Animal Physiology (22ZOOC33)

Structure of Skeletal Muscle

Structure of Skeletal Muscle

- **MUSCLE MASS**
- Muscle mass or muscle tissue is made up of a large number of individual **muscle cells or myocytes.**
- **The** muscle cells are commonly called muscle fibers because these cells are long and slender in appearance.
- Skeletal muscle fibers are multinucleated and are arranged parallel to one another with some connective tissue in between

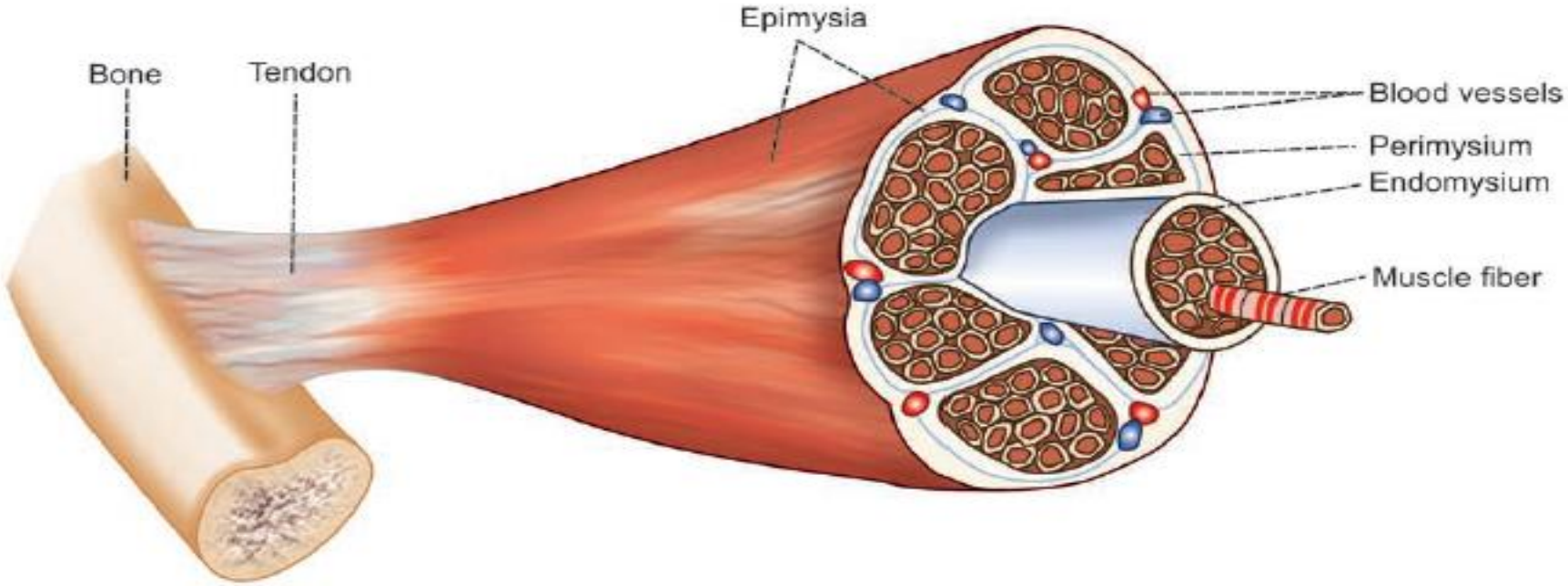


FIGURE 29.1: Structure of a skeletal muscle

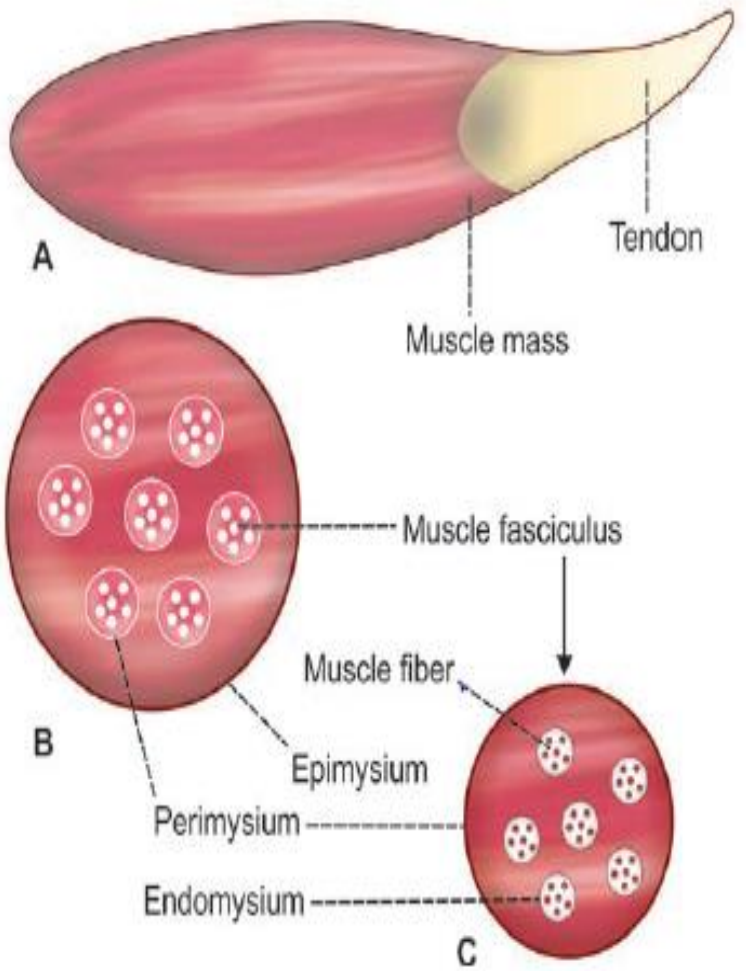


FIGURE 29.2: Diagram showing. A. Skeletal muscle mass; B. Cross-section of muscle; C. One muscle fasciculus.

- Muscle is separated from the neighboring tissues by a thick fibrous tissue layer known as **fascia.**
- Beneath the fascia, muscle is covered by a connective
- tissue sheath called **epimysium.**
- **In the muscle, the** muscle fibers are arranged in various groups called bundles or **fasciculi.**
- **Connective tissue sheath that** covers each fasciculus is called **perimysium.**
- **Each** muscle fiber is covered by a connective tissue layer called the **endomysium.**

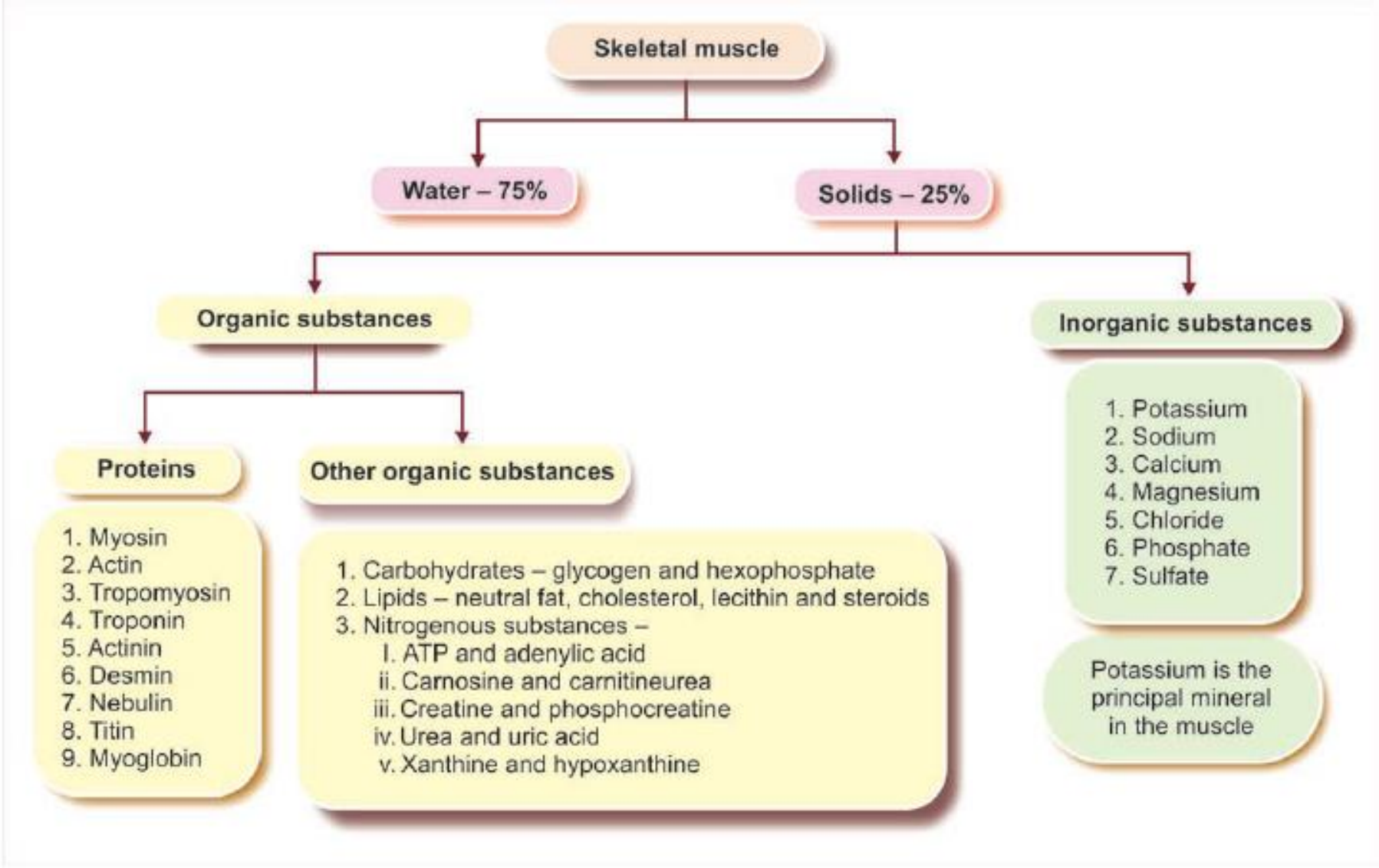


FIGURE 29.10: Composition of skeletal muscle

MYOSIN MOLECULE

- Each myosin filament consists of about 200 myosin molecules.
- Though about 18 classes of myosin are identified, only myosin II is present in the sarcomere.
- Myosin II is a globulin with a molecular weight of 480,000. Each myosin molecule is made up of 6 polypeptide chains, of which two are heavy chains and four are light.
- Molecular weight of each heavy chain is 200,000 (2 × 200,000 = 400,000).
- Molecular weight of each light chain is 20,000 (4 × 20,000 = 80,000).
- Thus, total molecular weight of each myosin molecule is 480,000 (400,000 + 80,000).

