AS-2866

B.Sc. (Hon's) (Fifth Semester) examination, 2013

ZOOLOGY

Paper: LZC-502

(Animal Physiology)

Maximum marks: 30

Section-A **10×1=10**

(Multiple choice questions)

Note: Question no. 1 is compulsory. Each carries equal 1 mark.

1. Choose the correct answer:

(i). **(b) Myosin**

(ii). **(c) M Line**

(iii). **(d) Inspiratory capacity**

(iv). **(d) Oxygen**

(v). **(a) Active transport**

(vi). **(a) Salivary juice and pancreatic juice**

(vii). **(d) Renal pyramid, renal cortex and renal column**

(viii). **(b) Nephron**

(ix). **(a) Spinal cord**

(x). **(c) Signal propagation**

Question No 213

Absorption in the Small Intestine

All the chemical and mechanical phases of digestion from the mouth through the small intestine are directed toward changing food into forms that can pass through the absorptive epithelial cells lining the mucosa and into the underlying blood and lymphatic vessels. These forms are monosaccharides (glucose, fructose, and galactose) from carbohydrates; single amino acids, dipeptides, and tripeptides from proteins; and fatty acids, glycerol, and monoglycerides from triglycerides. Passage of these digested nutrients from the gastrointestinal tract into the blood or lymph is called **absorption.**

Absorption of materials occurs via diffusion, facilitated diffusion, osmosis, and active transport. About 90% of all absorption of nutrients occurs in the small intestine; the other 10% occurs in the stomach and large intestine. Any undigested or unabsorbed material left in the small intestine passes on to the large intestine.

2a

Absorption of Monosaccharides

All carbohydrates are absorbed as monosaccharides. The capacity of the small intestine to absorb monosaccharides is huge—an estimated 120 grams per hour. As a result, all dietary carbohydrates that are digested normally are absorbed, leaving only indigestible cellulose and fibers in the feces. Monosaccharides pass from the lumen through the apical membrane via *facilitated diffusion* or *active transport.* Fructose, a monosaccharide found in fruits, is transported via *facilitated diffusion;* glucose and galactose are transported into absorptive cells of the villi via *secondary active transport* that is coupled to the active transport of Na^+ (Figure 24.20a). The transporter has binding sites for one glucose molecule and two sodium ions; unless all three sites are filled, neither substance is transported. Galactose competes with glucose to ride the same transporter. (Because both Na^+ and glucose or galactose move in the same direction, this is a *symporter.* Monosaccharides then move out of the absorptive cells through their basolateral surfaces via *facilitated diffusion* and enter the capillaries of the villi) (see Figure 24.20b).

2b

Absorption of Amino Acids, Dipeptides, and Tripeptides

Most proteins are absorbed as amino acids via *active transport* processes that occur mainly in the duodenum and jejunum. About half of the absorbed amino acids are present in food; the other half come from the body itself as proteins in digestive juices and dead cells that slough off the mucosal surface! Normally, 95–98% of the protein present in the small intestine is digested and absorbed. Different transporters carry different types of amino acids. Some amino acids enter absorptive cells of the villi via Na^+-dependent secondary active transport processes that are similar to the glucose transporter; other amino acids are actively transported by themselves. At least one symporter brings in dipeptides and tripeptides together with H^+; the peptides then are hydrolyzed to single amino acids inside the absorptive cells. Amino acids move out of the absorptive cells via diffusion and enter capillaries of the villus (Figure 24.20a, b). Both monosaccharides and amino acids are transported in the blood to the liver by way of the hepatic portal system. If not removed by hepatocytes, they enter the general circulation.

3

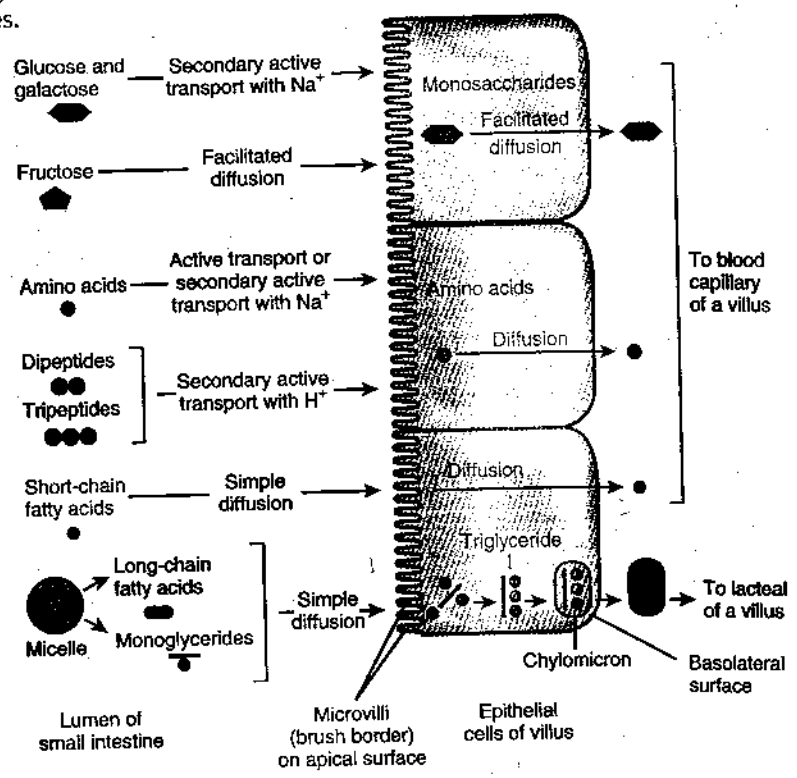
Absorption of Lipids

All dietary lipids are absorbed via *simple diffusion.* Adults absorb about 95% of the lipids present in the small intestine; due to their lower production of bile, newborn infants absorb only about 85% of lipids. As a result of their emulsification and digestion, triglycerides are mainly broken down into monoglycerides and fatty acids, which can be either short-chain fatty acids or long-chain fatty acids. Although short-chain fatty acids are hydrophobic, they are very small in size. Because of their size, they can dissolve in the watery intestinal chyme, pass through the absorptive cells via simple diffusion, and follow the same route taken by monosaccharides and amino acids into a blood capillary of a villus (Figure 24.20a). Long-chain fatty acids and monoglycerides are large and hydrophobic and have difficulty being suspended in the watery environment of the intestinal chyme. Besides their role in emulsification, bile salts also help to make these long-chain fatty acids and monoglycerides more soluble. The bile salts in intestinal chyme surround the long-chain fatty acids and monoglycerides, forming tiny spheres called **micelles** (mī-SELZ = small morsels), each of which is 2–10 nm in diameter and includes 20–50 bile salt molecules (Figure 24.20a). Micelles are formed due to the amphipathic nature of bile salts: The hydrophobic regions of bile salts interact with the long-chain fatty acids and monoglycerides, and the hydrophilic regions of bile salts interact with the watery intestinal chyme. Once formed, the micelles move from the interior of the small intestinal lumen to the brush border of the absorptive cells. At that point, the long-chain fatty acids and monoglycerides diffuse out of the micelles into the absorptive cells, leaving the micelles behind in the chyme. The micelles continually repeat this ferrying function as they move from the brush border back through the chyme to the interior of the small intestinal lumen to pick up more long-chain fatty acids and monoglycerides. Micelles also solubilize other large hydrophobic molecules such as fat-soluble vitamins (A, D, E, and K) and cholesterol that may be present in intestinal chyme, and aid in their absorption. These fat-soluble vitamins and cholesterol molecules are packed in the micelles along with the long-chain fatty acids and monoglycerides.

