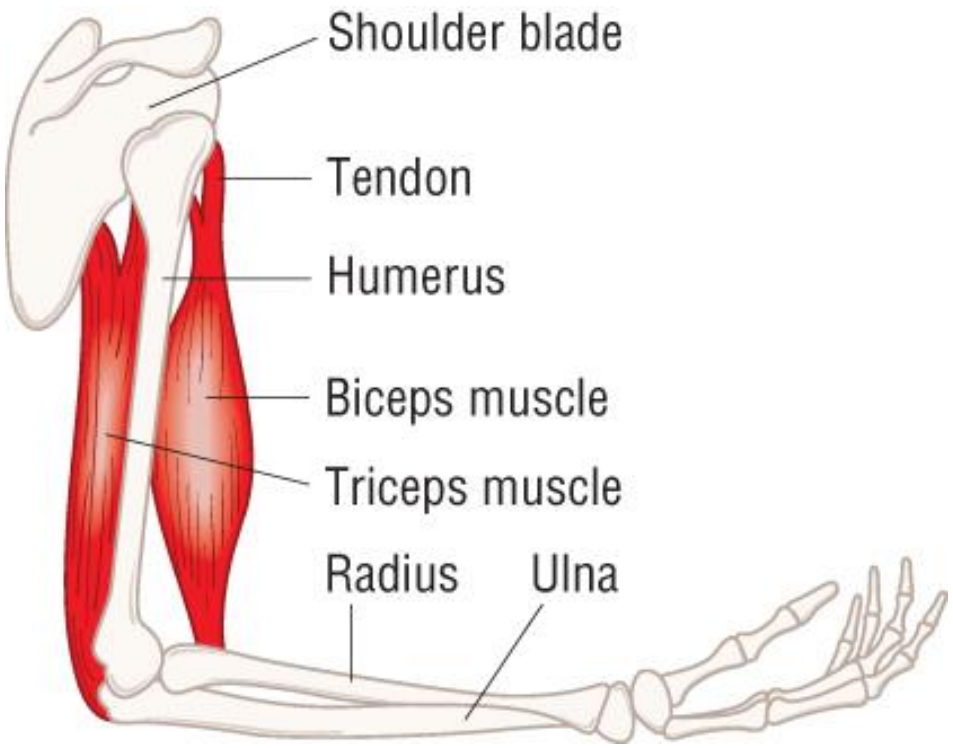
Muscles & Movement

- Describe how coordinated movement requires the action of skeletal muscles about joints.
- Compare and contrast the action of synapses and neuromuscular junctions.
- Outline the structural and functional differences between: *voluntary*; *involuntary*; and *cardiac muscle*.
- Explain the sliding-filament model of muscular contraction.
- Outline the role of ATP in muscular contraction and how the supply of ATP is maintained in muscles.

Voluntary Muscles

- Attached to skeleton via tendons.
  - Tough, inelastic collagen.
- Contraction initiated by nervous impulse.

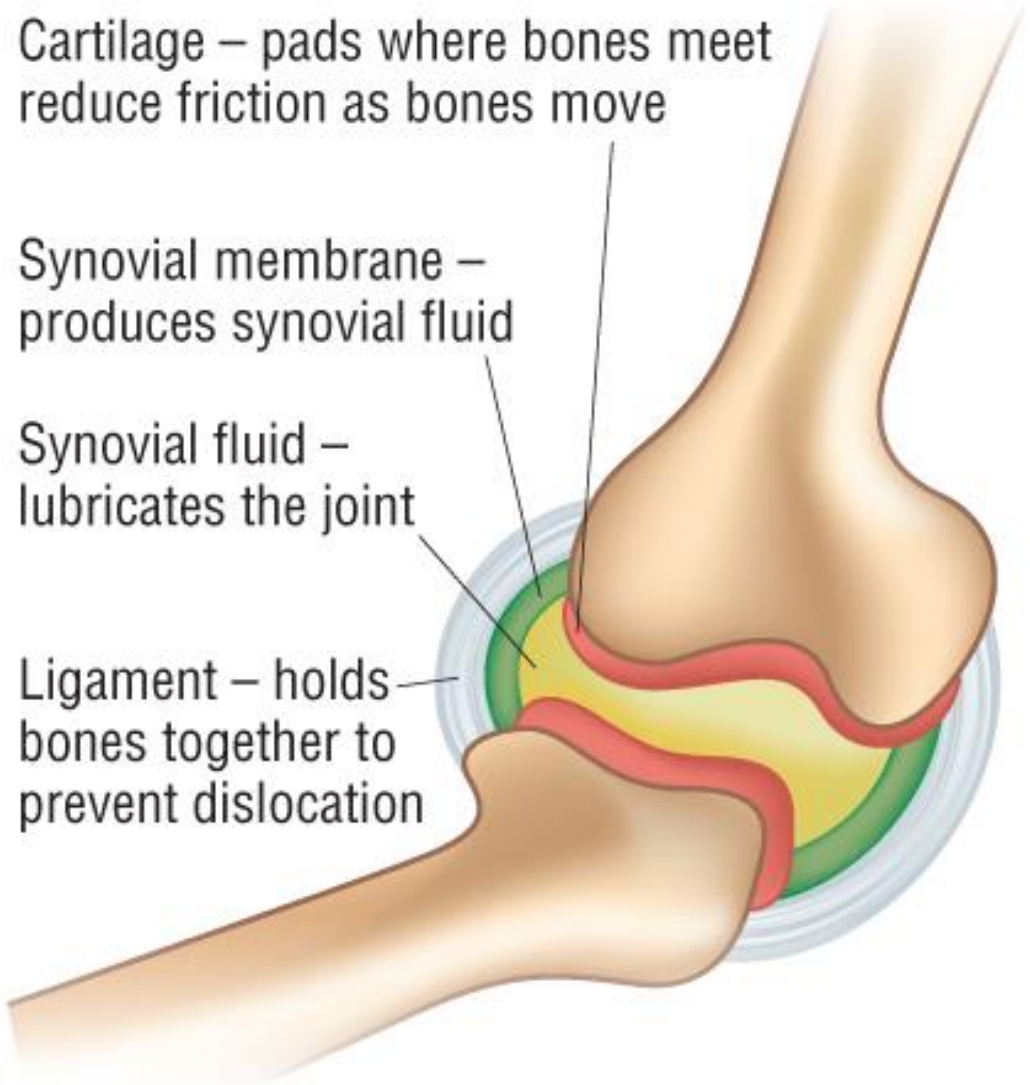


Antagonistic/Synergistic Muscles

- Movement of a body part requires:
  - Coordination of antagonistic pairs of muscles.
    - One contracts, one relaxes.
      - Eg. Bending the arm at the elbow.
  - Coordination of synergistic muscles.
    - Many muscles working together to produce the desired movement.
      - Eg. Moving the eyeball.

