UNIT 6 MOVEMENTS

Structure

6.1 INTRODUCTION

In the previous Block you have read about the physiology of nutrition, respiration, circulation, excretion and osmoregulation in animals. In this unit you will learn about physiology of movement in animals. Often we consider movement in connection with locomotion, i.e., an organism moving from place to place. It is a characteristic and fundamental property of animals. However, even animals that remain attached and never move about such as corals, sponges, etc. exhibit variety of movements. There are three basic mechanisms, that animals use to achieve motion. The three basic mechanisms of movements are :

i) **amoeboid movement,** ii) **ciliary movement,** and iii) **muscular movement.**

Amoeboid movement derives its name from the locomotion of amoeba. It involves extensive changes in cell shape, flow of cytoplasm and pseudopodial activity. **Ciliary locomotion** is the characteristic way in which ciliated protozoans such as *Paramecium* move. However, cilia are found in all animal phyla and serve a variety of functions. For example, cilia set up currents that effect movement through the water vascular systems of echinoderms. The respiratory tract of air breathing vertebrates is lined by the ciliated cells that slowly remove foreign particles that lodge on their surfaces. The sperms move with the aid of tail, which in principle acts like a cilium. **Muscular movement** is the fundamental mechanism used in the majority of animals for a variety of movements. Muscle has the ability to exert a force by shortening, called **'muscle contraction'.** This force is used for a variety of purposes. In this unit you will read about mechanism of amoeboid movement and ciliary movement, structure of the muscle and the mechanism of muscle contraction.

Objectives

After reading this unit you shall be able to :

- explain the physiology of amoeboid movement, ciliary and flagellar movement
- elucidate the structure of vertebrate muscle and explain the molecular basis of muscle contraction
- describe the mechanisms that regulate muscle contraction
- differentiate the structure and function of skeletal muscles, cardiac muscles and smooth muscles.

6.2 AMOEBOID MOVEMENT

Amoeboid movement is the characteristic of some protozoans, slime molds and vertebrate white blood cells. The movement of these is due to cytoplasmic streaming, change in cell shape and extension of pseudopodia. These changes are easily observed under the microscope, but the mechanisms involved in activating the movement are not well understood.

When an amoeba has to move, it stretches its arm-like extensions, the **pseudopodia,** into the required direction, and its cytoplasm flows into the newly formed pseudopodia. The newly formed pseudopodia gradually extend and enlarge so that the entire cell occupies the space where previously only a small pseudopodium began to form. As the cell moves, new pseudopodia are formed in the direction of the movement, while the posterior parts are withdrawn. It is not known with certainty how the extending and retracting of the pseudopodia takes place. It appears that there is a transaction of the regions of the cytoplasm from fluid-like **sol** to semi solid **gel** state. Now we shall study how amoeboid movement is accomplished by transactions of cytoplasm from sol to gel state.

Under the light microscope, we can find two regions in the cytoplasm of the amoeba; i) the central region, the **endoplasm** which is fluid like sol and ii) the **ectoplasm,** the region of the cytoplasm just beneath the plasma membrane, which is gel-like.

In the phase contrast microscope, we can see that the endoplasm contains abundant particles and membranous organellae, found in constant random motion, indicating their freedom of movement in the sol region of the cytoplasm. Ectoplasm contains a three-dimensional network of cross-linked **actin fibres,** and all other organelle are excluded from the region. This gel region apparently decides the shape of the pseudopodium and may transmit tension from the regions of cellular contraction to the sites of contact with the substratum. It is believed that the ectoplasm contains non-cross-linked actin filaments and probably **myosin filaments** also. As a pseudopodium elongates and the sol-like endoplasm streams into it, the region of the endoplasm near the tip of the pseudopodium apparently transforms into gel-like ectoplasm (Fig. 6.1). Simultaneously, the ectoplasm elsewhere in the cell transforms into sol-like endoplasm, probably by an uncrossing of linking actin fibres. Proteins such as **actin, fimbrin** and **fodrin** are involved in the sol-to-gel transition. They cross-link actin filaments and bundle them to each other. Crosslinking of actin filaments produces a network confining the movement of individual actin molecules and results in the semisolid gel state.

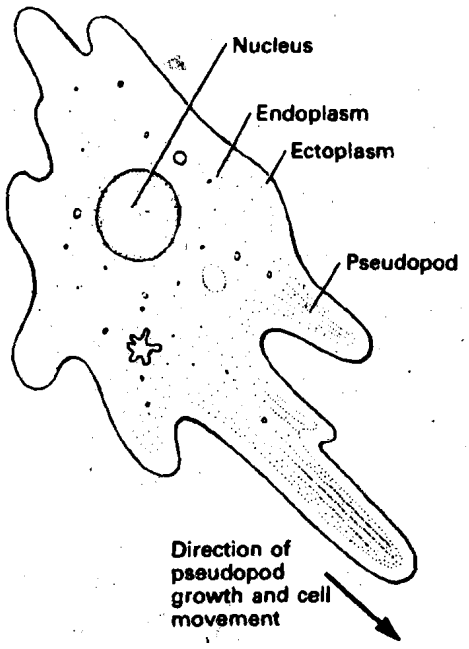
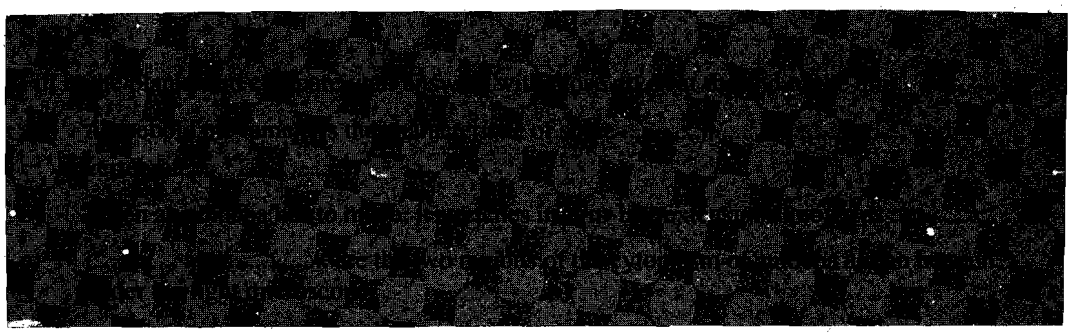


Fig. 6.1 : Schematic diagram of a moving amoeba cell, showing the sol-like endoplasm and the cortical gel-like ectoplasm.

Since actin and myosin are found in all eukaryotic cells, it thus appears that both cytoplasmic streaming and the formation of pseudopodia may depend on the interaction between myosin and actin filaments, similar to that found in muscle contraction. You will learn muscle contraction later in this unit.

It is not known how amoeboid movement is controlled or regulated. Amoebae cannot extend pseudopodia in all directions simultaneously and if they do they would be ripped apart! It has been shown however that the ability of many actin binding proteins to crosslink actin fibres is strongly dependant on both Ca^{2+} concentration and pH. Thus, Ca^{2+} and H^+ may regulate the sol-to-gel transition.