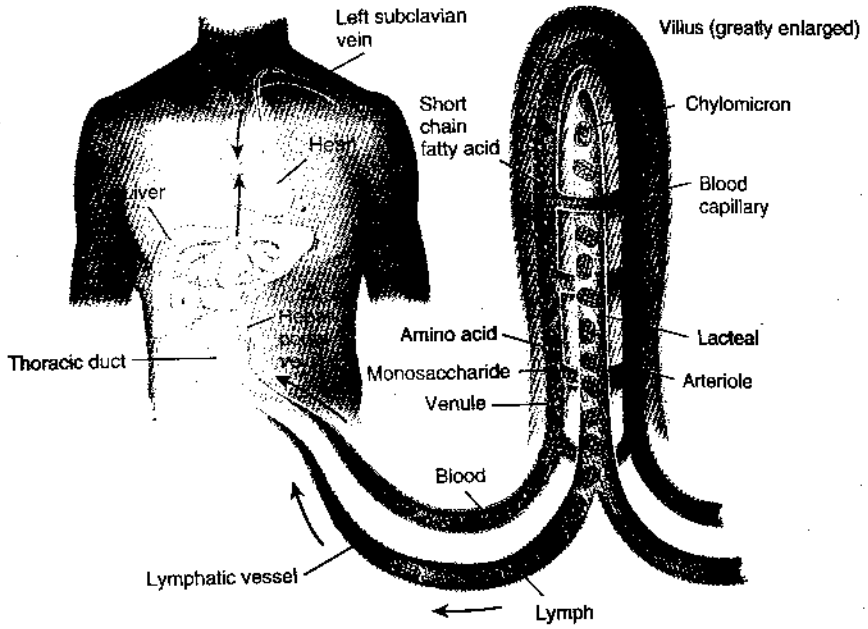
Once inside the absorptive cells, long-chain fatty acids and monoglycerides are recombined to form triglycerides, which aggregate into globules along with phospholipids and cholesterol and become coated with proteins. These large spherical masses, about 80 nm in diameter, are called **chylomicrons.** Chylomicrons leave the absorptive cell via exocytosis. Because they are so large and bulky, chylomicrons cannot enter blood capillaries—the pores in the walls of blood capillaries are too small. Instead, chylomicrons enter lacteals, which have much larger pores than blood capillaries. From lacteals, chylomicrons are transported by way of lymphatic vessels to the thoracic duct and enter the blood at the left subclavian vein

Figure 24.20 Absorption of digested nutrients in the small intestine. For simplicity, all digested foods are shown in the lumen of the small intestine, even though some nutrients are digested by brush-border enzymes.

Long-chain fatty acids and monoglycerides are absorbed into lacteals; other products of digestion enter blood capillaries.



(a) Mechanisms for movement of nutrients through absorptive epithelial cells of the villi



(b) Movement of absorbed nutrients into the blood and lymph

? A monoglyceride may be larger than an amino acid. Why can monoglycerides be absorbed by simple diffusion, but amino acids cannot?

Definition

In biochemistry, a **buffer** is that substance, **which resists or prevents any change in hydrogen ion (H^+ ion) concentration of the medium, in which it is present and maintains a relatively constant** *p*H. A buffer is usually a mixture of a weak acid with its salt of strong base or weak base with its salt of strong acid.

Buffer System in the Body

Different biological reactions within the body of all living organisms, are completed at different *p*H. Therefore, there exists variation in *p*H in different parts of the body in an organism. But *p*H in a specific part of the body is quite constant. To maintain a constant *p*H different parts of body possess different buffer systems. These are :—

1. **Blood Buffers**
   - A. **Blood Buffers in Plasma**
   1. Carbonic acid-bicarbonate system ($H_2CO_3/NaHCO_3$)
   2. Inorganic phosphate buffer system (Na_2HPO_4/NaH_2PO_4)
   3. Plasma proteins
   - B **Blood buffers in R. B. C.**
   1. Haemoglobin—(i) Reduced haemoglobin
      HHb (acidic)
      KHb (alkaline)
      (ii) Oxyhaemoglobin
      $HHbO_2$
      $KHbO_2$
   2. Bicarbonates
   3. Phosphates (both organic and inorganic phosphates)
2. **Muscle Buffers**
   1. **Proteins** of the muscles.
   2. **Creatin** acts as a strong base and remains combined with phosphoric acid.
   3. **Ammonia** liberated from adenylic acid.

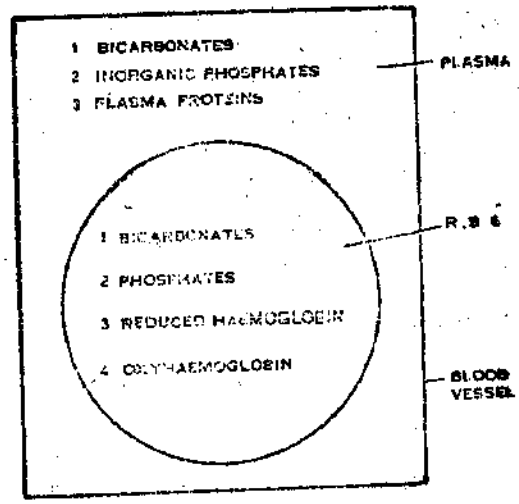


Fig. 9·1. Blood buffers.

3. **Urine Buffers**
   1. Carbonic acid-bicarbonate
   2. Dibasic phosphate
   3. Phosphoric acid-phosphate

Buffer Activity

1. **Bicarbonate buffers—Carbonic acid and sodium bicarbonate** form the most important buffer system. Carbonic acid is weak acid. It ionizes according to the following equations :

$$H_2CO_3 \rightleftharpoons H^+ + HCO_3^-$$

In any solution, at a given *p*H (H^+ ion concentration) an equilibrium is established between the undissociated molecules of the acid and its dissociated ions. If some sodium bicarbonate ($NaHCO_3$) is added to this solution, it ionizes as follows :

$$NaHCO_3 \rightarrow Na^+ + HCO_3$$

$NaHCO_3$ is a salt of strong base and ionizes completely, increasing the concentration of HCO_3^- ions in the solution. To maintain the original *p*H and HCO_3^- ion concentration, the excess of HCO_3^- ions combines with H^+ ions forming undissociated H_2CO_3 molecules.

At new equilibrium, the ionic balance is established according to the combined equations :

$$H_2CO_3 \rightleftharpoons H^+ + HCO_3^-$$
$$NaHCO_3 \rightleftharpoons Na^+ + HCO_3^-$$

1. Acts as alkali buffer—If a strong acid (HCl) is added to this buffer system, its dissociation will increase H^+ ion concentra-

tion (acidity) of the medium as follows :

$$HCl \rightleftharpoons H^+ + Cl^-$$

The bicarbonate ions of the buffer system remove excess of H^+ ions from the medium.

$$HCO_3^- + H^+ \rightarrow \underset{\text{undissociated}}{H_2CO_3}$$

More bicarbonates are added by the ionization of $NaHCO_3$.

$$NaHCO_3 \rightleftharpoons Na^+ + HCO_3^-$$

Thus the bicarbonate buffer system resists the change in the *pH* of medium and maintains a constant H^+ ion concentration in the medium.

2. **Acts as acid buffer**—If **strong base** (NH_4OH) is added to this buffer system it adds OH^- ions. The OH^- ions immediately combine with H^+ ions in the medium. The removal of H^+ ions

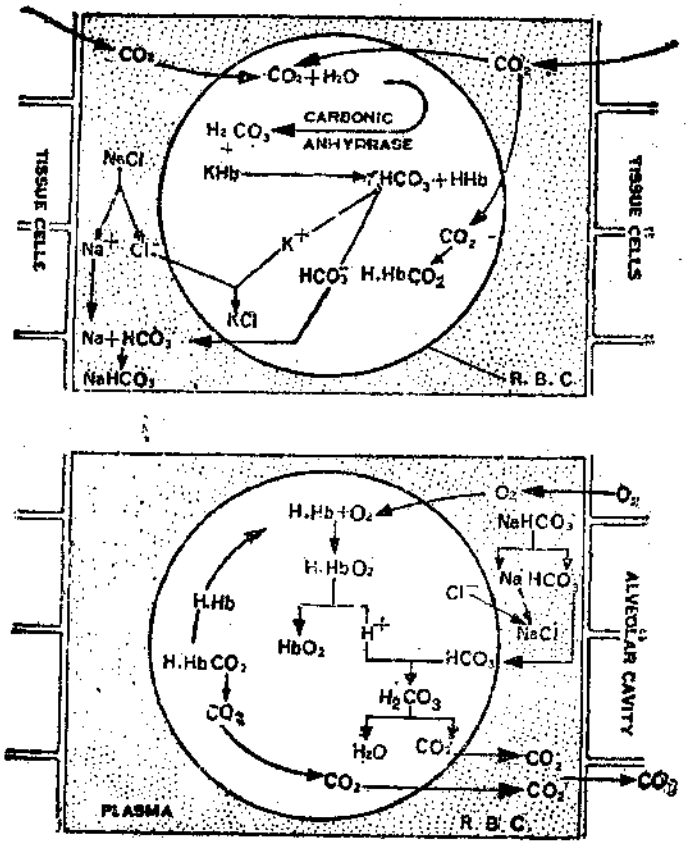


Fig. 7·4. Chloride shift and maintenance of constant *p*H.

cause more H_2CO_3 to ionize. The ionization of H_2CO_3 releases free H^+ ions to restore the original concentration of H^+ ions.

$$NH_4OH \rightleftharpoons NH_4^+ + OH^-$$

$$H^+ + OH^- \rightleftharpoons HOH$$

$$H_2CO_3 \rightleftharpoons H^+ + (HCO_3)^-$$

Thus carbonic acid resists the alkalinity of the medium and tries to maintain a constant *p*H.