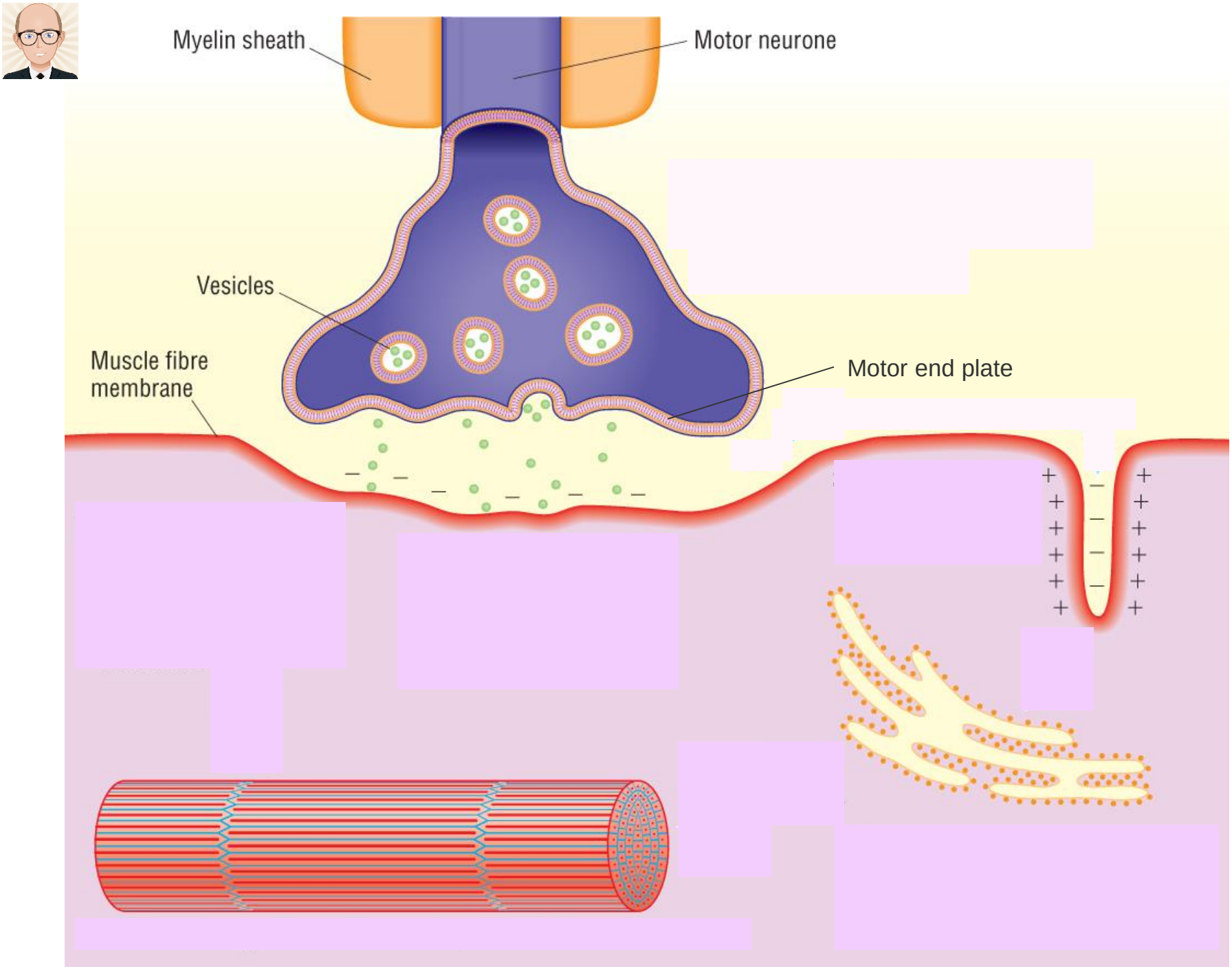
Joints

- Synovial joints:
  - Elbow, knee, etc.
  - Large degree of movement is possible.



The Neuromuscular Junction

- Connects neurones to muscles.
- Action potential stimulates muscle contraction.
- Has similarities to a synapse.


Myelin sheath
Motor neurone
Vesicles
Muscle fibre membrane
Motor end plate

Structure of a Chemical Synapse

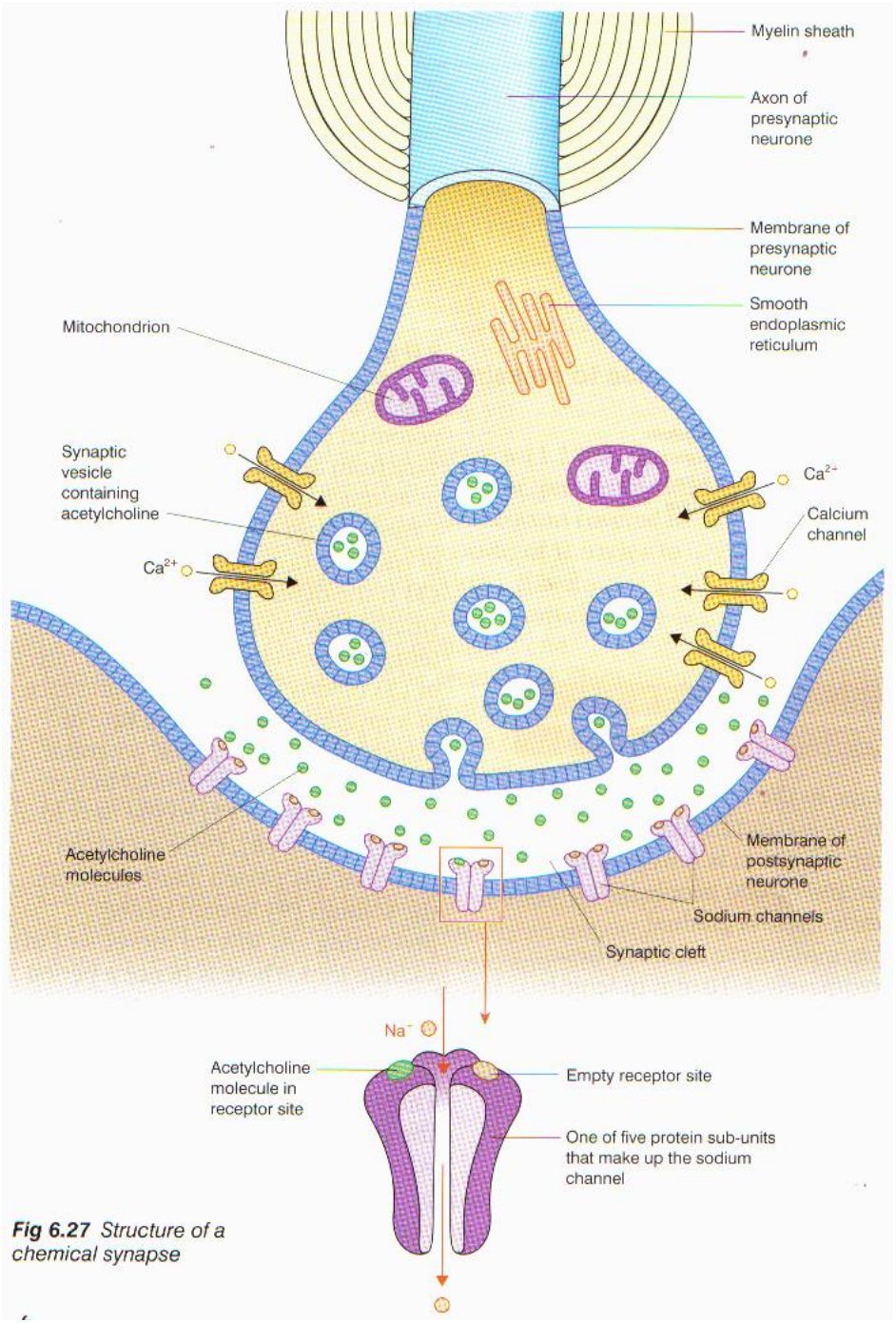


Fig 6.27 *Structure of a chemical synapse*

Compare & Contrast

	Neuromuscular Junction	Synapse
Similarities	Ca^{2+} channels open in response to action potential. Ca^{2+} enters, causing vesicles to fuse with membrane. Neurotransmitter enters synaptic cleft & diffuses across. Neurotransmitter binds to Na^{+} channels causing new AP. Action potential spreads over post synaptic membrane. Neurotransmitter hydrolysed & reabsorbed by presynaptic knob.	
Differences	Action potential spreads into T tubules. Causing Ca^{2+} release from sarcoplasmic reticulum leading to contraction. End of the motor neurone is called Motor End Plate.	Action potential continues along membrane of post synaptic neurone. End of the motor neurone is called Presynaptic Knob.