Portions of Myosin Molecule

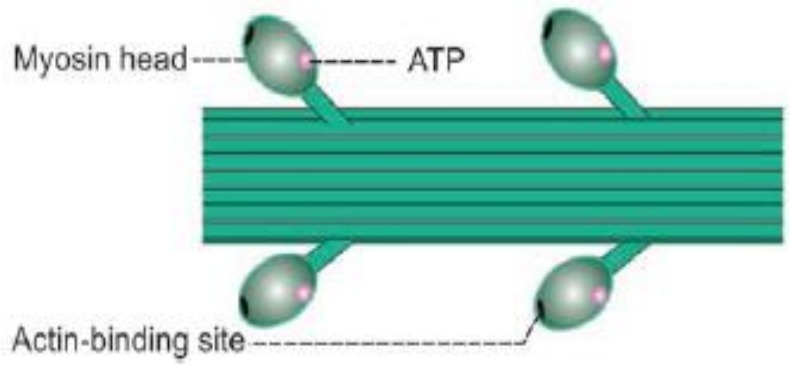


FIGURE 29.6: Diagram showing myosin filament.
ATP = Adenosine triphosphate.

- Each myosin molecule has two p
- 1. Tail portion
- 2. Head portion.
- *Tail portion of myosin molecule*
- It is made up of two heavy chains, which twist around
- each other in the form of a double helix.
- *Head portion of myosin molecule*
- At one end of the double helix, both the heavy chains
- turn away in opposite directions and form the globular head portion. Thus the head portion has two parts.
- Two light chains are attached to each part of the

- Each myosin head has two attachment sites.
- One site is for actin filament and the other one is for one ATP molecule.
- Myosin head is absent in the central part of myosin filament, i.e. in the 'H' zone.

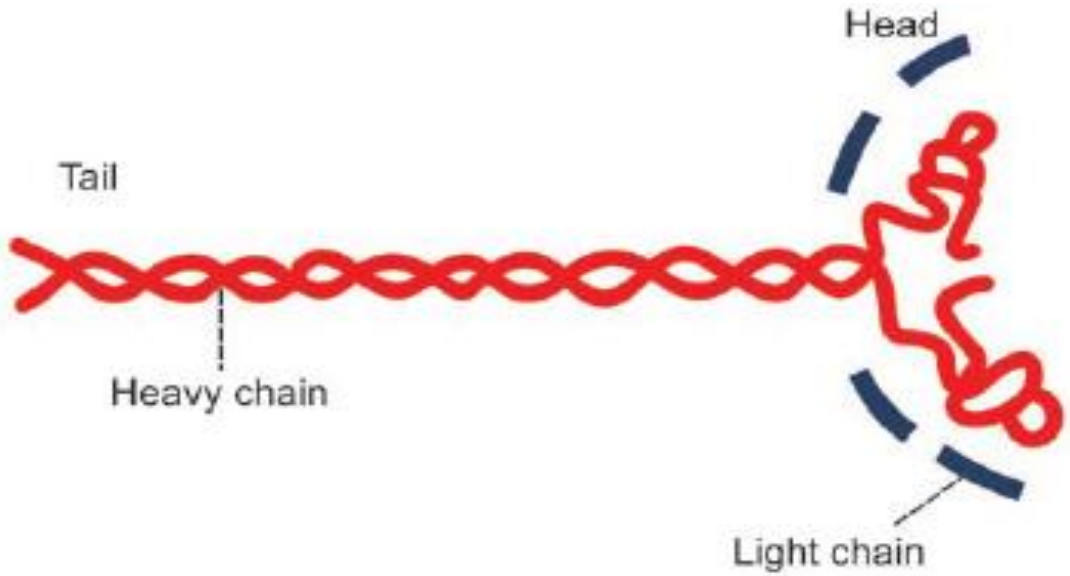


FIGURE 29.7: Myosin molecule formed by two heavy chains and four light chains of polypeptides

- **ACTIN MOLECULE**
- Actin molecules are the major constituents of the thin
- actin filaments.
- Each actin molecule is called **F-actin** and it is the polymer of a small protein known as **G-actin.**
- There are about 300 to 400 actin molecules in each actin filament.
- The molecular weight of each molecule is 42,000.
- The actin molecules in the actin filament are also arranged in the form of a double helix.
- Each Factin molecule has an active site to which the myosin head is attached.

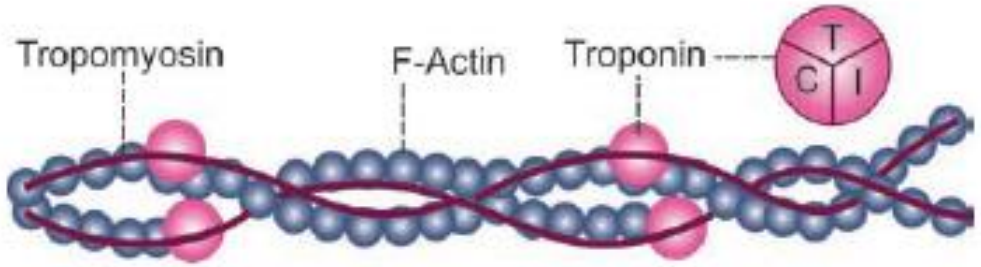


FIGURE 29.8: Part of actin filament.
Troponin has three subunits, T, C and I.

- **TROPOMYOSIN**
- About 40 to 60 tropomyosin molecules are situated along the double helix strand of actin filament.
- Each tropomyosin molecule has the molecular weight
- of 70,000.
- In relaxed condition of the muscle, the tropomyosin molecules cover all the active sites of Factin molecules.

TROPONIN

It is formed by three subunits:

1. Troponin I, which is attached to Factin
2. Troponin T, which is attached to tropomyosin
3. Troponin C, which is attached to calcium ions.

OTHER PROTEINS OF THE MUSCLE

- In addition to the contractile proteins, the sarcomere
- contains several other proteins such as:
- 1. **Actinin, which attaches actin filament to 'Z' line.**
- 2. **Desmin, which binds 'Z' line with sarcolemma.**
- 3. **Nebulin, which runs in close association with and**
- parallel to actin filaments.
- 4. **Titin, a large protein connecting 'M' line and 'Z' line.**Each titin molecule forms **scaffolding (framework)**for sarcomere and provides elasticity to the muscle. When the muscle is stretched, the titin unfolds itself.
- However, if the stretching is more, it offers resistance and protects the sarcomere from overstretching.
- 5. **Dystrophin, a rodshaped** large protein that connects
- actin filament to **dystroglycan.**
- **Dystroglycan is a** transmembrane protein, present in the sarcolemma.
- Dystro phin and dystroglycan form dystrophindystroglycan
- or dystrophinglycoprotein complex.

- **MYOFIBRIL**
- Myofibrils or myofibrillae are the fine parallel filaments
- present in sarcoplasm of the muscle cell.
- Myofibrils run through the entire length of the muscle fiber.
- In the cross-section of a muscle fiber, the myofibrils appear like small distinct dots within the sarcoplasm.
- Diameter of the myofibril is 0.2 to 2 μ.
- The length of a myofibril varies between 1 cm and 4 cm, depending upon the length of the muscle fiber (Table 29.1).
- In some muscle fibers, some of the myofibrils are arranged in groups called Cohnheim's areas or fields

TABLE 29.1: Dimensions of structures in skeletal muscle

Structure	Length	Diameter
Muscle fiber	1 cm to 4 cm	10 μ to 100 μ
Myofibril	1 cm to 4 cm	0.2 μ to 2 μ
Actin filament	1 μ	20 Å
Myosin filament	1.5 μ	115 Å

- **MUSCLE FIBER**
- Each muscle cell or muscle fiber is cylindrical in shape.
- Average length of the fiber is 3 cm.
- It varies between 1 cm and 4 cm, depending upon the length of the muscle. The diameter of the muscle fiber varies from 10 μ to 100 μ.
- The diameter varies in a single muscle.
- Muscle fibers are attached to a tough cord of connective tissue called **tendon.**
- **Tendon is in turn** attached to the bone. Tendon of some muscles is thin, flat and stretched but tough. Such type of tendon is called **aponeurosis.**

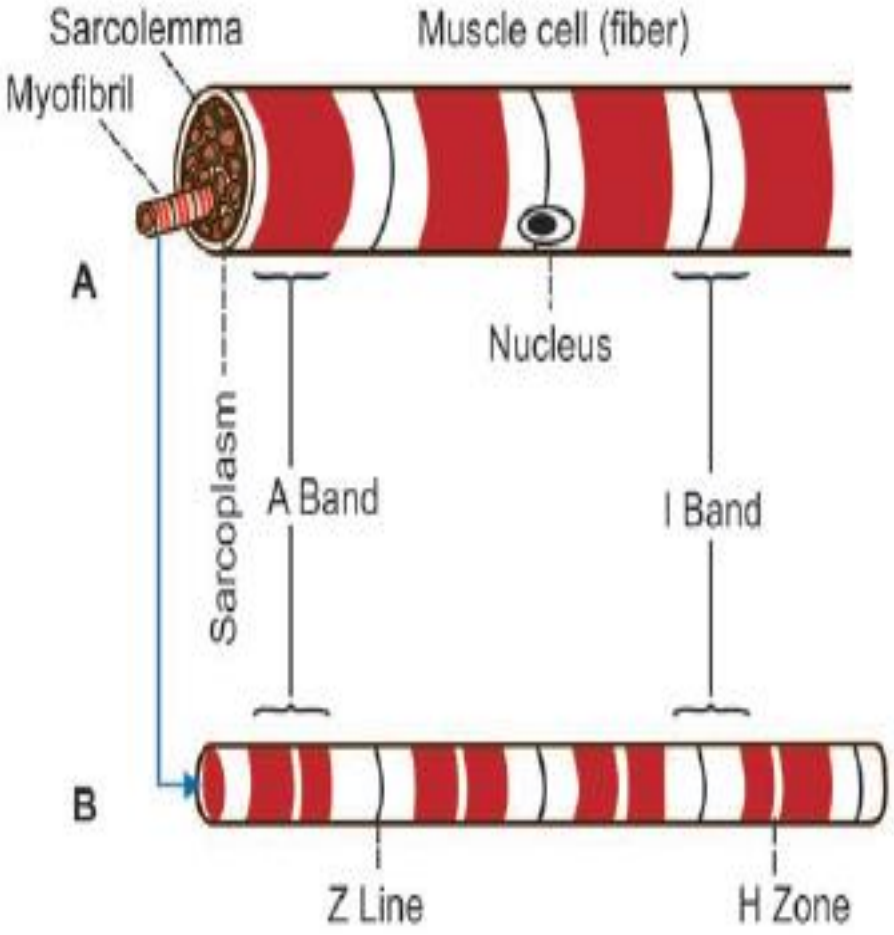


FIGURE 29.3: A. One muscle cell; B. One myofibril.