A. Blood Buffers

1. **Bicarbonate buffers** ($NaHCO_3/H_2CO_3$)—These are called alkali reserves of plasma. Their functioning has already been discussed.

2. **Phosphate buffers** (Na_2HPO_4/NaH_2PO_4)—Phosphates present in blood are **alkaline phosphate** (Na_2HPO_4) and **acid phosphate** (NaH_2PO_4). When acid enters the blood Na_2HPO_4 ionizes further to remove excess of H^+ ions :—

$$\text{Lactic acid} + Na_2HPO_4 \rightleftharpoons \text{Na-lactate} + NaH_2PO_4$$

Similarly, when an alkali enters the blood, it is buffered by acid phosphate NaH_2PO_4.

3. **Haemoglobin**—Haemoglobin is one of the most important buffers of the blood. Each molecule of haemoglobin contains 38 molecules of histidine. Histidine molecules of haemoglobin contain **imidazole**. The imidazole contains **an imidazole nitrogen group** It dissociates in the acidic medium and conjugates in alkaline medium.

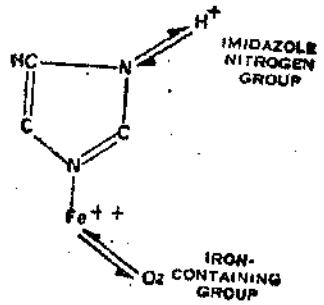


Fig. 7·5. Imidazol group in the histidine of haemoglobin.

When CO_2 enters the blood stream, H_2CO_3 is formed largely inside the RBCs due to the presence of carbonic anhydrase. This increases acidity. The alkaline reduced haemoglobin-KHb reacts with H_2CO_3 producing $KHCO_3$ and HHb —acid reduced haemoglobin :—

$$KHb + H_2CO_3 \rightarrow KHCO_3 + HHb$$

$KHCO_3$ is a salt of strong base and ionizes into $K^+ + HCO_3^-$ ions. Thus concentration of HCO_3^- ions in RBCs increase and it tends to become alkaline. The Cl^- ions from the blood plasma diffuse into the RBC and combine with K^+ ions to form KCl, HCO_3 ions from RBC diffuse out into the plasma, where these combine with Na^+ and form $NaHCO_3$.

4. **Plasma Proteins**—Proteins contain large number of acidic and base groups due to the presence of COOH and NH_2 groups. In acidic medium their basic NH_2^+ groups combine with H^+ ions and in alkaline medium, the acidic COOH—groups give H^+ ions and maintain a constant *p*H.

Question No (5)

PULMONARY VENTILATION

OBJECTIVE

The process of gas exchange in the body, called **respiration,** has three basic steps:

1. **Pulmonary ventilation** (*pulmon-* = lung), or **breathing,** is the inhalation (inflow) and exhalation (outflow) of air and involves the exchange of air between the atmosphere and the alveoli of the lungs.
2. **External (pulmonary) respiration** is the exchange of gases between the alveoli of the lungs and the blood in pulmonary capillaries across the respiratory membrane. In this process, pulmonary capillary blood gains O_2 and loses CO_2.
3. **Internal (tissue) respiration** is the exchange of gases between blood in systemic capillaries and tissue cells. In this step the blood loses O_2 and gains CO_2. Within cells, the metabolic reactions that consume O_2 and give off CO_2 during the production of ATP are termed *cellular respiration* (discussed in Chapter 25).

In pulmonary ventilation, air flows between the atmosphere and the alveoli of the lungs because of alternating pressure differences created by contraction and relaxation of respiratory muscles. The rate of airflow and the amount of effort needed for breathing is also influenced by alveolar surface tension, compliance of the lungs, and airway resistance.

Pressure Changes During Pulmonary Ventilation

Air moves into the lungs when the air pressure inside the lungs is less than the air pressure in the atmosphere. Air moves out of the lungs when the air pressure inside the lungs is greater than the air pressure in the atmosphere.

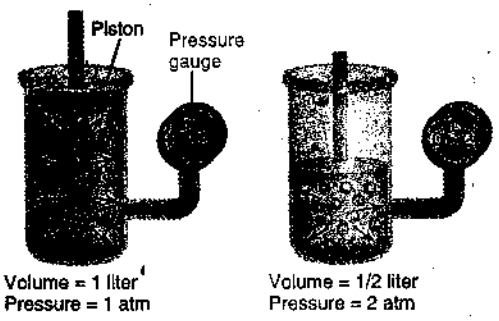
Inhalation

Breathing in is called **inhalation (inspiration).** Just before each inhalation, the air pressure inside the lungs is equal to the air pressure of the atmosphere, which at sea level is about 760 millimeters of mercury (mmHg), or 1 atmosphere (atm). For air to flow into the lungs, the pressure inside the alveoli must become lower than the atmospheric pressure. This condition is achieved by increasing the size of the lungs.

The pressure of a gas in a closed container is inversely proportional to the volume of the container. This means that if the size of a closed container is increased, the pressure of the gas inside the container decreases, and that if the size of the container is decreased, then the pressure inside it increases. This inverse relationship between volume and pressure, called **Boyle's law,** may be demonstrated as follows (Figure 23.12): Suppose we place a gas in a cylinder that has a movable piston and a pressure gauge, and that the initial pressure created by the gas molecules striking the wall of the container is 1 atm. If the piston is pushed down, the gas is compressed into a smaller volume, so that the same number of gas molecules strike less wall area. The gauge shows that the pressure doubles as the gas is compressed to half its original volume. In other words, the same number of molecules in half the volume produces twice the pressure. Conversely, if the piston is raised to increase the volume, the pressure decreases. Thus, the pressure of a gas varies inversely with volume.

Figure 23.12 Boyle's law.

The volume of a gas varies inversely with its pressure.



If the volume is decreased from 1 liter to 1/4 liter, how would the pressure change?

