6
Cardio Vascular System

Heart

Heart is a central pumping organ. It receives and pumps out blood to the whole body.

1. *Shape :* Conical or roughly heart shaped (like heart of playing cards)
2. *Size :* 12 cm from base to apex.
   6 cm antero-posteriorly.

Situation : Situated in the middle mediastinum in between the two lungs, obliquely placed behind the body of sternum and the adjoining portions of ribs and cartilages: about one third of it is on the right side and two third of it on the left side of the middle.

Parts of the heart

Heart has got four chambers, with some openings. The chambers are-

a. Right atrium
b. Left atrium — *Receiving chambers*

c. Right ventricle
d. Left ventricle. — *Distributing chambers*

Openings of the heart

Heart has got four openings :

a. *Right atrioventricular opening :* Lies between right atrium & right ventricle and is guarded by right atrioventricular (tricuspid) valve.

b. *Left atrioventricular opening :* Lies between left atrium and left ventricle and is guarded by left atrioventricular or mitral (bicuspid) valve.

c. *Pulmonary opening :* Lies between right ventricle & pulmonary trunk and guarded by semilunar valve.

d. *Aortic opening :* Lies between Left ventricle and ascending aorta and is guarded by semilunar valve.

Valves of the heart

There are four valves in heart -

a. *Right atrioventricular valves :* It is also called tricuspid as it has three cusps. The cusps are -
   i. Anterior or infundibular
   ii. Posterior or marginal
   iii. Medial or septal

b. *Left atrio-ventricular valve :* It is also called mitral (due to its resemblance to a bishop mitre) or bicuspid valve as it has two cusps. The cusps are -
   i. Anterior or aortic
   ii. Posterior

c. *Semilunar valves :* Two in number, one in aortic and another in pulmonary opening. They each have three cusps. They are so called as semilunate in shape.

Functions of valves :

1. Valves help in the flow of blood in one direction.
2. Prevent regurgitation of back flow of blood by their direction during opening and closure.
3. They are concerned with the production of heart sound.

Precardium : The area of the anterior chest wall that overlies the heart is called precardium.

Nerve Supply

Q. 00. **What is the nerve supply of heart.**

Ans. *Nerve supply to the heart are* :

i. *Parasympathetic nerve* :
   a. *Nerve fibers* : Arise from nucleus abbiguus (medulla) → join vagus and finally terminate on heart;
   b. *Neurotransmitter* : Acetylcholine.
   c. *Effect* : Cardiac inhibition.

ii. *Sympathetic nerve* :
   a. *Nerve fibers* : Arise from LHC of upper thoracic but specially from T_3 & T_4; LHCs are controlled by VMC (medulla)
   b. *Neurotransmitter* : Noradrenalin
   c. *Effect* : Cardiac stimulation.

Cardiac or rather cardiovascular reflexes : operate via sympathetic and to some limited extent by parasympathetic nerves. Well known reflexes include -

i. *broreceptor*
ii. *chemoreceptor*
iii. *atrial* and
iv. *Bezold Jarisch reflexes.*

All these reflexes change heart rate, many of them change the blood pressure and some even the respiration; all of them restore the homeostasis.

Q. 06. ***List the effects of stimulation of sympathetic and parasympathetic systems on the heart.**

	Parasympathetic	*Sympathetic*
Heart (β_1, β_2) : a. *S-A node*	Decrease in heart rate, vagal arrest.	Increase in heart rate

	Parasympathetic	*Sympathetic*
b. *Atria*	Decrease in contractility and (usually) increase in conduction velocity.	Increase in contractility and conduction velocity
c. *A-V node*	Decrease in conduction velocity	Increase in conduction velocity.
d. *His-Purkinje system*	Decrease in conduction velocity	Increase in conduction velocity.
e. *Ventricles*	Decrease in contractility.	Increase in contractility
Arterioles : a. Coronary	Dilatation	Constriction (α_1, α_2) Dilation (β_2).

Q. ***Heart receives its blood supply during diastole- Explain.***

Ans. During diastole of heart, cardiac muscles are relaxed, intramural tension decreases and coronary blood vessels are less compressed. So, blood can easily enters into coronary vessels and supply blood. On the other hand, during systole, due to increase cardiac muscular tension, coronary vessels are compressed. So, heart gets its boold supply during diastole.