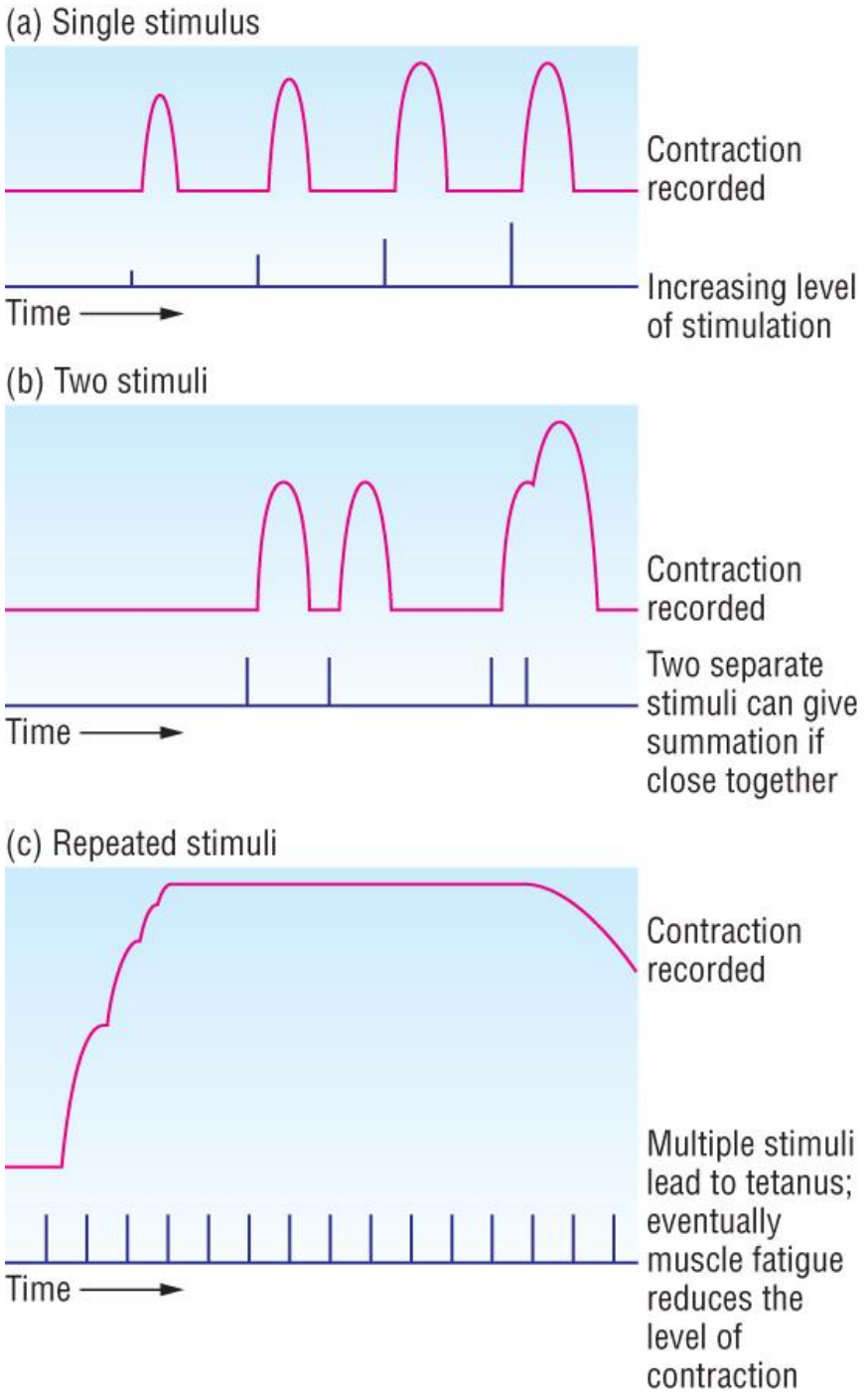
How can we control the strength of our muscle contractions?

These actions use some of the same muscles

- Many motor neurones stimulate a single muscle.
  - Each neurone branches into a few neuromuscular junctions.
  - Each action potential causes contraction of just a small number of muscle cells – the Motor Unit.
  - The more motor units stimulated, the stronger the contraction.
    - Called Gradation of Response.