Sarcomere

- Sarcomere is defined as the structural and functional unit of a skeletal muscle.
- It is also called the basic contractile unit of the muscle.
- ***Extent***
- Each sarcomere extends between two 'Z' lines of myofibril. Thus, each myofibril contains many sarcomeres arranged in series throughout its length. When the muscle is in relaxed state, the average length of each sarcomere is 2 to 3 μ.
- ***Components***
- Each myofibril consists of an alternate dark 'A' band and light 'I' band (Fig. 29.4). In the middle of 'A' band, there is a light area called **'H' zone (H = hell = light - in German,** H = Henson – discoverer). In the middle of 'H' zone lies the middle part of myosin filament.
- This is called **'M' line** (in German-mittel = middle). 'M' line is formed by myosin binding proteins.

- **ELECTRON MICROSCOPIC STUDY OF SARCOMERE**
- Electron microscopic studies reveal that the sarcomere
- consists of many threadlike structures called m**yofilaments.**
- Myofilaments are of two types:
- 1. Actin filaments
- 2. Myosin filaments.

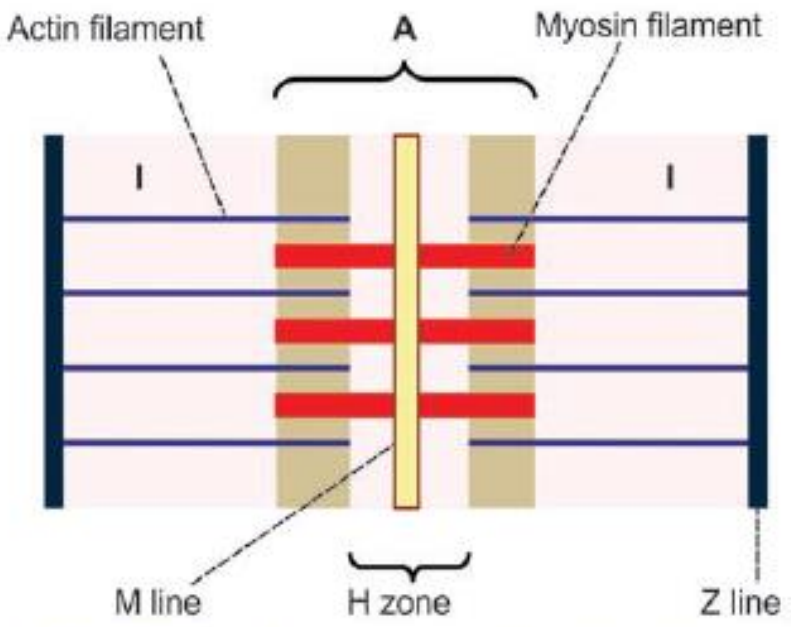


FIGURE 29.4: Sarcomere. A = A band, I = I band.

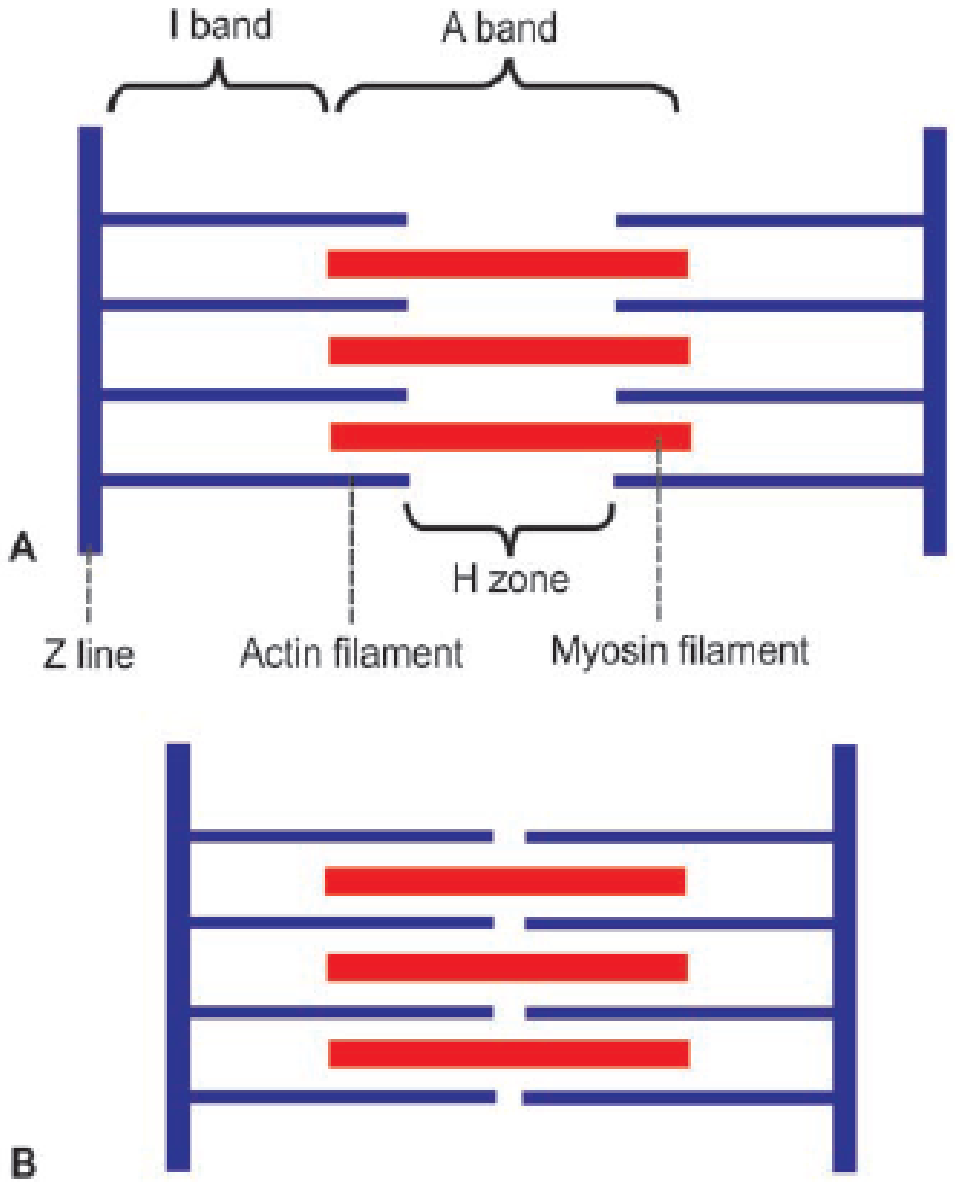


FIGURE 29.5: Sarcomere in resting muscle A. Contracted muscle; B. During contraction; Z lines come close, H zone and I band are reduced and no change in A band.

CONTRACTILE ELEMENTS (PROTEINS) OF MUSCLE

- Myosin filaments are formed by myosin molecules.
- Actin filaments are formed by three types of proteins
- called actin, tropomyosin and troponin.
- These four proteins together constitute the contractile proteins or the contractile elements of the muscle.

- **SARCOTUBULAR SYSTEM**
- Sarcotubular system is a system of membranous tructures in the form of vesicles and tubules in the sarco plasm of the muscle fiber.
- It surrounds the myofibrils embedded in the sarcoplasm

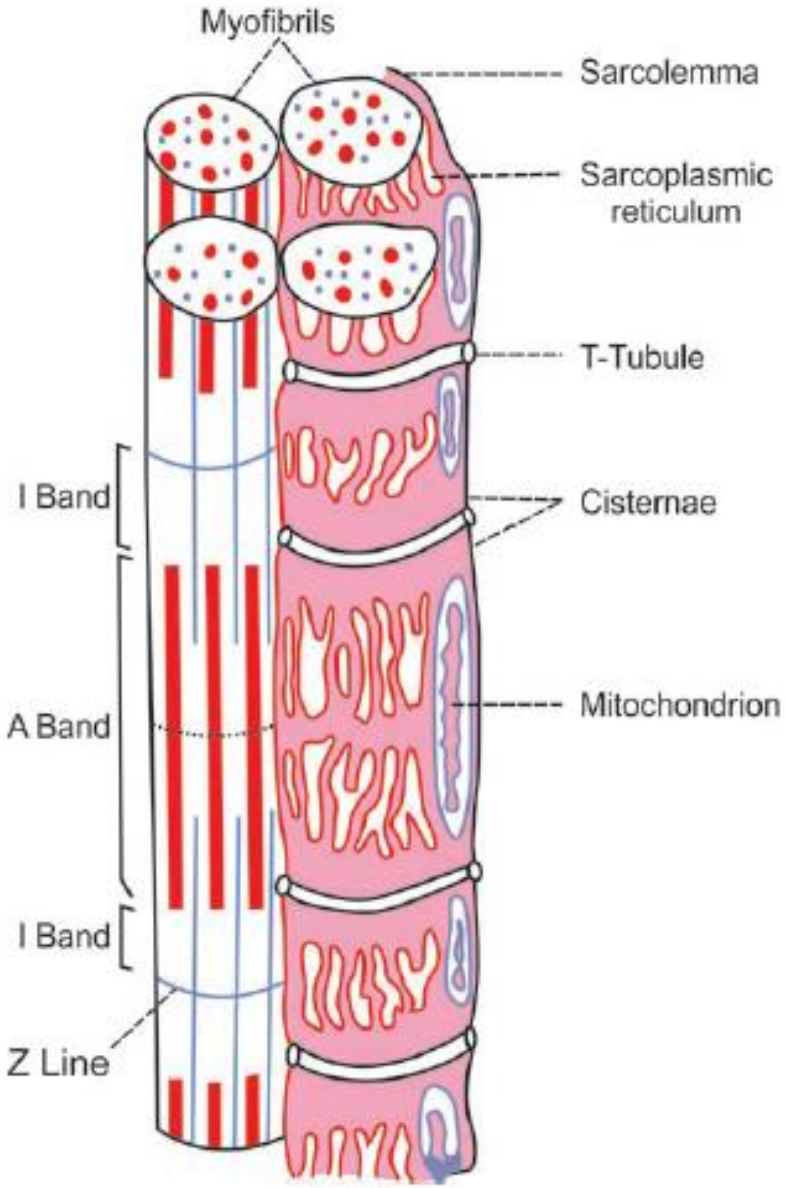


FIGURE 29.9: Diagram showing the relation between sarcotubular system and parts of sarcomere. Only few myofilaments are shown in the myofibril drawn on the right side of the diagram.