In the low molar concentration of Ca^{2+} (≈ ppm range), when pH is lowered to 6.8, the cytoplasm of amoeba sets as a gel. Conversely, solation of the gel is induced by raising the pH or Ca^{2+} concentration. Studies have implicated involvement of a **gelsolin** or **villin** protein in the gel-to-sol transition, because these proteins fragment actin filaments in the presence of micromolar concentration of (10^{-6} moles) Ca^{2+}. It has been suggested that directed growth of pseudopodia is due to differences in Ca^{2+} or H^+ concentration among various regions of the cytoplasm; whether this is the case, however remains to be determined. In the next section you will read about ciliary and flagellar movements.



6.3 CILIARY AND FLAGELLAR MOVEMENTS

Cilia and flagella or their derivatives occur in all animal phyla. They constitute the primary locomotory structures of many protozoa and of several kinds of metazoan cells, for example, sperm, ciliated epithelia of invertebrates and vertebrates. Derivatives of cilia occur in a wide variety of photoreceptor, mechanoreceptor and chemoreceptor cells.

Cilia and flagella have a similar internal structure; the difference lies in their beating patterns which are illustrated in Fig. 6.2. A flagellum like a tail of sperm, beats with a symmetrical undulation that is propagated as a wave along the flagellum (Fig. 6.2a). A cilium, in contrast beats asymmetrically with a fast or dash-like stroke in one direction, followed by a slower recovery motion in which the bending cilium returns to the original position (Fig. 6.2b). In flagellar motion, water is propelled parallel to the long axis of the flagellum; in ciliary motion water is propelled parallel to the surface that bears the cilia.

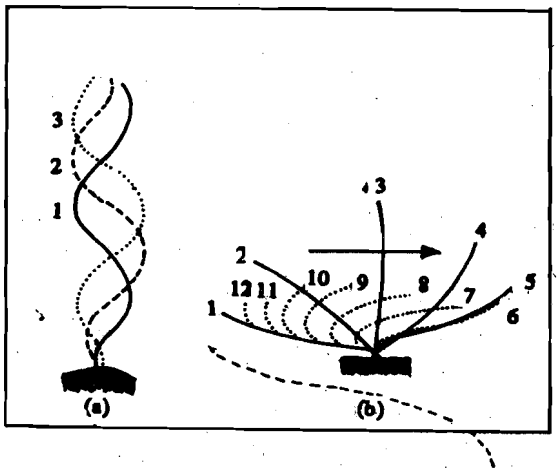


Fig. 6.2 : a) The typical beat of a flagellum propels water parallel to the main axis of the flagellum (arrow).
b) The beating of a cilium (right) propels water parallel to the surface to which the cilium is attached.

Structure of the Cilia and Flagella

Cilia and flagella are hair-like cell organelles. The diameter (0.2 μm) and internal structure of cilia and flagella is similar to each other but these structures differ in length. Cilia are generally less than 15 μm in length while flagella may be as long as 200 μm. The internal structure and the molecular composition of cilia and flagella have been well studied by electron microscopy and biochemical techniques. In Fig. 6.3 you can see that the covering membrane of the cilium or flagellum is continuous with the plasma membrane of the cell. It is actually the evagination of the plasma membrane. The cilia is attached to the body of the organism by a **basal body** or **kinetosome.**

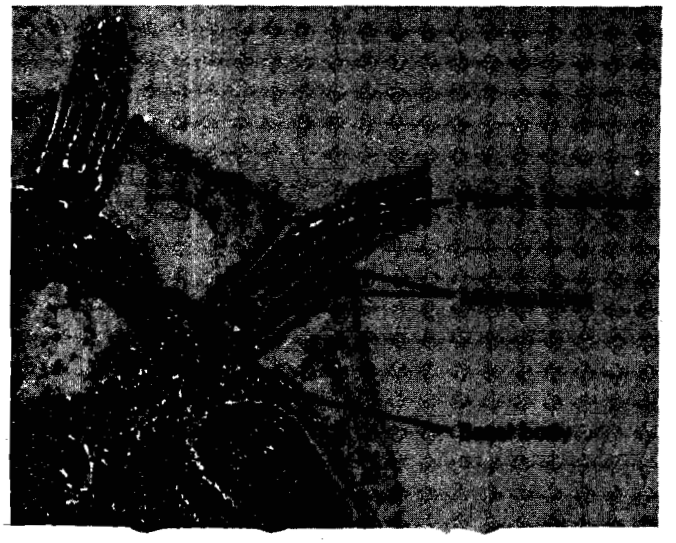


Fig. 6.3 : 'ectron micrograph of flagella showing the plasma membrane, microtubules and the basal body.

The main ı 'ernal structures of a cilium are **microtubules**, which extend from the base to the tip. The ıicrotubules are arranged in 9 + 2 configuration, consisting of nine outer doublets sun ınding two single central microtubules (Fig. 6.4).

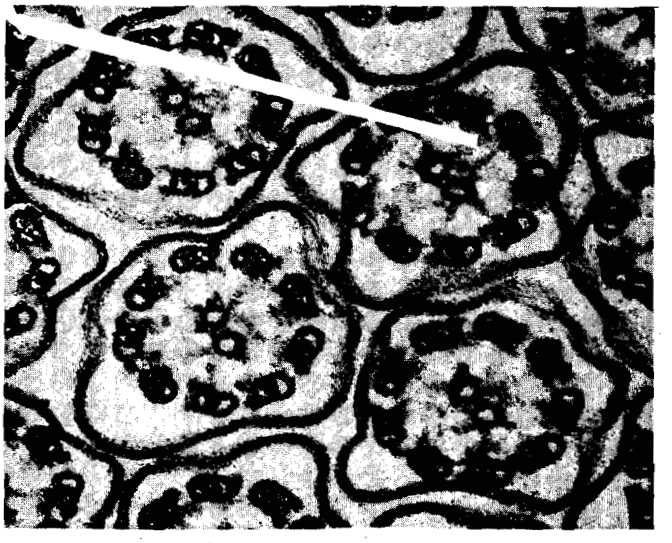


Fig. 6.4 : Cross-sections of the flagella showing the characteristic 9 + 2 arrangement of the filaments in each flagellum.

Each microtubule is a hollow cylinder composed of polymers of the globular proteins-**tubulins.** The outer doublets each consists of a complete tubule (the A tubule) with 13 subunits and an attached incomplete B tubule containing only 10 or 11 subunits (Fig. 6.5). Each A tubule bears two **side arms,** called **dynein arms** that project laterally towards the B tubule of the next doublet. There is a series of radial spokes which extend from the A sub-tubule to the central pair of microtubules. The outer doublets are connected circumferentially by **nexin links** (see Fig. 6.5). The entire array of microtubules and associated arms and links is called the **axoneme.** The nine peripheral doublets merge at the base to form a hollow tube that forms the basal body.