Investigating frog muscle contraction

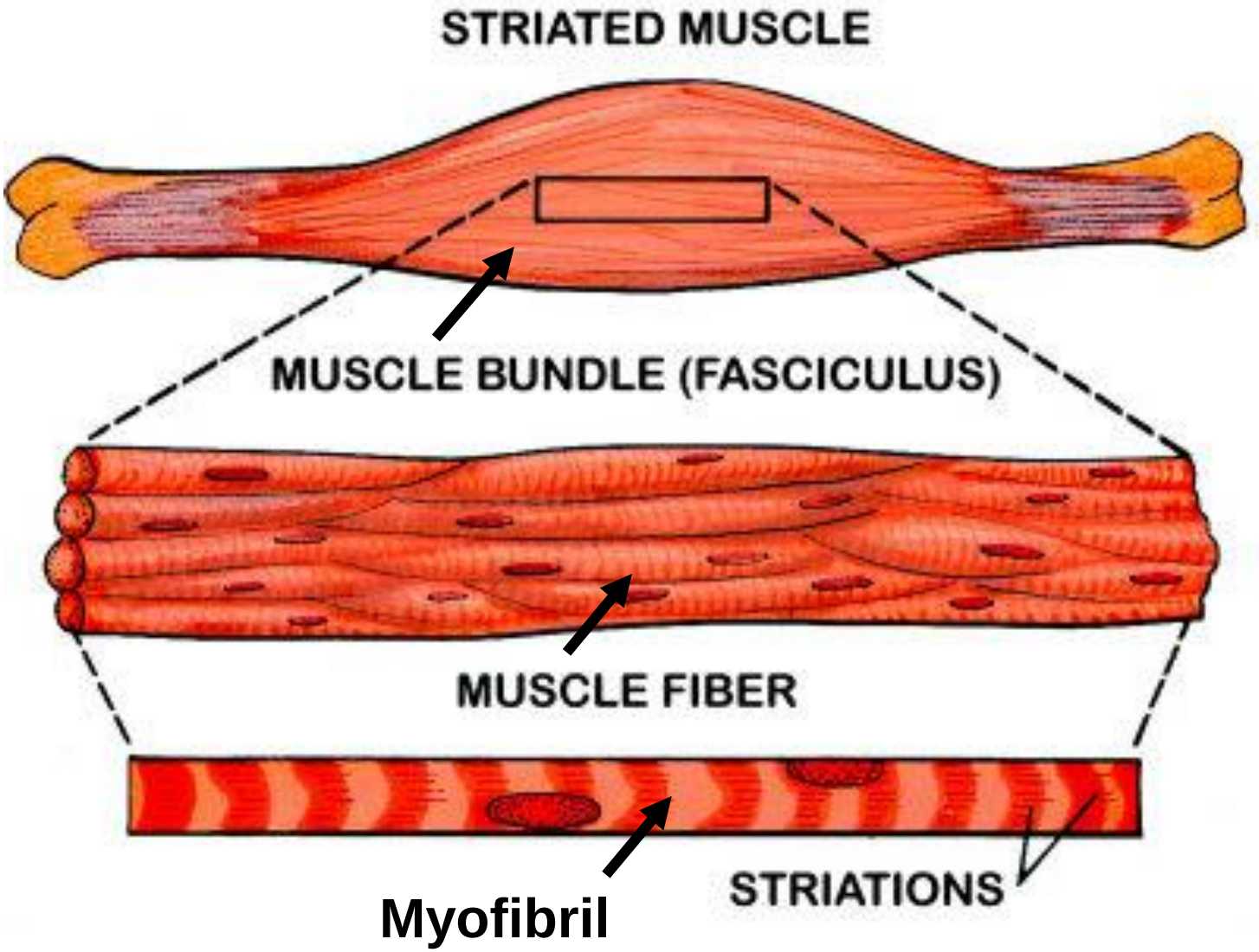
- Single stimulus produces single contraction. Strength of contraction is related to strength of stimulus until maximum contraction is reached.
- If stimulus frequency is increased, contraction strength increases beyond maximum for single stimulus – summation.
- Repeated large stimuli lead to sustained powerful contraction – tetanus.
  - Until muscle fatigue occurs.



Types of Muscle

- Voluntary muscle
  - Also called skeletal muscle or striated muscle
- Cardiac muscle
- Involuntary muscle
  - Also called smooth muscle

Voluntary (Striated) Muscle

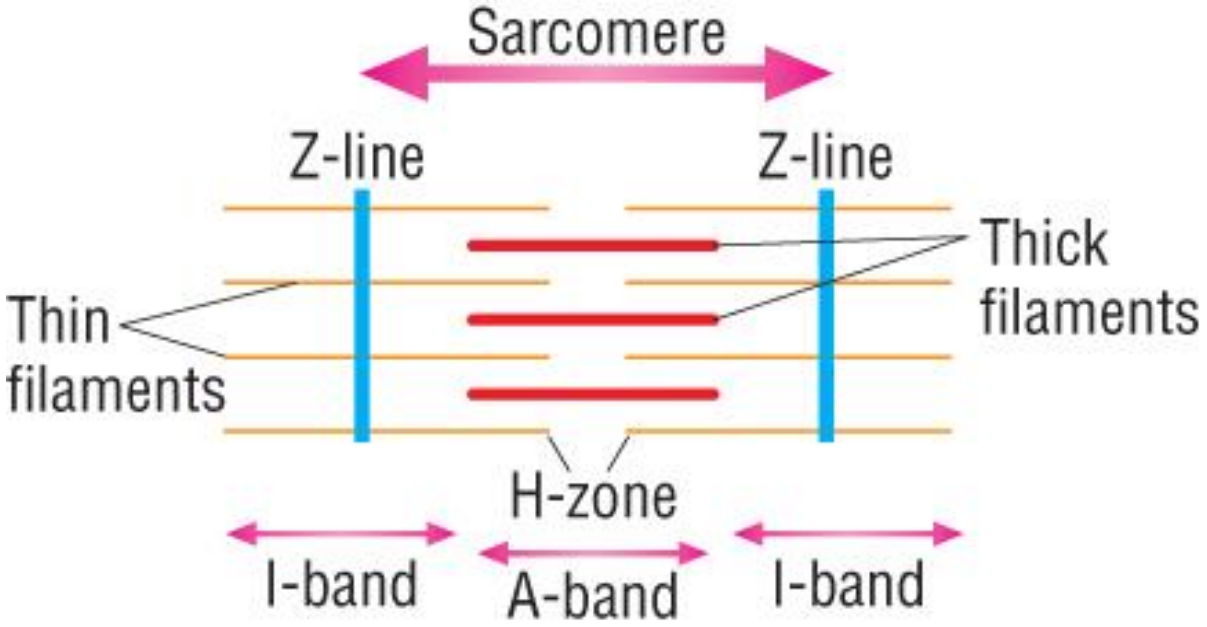


Myofibrils

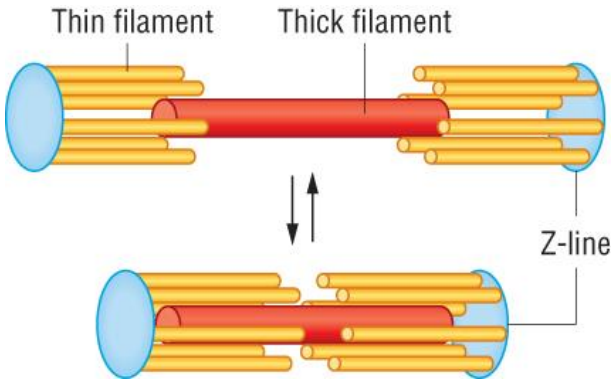
- Organelles within the muscle fibres.
- The contractile elements of the muscle.
- Have a striated (banded) pattern.
  - These bands have names.
  - They are due to the myofilaments that make up myofibrils

Myofilaments

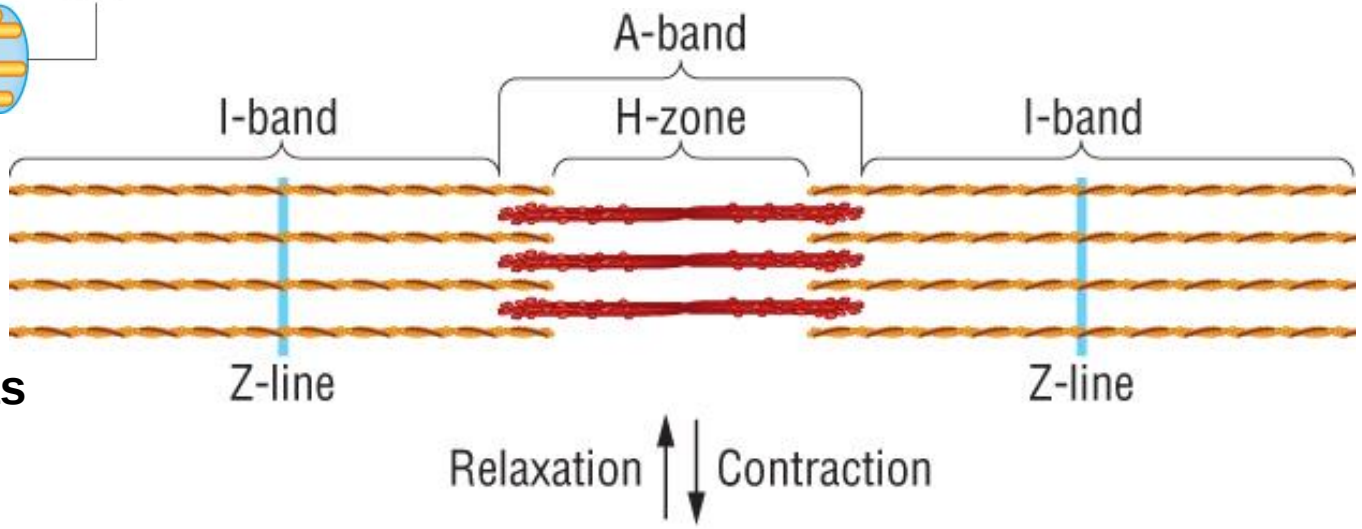
- Arranged as units called sarcomeres.
- Two different types of myofilament:
  - Thick filament
  - Thin filament
- Thick & thin filaments overlap, producing the banding patterns.