- **STRUCTURES CONSTITUTING THE SARCOTUBULAR SYSTEM**
- Sarcotubular system is formed mainly by two types
- 1. Ttubules 2. Ltubules or sarcoplasmic reticulum.
- ***T-Tubules***
- T tubules or transverse tubules are narrow tubules formed by the invagination of the sarcolemma.
- These tubules penetrate all the way from one side of the muscle fiber to an another side.
- That is, these tubules penetrate the muscle cell through and through.
- Because of their origin from sarcolemma, the Ttubules open to the exterior of the muscle cell. Therefore, the ECF runs through their lumen.

- ***L-Tubules or Sarcoplasmic Reticulum***
- Ltubules or longitudinal tubules are the closed tubules
- that run in long axis of the muscle fiber, forming
- **sarcoplasmic reticulum.**
- **These tubules form a closed** tubular system around each myofibril and do not open to exterior like T-tubules
- L-tubules correspond to the endoplasmic reticulum of other cells. At regular intervals, throughout the length of the myofibrils, the L-tubules dilate to form a pair
- of lateral sacs called terminal **cisternae.**
- **Each pair of** terminal cisternae is in close contact with T-tubule.
- The T-tubule along with the cisternae on either side is called the triad of skeletal muscle.

- ***Function of L-Tubules***
- Ltubules store a large quantity of calcium ions.
- When action potential reaches the cisternae of L-tubule,
- The calcium ions are released into the sarcoplasm. Calcium ions trigger the processes involved in contraction of the muscle.
- The process by which the calcium ions cause contraction of muscle is called excitation contraction coupling

Animal Physiology (22ZOOC33)

Mechanism of Muscle Contraction

Mechanism of Muscle Contraction

- The muscle contracts when it is stimulated.
- Contraction of the muscle is a physical or mechanical event. In addition, several other changes occur in the muscle.
- Changes taking place during muscular contraction:
- 1. Electrical changes
- 2. Physical changes
- 3. Histological (molecular) changes
- 4. Chemical changes
- 5. Thermal changes.

- **ELECTRICAL CHANGES DURING MUSCULAR CONTRACTION**
- Electrical events occur in the muscle during resting condition as well as active conditions.
- Electrical potential Changes during Muscular Contraction in the muscle during resting condition is called resting membrane potential.
- Electrical changes that occur in active conditions, i.e. when the muscle is stimulated are together called action potential.
- Electrical potentials in a muscle (or any living tissue) are measured by using a cathode ray oscilloscope or computerized polygraph

- When two electrodes are connected to a cathode ray through a suitable amplifier and placed over the surface of the muscle fiber, there is no potential difference, i.e. there is zero potential difference.
- But, if one of the electrodes is inserted into the interior of muscle fiber, potential difference is observed across the sarcolemma (cell membrane).
- There is negativity inside and positivity outside the muscle fiber.
- This potential difference is constant and is called resting membrane potential.
- The condition of the muscle during resting membrane
- potential is called **polarized state.**
- **In human** skeletal muscle, the resting membrane potential is –90 mV.

Ionic Basis of Resting Membrane Potential

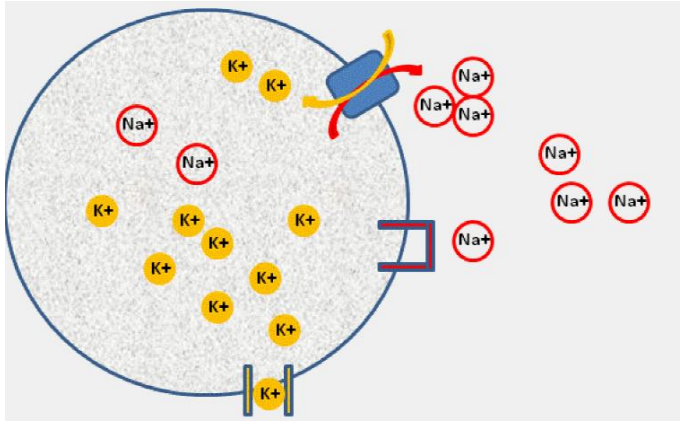
- Development and maintenance of resting membrane potential in a muscle fiber or a neuron are carried out by movement of ions, which produce ionic imbalance across the cell membrane.
- This results more positivity outside and more negativity inside the cell.

Ionic imbalance is produced by two factors:

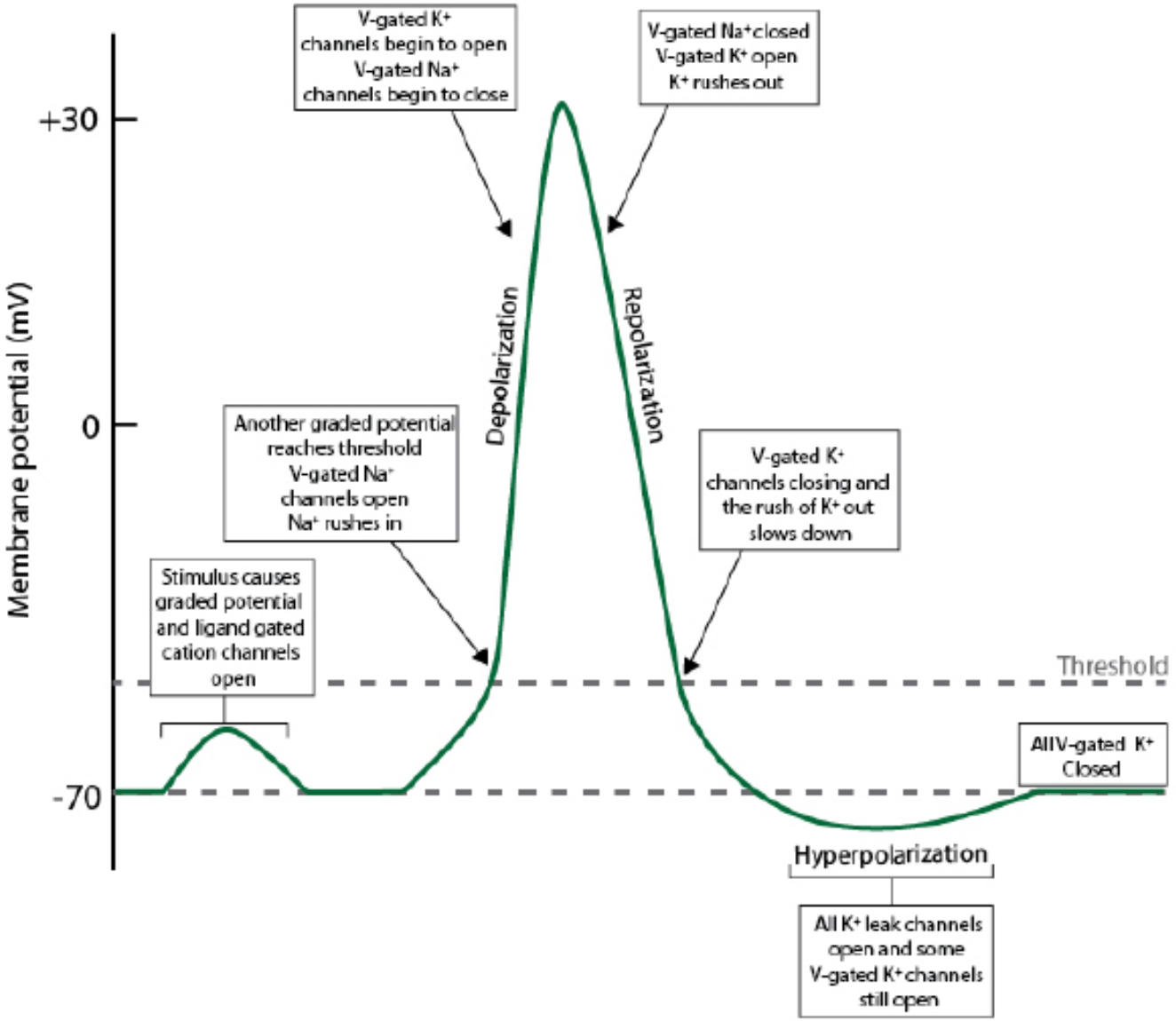
- 1. Sodium potassium pump
- 2. Selective permeability of cell membrane.

1. *Sodium-potassium pump*

- Sodium and potassium ions are actively transported in opposite directions across the cell membrane by means of an electrogenic pump called sodium potassium pump.
- It moves three sodium ions out of the cell and two potassium ions inside the cell by using energy from ATP.
- Since more positive ions (cations) are pumped outside than inside, a net deficit of positive ions occurs inside the cell.
- It leads to negativity inside and positivity outside the cell



- ***Selective permeability of cell membrane***
- Permeability of cell membrane depends largely on the
- transport channels.
- The transport channels are selective for the movement of some specific ions.
- Their permeability to these ions also varies.
- Most of the channels are **gated channels and the specific ions can move** across the membrane only when these gated channels are opened.
- Two types of channels are involved:
- i. Channels for major anions like proteins
- ii. Leak channels.