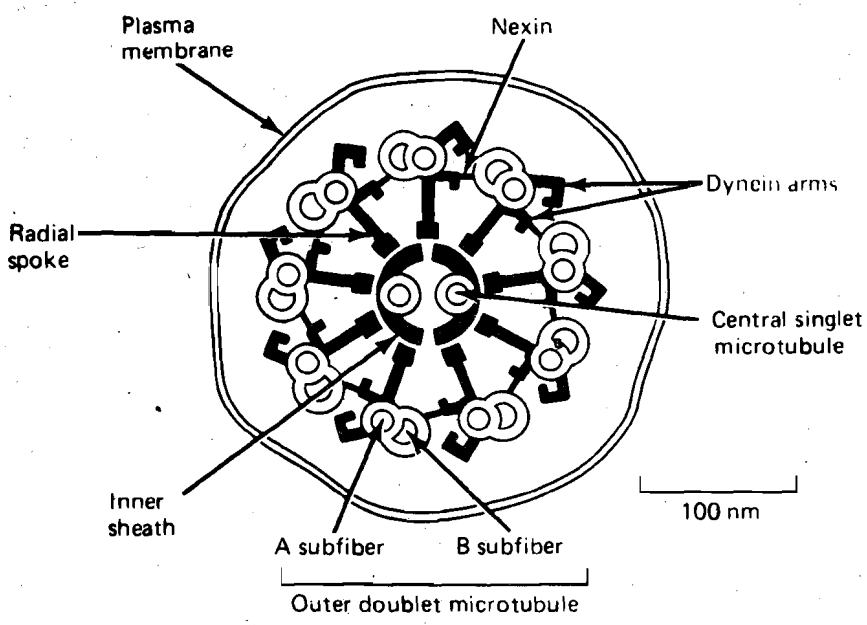


Fig. 6.5 : Diagram showing the internal structure of a cilium in cross-section.

Mechanism of Movement

Three types of mechanisms have been suggested to explain the mechanism of movement of cilia and flagella. These are :

i) the flagellum moves passively, much like a whip, by forces exerted at its base;

ii) the elements along the inner curvature of a propagating wave contract while the opposite side does not. Such a type of contraction takes places alternately on the inner curvature of a propagating wave on opposite side to bend the cilium or flagellum from side to side, and

iii) the thin filaments of the cilium do not change shape, but move past one another to produce a curvature, similar to the sliding filaments during muscle contraction (Fig. 6.6).

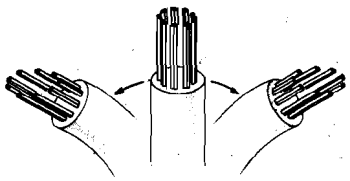


Fig. 6.6 : Diagram of sliding microtubules during beating of a cilium. The membrane of the distal portion of the cilium is removed, exposing the 9 outer doublets. In the straight cilium (center) all of the tubules end at the same point. During beating, the outer doublets slide past one another (left and right) causing a bending of the cilium and a displacement at the end of the tubules.

Electron microscopic studies demonstrate that bending of flagellum occurs when the extending dynein arms attach to the neighbouring B-tubule, inducing sliding movement. The dynein arms seem to "walk" along the cilium, presumably by the attachment of radial spokes to the central microtubule to constrain the sliding. The radial spokes and the nexin links are required to convert the sliding movement into typical bending of the cilium or the flagellum. The energy for the sliding movement is provided by the hydrolysis of ATP (Fig. 6.7).

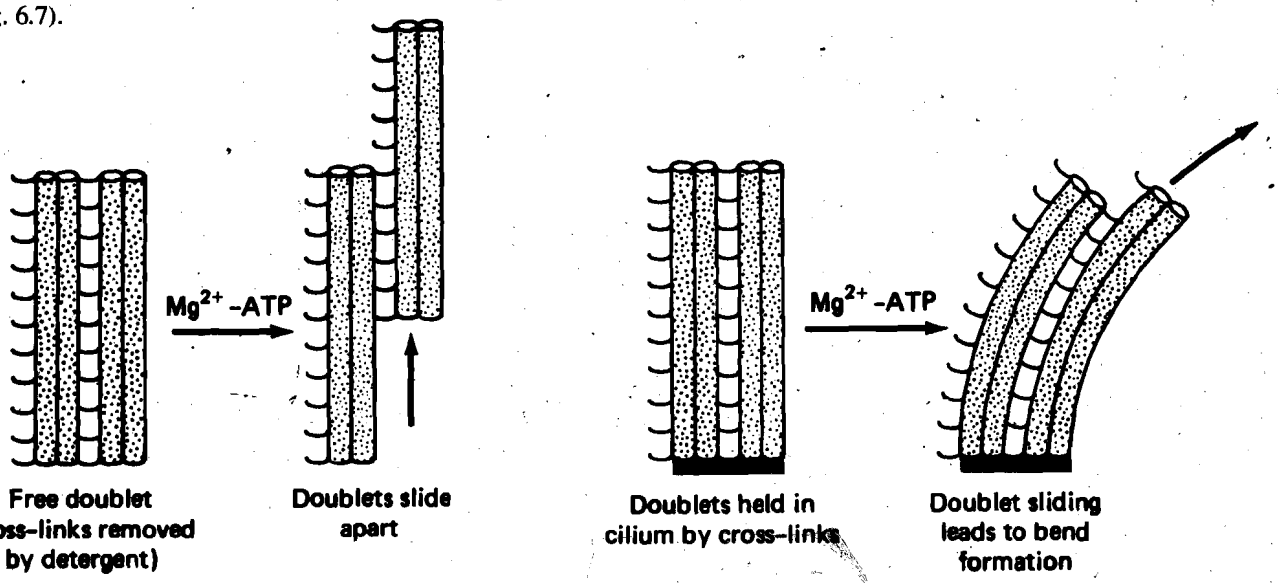


Fig. 6.7 : Diagram illustrating the experimental demonstration of sliding of outer microtubule doublets *in vitro* as well as the bending of the doublets if they are bound together at one end. The requirement of the Mg^{2+} and ATP is also shown.

6.4 MUSCLE AND MOVEMENTS

In the earlier section you have read about amoeboid movement, ciliary and flagellar movement. In this section you will learn how muscles are involved in the movement.

Muscle cells are found in almost all the phyla of the animal kingdom except the phylum protozoa. Contraction and relaxation of these muscles brings about movement in the organisms. In vertebrates there are three types of muscles : **skeletal** muscles, **cardiac** muscles and **smooth** muscles. Skeletal muscles are attached to the bones in the arms, legs and the spinal cord and produce activities such as walking, movement of head, hands, etc. Cardiac muscles are the muscles of the heart. These are specialised for continuous contractions of the heart, needed in pumping of the blood. Smooth muscles are present in the walls of internal organs such as the large and small intestine, the gall bladder and large blood vessels. Contraction and relaxation of smooth muscles control the diameter of blood vessels and also propel food along the gastro-intestinal tract. Under the microscope the skeletal muscles and the cardiac muscles exhibit transverse light and dark bands alternating with each other. Therefore, the skeletal muscles and the cardiac muscles are also called **striated muscles**. The smooth muscles do not have striations. You will read more about the structure of these three types of muscles in Sub-section 6.4.1.

Skeletal muscles are usually called **voluntary** because, the muscles of the limb and trunk are under control of the will.