Sliding Filament Model



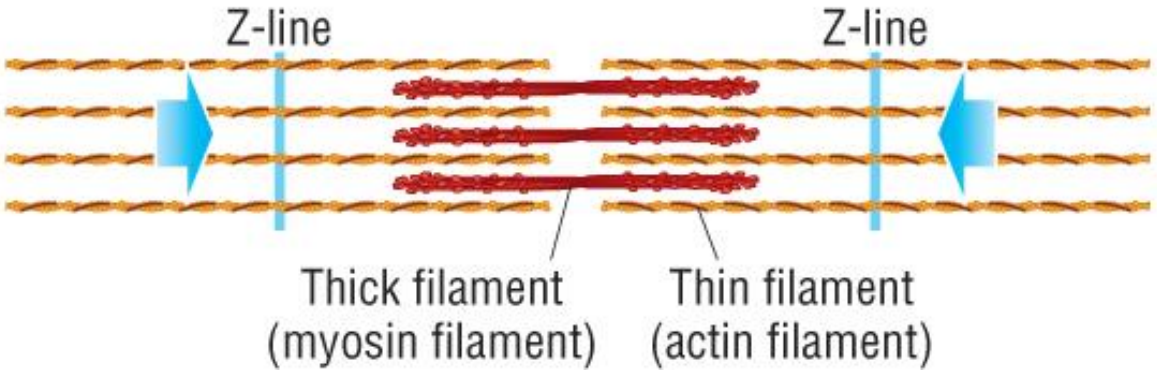
Thick filament (myosin) is surrounded by 6 thin filaments (actin).



Banding changes as filaments overlap.

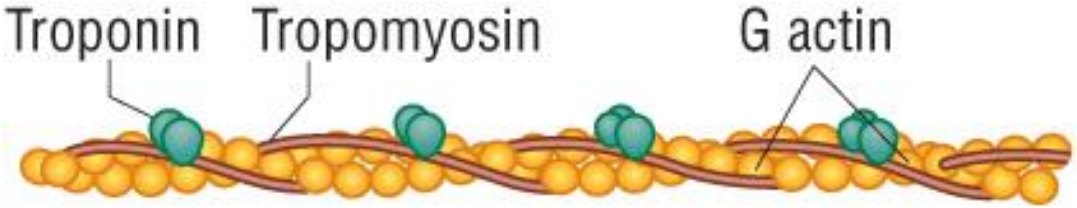
Relaxation ↑ ↓ Contraction

Which bands/zones change in size as the myofibril contracts?



Actin & Myosin

- Actin filaments:
  - Many G-actin monomers joined to form F-actin polymers.
  - Two F-actin polymers coil around each other.
  - Two tropomyosin molecules coil with the F-actin.
  - Troponin has 3 binding sites – one to actin, one to tropomyosin and one to Ca^{2+} ions.
- Myosin filaments:
  - Bundles of myosin molecules, each with a tail end and a head end.
  - Thick filaments made of many myosin molecules.

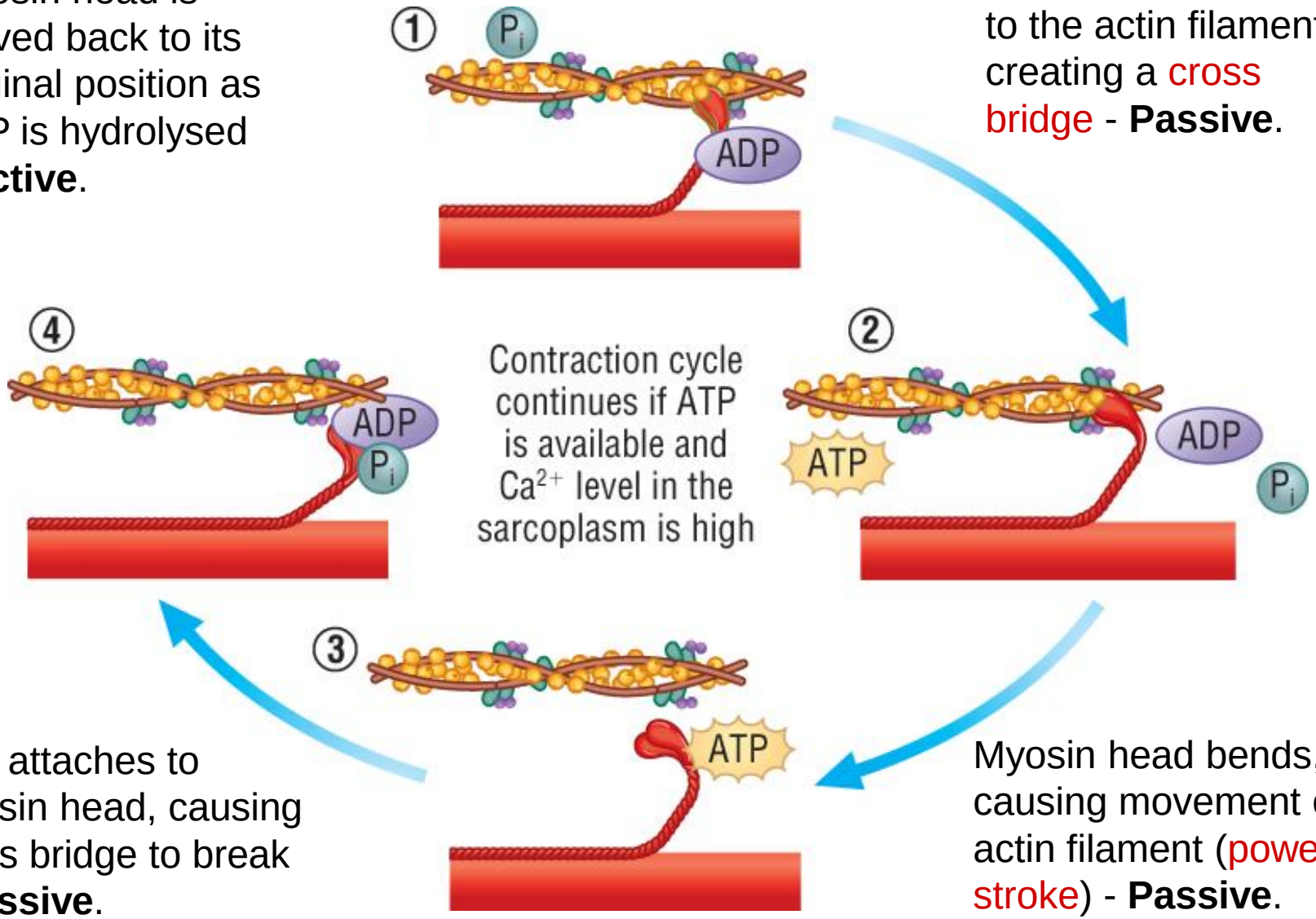


Thin actin filament

Thick myosin filament

Contraction Action

Myosin head is moved back to its original position as ATP is hydrolysed - **Active**.