Heart Muscles

Physiology of cardiac muscle :The heart is composed of three major types of cardiac muscle -

a. Atrial muscle
b. Ventricular muscle
c. *Spcialized excitatory and conductive muscle fibres-*
   i. The sinus node (sino-atrial or SA node)
   ii. The internodal pathway
   iii. The A-V node (Atrio-ventricular node)
   iv. The A-V bundle
      1. Penetrating portion
      2. Distal portion
   v. The left and right bundles of purkinje fibers.

N.B. SA node, internodal pathways, AV node, Bundle of His & its branches, purkinje fibers *(Ref. Ganong 22th ed)*

The atrial and ventricular muscle contract, in which the duration of contraction is much longer.

The specialized excitatory and conductive muscle fibers contract very feebly as they contain few contractile fibrils: and exhibit rhythmicity and varying rates of conduction.

(Ref. Guyton 11th ed; page 103)

Functional anatomy of muscle

Myocardial cells (=fibers) are of 2 types-

i. *Pace maker and conducting cells* (=SAN,AV junctional tissue , bundle of His and its branches, purkinje's fibers) : They generate impulse and conduct the impules

ii. *Working myocardial cells* (WMC) : Which contract. WMC consists of myofibrils; each myofibril cosists of 4 types of myofilaments :
   i. Actin
   ii. Myosin
   iii Tropomyosin
   iv. Troponin.

At rest, the active sites of actin (where myosin heads are to be attached during contraction), are covered by tropomyosin.

2 types of tubules are seen in sarcolemma (=cell membrane of muscle fiber)- i. T tubules which communicate with the ECF, and ii. L tubules (=sarcoplasmic reticulum) which do not communicate with the exterior. T tubules occur near the Z lines (which form the boundaries of sarcomere) and near them L tubules form cisterns (or triads).

Syncytium

It means a group of cells in which the protoplasm of one cell is continuous with that of adjoining cells such as the mesenchyme cells of embroys, striated muscle fibre.

Heart muscle cells are large and branching. They are connected to each other by angulated dark areas crossing the cardiac muscles fibre are called intercalated discs; however, they are actually cell membranes that separate individual cardiac muscle cells from each other. That is cardiac muscle fibres are made up of many individual cells connected in series with each other. The intercalated disc exerts a very little resistance (1/400th of normal), which resembles that there is no membrane in between the cardiac muscle cells, because the cell membranes fuse with each other and form very permeable junctions (*gap junction*) that allow relatively free diffusion of ions. Therefore, from a functional point of view, ions move with ease along the axes of the cardiac muscle fibres so that action potentials travel from one cardiac muscle cell to another, past the intercalated discs, with only slight hindrance. Therefore, cardiac muscle is a syncytium.

The heart is composed of two separate syncytiums; the atrial syncytium that constitute the walls of two atria & the ventricular syncytium that constitute the wall of two venticule.

(Ref. Guyton & Hall-11th Edition; page 103, 104)

Junctional tissues of heart

Cardiac muscle consist especially of certain specialized structures which are responsible for initiation and transmission of cardiac impulses at a regular and faster rate than rest of the muscle. These are called junctional tissue of heart.

Junctional tissues are :

i. The sinus node (Sino-atrial or SA node)

ii. The internodal atrial pathway.
iii. The AV node (Atrio-ventricular node)
iv. The bundle of His and its branches.
v. Purkinje system.

SA node or sino-atrial node

Situation : It is situated in the right atrium at the junction of superior venacava and right auricular appendage. It extends downwards along the sulcus terminalis for about 2 cm.

Histology : Muscle fibre are thin, elongated, fusi form, longitudi nally straited and inte rcrossing with one another with a plexiform manner. There is distin ctive cell type known as P cell, which is centrally located and stellate in appearance, related to pace maker activity.

Blood supply :

i. Artery :
Mainly by right coronary and some times from left coronary artery.
ii. Vein : Primitive right great vein, later which become superior venacava.

Nerve supply :

i. Para sympathetic : By right vagus nerve.
ii. Sympathetic : Thoracic 1-5 segment.

Functions :

i. It can initiate the physiological cardiac impulse before any other part of heart.
ii. It is called 'pace maker'.
iii. It can discharge impulse at a rate of about 70-80 impulse/min.