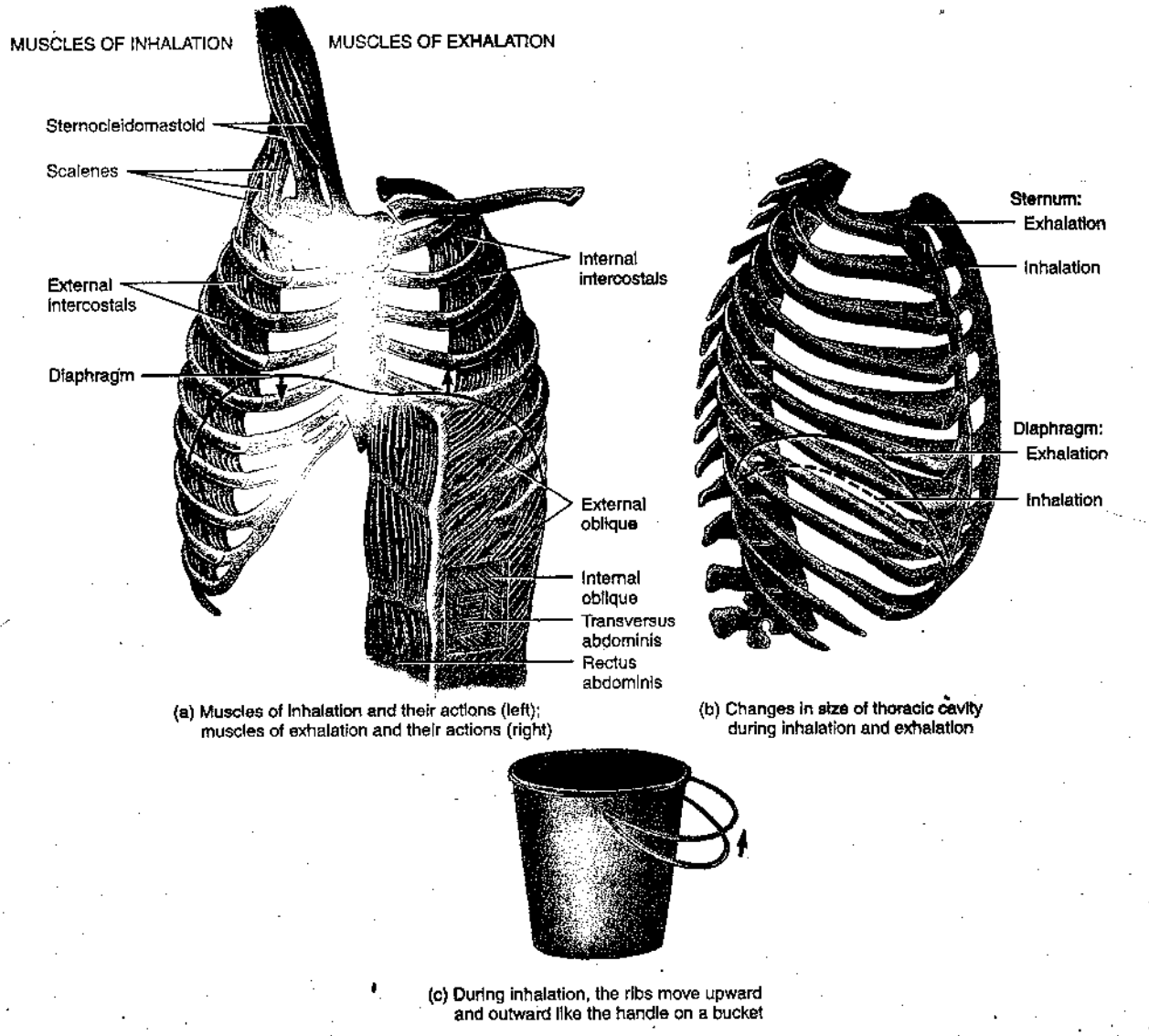
Differences in pressure caused by changes in lung volume force air into our lungs when we inhale and out when we exhale. For inhalation to occur, the lungs must expand, which increases lung volume and thus decreases the pressure in the lungs to below atmospheric pressure. The first step in expanding the lungs during normal quiet inhalation involves contraction of the main muscles of inhalation, the diaphragm and external intercostals (Figure 23.13).

The most important muscle of inhalation is the diaphragm, the dome-shaped skeletal muscle that forms the floor of the thoracic cavity. It is innervated by fibers of the phrenic nerves, which emerge from the spinal cord at cervical levels 3, 4, and 5. Contraction of the diaphragm causes it to flatten, lowering its dome. This increases the vertical diameter of the thoracic cavity. During normal quiet inhalation, the diaphragm descends about 1 cm (0.4 in.), producing a pressure difference of 1–3 mmHg and the inhalation of about 500 mL of air. In strenuous breathing, the diaphragm may descend 10 cm (4 in.), which produces a pressure difference of 100 mmHg, and the inhalation of 2–3 liters of air. Contraction of the diaphragm is responsible for about 75% of the air that enters the lungs during quiet breathing. Advanced pregnancy, excessive obesity, or confining abdominal clothing can prevent complete descent of the diaphragm.

The next most important muscles of inhalation are the external intercostals. When these muscles contract, they elevate the ribs. As a result, there is an increase in the anteroposterior and lateral diameters of the chest cavity. Contraction of the external intercostals is responsible for about 25% of the air that enters the lungs during normal quiet breathing.

Figure 23.13 Muscles of inhalation and exhalation and their actions. The pectoralis minor muscle (not shown here) is illustrated in Figure 11.14a on page 371.

⑥ During deep, labored breathing, accessory muscles of inhalation (sternocleidomastoids, scalenes, and pectoralis minors) participate.



(a) Muscles of inhalation and their actions (left); muscles of exhalation and their actions (right)

(b) Changes in size of thoracic cavity during inhalation and exhalation

(c) During inhalation, the ribs move upward and outward like the handle on a bucket

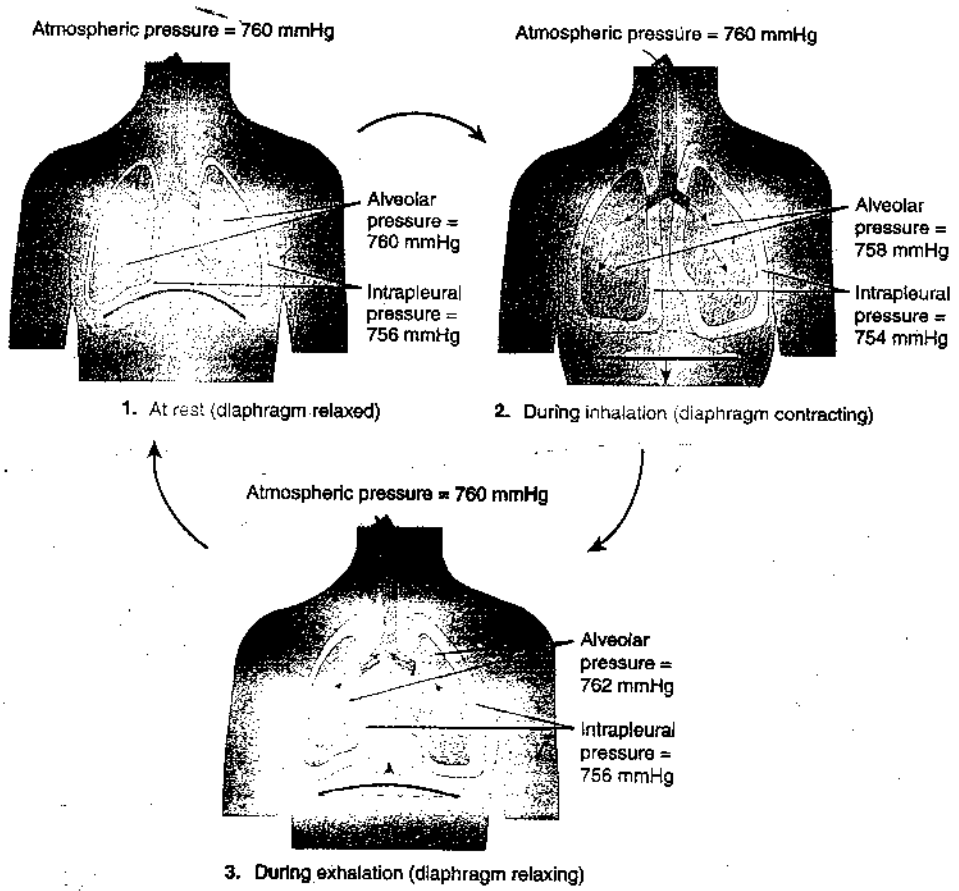
? Right now, what is the main muscle that powers your breathing?

During quiet inhalations, the pressure between the two pleural layers in the pleural cavity, called **intrapleural (intrathoracic) pressure,** is always subatmospheric (lower than atmospheric pressure). Just before inhalation, it is about 4 mmHg less than the atmospheric pressure, or about 756 mmHg at an atmospheric pressure of 760 mmHg (Figure 23.14). As the diaphragm and external intercostals contract and the overall size of the thoracic cavity increases, the volume of the pleural cavity also increases, which causes intrapleural pressure to decrease to about 754 mmHg. During expansion of the thorax, the parietal and visceral pleurae normally adhere tightly because of the subatmospheric pressure between them and because of the surface tension created by their moist adjoining surfaces. As the thoracic cavity expands, the parietal pleura lining the cavity is pulled outward in all directions, and the visceral pleura and lungs are pulled along with it.