Myosin head attaches to the actin filament creating a cross bridge - **Passive**.

Myosin head bends, causing movement of the actin filament (power stroke) - **Passive**.

ATP attaches to myosin head, causing cross bridge to break - **Passive**.

Initiation of Contraction

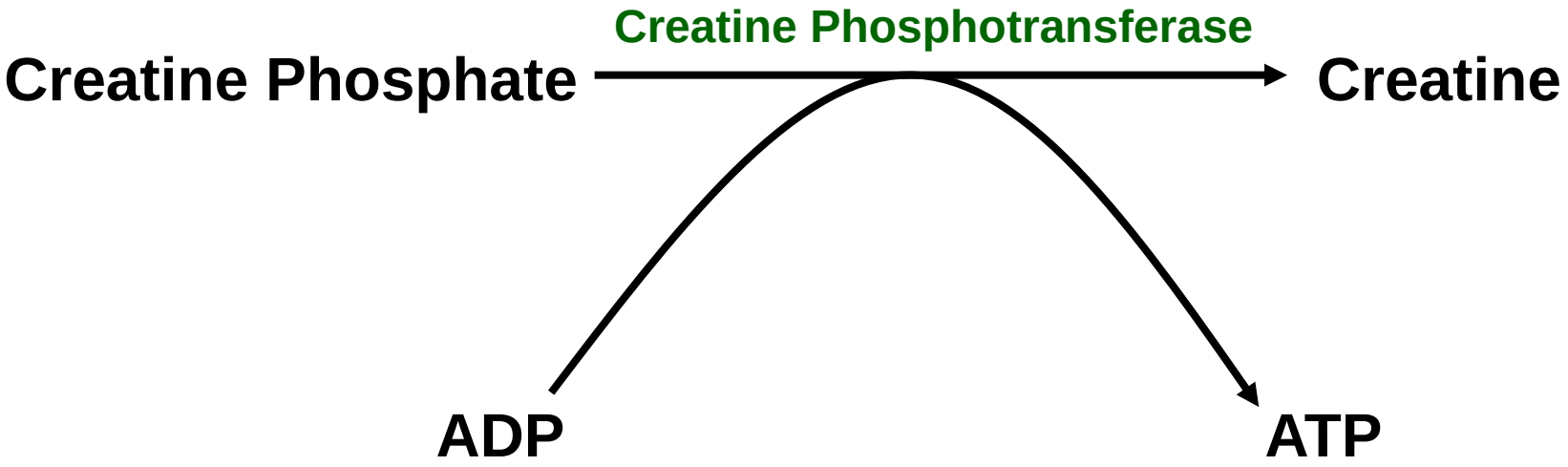
- When relaxed, myosin head binding sites on the actin filaments are covered by the tropomyosin molecule.
- A motor action potential causes Ca^{2+} ions to be released from the sarcoplasmic reticulum.
- Ca^{2+} ions bind to troponin, causing it to change shape.
- This shape change moves tropomyosin out of the way.
- Actin-myosin binding sites are now uncovered and myosin heads can bind & bend.
- After nervous impulse has passed, Ca^{2+} ions are actively transported back into sarcoplasmic reticulum & tropomyosin covers the binding sites again.
- Muscle relaxes.

ATP Supply

- Muscle fibres have enough ATP for 1-2 seconds of contraction only.
  - So it must be recycled quickly.

- 3 methods of recycling ATP:
  - Aerobic respiration
    - Dependent on oxygen supply.
  - Anaerobic respiration
    - Fast acting but produces lactic acid.
  - Creatine phosphate
    - Phosphates “borrowed” from this molecule.

Creatine Phosphate (or Phosphocreatine)



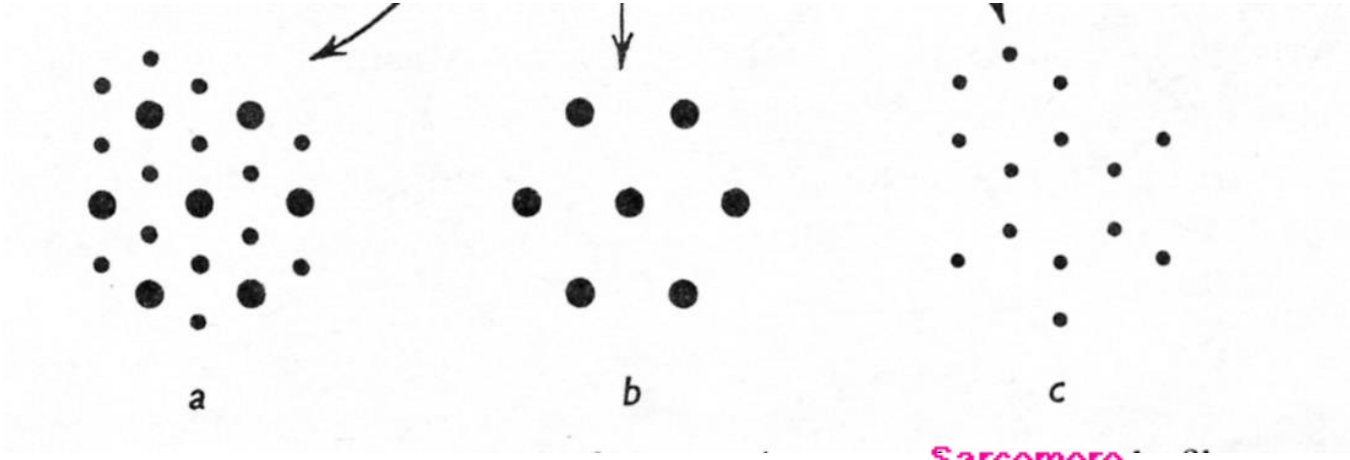
- This will give a muscle another 2 – 4 seconds contraction in times of need.
- Creatine phosphate will be replenished once the ATP supply increases.