Your leg or your hand will move only if you wish to move them. However, some movements like breathing do take place without our control. Smooth muscles are not under the control of the conscious mind, and therefore are called **involuntary**. Their contractions are usually slower than those of skeletal muscles and these movements normally take place without our knowledge. In the next subsection you shall learn about the structure of skeletal muscles.

6.4.1 Structure of Vertebrate Skeletal Muscles

Vertebrate skeletal muscles are composed of a large number of long, cylindrical and multinucleated cells, called **muscle fibres** arranged parallel to each other. The fibres contain longitudinally arranged elements called **myofilaments**. The myofilaments are organised into **myofibrils.** The fibres measure between 0.1 to 0.01 mm in diameter and several centimetres long. The myofibrils have characteristic cross striations called **Z-lines,** which are repeated at regular intervals. The region between two Z-lines is called a **sarcomere,** which is a functional unit of a myofibril. Thus a myofibril consists of longitudinally repeating sarcomeres. The Z-lines of adjacent myofibrils are lined up with each other, forming alternating A-bands and I-bands. There is a lighter region in the middle of the A-band called the H-Zone.These bands appear continuous for all the fibrils of a muscle fibre and it is this alignment of banding that gives the fibre its striated appearance (Fig. 6.8).

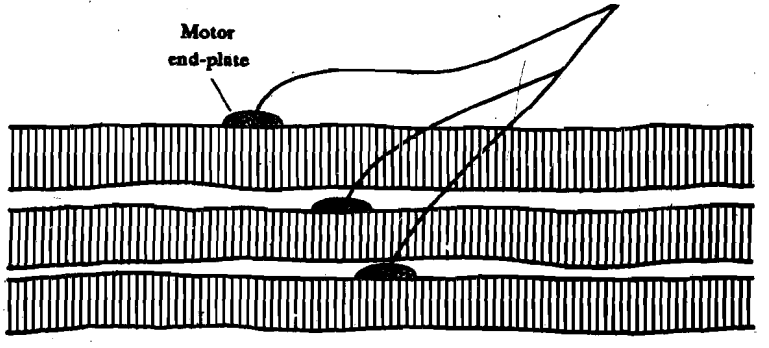


Fig. 6.8 : Schematic diagram of striated (skeletal) muscle and its components.

Myofibrils consist of two kinds of myofilaments. The **thick filaments** and the **thin filaments**. The thick filaments are made up of **myosin** and the thin filaments are of **actin** and the regulatory proteins **troponin** and **tropomysin**. The thick filaments are confined to A-band whereas the thin filaments extend from the Z-line and enter into A-band between thick filaments. The I-band contains only thin filaments. In the region of overlap between thick and thin filaments, the adjacent thick and thin filaments interact with each other by forming cross bridges. The cross bridges are projections of myosin molecules whose interaction with actin molecules generate the force for the muscle contraction.

The muscle fibre is surrounded by a cell membrane called **sarcolemma.** The sarcolemma connects with a complex system of transverse tubules, called **T-system** that runs across the muscle cells near the Z-lines. The T-tubules appear as invagination of the sarcolemma into the interior of the fibre. The muscle fibre is also surrounded by a sleeve-like structure called **sarcoplasmic reticulum,** which is involved in the initiation of muscle contraction (Fig 6.9).

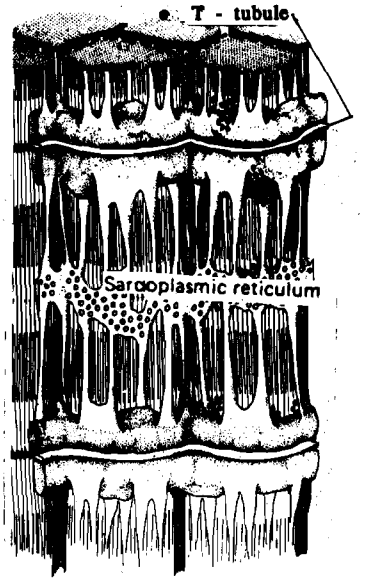


Fig. 6.9 : Shows the system of T-tubules and the sarcoplasmic reticulum that surrounds the striated muscles.

6.4.2 Mechanism of Muscle Contraction

Contraction is the function of a muscle. It is a physical activity generating force. It causes shortening of the muscle. The widths of the I-bands and H-zone both decrease but the width of the A-band remains constant. Apparently the lengths of the myosin and actin filaments also do not change. This shows that muscle shortens in contraction without either of the kinds of filaments changing length. Instead contraction of the muscle fibre is brought about by sliding of the filaments past each other in their region of overlap. These observations were first made in 1954 by two independent teams, H.E. Huxley and Hanson and A.F. Huxley and Niedergerke, and they proposed it as **"sliding filament model"** which has been experimentally confirmed. In Fig. 6.10 you can note the relation of the myofilaments during shortening of two sarcomers.

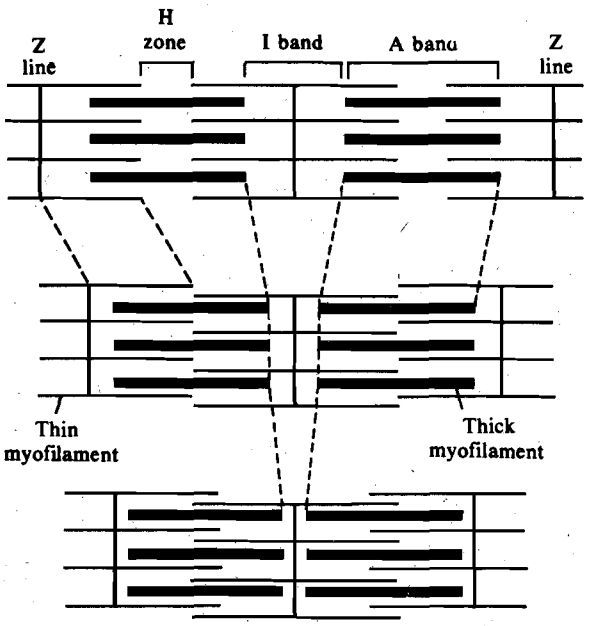


Fig. 6.10 : The sliding-filament hypothesis.

The process of sliding of thick and thin filaments past each other involves the cross bridges which extend from the thick filaments to contact the thin filaments. To understand how these cross bridges generate the force of contraction, it is necessary to study the molecular structure of actin and myosin filaments.

6.4.3 Molecular Basis of Muscle Contraction

You have studied in Sub-section 6.4.1 that the thick filaments are composed of protein myosin and the thin filaments contain primarily protein action. Myosin molecules are very large proteins, each consisting of a double-headed globular region joined to a long rod or tail (Fig. 6.11).

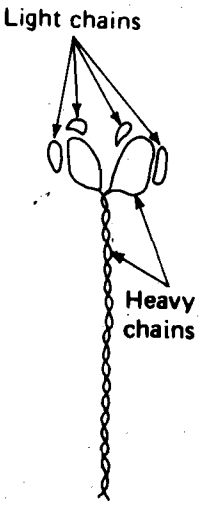


Fig. 6.11 : Myosin molecule : It consists of two heavy chains and four light chains.