As the volume of the lungs increases in this way, the pressure inside the lungs, called the **alveolar (intrapulmonic) pressure,** drops from 760 to 758 mmHg. A pressure difference is thus established between the atmosphere and the alveoli. Because air

Figure 23.14 Pressure changes in pulmonary ventilation. During inhalation, the diaphragm contracts, the chest expands, the lungs are pulled outward, and alveolar pressure decreases. During exhalation, the diaphragm relaxes, the lungs recoil inward, and alveolar pressure increases, forcing air out of the lungs.

Air moves into the lungs when alveolar pressure is less than atmospheric pressure, and out of the lungs when alveolar pressure is greater than atmospheric pressure.



How does the intrapleural pressure change during a normal, quiet breath?

always flows from a region of higher pressure to a region of lower pressure, inhalation takes place. Air continues to flow into the lungs as long as a pressure difference exists. During deep, forceful inhalations, accessory muscles of inspiration also participate in increasing the size of the thoracic cavity (see Figure 23.13a). The muscles are so named because they make little, if any, contribution during normal quiet inhalation, but during exercise or forced ventilation they may contract vigorously. The accessory muscles of inhalation include the sternocleidomastoid muscles, which elevate the sternum; the scalene muscles, which elevate the first two ribs; and the pectoralis minor muscles, which elevate the third through fifth ribs. Because both normal quiet inhalation and inhalation during exercise or forced ventilation involve muscular contraction, the process of inhalation is said to be *active*.

Figure 23.15a summarizes the events of inhalation.

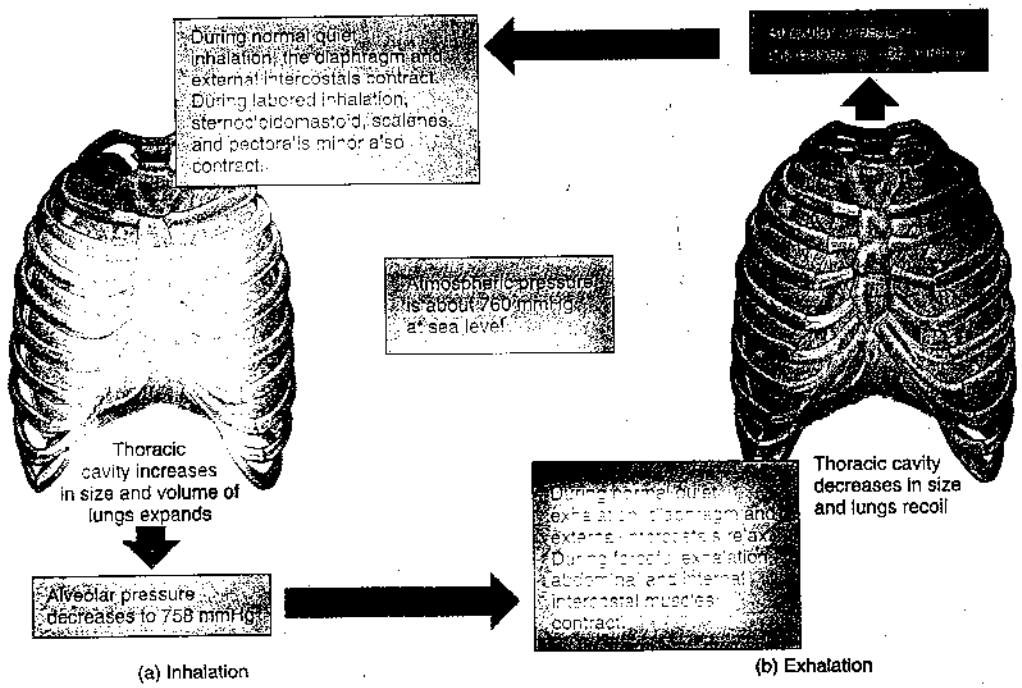
Exhalation

Breathing out, called **exhalation (expiration)**, is also due to a pressure gradient, but in this case the gradient is in the opposite direction: The pressure in the lungs is greater than the pressure of the atmosphere. Normal exhalation during quiet breathing, unlike inhalation, is a *passive process* because no muscular contractions are involved. Instead, exhalation results from **elastic recoil** of the chest wall and lungs, both of which have a natural tendency to spring back after they have been stretched. Two inwardly directed forces contribute to elastic recoil: (1) the recoil of elastic fibers that were stretched during inhalation and (2) the inward pull of surface tension due to the film of alveolar fluid.

Exhalation starts when the inspiratory muscles relax. As the diaphragm relaxes, its dome moves superiorly owing to its elasticity. As the external intercostals relax, the ribs are

Figure 23.15 Summary of events of inhalation and exhalation.

Inhalation and exhalation are caused by changes in alveolar pressure.



? What is normal atmospheric pressure at sea level?

depressed. These movements decrease the vertical, lateral, and anteroposterior diameters of the thoracic cavity, which decreases lung volume. In turn, the alveolar pressure increases to about 762 mmHg. Air then flows from the area of higher pressure in the alveoli to the area of lower pressure in the atmosphere (see Figure 23.14).

Exhalation becomes active only during forceful breathing, as occurs while playing a wind instrument or during exercise. During these times, muscles of exhalation—the **abdominals** and internal intercostals (see Figure 23.13a)—contract, which increases pressure in the abdominal region and thorax. Contraction of the abdominal muscles moves the inferior ribs downward and compresses the abdominal viscera, thereby forcing the diaphragm superiorly. Contraction of the internal intercostals, which extend inferiorly and posteriorly between adjacent ribs, pulls the ribs inferiorly. Although intrapleural pressure is always less than alveolar pressure, it may briefly exceed atmospheric pressure during a forceful exhalation, such as during a cough.

Figure 23.15b summarizes the events of exhalation.

Other Factors Affecting Pulmonary Ventilation

As you have just learned, air pressure differences drive airflow during inhalation and exhalation. However, three other factors affect the rate of airflow and the ease of pulmonary ventilation: surface tension of the alveolar fluid, compliance of the lungs, and airway resistance.

Surface Tension of Alveolar Fluid

As noted earlier, a thin layer of alveolar fluid coats the luminal surface of alveoli and exerts a force known as **surface tension.** Surface tension arises at all air–water interfaces because the polar water molecules are more strongly attracted to each other than they are to gas molecules in the air. When liquid surrounds a sphere of air, as in an alveolus or a soap bubble, surface tension produces an inwardly directed force. Soap bubbles "burst" because they collapse inward due to surface tension. In the lungs, surface tension causes the alveoli to assume the smallest possible diameter. During breathing, surface tension must be overcome to expand the lungs during each inhalation. Surface tension also accounts for two-thirds of lung elastic recoil, which decreases the size of alveoli during exhalation.

The surfactant (a mixture of phospholipids and lipoproteins) present in alveolar fluid reduces its surface tension below the surface tension of pure water. A deficiency of surfactant in premature infants causes *respiratory distress syndrome,* in which the surface tension of alveolar fluid is greatly increased, so that many alveoli collapse at the end of each exhalation. Great effort is then needed at the next inhalation to reopen the collapsed alveoli.

Question NO (6)

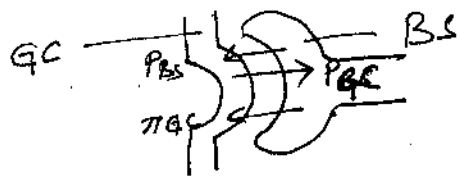
Fluid in Bowman's space normally contain no cells but contain all plasma substance except protein. Renal corpuscles restrict the movement of high molecular weight substance thus they are ~~not~~ filtered with water and low molecular weight substances. corpuscular membrane is negatively charged, so they oppose the movement of these plasma protein. ~~the~~ The unequal distribution of protein causes the water concentration of the plasma to be slightly less than the fluid in the Bowmans space. The difference in water concentration favors fluid movement by bulk flow from Bowman's space into the glomerular capillaries osmotic force ~~also~~ oppose filtration due to the presence of protein. ~~in glomerular~~ Thus net glomerular filtration ~~pressure~~ depends upon theree forces.

	mmHg
Favoring filtration	
Glomerular capillary blood pressure (P_{GC})	60
Opposing filtration	
Fluid pressure in Bowman's ~~space~~ (P_{BS})	15
Osmotic force due to protein in plasma (π_{GC})	29

Net Glomerular filtration pressure $= P_{GC} - P_{BS} - \pi_{GC}$

$= 60 - 15 - 29$

$= +16$



The volume of fluid filtered from the glomeruli into Bowman's space per unit time is known as glomerular filtration rate (GFR). GFR is directly proportional to the membrane permeability and surface area. Average GFR of a healthy person is about 180 L/day or 125 ml/min. Only 20% of the plasma that enters the glomerular capillary is filtered into Bowman's space.