Muscle Fatigue

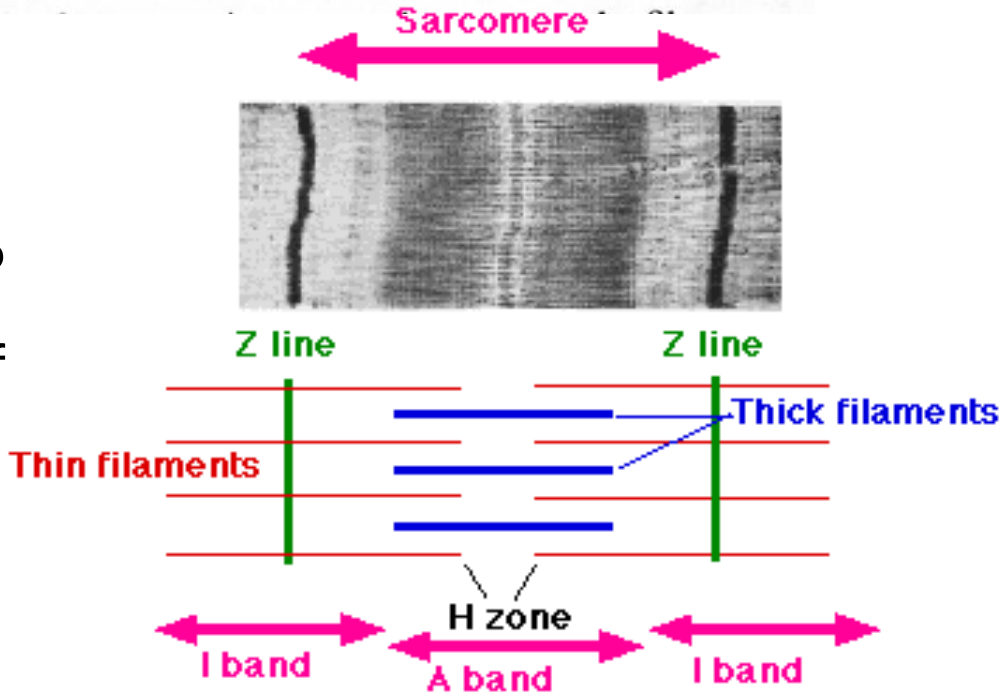
- A state of exhaustion.
  - Produced by strenuous muscular activity.

- Caused by:
  - Lack of ATP
  - Depletion of glycogen stores
  - High levels of lactate

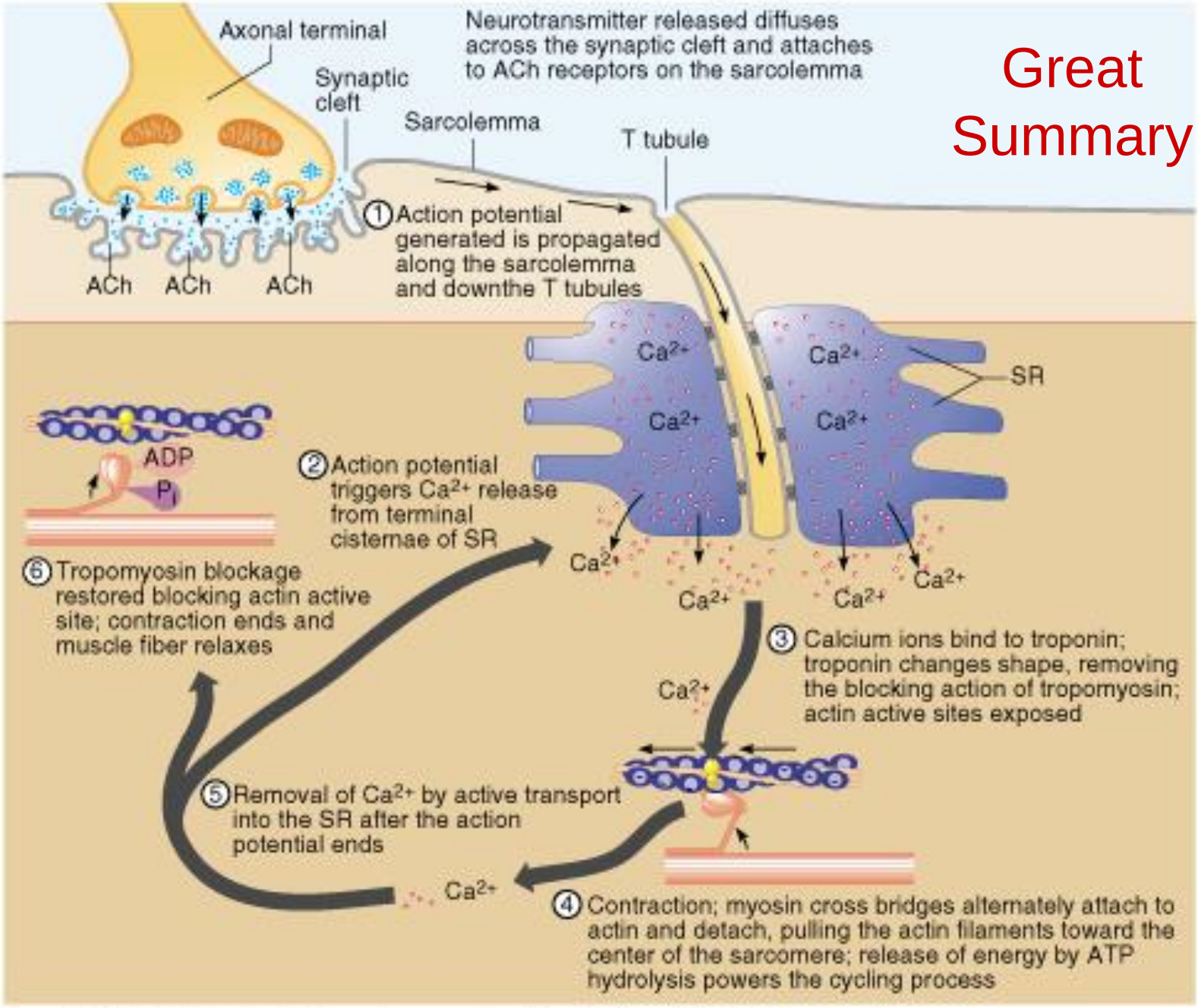
Review



- Where on the sarcomere are these sections taken from?
  - A is the outer edge of A band.
  - B is the H zone.
  - C is the I band.



Great Summary



Challenge

The head groups at one end of a thick filament are orientated in the opposite direction to the head groups at the other. Consequently, when both ends form cross-bridges and pull against the thin filaments, the resulting movement pulls the Z-lines towards each other, thereby shortening the length of the sarcomere.

(a) Explain why there is a maximum possible level of muscle contraction.

(b) (b) Suggest why there is a region of the thick filament that has no head groups.

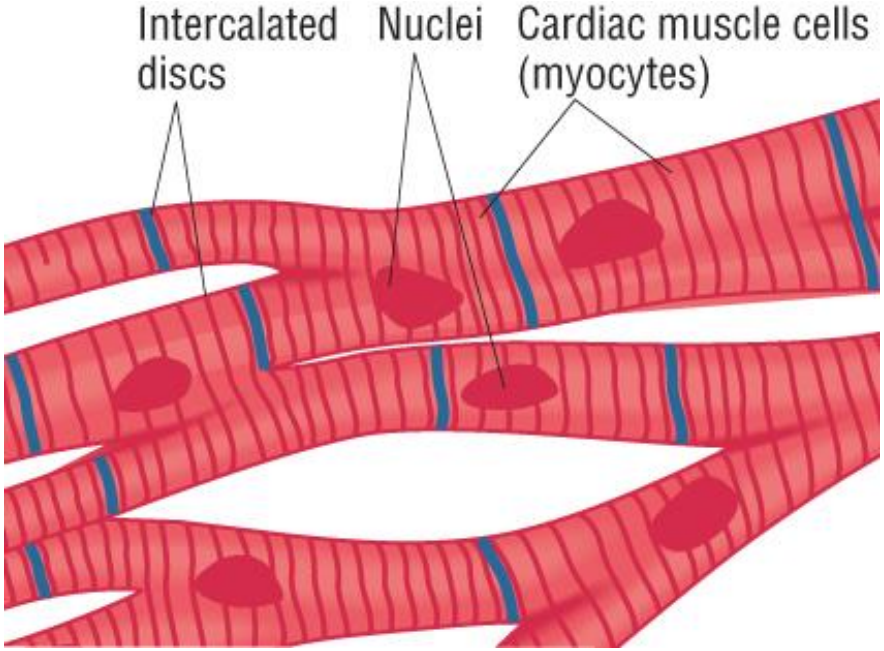
(c) Myosin head groups have ATPase capability – why is this important?