As you see in Figure 6.11 that a myosin molecule contains two **heavy** chains; part of a heavy chain makes up one head and the other part extends the length of the tail. Myosin heads also contain four smaller **light** chains. Thus, each myosin molecule consists of six polypeptide chains. The tails of many myosin molecules together make up the thick filament. Whereas the globular heads project to the side forming the cross bridges.

The myosin molecules in a thick filament have their head-ends oriented towards the end of the filaments and their tails pointing towards the middle. As a result there is a short bare zone devoid of cross bridges at the middle of the thick filaments (Fig. 6.12).

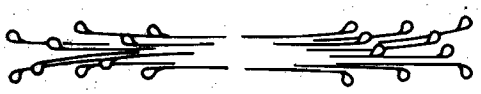


Fig. 6.12 : Polarity of myosin molecules in a thick filament.

The thin filaments which contain actin molecules have a different arrangement. The thin filaments contain two chains of actin molecules wound around each other in a helix (Fig. 6.13).

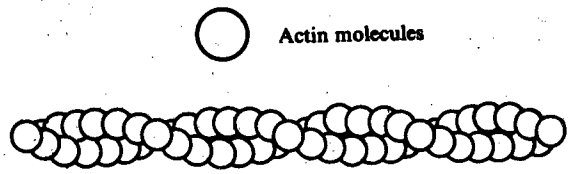


Fig. 6.13 : The thin filament.

Actin molecules of the thin filaments are also oriented in a specific manner. All the molecules on one side of the Z-line have one orientation and all those on the other side have the opposite polarity. Hence, the polarities of both the actin and myosin molecules are reversed on the opposite sides of the middle of a sarcomere. Fig. 6.14 illustrates arrangement of thick and thin filaments in a muscle fibre.

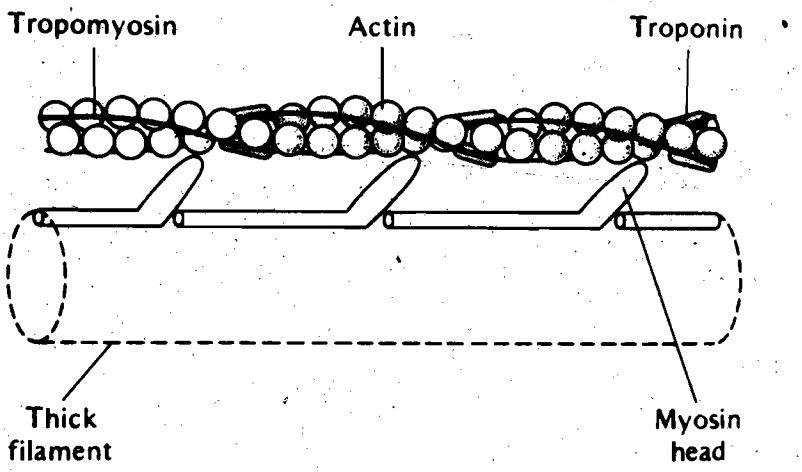


Fig. 6.14 : The thick filament consists of the protein myosin with its heads protruding towards the actin strands.

After learning about the molecular structure of both myosin and actin fibres, we shall now learn about the mechanism of muscle contraction. The immediate source of energy for a muscle contraction is adenosine triphosphate (ATP). The energy required to move myosin and actin filaments past each other comes from the binding and splitting of ATP by the globular heads of myosin molecules. These heads which form the cross bridges, cyclically attach to actin molecules and then swivel, acting as oars that pull the actin and myosin molecules past each other and thereby help in the sliding movement. The globular subunit of myosin has two active sites, one for actin and the other for ATP. In the cross bridging cycle, the globular head binds ATP and splits it to ADP + Pi in the presence of Mg^{2+}, but does not release the ADP and Pi. The energy released is stored in the myosin ADP complex. This complex then binds actin, forming actin-myosin-ADP-Pi-complex. In the next step, ADP and Pi are released by myosin and the myosin head changes the conformation, pulling the attached actin towards the middle of the myosin filament. The myosin head then binds to a new ATP, triggering its release from actin. Subsequently, the new ATP is hydrolysed, blocking the myosin head in position to bind another actin molecule (Fig. 6.15).

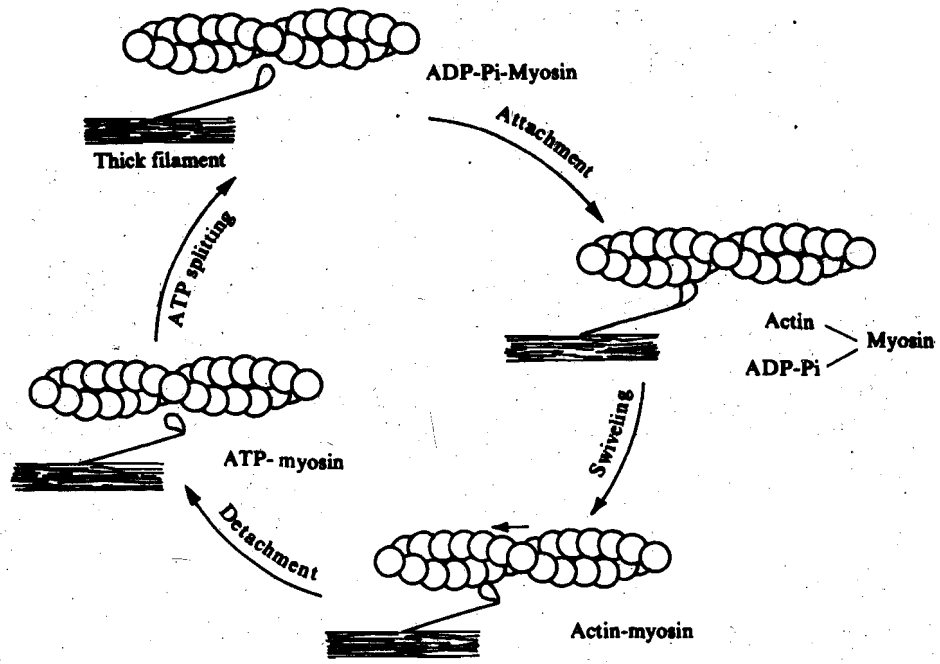


Fig. 6.15 : Proposed molecular events of a single cross bridge cycle. The thick and thin filaments move relative to each other by the swivelling action of the myosin head. The initial state in a relaxed muscle fibre is at the top of the figure.

6.4.4 Control of Contraction by Calcium and Regulatory Proteins

You have read in the earlier sections that the thin filaments of myofibril consist of actin and regulatory proteins, **troponin** and **tropomyosin**. Tropomyosin is a long protein coiled along the groove between the two chains of actin filament. Troponin is also found on the actin filaments. It is located at regular intervals along the actin filaments.