Arginase is found in all the tissues in elasmobranch fishes and liver cells of mammals.

Ornithine cycle was described by **Krebs** and **Henseleit** (1932). They demonstrated the action of arginase in urea formation by using slices of rat liver.

Formation of Urine

The urine is formed inside the kidneys from the nitrogenous wastes produced during catabolic process in the body. The following three processes or steps occur during urine formation :—

1. Filtration, 2. Selective reabsorption, and 3. Tubular secretion.

1. Ultrafiltration—The Bowman's capsules of kidney act as ultra-filters and lie in close contact with the glomerulus. The blood flowing through the afferent arterioles contains urea, water, several salts and blood proteins dissolved in the plasma. It is separated from the cavity of renal tubule of Bowman's capsule only by two very thin membranes—the endothelial layer of blood capillaries and the epithelial layer of Bowman's capsule. These two layer are one cell thick and lie in close coantact. The diameter of afferent arteriole is more than the efferent arteriole. Therefore, the amount of blood which enters the afferent arteriole in a definite time is not

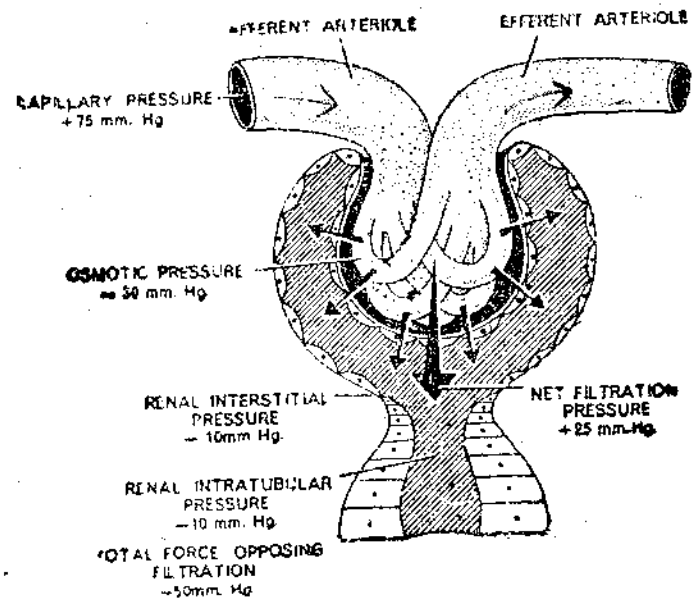


Fig. 6·3. Diagrammatic representation of ultrafiltration in Bowman's capsule of urinferous tubule.

drained out so the hydrostatic pressure of blood in the capillary network of glomerulus increases.

This **capillary pressure** in glomerulus is about 75 mm Hg. This pressure has to overcome the osmotic pressure of the plasma, which works to retain constituents within the capillary walls. The **osmotic pressure** of plasma is about 30 mm Hg. The renal **interstitial pressure** on the capillaries together with the resistance to flow in the uriniferous tubules (*i.e.* **intratubular pressure**) contribute a a pressure of about 20 mm Hg. This also works against the capillary pressure. Therefore, the **net filtration pressure** responsible for the filtration is about 25 mm. Hg. As a result, water and diffusible solute molecules are forced across the membrane from the blood plasma into the Bowman's capsule. This process is known as **ultrafiltration**. As a result of **ultrafiltration** almost all the substances dissolved in plasma filter out into the cavity of Bowman's capsule along with water except the blood corpuscles, colloids and certain proteins. This filtered liquid is known as **nephric filtrate** or **glomerul r filtrate**.

Summary of filtration Pressure in Glomeralus

1. **GHP**—75 mm. Hg
   (Glomerular Hydrostatic Pressure)
2. **BCOP**
   (Blood Colloidal Osmotic Pressure)—30 mm. Hg.
3. Renal Interstitial Pressure—10 mm. Hg.
4. Renal Intratubular Pressure—10 mm. Hg.
5. Net Filtration Pressure = 75—(30+10+10 mm. Hg.)
   = 75—50 mm. Hg.
   = 25 mm. Hg.

2. Selective Absorption—Since the function of nephron is exclusively of a filter, not only urea but even the useful substances like glucose, amino acid, inorganic salts, PO_4 ions and vitamin C, etc. also diffuse out into the nephric filtrate. Their removal from the plasma along with urea will be harmful to the body. Therefore, the useful substances should be reabsorbed into the blood. For this purpose, the efferent arterioles form a network of capillaries around the neck and body of the uriniferous tubule (see fig. 6·3).

Different useful substances filtered into the nephric filtrate are reabsorbed in the uriniferous tubule at different regions along its length. The water is reabsorbed in the descending limb of Henle's loop and in the proximal convoluted tubule, whereas the glucose, sodium carbonate, sodium chloride and other useful substances are absorbed in the glandular portion of uriniferous tubules. Approximately all the glucose is reabsorbed into the blood in the proximal convoluted tubule. These are passed to the capillaries of efferent arteriole and, thus, is returned back to the blood. In the ascending

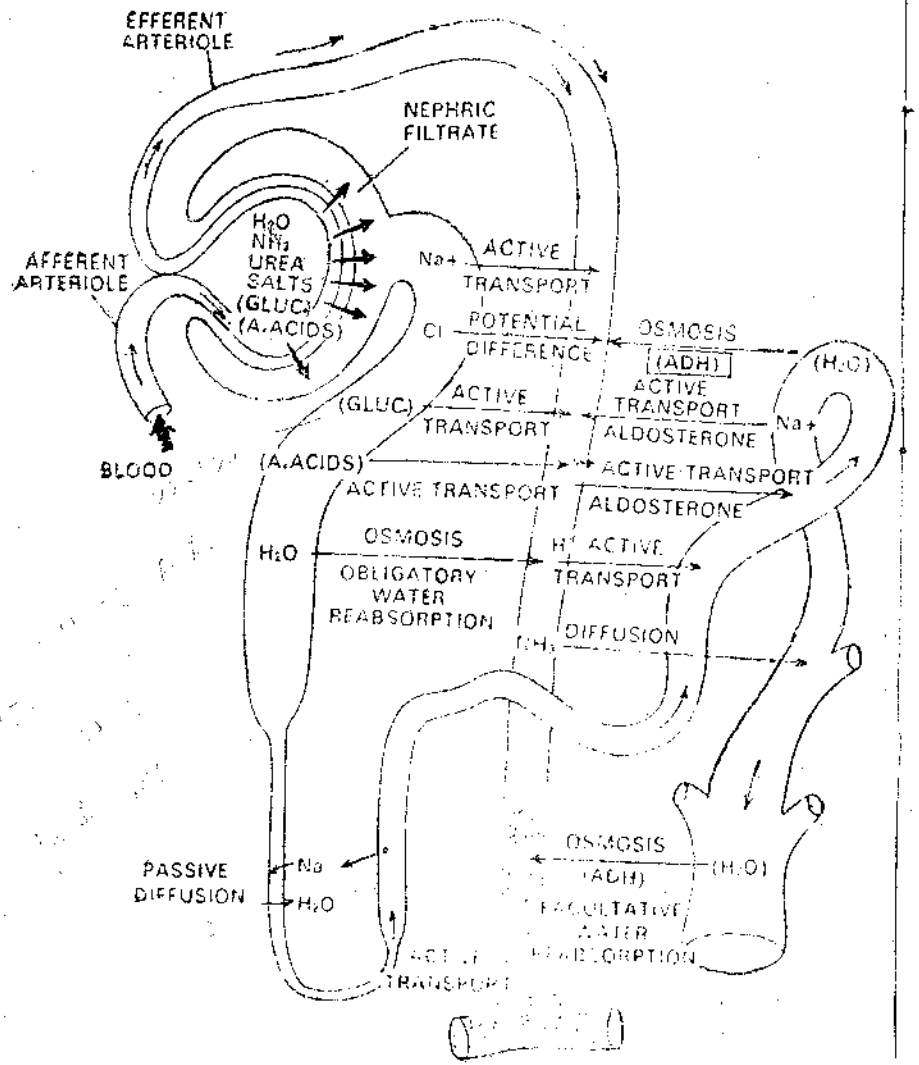


Fig. 6·4. Diagrammatic represention of selective absorption in the uriniferous tubule and changes occurring in th composition of urine.

limb, active loss of sodium chloride takes place without water. As a result the filtrate now becomes hypo-osmotic. The nephric filtrate at this stage is left with urea, little amount of water and certain salts.