V-gated K+ channels begin to open V-gated Na+ channels begin to close
V-gated Na+ closed V-gated K+ open K+ rushes out
+30
0
-70
Membrane potential (mV)
Depolarization
Repolarization
Another graded potential reaches threshold V-gated Na+ channels open Na+ rushes in
V-gated K+ channels closing and the rush of K+ out slows down
Stimulus causes graded potential and ligand gated cation channels open
Threshold
All V-gated K+ Closed
Hyperpolarization
All K+ leak channels open and some V-gated K+ channels still open

- **ACTION POTENTIAL CURVE**
- Action potential curve is the graphical registration of electrical activity that occurs in an excitable tissue such as muscle after stimulation.
- It shows three major parts:
- 1. Latent period
- 2. Depolarization
- 3. Repolarization.
- Resting membrane potential in skeletal muscle is -90 mV and it is recorded as a straight baseline.

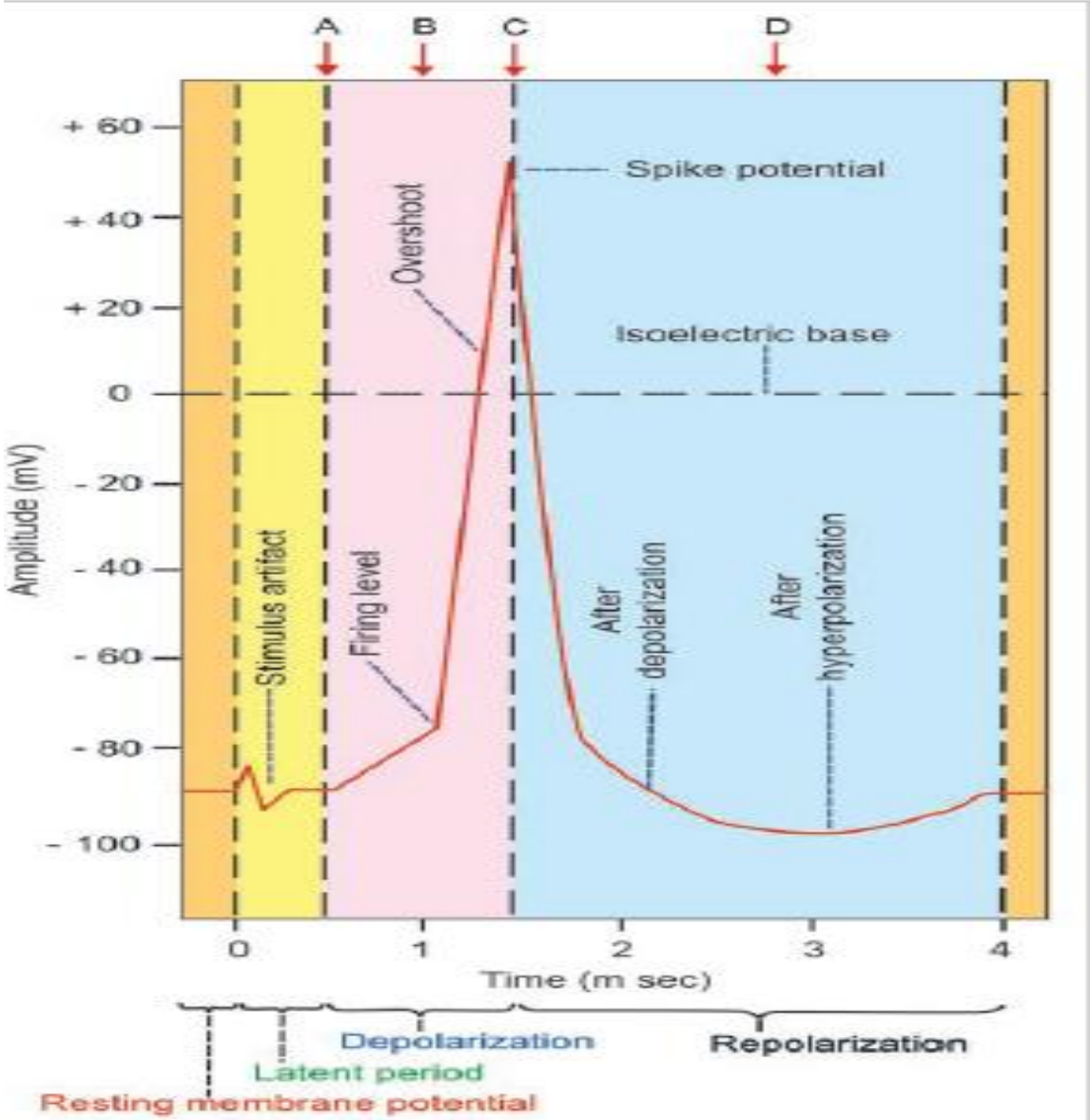


FIGURE 31.2: Action potential in a skeletal muscle
A = Opening of few Na^+ channels
B = Opening of many Na^+ channels
C = Closure of Na^+ channels and opening of K^+ channels
D = Closure of K^+ channels

- *Stimulus artifact*
- When a stimulus is applied, there is a slight irregular deflection of baseline for a very short period.
- This is called stimulus artifact.
- The artifact occurs because of the disturbance in the muscle due to leakage of current from stimulating electrode to the recording electrode.
- The stimulus artifact is followed by latent period.

- *2. Depolarization*
- Depolarization starts after the latent period. Initially, it is very slow and the muscle is depolarized for about 15 mV.
- *Firing level and depolarization*
- After the initial slow depolarization for 15 mV (up to –75 mV), the rate of depolarization increases suddenly.
- In which point depolarization increases suddenly is called firing level.
- *Overshoot*
- From firing level, the curve reaches **isoelectric potential (zero potential) rapidly and then shoots up (overshoots)** beyond the zero potential **(isoelectric base) up to +55**mV. It is called overshoot.

- ***3. Repolarization***
- When depolarization is completed (+55 mV), the repolarization starts. Initially, the repolarization occurs rapidly and then it becomes slow.
- *Spike potential* Rapid rise in depolarization and the rapid fall in repolarization are together called spike potential.
- It lasts for 0.4 millisecond.
- *After depolarization or negative after potential* Rapid fall in repolarization is followed by a slow repolarization.
- It is called after depolarization or negative after potential. Its duration is 2 to 4 milliseconds.

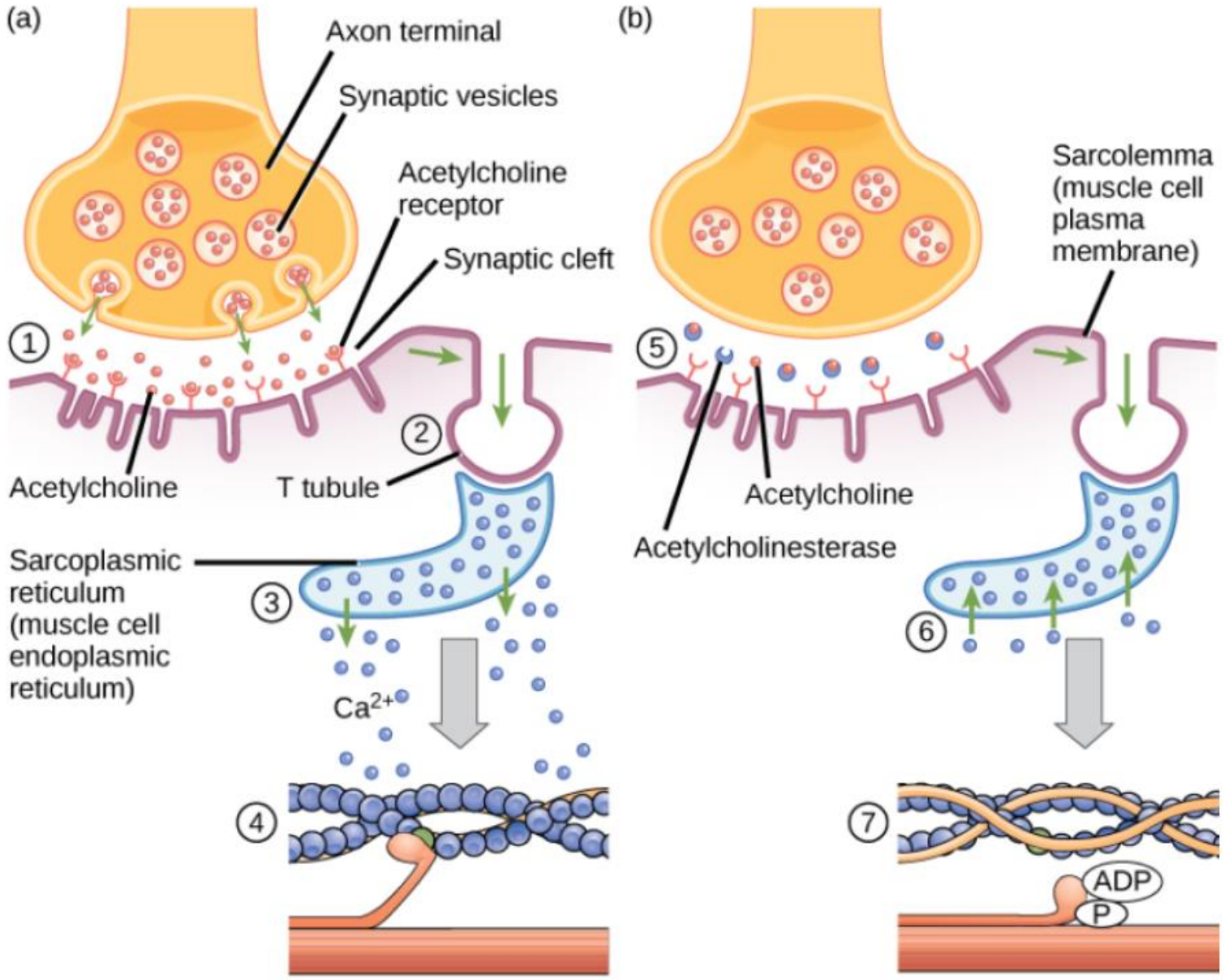
- **Physical Changes During Muscular Contraction**
- Physical change, which takes place during muscular contraction, is the change in length of the muscle fibers or change in tension developed in the muscle.
- Depending upon this, the muscular contraction is classified into two types namely isotonic contraction and isometric contraction
- **Histological Changes During Muscular Contraction**
- **ACTOMYOSIN COMPLEX**
- In relaxed state of the muscle, the thin actin filaments from opposite ends of sarcomere are away from each other leaving a broad ‘H’ zone.
- During contraction of the muscle, actin (thin) filaments glide over myosin (thick) filaments and form actomyosin complex

Molecular Basis Of Muscular Contraction

- Molecular mechanism is responsible for formation of
- actomyosin complex that results in muscular contraction.
- It includes three stages:
- 1. Excitation-contraction coupling.
- 2. Role of troponin and tropomyosin.
- 3. Sliding mechanism.