At the resting state of the muscle, tropomyosin prevents the interaction of myosin head with the actin filament by blocking the cross bridges binding sites of actin molecules (Fig. 6.16a). The troponin which acts as a controlling protein has a high affinity for calcium ion. It has a binding site for Ca^{2+}. When a muscle is stimulated, the calcium ion concentration within the muscle fibre rises abruptly; the calcium ions bind to troponin and bring about conformational changes in both troponin and tropomyosin molecules. This effect induces the movement of tropomyosin to uncover the binding sites and allows cross bridges of the myosin to bind to the actin filament. Thus, calcium ion acts as the physiological regulator of muscle contraction (Fig. 6.16b).

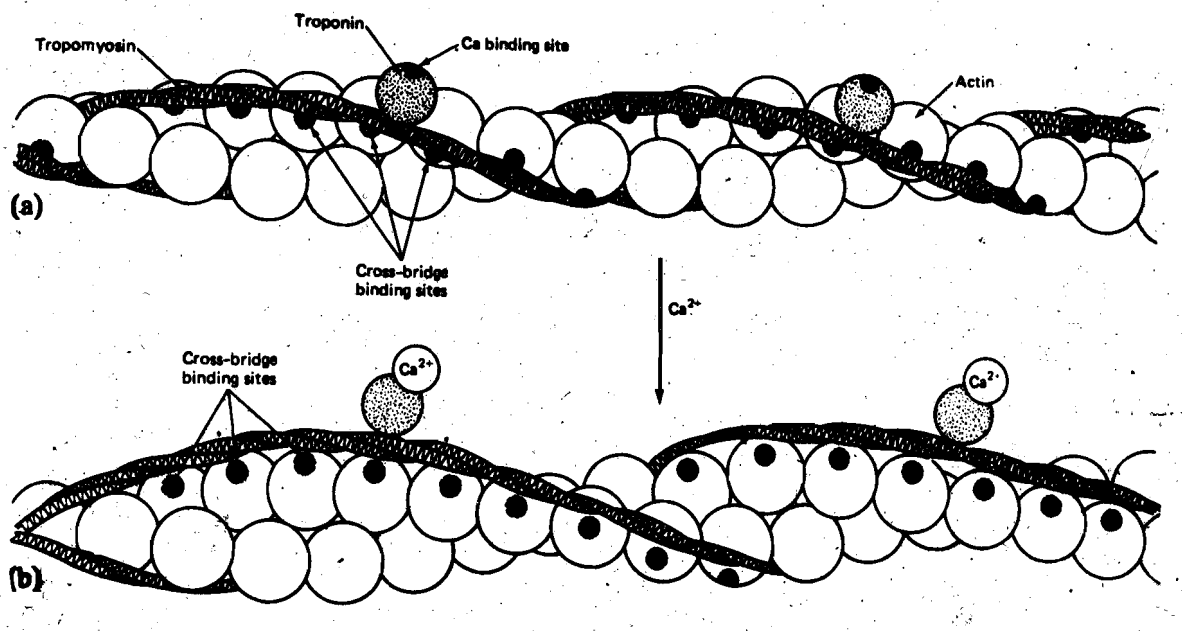


Fig. 6.16 : Actin-linked regulation of contraction in vertebrate skeletal muscle. In the absence of calcium ions, tropomyosin blocks the cross bridge binding sites of actin molecules. Calcium ions bind to troponin inducing the movement of tropomyosin to uncover the binding sites and allowing cross bridges to bind to the thin filaments.

6.4.5 Initiation of Muscle Contraction

After studying the mechanisms of muscle contraction you must be interested to know how muscle contraction is initiated.

Muscle contraction is stimulated by nerve impulse. Muscles are associated with nerve endings. The junction of the nerve end and the muscle is called **neuromuscular junction** or **motor end plate.**

You have read in the earlier section that contraction of the muscle is triggered by the presence of Ca^{2+} that bind to troponin. Ca^{2+} are stored in the sarcoplasmic reticulum.

Nerve impulse produces depolarisation of the sarcolemma which propagates rapidly over the entire surface of the fibre and also propagates along T-tubular membranes into the interior fibre. This depolarisation is referred to as **excitation** of the fibre. In resting muscle Ca^{2+} is largely confined to the lateral sacs of the sarcoplasmic reticulum. Depolarisation of tubules induces release of Ca^{2+} from the sarcoplasmic reticulum. Ca^{2+} rapidly diffuses to the adjacent myofilament and bind to troponin. Binding of Ca^{2+} to troponin changes the conformation of both troponin and tropomyosin molecules exposing the myosin binding sites of the actin filament. The globular heads of the myosin molecules then bind to the myosin binding sites on the actin forming cross bridges. The cross bridges move thick and the thin filaments relative to each other resulting in contraction of the fibre. Relaxation of the muscle fibre results from the recovery of Ca^{2+} back into the sarcoplasmic reticulum. This is done by an ATP dependent Ca^{2+} pump. Decrease in the concentration of Ca^{2+} dissociates Ca^{2+} from troponin and then tropomyosin inhibits contraction (Fig. 6.17).

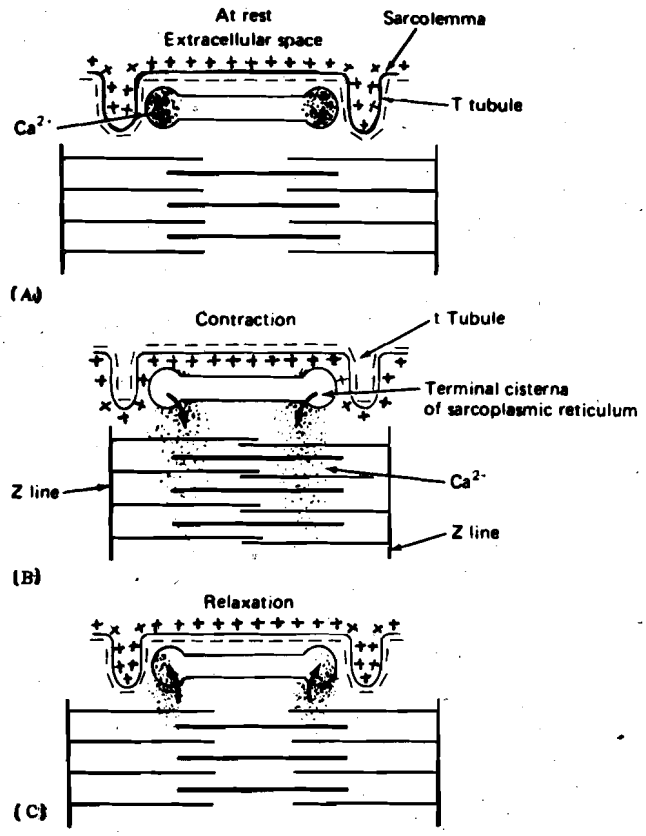


Fig. 6.17 : Diagram showing how calcium release and recovery control the sliding of actin and myosin filaments. (A) T-tubules continuous with the sarcolemma (plama membrane) penetrate the interior of the muscle fibre. (B) When action potentials pass down the T-tubules, calcium ions are released from terminal cisternae of the sarcoplasmic reticulum. Calcium permits cross bridge interaction and sliding of actin and myosin filaments. (C) Relaxation results from the recovery of calcium ions by active transport back into the terminal cisternae.