3. Secretion

The excretory products left in the blood capillaries are secreted into the uriniferous tubules by diffusion. The creatine, uric acid and potassium are secreted from the blood into the nephric filtrate. These mix with the nephric filtrate. This filtrate is known as **urine.**

Horomonal Control of Excretion

The process of excretion of salts and water from the blood is controlled by hormones secreted by the adrenal cortex, a part of adrenal glands. In general, these hormones are known as cortical hormones. These exercise a major control over metabolism of water and salts. The effect of different cortical hormones is shown in fig. 6·5. If there is lack of some cortical hormones, sodium and water are excreted in excessive amount and potassium is lost. The loss of salts and water from blood results in a lowering of blood pressure and disturbing the functioning of kidney tubules and failure of filtration in glomerulus.

The action and secretion of cortical hormones is governed by **adrenocortrophic** (ACTH) **hormones** secreted by the pars distalis of pituitary gland.

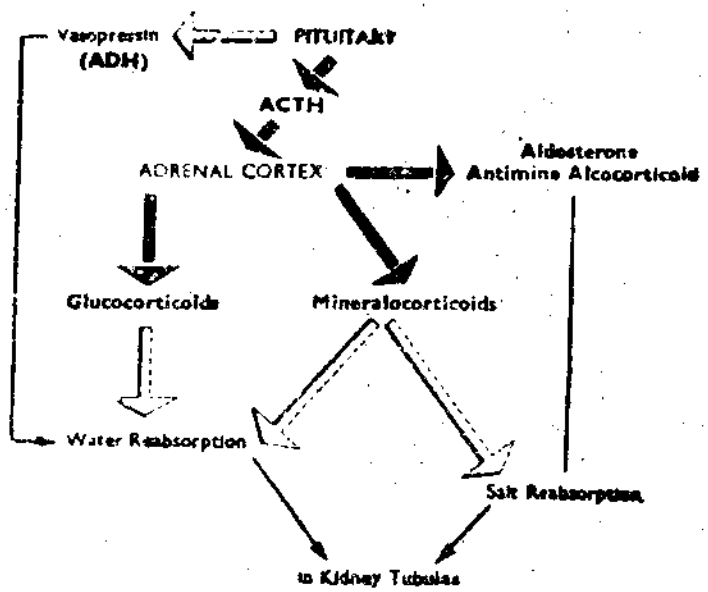


Fig. 6.5. Diagrammatic representation of water reabsorption and salt reabsorption in kidney tubules. Solid lines indicate activating influence and broken arrows an inhibiting influence on the process of excretion.

(7) Question No (7)

Active Transport of Sodium and Potassium Ions Through the Membrane—The Sodium-Potassium (Na^+-K^+) Pump. First, let us recall from Chapter 4 that all cell membranes of the body have a powerful Na^+-K^+ pump that continually transports sodium ions to the outside of the cell and potassium ions to the inside, as illustrated on the left-hand side in Figure 5-4. Further, note that this is an *electrogenic pump* because more positive charges are pumped to the outside than to the inside (three Na^+ ions to the outside for each two K^+ ions to the inside), leaving a net deficit of positive ions on the inside; this causes a negative potential inside the cell membrane.

The Na^+-K^+ pump also causes large concentration gradients for sodium and potassium across the resting nerve membrane. These gradients are the following:

Na^+ (outside): 142 mEq/L

Na^+ (inside): 14 mEq/L

K^+ (outside): 4 mEq/L

K^+ (inside): 140 mEq/L

The ratios of these two respective ions from the inside to the outside are

$$Na^+_{inside}/Na^+_{outside} = 0.1$$

$$K^+_{inside}/K^+_{outside} = 35.0$$

Leakage of Potassium Through the Nerve Membrane. The right side of Figure 5-4 shows a channel protein, sometimes called a *"tandem pore domain," potassium channel,* or *potassium (K^+) "leak" channel,* in the nerve membrane through which potassium can leak even in a resting cell. The basic structure of potassium channels was described in Chapter 4 (Figure 4-4). These K^+ leak channels may also leak sodium ions slightly but are far more permeable to potassium than to sodium, normally about 100 times as permeable. As discussed later, this differential in permeability is a key factor in determining the level of the normal resting membrane potential.

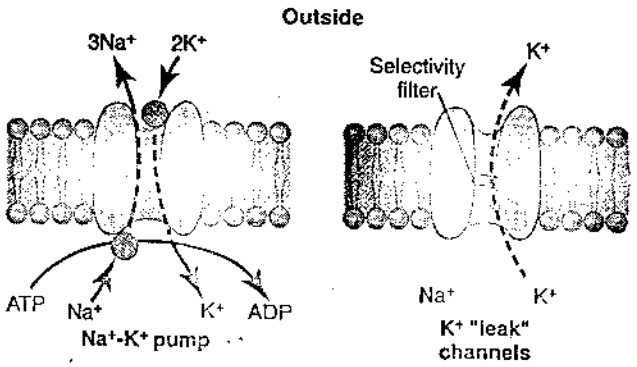


Figure 5-4 Functional characteristics of the Na^+-K^+ pump and of the K^+ "leak" channels. ADP, adenosine diphosphate; ATP, adenosine triphosphate. The K^+ "leak" channels also leak Na^+ ions into the cell slightly, but are much more permeable to K^+.

Resting Membrane Potential of Nerves

The resting membrane potential of large nerve fibers when not transmitting nerve signals is about −90 millivolts. That is, the potential *inside the fiber* is 90 millivolts more negative than the potential in the extracellular fluid on the outside of the fiber. In the next few paragraphs, the transport properties of the resting nerve membrane for sodium and potassium and the factors that determine the level of this resting potential are explained.

Origin of the Normal Resting Membrane Potential

Figure 5-5 shows the important factors in the establishment of the normal resting membrane potential of −90 millivolts. They are as follows.

Contribution of the Potassium Diffusion Potential. In Figure 5-5*A*, we make the assumption that the only movement of ions through the membrane is diffusion of potassium ions, as demonstrated by the open channels between the potassium symbols (K^+) inside and outside the membrane. Because of the high ratio of potassium ions inside to outside, 35:1, the Nernst potential corresponding to this ratio is −94 millivolts because the logarithm of 35 is 1.54, and this multiplied by −61 millivolts is −94 millivolts. Therefore, if potassium ions were the only factor causing the resting potential, the resting potential *inside the fiber* would be equal to −94 millivolts, as shown in the figure.