Pace maker : Means regulation of motion. SA node is called pace maker of heart. Because it can initiate normal physiological impulse at first and the rate of discharge is greater than any other part of the heart.

Internodal pathway

It is the pathway that coduct the impulse from the SA node to AV node and left atrium. These internodal tracts contain purkinje type of fibres. Goldman has described three inter nodal pathways-

a. Anterior internodal tract of *Bachman*
b. Middle internodal tract of *Wenckebach*
c. Posterior internodal tract of *Thorel.*

Anterior internodal tract : After coming out from SA node curves round the superior venacava and anterior wall of right atrium it bifurcates into two brances -

i. One knwon as Bachmann's Bundle extends along the dorsal aspect of interatrial band and supplies to left atrium.
ii. Another goes to AV node and merges with other conducting system.

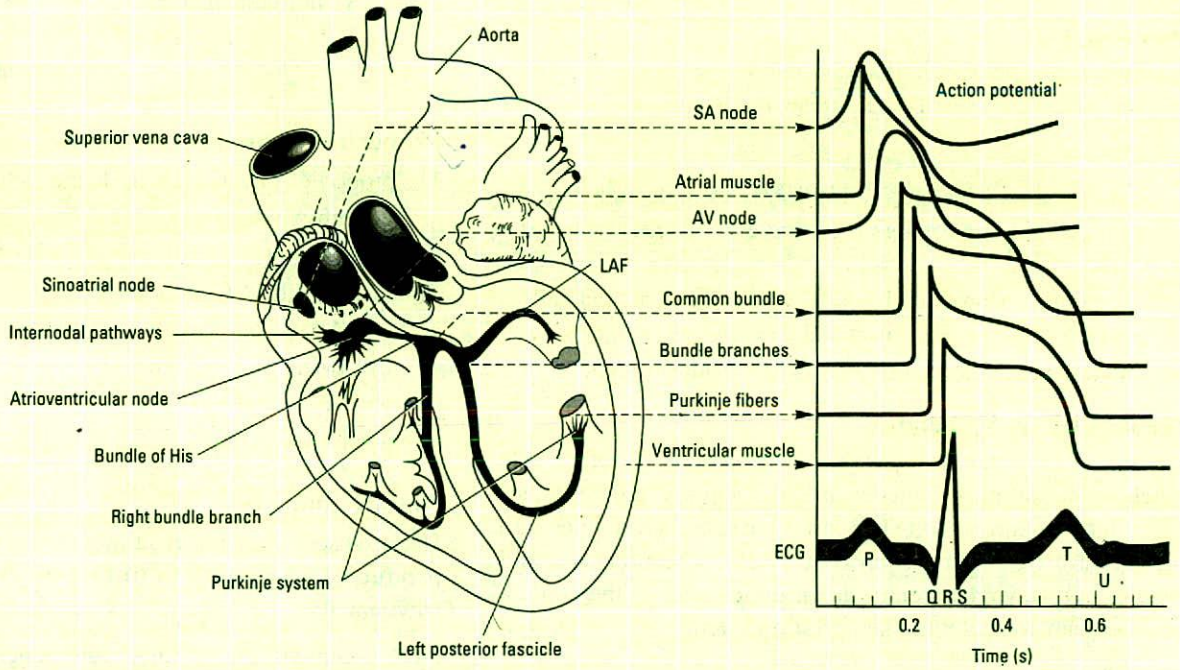


Fig.6-1. *Junctional tissue of the heart.*

Middle internodal tract : Arises from postero- dorsal margin of SA node and grazes the inter atrial septum to reach AV node.

Posterior internodal tract : (Thorel's tract) - Passes along the crista terminalis to reach AV node.

Function :

These internodal tracts serve as preperential pathways for conduction of impulse from SA node to AV node and left atrium.

AV node or Atrioventricular node

Situation : It is situated at the posterior and right border of the interatrial septum near the opening of the coronary sinus.

AV node is normally the only coducting path- way between the atria and the ventricle.

Histology : The cells of AV nodes are cardiac muscle fibre but have a few myofibrils.

Blood supply :

i. Artery : about 90% by right coronary artery and remaining by left coronary artery.
ii. Vein : Into coronary sinus.