Neuronal Control of Skeletal Muscles

- Communication occurs between nerves and muscles through neurotransmitters.
- Neuron action potentials cause the release of neurotransmitters from the synaptic terminal into the synaptic cleft, where they can then diffuse across the synaptic cleft and bind to a receptor molecule on the motor end plate.
- The motor end plate possesses junctional folds—folds in the sarcolemma that create a large surface area for the neurotransmitter to bind to receptors.
- The receptors are actually sodium channels that open to allow the passage of Na+ into the cell when they receive a neurotransmitter signal.


(a)
Axon terminal
Synaptic vesicles
Acetylcholine receptor
Synaptic cleft
1
Acetylcholine
2
T tubule
Sarcoplasmic reticulum (muscle cell endoplasmic reticulum)
3
Ca^{2+}
4
(b)
Sarcolemma (muscle cell plasma membrane)
5
Acetylcholine
Acetylcholinesterase
6
7
ADP
P

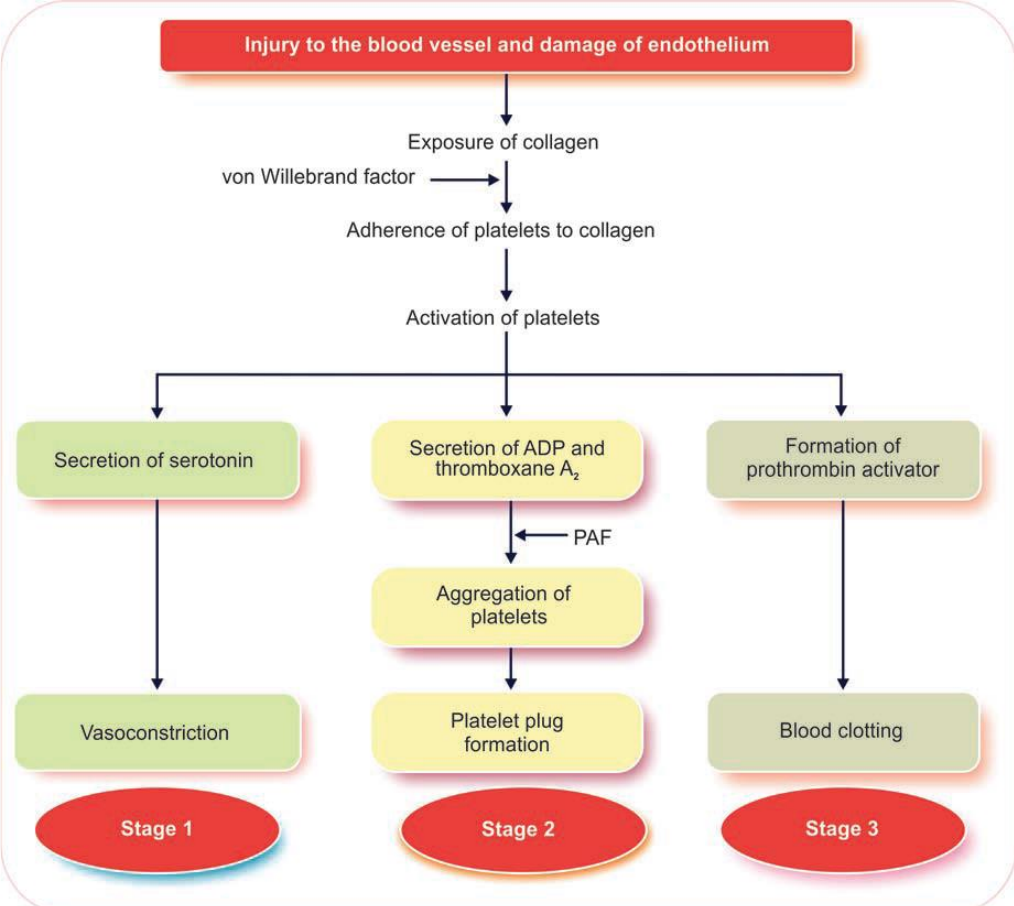
ANIMAL PHYSIOLOGY
(22ZOOC33)

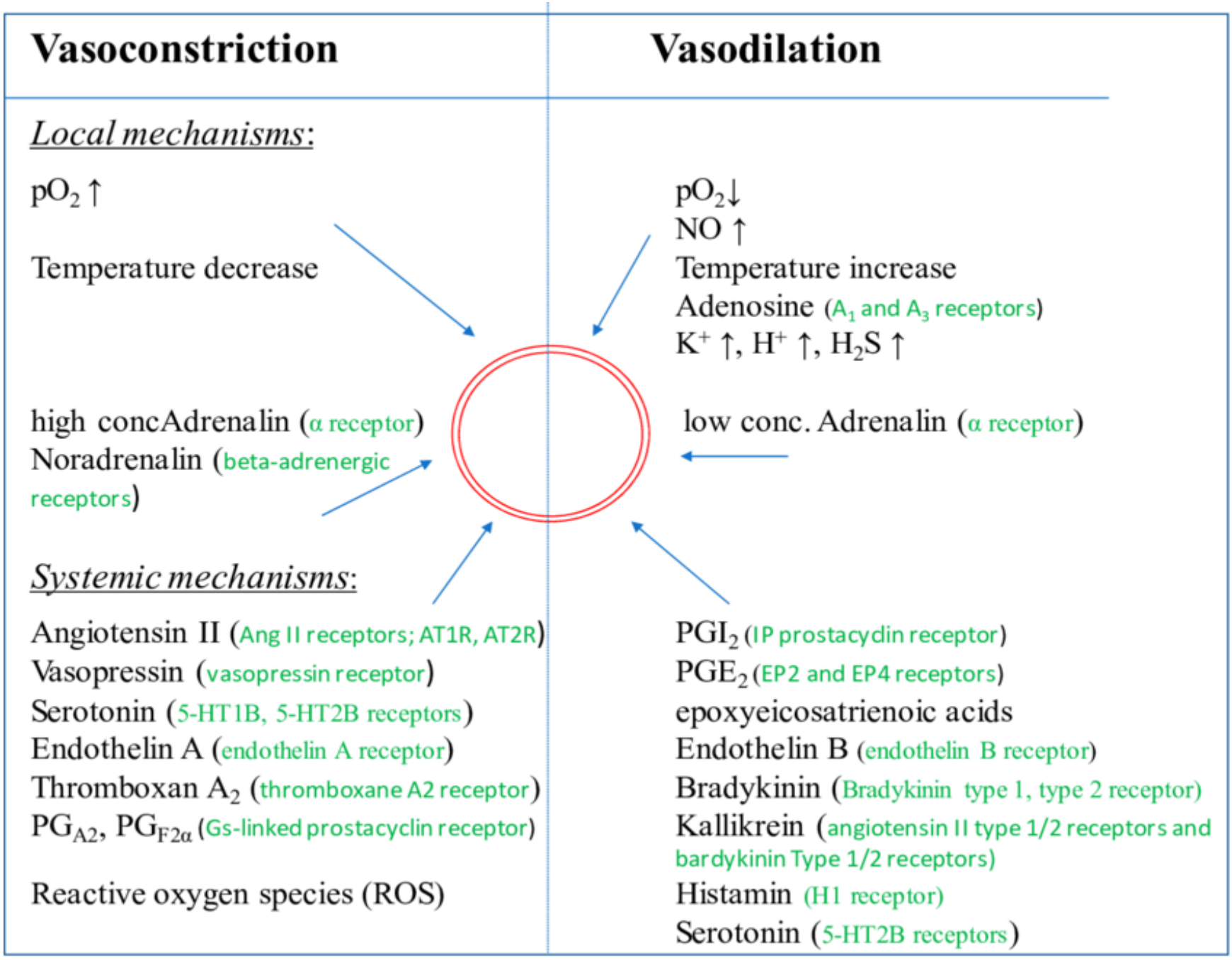
Coagulation of Blood

- Arrest or stoppage of bleeding

STAGES OF HEMOSTASIS

- When a blood vessel is injured, the injury initiates a series of reactions, resulting in hemostasis.
- It occurs in three stages:
- 1. Vasoconstriction
- 2. Platelet plug formation
- 3. Coagulation of blood