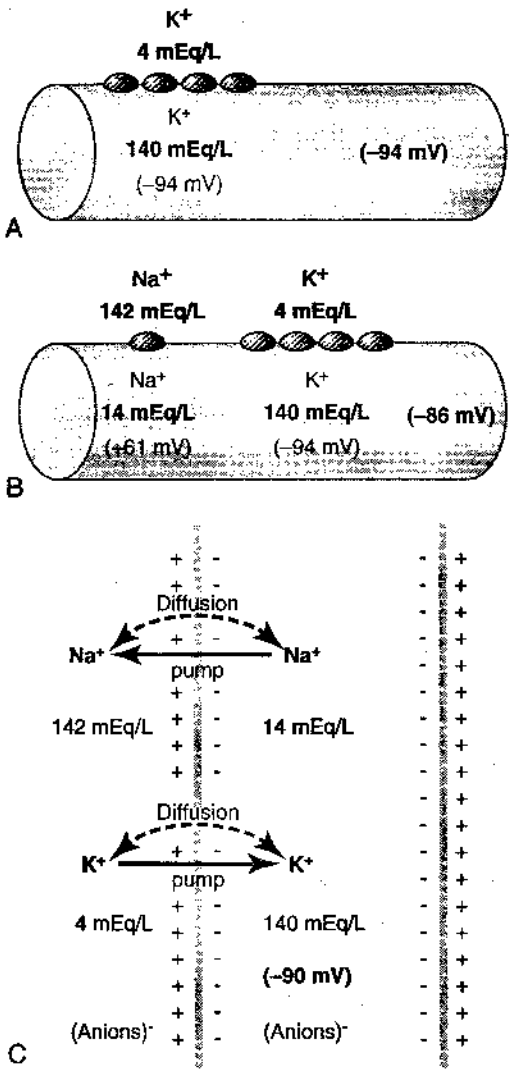


Figure 5-5 Establishment of resting membrane potentials in nerve fibers under three conditions: *A*, when the membrane potential is caused entirely by potassium diffusion alone; *B*, when the membrane potential is caused by diffusion of both sodium and potassium ions; and *C*, when the membrane potential is caused by diffusion of both sodium and potassium ions plus pumping of both these ions by the Na^+-K^+ pump.

Contribution of Sodium Diffusion Through the Nerve Membrane. Figure 5-5*B* shows the addition of slight permeability of the nerve membrane to sodium ions, caused by the minute diffusion of sodium ions through the K^+-Na^+ leak channels. The ratio of sodium ions from inside to outside the membrane is 0.1, and this gives a calculated Nernst potential for the inside of the membrane of +61 millivolts. But also shown in Figure 5-5*B* is the Nernst potential for potassium diffusion of −94 millivolts. How do these interact with each other, and what will be the summated potential? This can be answered by using the Goldman equation described previously. Intuitively, one can see that if the membrane is highly permeable to potassium but only slightly permeable to sodium, it is logical that the diffusion of potassium contributes far more to the membrane potential than does the diffusion of sodium. In the normal nerve fiber, the permeability of the membrane to potassium is about 100 times as great as its permeability to sodium. Using this value in the Goldman equation gives a potential inside the membrane of −86 millivolts, which is near the potassium potential shown in the figure.

Contribution of the Na^+-K^+ Pump. In Figure 5-5*C*, the Na^+-K^+ pump is shown to provide an additional contribution to the resting potential. In this figure, there is continuous pumping of three sodium ions to the outside for each two potassium ions pumped to the inside of the membrane. The fact that more sodium ions are being pumped to the outside than potassium to the inside causes continual loss of positive charges from inside the membrane; this creates an additional degree of negativity (about −4 millivolts additional) on the inside beyond that which can be accounted for by diffusion alone. Therefore, as shown in Figure 5-5*C*, the net membrane potential with all these factors operative at the same time is about −90 millivolts.

In summary, the diffusion potentials alone caused by potassium and sodium diffusion would give a membrane potential of about −86 millivolts, almost all of this being determined by potassium diffusion. Then, an additional −4 millivolts is contributed to the membrane potential by the continuously acting electrogenic Na^+-K^+ pump, giving a net membrane potential of −90 millivolts.

Nerve Action Potential

Nerve signals are transmitted by *action potentials*, which are rapid changes in the membrane potential that spread rapidly along the nerve fiber membrane. Each action potential begins with a sudden change from the normal resting negative membrane potential to a positive potential and then ends with an almost equally rapid change back to the negative potential. To conduct a nerve signal, the action potential moves along the nerve fiber until it comes to the fiber's end.

Muscle Protein Question No (8)

Myofibrils are built from three kinds of protein
(a) Contractile protein - which generate force during contraction.
(b) Regulatory protein - which help switch the contraction process on and off.
(c) Structural protein - which keeps the thick and thin filament in the proper alignment, give the myofibril elasticity.

(a) The two contractile protein in muscles are myosin and actin, which are the main component of thick and thin filament. Myosin functions as a motor protein in all three types of tissue.. Motor protein pull or push the various Cellular structures to achieve movement by converting the chemical energy in ATP to the mechanical energy of motion or the product of force. In skeletal muscle about 300 molecules of myosin form a single thick filament. Each myosin molecule is shaped like two golf clubs twisted together. The myosin tail points towards the M lin in the center of the sarcomere. Tails of neighboring myosin molecule lie parallel

thick filament. The two projections of each myosin molecule (heads) are called myosin heads.

Thin filament are anchored to Z discs. Their main component of the protein is actin. Individual actin molecules join to form an actin filament that is twisted into a helix (On each actin molecule a myosin binding site is present, where a myosin head can attach)

(b) The regulatory proteins are present in smaller amount. The regulatory protein are tropomyosin and troponin. These proteins are the part of thin filament.

In relaxed condition, the tropomyosin cover the myosin binding sites on actin.

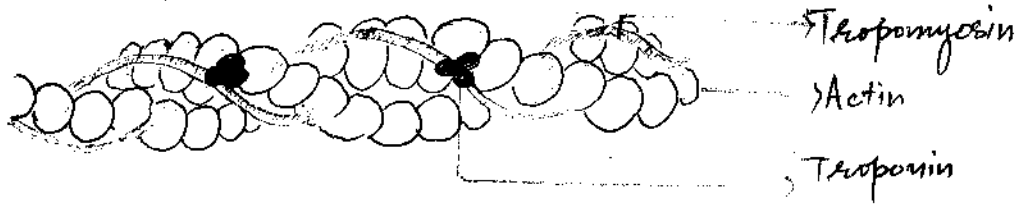
The myosin heads of thick filaments are held in place where Troponin molecules are present on the tropomyosin strand.

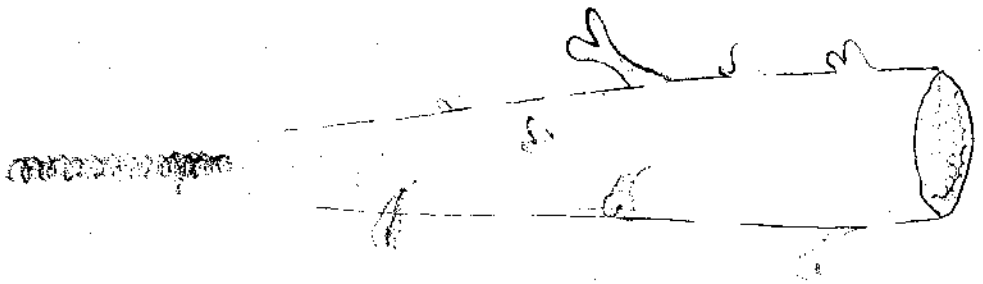
During contraction the myosin heads binds to actin and form cross bridge.

Contractile Protein : Myosin, Actin

Regulatory protein : Tropomyosin and Troponin.

Structural protein : Titin





The myosin actin crossbridges rotate towar center of the sarcomere.

(c) The structural protein are the very important protein found in the muscle. These are present in very less amount but contribute a major role in the alignment, stability, elasticity and extensibility of myofibrils. Few importa structural proteins are – titin, a-actinin, myomesin, nebulin and dystrophin.

Titin is the third most plentyful protein in skeletal muscle after actin and myosin. Titin molecule is huge in size and its molecular weight is about 3 millio dalton. Titin is 50 times larger than an average sized protein. Each titin molecule spans half a sarcomere, from a Z line to M line. In relaxed muscle the distance covered by a titin molecule is about 1 to 1·2 um. Each titin molecule connets a Z line to the M line of the sarcomere, there by helping stabilize the position of thick filament

Titin molecule that extends from the Z line to the begining of thick filament is very elastic. It can stretch at least four times its resting length and then spring back.

Titin helps the sarcomere return to its resting length after a muscle has contracted or stretched and prevent the over extension of sarcomere. thus maintains the central location of the A band.

α-actinin – α-actinin is the dense material that is found on the Z line. α-actinin of Z line binds to actin molecules of the thin filament and to titin.

Myomesin — Molecules of myomesin form the M line. The M line protein binds to titin and connect adjacent thick filaments to one another.

Nebulin – Nebulin is a long, nonelastic protein, wrapped around the entire length of each thin filament.

Dystrophin – It is a cytoskeletal protein that links thin filaments of the sarcomere to the integral membrane protein of the sarcolemma.