Nerve supply :

i. Para sympathetic : Left vagus nerve
ii. Sympathetic : T_1 - T_S sympathetic ganglion.

Functions :

i. It receives impulse originating form SA node and transmit it to the ventricle through the bundle of His at a rate of 0.1 m/sec.
ii. AV node can produce impulse when SA node can fail to produce impulse. So, it is called " reserve pace- maker".
iii. The rate of AV nodal impulse 40-60 impulse/minute.

Bundle of His or AV bundle

Situation : It extends from AV node. It gives off a left bundle branch at the top of the inter ventricular septum and continues as the right bundle branch. The left bundle branch divides into anterior fascicle and a posterior fascicle. The branches and fascicles run subendocardialy down either side of the septum and come into contact with the purkinje system.

Function :

i. *Conductions :* It's main function is to conduct the atrial impulse into ventricle.
ii. *Rhythmicity :* When SA node and AV node fails, the Bundle of His can originate cardiac impulse 30-36 impulse/minute.

Purkinje fiber

It arises from the branches of the Bundle of His, spread from the interventricular septum directly to the papillary muscle and then to the lateral walls of the ventricle ending ultimately with the subendocardial net work. It fibers spread to all parts of the ventricular myocardium.

Functions :

i. To conduct impulso quickly to overy part of ventricular muscle fibre.
ii. It can also initiate impulse in case of atrio-ventricular dissociation at a rate of 15 to 40 impulse/ min.

Cardiac ion channels :

i. *Voltage-gated channels*
   Na^+
   TCa^{++}
   LCa^{++}
   K^+
   Inward rectifying
   Delayed rectifying
   Transient outward
ii. *Ligand-gated K^+ channels* :
   Ca^+ activated
   Na^+ activated
   ATP-sensitive
   Acetylcholine-activated
   Arachidonic acid-activated.

(Ref. Ganong 21th edition, Page 80)

Properties of the heart muscles

The properties of caridac muscle are as follows :

i. Autorhythmicity
ii. Conductivity
iii. Excitability & Contractility
   a. All or none law.
   b. Frank Starling law
iv. Refractory period
v. Tonicity.

Autorhythmicity

Heart muscle does not require any stimulation from outside to produce its impulse (automaticity) at regular intervals (rhythmicity).

Autorhythmicity, also called automaticity. Rhythmicity means that the heart muscle is endowed with the property of generating its own impulse at regular intervals (and these impulses, as will be shown afterwards, can be propagated).

Site of impulse generation : The SAN (sinuatrial node) and the junctional tissues (i.e, the conducting system) can generate the impulses but the working myocardial cells cannot. Normally, however, SAN generates the impulses while other parts of the junctional tissue only propagate the impulse (but does not generate).

Mechanism of sinus nodal rhythmicity : The potential of the sinus nodal fiber between discharges has a negativity of only -55 to -60 millivolts in comparison with -85 to -90 millivolts for the ventricular muscle fiber. The cause of this reduced negativity is that the cell membranes of the sinus fibers are naturally leaky to sodium ions. At this level of negativity, the fast sodium channels have mainly become "inactivated," which means that they have become blocked.

Self-excitation of sinus nodal fibers : Because of the high sodium ion concentration in the extracellular fluid as well as the negative electrical charge inside the resting sinus nodal fibers, the positive sodium ions outside the fibers even normally tend to leak to the inside. Furthermore, the resting nodal fibers

have a moderate number of channels that are already open to the sodium ions. Therefore, influx of positively charged sodium ions causes a rising membrane potential. When it reaches a threshold voltage of about -40 millivolts, the calcium-sodium channels become activated, leading to rapid entry of both calcium and sodium ions, thus causing the action potential. Then the calcium-sodium channels become inactivated (that is, they close) within about 100 to 150 milliseconds after opening, and at about the same time, greatly increased numbers of potassium channels open. Therefore, the influx of calcium and sodium ions through the calcium-sodium channels ceases, while at the same time large quantities of positive potassium ions diffuse out of the fiber, thus terminating the action potential. Furthermore, the potassium channels remain open for another few tenths of a second, carrying a great excess of positive potassium charges out of the cell, which temporarily causes considerable excess negativity inside the fiber; this is called hyperpolarization. This hyperpolarization initially carries the "resting" membrane potential down to about -55 to -60 millivolts at the termination of the action potential.