(d) Suggest whether thin filaments are polar and explain your answer.

(e) Suggest why rigor mortis (muscle contraction and stiffening) occurs three to four hours after death.

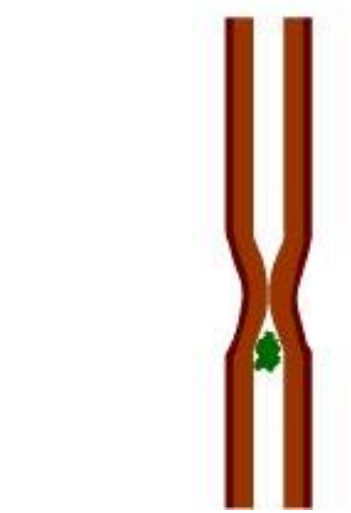
Cardiac Muscle

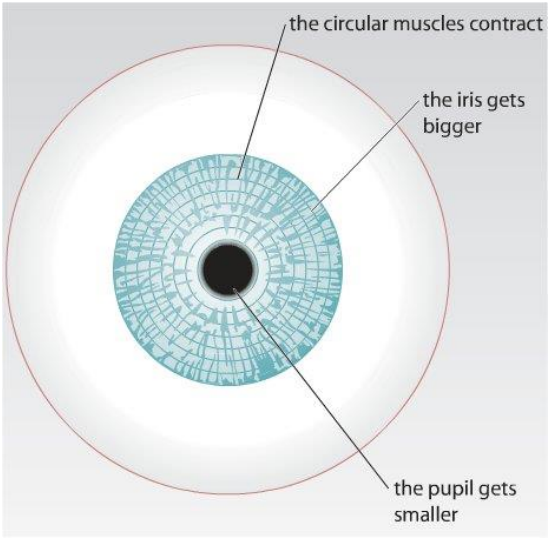
- Powerful contractions, no fatigue.
- Similar structure to voluntary muscle.
- Branched muscle fibres.
  - Action potentials quickly spread throughout.
- Some fibres are myogenic (SA node).

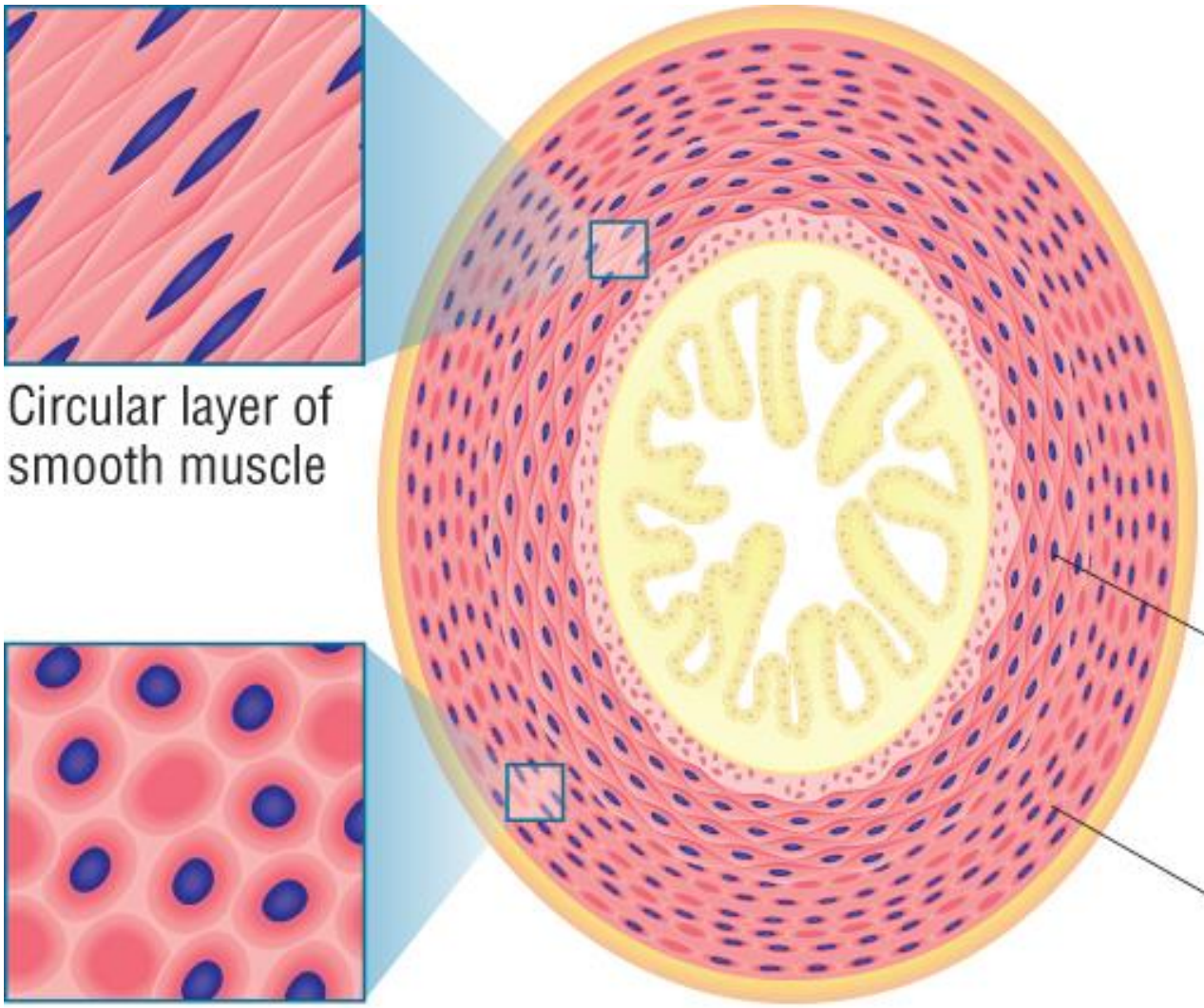


Involuntary (Smooth) Muscle

- Stimulated by autonomic motor neurones.
- No striations.
- Slow contractions, easily fatigued.
- Various locations in the body:
  - Walls of intestines
  - Iris of the eye
  - Walls of arteries/arterioles
  - Wall of uterus
  - Stomach & anal sphincters






Circular layer of smooth muscle
Longitudinal layer of smooth muscle
The circular layer runs around the intestine and its contraction causes segmentation
The longitudinal layer runs along the intestine; it causes wave-like contractions

Write a question for which the answer is…

Synovial joint

Power stroke

Cross-bridge

Myogenic

Sarcomere

Skeletal muscle

Synapse

Striated muscle

Actin

Gradation of response

Myosin

Neuromuscular junction

ATP

Sliding filament

Cardiac muscle

Voluntary muscle