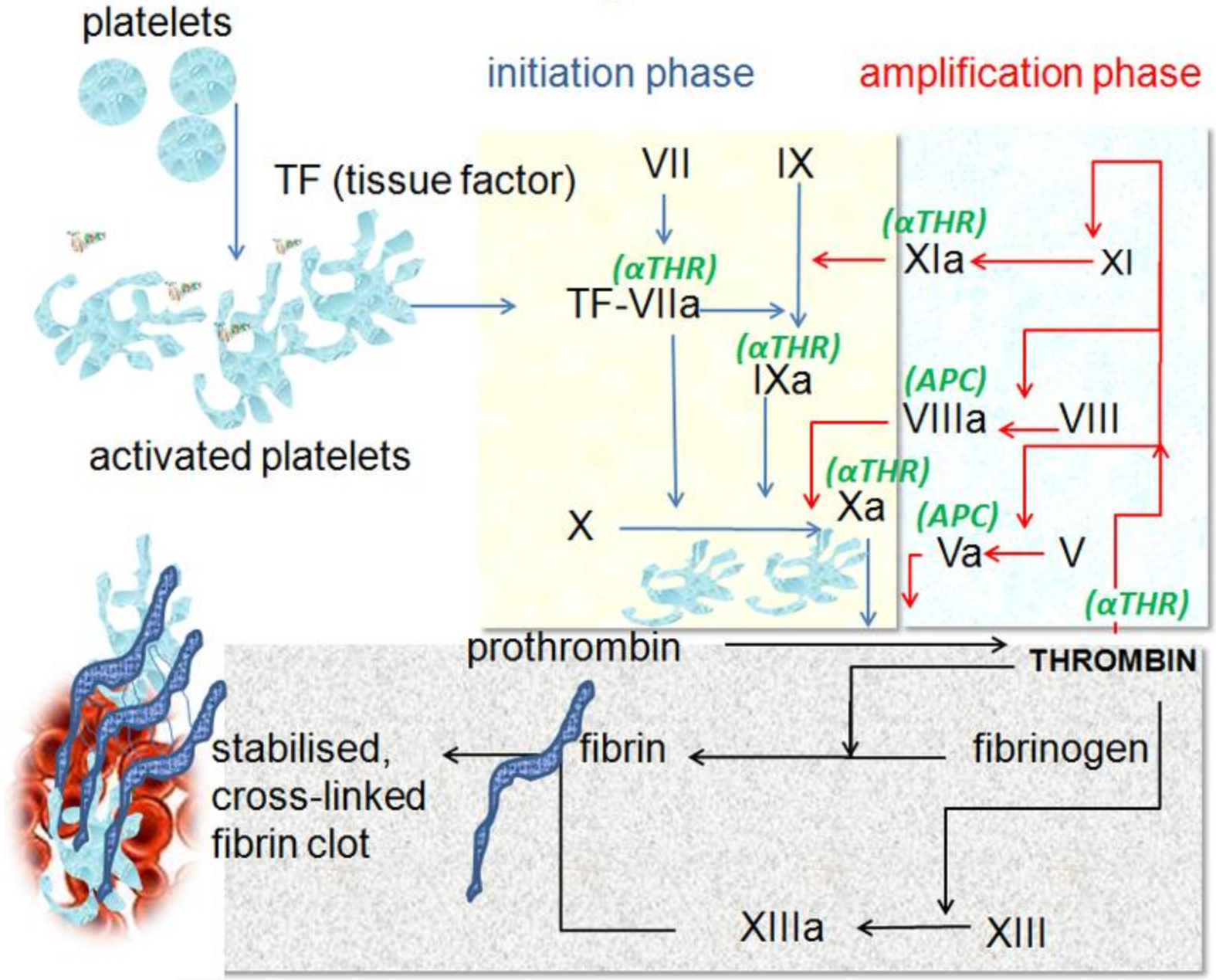



Vasoconstriction
Vasodilation
Local mechanisms:
pO2 ↑
Temperature decrease
pO2↓
NO ↑
Temperature increase
Adenosine (A1 and A3 receptors)
K+ ↑, H+ ↑, H2S ↑
high concAdrenalin (α receptor)
Noradrenalin (beta-adrenergic receptors)
low conc. Adrenalin (α receptor)
Systemic mechanisms:
Angiotensin II (Ang II receptors; AT1R, AT2R)
Vasopressin (vasopressin receptor)
Serotonin (5-HT1B, 5-HT2B receptors)
Endothelin A (endothelin A receptor)
Thromboxan A2 (thromboxane A2 receptor)
PGA2, PGF2α (Gs-linked prostacyclin receptor)
Reactive oxygen species (ROS)
PGI2 (IP prostacydin receptor)
PGE2 (EP2 and EP4 receptors)
epoxyeicosatrienoic acids
Endothelin B (endothelin B receptor)
Bradykinin (Bradykinin type 1, type 2 receptor)
Kallikrein (angiotensin II type 1/2 receptors and bardykinin Type 1/2 receptors)
Histamin (H1 receptor)
Serotonin (5-HT2B receptors)

PLATELET PLUG FORMATION

- Platelets get adhered to the collagen of ruptured blood vessel and secrete **adenosine diphosphate (ADP) and throm boxane A2**.
- These two substances attract more and more platelets and activate them.
- All these platelets aggregate together and form a loose temporary platelet plug or temporary hemostatic plug, which closes the ruptured vessel and prevents further blood loss.
- Platelet aggregation is accelerated by plateletactivating factor (PAF).

Blood coagulation *in vivo*



- **Coagulation of blood** occurs through a series of reactions due to the activation of a group of substances.
- Substances necessary for clotting are called **clotting factors.**

Clotting Factors	
Factor I	Fibrinogen
Factor II	Prothrombin
Factor III	Thromboplastin (Tissue factor)
Factor IV	Calcium
Factor V	Labile factor (Proaccelerin or accelerator globulin)
Factor VI	Presence has not been proved
Factor VII	Stable factor
Factor VIII	Antihemophilic factor (Antihemophilic globulin)
Factor IX	Christmas factor
Factor X	Stuart-Prower factor
Factor XI	Plasma thromboplastin antecedent
Factor XII	Hageman factor (Contact factor)
Factor XIII	Fibrin-stabilizing factor (Fibrinase).

SEQUENCE OF CLOTTING MECHANISM

- **ENZYME CASCADE THEORY**
- Most of the clotting factors are proteins in the form of enzymes.
- Normally, all the factors are present in the form of inactive **proenzyme.**
- These proenzymes must be activated into enzymes to **enforce clot formation**.
- It is carried out by a **series of proenzyme-enzyme conversion reactions**.
- First one of the series is converted into an active enzyme that activates the second one, which activates the third one; this continues till the final active enzyme thrombin is formed.
- Enzyme cascade theory explains how various reactions, involved in the conversion of proenzymes to active enzymes take place in the form of a cascade.
- **Cascade refers to a process that occurs through a series of steps, each step initiating the next, until the final step is reached.**

Stages of Blood Clotting

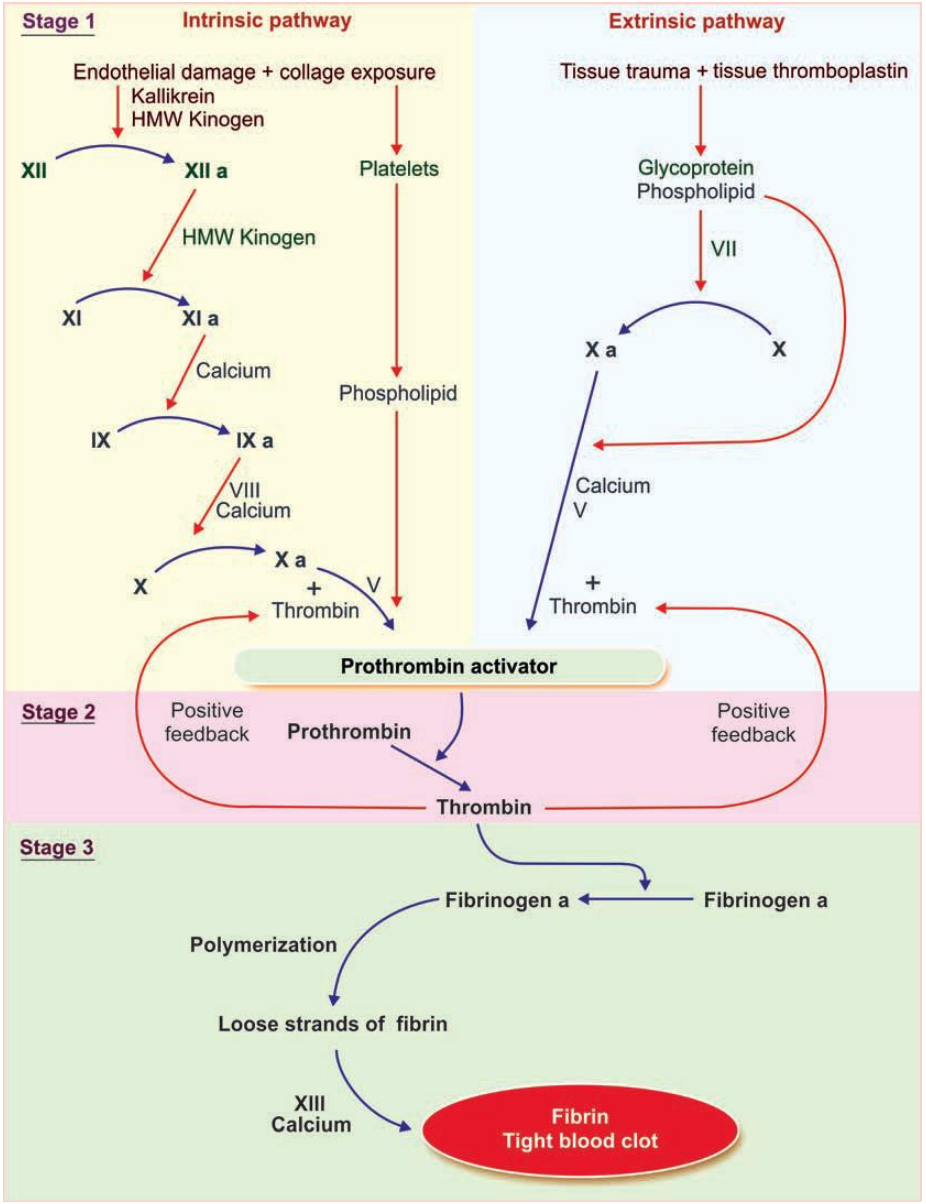
- Blood clotting occurs in three stages:
  - 1. Formation of prothrombin activator
  - 2. Conversion of prothrombin into thrombin
  - 3. Conversion of fibrinogen into fibrin

STAGE 1: FORMATION OF PROTHROMBIN ACTIVATOR

- Blood clotting commences with the formation of a substance called **prothrombin activator**, which converts **prothrombin into thrombin**.
- Its formation is initiated by substances produced either within the blood or outside the blood.
- Thus, formation of prothrombin activator occurs through two pathways:
  - i. Intrinsic pathway
  - ii. Extrinsic pathway.

i. *Intrinsic Pathway for the Formation of Prothrombin Activator*

- In this pathway, the formation of prothrombin activator is initiated by platelets, which are within the blood itself



ii. *Extrinsic Pathway for the Formation of Prothrombin Activator*

- In this pathway, the formation of prothrombin activator is initiated by the tissue thromboplastin, which is formed from the injured tissues.

STAGE 2: CONVERSION OF PROTHROMBIN INTO THROMBIN

- Blood clotting is all about thrombin formation.
- Once thrombin is formed, it definitely leads to clot formation.