6.4.6 Energetics of Muscle Contraction

You have studied in the above sections that ATP is the immediate source for muscle contraction. It is required for the process of relaxation also. Muscle contains only enough ATP to sustain contraction for a few seconds. You have read in Units 10 and 11 of LSE-01;

Cell Biology that **glycolysis** and **oxidative phosphorylation** are the source of ATP, but these multistep pathways do not increase their rates immediately to supply ATP for muscle contraction. ATP required for the muscle contraction is generated immediately from one of the two energy rich compounds **creatinine phosphates** in vertebrates and **arginine phosphate** in invertebrates. These energy rich compounds are known as **phosphogens.** In vertebrate muscle creatinine phosphate rephosphorylates ADP in a reversible reaction.

Creatinin Phosphate + ADP $\rightleftharpoons$ Creatinin + ATP

If ATP becomes depleted, the muscle is unable to keep contracting and this process is known as **muscle fatigue**. Fig. 6.18 illustrates biochemical pathways that produce ATP **during vertebrate muscle contraction.**

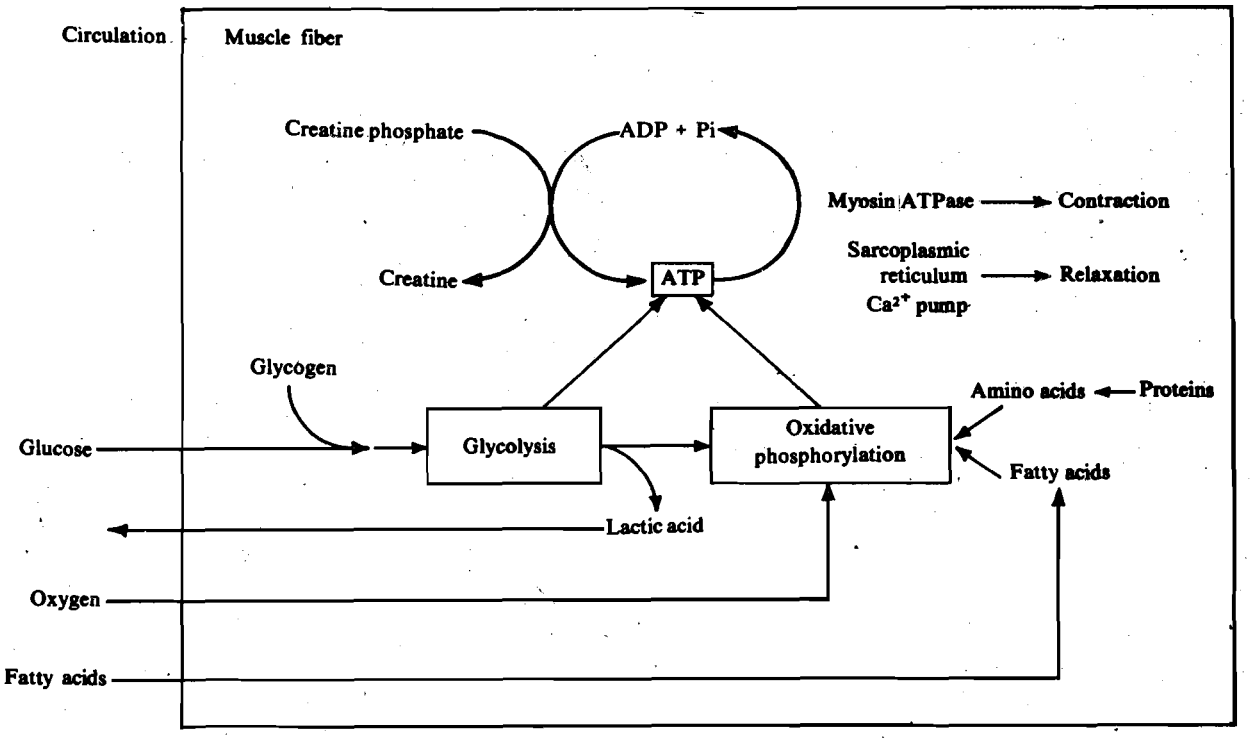
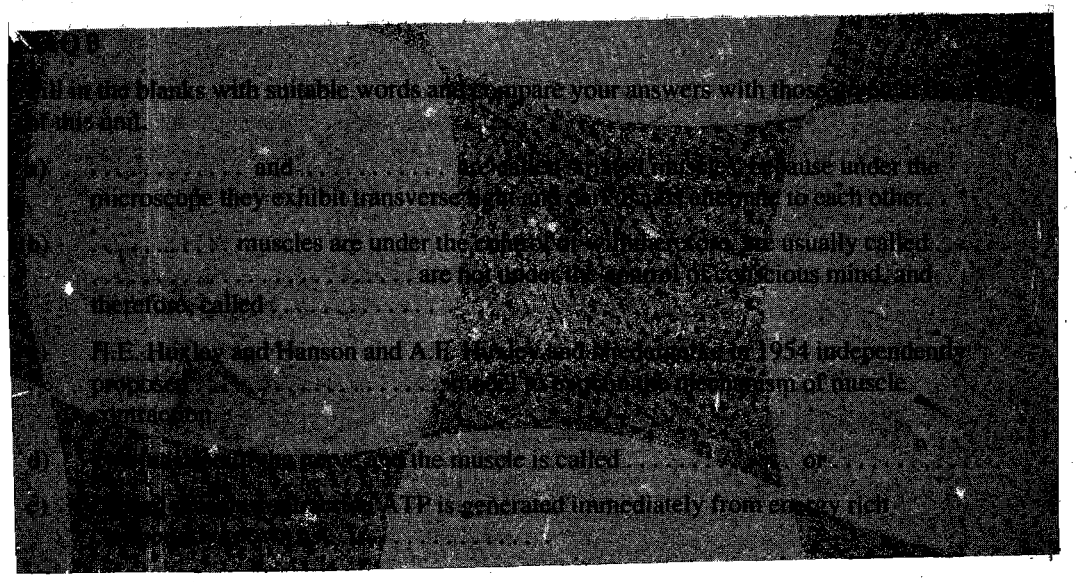


Fig. 6.18 : Biochemical pathways producing ATP utilised during vertebrate muscle contraction.



6.5 CARDIAC MUSCLES AND SMOOTH MUSCLES

In the earlier section you have read about the structure and function of skeletal muscles. In this section you will read about vertebrate cardiac muscles and smooth muscles.

