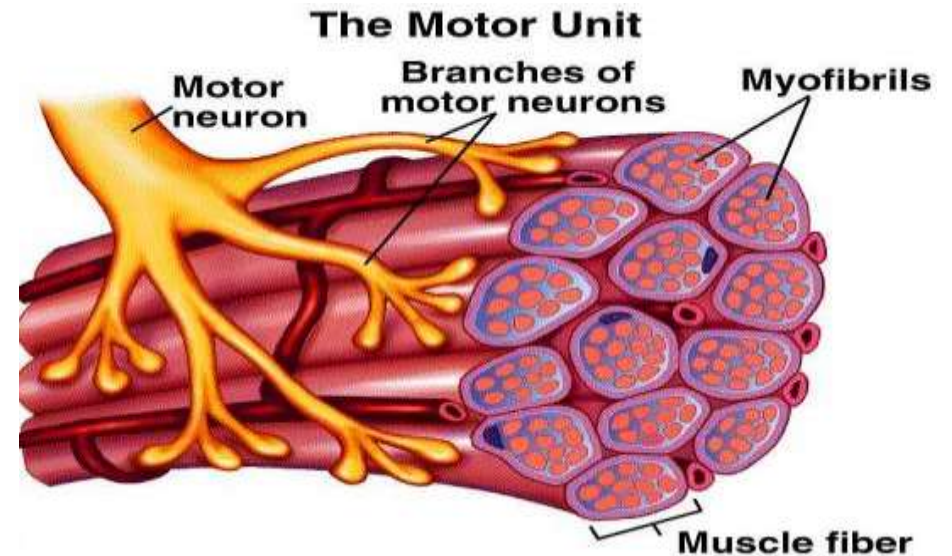
# 1. All-or-None Response

Fibers do NOT contract partially, they either do or they don't

What do you do at the muscle cell level when you want to pick up a pencil instead of a 50 lb dumbbell?

## 2. Motor Unit

The muscle fiber + the motor neuron



## 3. Recruitment

more and more fibers contract as the **INTENSITY** of the stimulus increases

## 4. Muscle Tone

Sustained contraction of individual fibers, even when muscle is at rest





5. **Hypertrophy** - muscles enlarge (working out or certain disorders)

6. **Atrophy** - muscles become small and weak due to lack of use



7. **Muscle Fatigue** - muscle loses ability to contract after prolonged exercise OR strain
8. **Muscle Cramp** - a sustained involuntary contraction
9. **Oxygen Debt** - oxygen is used to create ATP, -- not have enough oxygen **causes Lactic Acid** to accumulate in the muscles → Soreness