STAGE 3: CONVERSION OF FIBRINOGEN INTO FIBRIN

- The final stage of blood clotting involves the conversion of fibrinogen into fibrin by thrombin

Sequence of Events in Stage 3

- i. Thrombin converts inactive fibrinogen into activated fibrinogen due to loss of 2 pairs of polypeptides from each fibrinogen molecule. The activated fibrinogen is called **fibrin monomer.**
- ii. Fibrin monomer polymerizes with other monomer molecules and form loosely arranged strands of fibrin
- iii. Later these loose strands are modified into dense and tight fibrin threads by fibrin-stabilizing factor (factor XIII) in the presence of calcium ions. All the tight fibrin threads are aggregated to form a meshwork of **stable clot.**

Function of blood

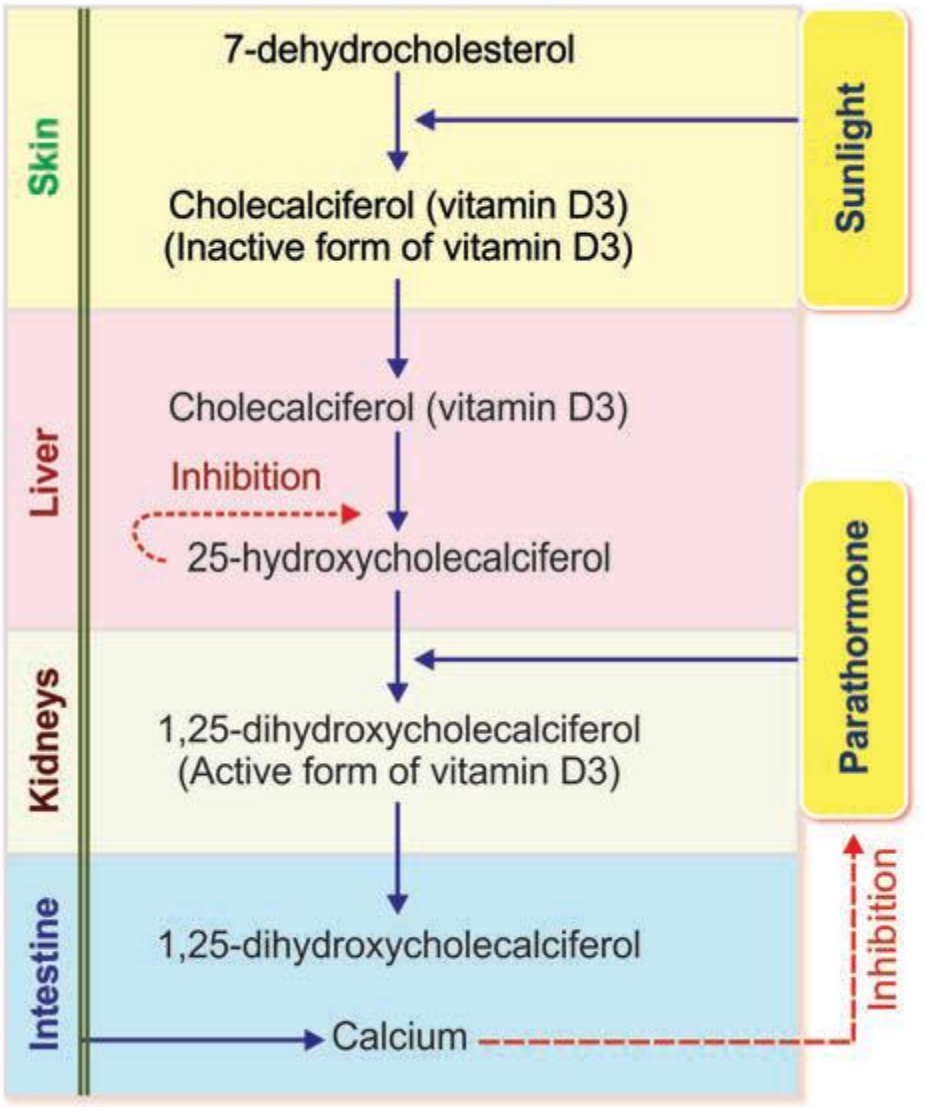
- The main function of the blood is to maintain **intracellular homeostasis** by:
  - a). Carries 02 and nutrients (glucose, amino acids, lipids, and vitamins) to the cells.
  - b). Carries C02 and other wastes (nitrates, creatine, nucleic acid) away from the cell.
- 2. **Providing intercellular communication in the body:** carries hormones (secreted by endocrine glands) to the target organs. 3. **Protection and defense:** it allows cells and immunological proteins to transport from place to place where need them.
- 4. **Self repair mechanism:** clotting cascade.

Role of calcium ions

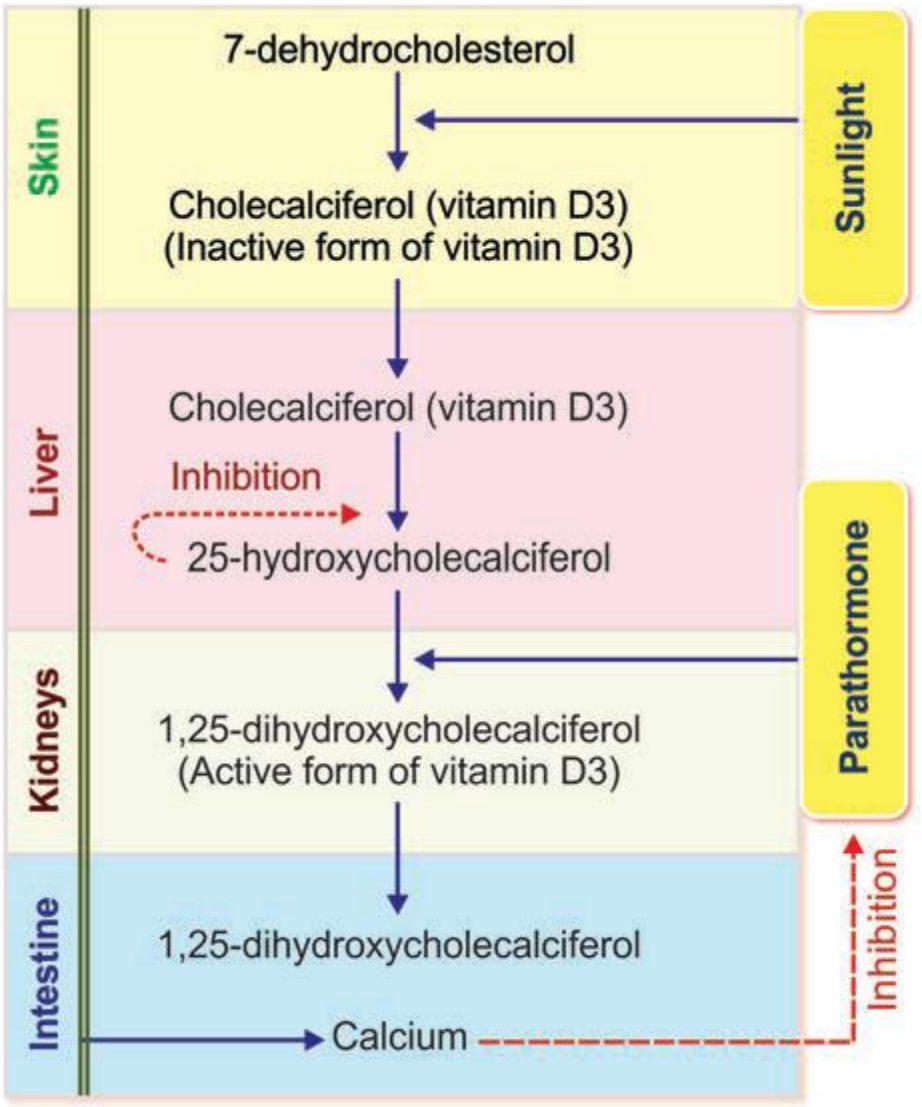
- *Activation of vitamin D*
- There are various forms of vitamin D. But, the most important one is vitamin D3. It is also known as **cholecalciferol.**
- Vitamin D3 is synthesized in the skin from 7-dehydrocholesterol, by the action of **ultraviolet rays** from the **sunlight.** It is also obtained from dietary sources.
- The activation of vitamin D3 occurs in two steps
- *First step*
- Cholecalciferol (vitamin D3) is converted into 25-hydroxycholecalciferol in the liver. This process is limited

Role of calcium ions

- ***First step***
- Cholecalciferol (vitamin D3) is converted into 25- hydroxycholecalciferol in the liver.
- This process is limited and is inhibited by 25-hydroxycholecalciferol itself by feedback mechanism.
- This inhibition is essential for two reasons:
  - i. Regulation of the amount of active vitamin D
  - ii. Storage of vitamin D for months together.
- If vitamin D3 is converted into 25-hydroxycholecalciferol, it remains in the body only for 2 to 5 days. But vitamin D3 is stored in liver for several months



- ***Second step***
- 25-hydroxycholecalciferol is converted into 1,25-
- dihydroxycholecalciferol **(calcitriol)** in kidney. It is the
- active form of vitamin D3. This step needs the presence
- of PTH.



Role of Calcium Ion

- ***in Regulating 1, 25-Dihydroxycholecalciferol***
- When blood calcium level increases, it inhibits the formation of 1,25-dihydroxycholecalciferol. The mechanism involved in the inhibition of the formation of 1,25- dihydroxycholecalciferol is as follows:
- i. Increase in calcium ion concentration directly suppresses the conversion of 25-hydroxycholecalciferol into 1,25-dihydroxycholecalciferol. This effect is very mild
- ii. Increase in calcium ion concentration decreases the PTH secretion, which in turn suppresses the conversion of 25-hydroxycholecalciferol into 1,25-dihydroxycholecalciferol.

- This regulates the calcium ion concentration of plasma itself indirectly, i.e. when the PTH synthesis is inhibited, the conversion of 25-hydroxycholecalciferol into 1,25-hydroxycholecalciferol is also inhibited.
- Lack of 1,25-dihydroxycholecalciferol, decreases the absorption of calcium ions from the intestine, from the bones and from the renal tubules as well.
- This makes **the calcium level** in the plasma to fall back to normal.

Actions of 1, 25-Dihydroxycholecalciferol

- 1. It increases the absorption of calcium from the intestine, by increasing the formation of calcium binding proteins in the intestinal epithelial cells.
- These proteins act as carrier proteins for facilitated diffusion, by which the calcium ions are transported.
- The proteins remain in the cells for several weeks after 1,25-dihydroxycholecalciferol has been removed from the body, thus causing a prolonged effect on calcium absorption
- 2. It increases the synthesis of calcium-induced ATPase in the intestinal epithelium
- 3. It increases the synthesis of alkaline phophatase in the intestinal epithelium
- 4. It increases the absorption of phosphate from intestine along with calcium