Cardiac Muscles

Cardiac muscles exhibit cross-banded appearance under the microscope similar to the skeletal muscles. Therefore, these are also called striated muscles, but the striations are not aligned as found in the skeletal muscles. Therefore, the striated appearance is less distinct in them. These also contain actin and myosin filaments. Cardiac muscle fibres are smaller in size than the skeletal muscle fibres. They are mononucleated but contain abundant mitochondria. Sarcoplasmic reticulum and T- tubules may be well developed or absent (Fig. 6.19)

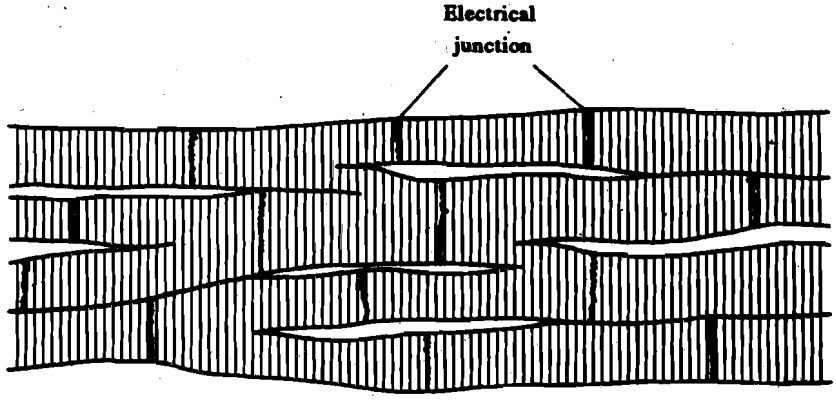


Fig. 6.19 : Cardiac Muscles.

The functional properties of cardiac muscle differ from the skeletal muscle in two important respects. One is that, when a contraction starts in one area of the heart muscle mass, it rapidly spreads throughout the muscle mass. During contraction the cell membrance of the cardiac muscle fibres undergoes electric changes, known as **action potentials.** The second peculiarity of the heart muscle is that, the cell membrane after completion of an action potential, remains in a refractory state for a long enough time to allow the muscle to relax. Because of this refractory period, the cardiac muscle cannot go into a sustained contraction. The refractory period is thus essential for the alternation between contraction and relaxation vis-a-vis normal rhythmic contraction of the heart.

Smooth Muscles

Smooth muscles do not have transverse striations like those of skeletal and cardiac muscles. You have already studied in Section 6.4 that smooth muscles line the blood vessels, digestive tract, urinary bladder, uterus etc. These are also present in the iris and skin.

Smooth muscles are small and spindle shaped. Each muscle has a single nucleus. The cytoplasm of the smooth muscle cells contains actin and myosin filaments arranged in a random manner. Despite the lack of internal organisation of the filaments, there is cross bridge formation between the actin and myosin filaments. The contraction mechanism in smooth muscles is similar to the sliding filament type of contraction as found in the skeletal muscles (Fig. 6.20).

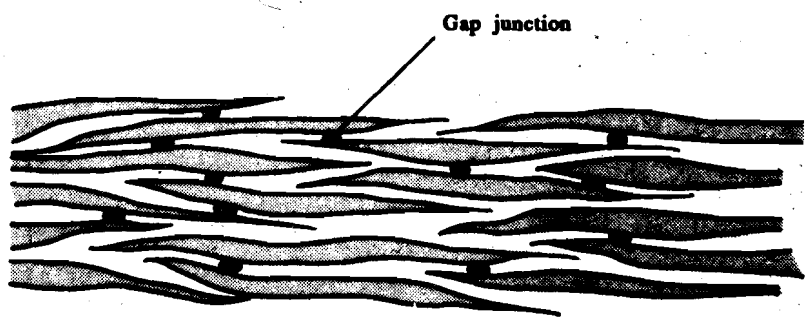


Fig. 6.20 : Smooth Muscles.

In order to contract, smooth muscle need not be stimulated through the nerves. They show spontaneous rhythmic contractions that can vary in both frequency and intensity. This spontaneous activity of smooth muscle, however, can be modified by nerves as well as by hormones such as **epinephrine** and **norepinephrine.** A distinctive feature of vertebrate

smooth muscle is the slowness of response. Another important property of it is that the smooth muscle can maintain contraction for prolonged periods with very little energy expenditure.

6.6 SUMMARY

In this unit you have read about the physiology of movement in animals. You have read that :

- movement in amoeba is due to cytoplasmic streaming, change in the cell shape and extension of pseudopodia,
- cilia and flagella have a similar internal structure consisting of microtubules in 9+2 configuration. The filaments of the cilium move past one another to produce movement similar to the sliding filaments of muscle contraction,
- in vertebrates, there are three types of muscles: skeletal muscles, cardiac muscles and smooth muscles. Skeletal muscles and cardiac muscles exhibit transverse light and dark band alternating with each other. Therefore, they are known as striated muscles. Smooth muscles do not exhibit striations,
- contraction of the muscle fibre is brought about by sliding of actin and myosin filaments past each other. This is brought about by formation of cross bridges by the globular heads of the myosin filament with the actin filament which then swivel,
- ATP is the immediate source of energy for muscle contraction,
- calcium, troponin and tropomyosin regulate contraction of the muscle.

6.7 TERMINAL QUESTIONS

1) Explain briefly in the space given below the mechanism of movement of cilia.

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2) Explain briefly in the space given below the role of myosin in muscle contraction.

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3) Explain briefly in the space given below the role of calcium in the regulation of muscle contraction.

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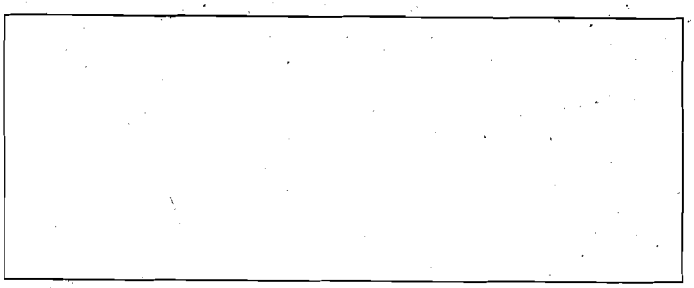
4) Illustrate the biochemical pathways that produce ATP during vertebrate muscle contraction.



6.8 ANSWERS

Self-assessment Questions

1) a) Protozoans, slime molds, white blood cells
   b) Pseudopodia
   c) ectoplasm, endoplasm
2) a) kinetosome
   b) 9 + 2
   c) A, outer doublets
3) a) striated muscles, cardiac
   b) skeletal muscles, voluntary, smooth muscle, involuntary
   c) sliding filament model
   d) neuromuscular junction or motor end-plate
   e) phosphogens

Terminal Questions

1) Electron miscroscopic studies demonstrated that in order to move, the filaments of the cilium do not change shape, but move past one another to produce a curvature, similar to the sliding filaments of muscle contraction. This is done by attatchment of the dynein arms to the neighbouring tubule and walk along it, inducing sliding movement.
2) A myosin molecule contains two heavy chains; part of a heavy chain makes up one head and the other part forms the tail. The heads of the myosin molecules form cross bridges with the actin molecules. These cross bridges cyclically attatch to actin molecules and swivel, acting as oars that pull the actin and myosin filaments past each other affecting sliding movement.
3) The thin filaments of the myofibril consist of actin and two regulatory proteins, troponin and tropomyosin. In a resting state of the muscle, tropomyosin blocks the cross bridges binding sites of the actin molecules. When a muscle is stimulated the calcium ion concentration of the muscle fibre increases. The calcium ions bind to the troponin molecules. Binding of calcium ions to the troponin causes conformational changes in both troponin and tropomyosin, thereby uncovering the cross bridges binding sites of the actin molecules, affecting muscle contraction.
4) Please refer Fig. 6.16.