During the next few tenths of a second after the action potential is over, progressively more and more of the potassium channels begin to close. Now the inward-leaking sodium ions once again over balance the outward flux of potassium ions, which causes the "resting" potential to drift upward once more, finally reaching the threshold level for discharge at a potential of about -40 millivolts. Then the entire process begins again: self-excitation, recovery from the action potential, hyperpolarization after the action potentials is over, drift of the "resting" potential again to threshold, then re-excitation again to elicit another cycle. This process continues indefinitely throughout a person's life.

(Ref. Guyton & Hall-11th Edition; Page 116, 117, 118)

Q. 03. **What is pacemaker potential?**

Ans. Pacemaker potential :

i. *Definition* : Rhythmically dicharging cells have a membrane potential that after each impulse, declines to the firing level. This is called the pace maker potential or pre potential. It triggers the next impulse.

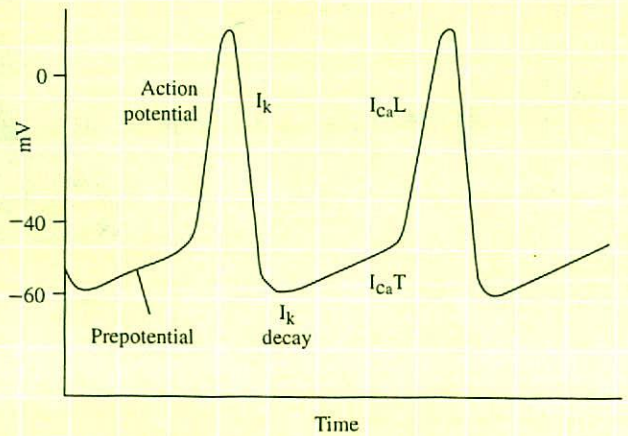


Fig: 6-2. *Rhythmical discharge of a sinus nodal fiber. Also, the sinus nodal action potential is compared with that of a ventricular muscle fiber.*

ii. *Cause* :

a. *First part of the prepotential* : At the peak of each impulse, I_k begins and brings about repolarizanon. I_k then declines, and as K^+ efflux decreases, the membrane begins to depolarize, forming the first part of the prepotential.

b. 2nd part of the prepotential : Ca^{++} channels then open. These are of two types in the heart, the T (for transient) channels and the L (for long-lasting) channels. The calcium current (I_{Ca}) due to opening of T channels completes the prepotential, and I_{Ca} due to opening of L channels produces the impulse. Other ion channels are also involved, and there is evidence that local Ca^{++} release from the sarcoplasmic reticulation (***Ca++ sparks***) occurs during the prepotential.

The action potentials in the SA and AV nodes are largely due to Ca^{++}, with little contribution by Na^+ influx. Consequently there is no sharp, rapid depolarizing spike before the plateau, as there is in other parts of the conduction system and the atrial and ventricular fibers. In addition, prepotentials are normally prominent only in the SA and AV nodes. However, there are 'latent pacemakers' in other portions of the conduction system that can take over when the SA and AV nodes are depressed or conduction from them is blocked. Atrial and ventricular muscle fibers do not have prepotentials, and they discharge spontaneously only when injured or abnormal.

(Ref. Ganong 22th edition; page 548)

Conductivity

Impulse generated from SA node of heart transmit to its epicardial surface through junctional tissue in it.

Q. ***Describe how cardiac impulses is conducted through out the heart?***

Ans. The impulse is originated in the SA node at the rate of 70-80 imp/min. From the SA node the impulse then passes to the AV node through the internodal pathway approximately 0.04 sec. after its oirigin in the SA node. There are three internodal pathway -

i. Anterior.
ii. Middle.
iii. posterior.

The impulse then reaches to the junctional fibres of AV node. From the junctional fibers the impulse reaches to the AV nodal fibers. It takes about 0.06 sec. The impulse then travels through AV nodal fibres to the transitional fibers. There is a delay of about 0.1 second (AV nodal delay) before excitation spreads to ventricle.

From transitional fibers the impulse is transmitted along Bundle if His. The impulse from the Bundle of His passes quickly through the right and left bundle branches.

From the branches of the Bundle of His the impulse quickly passes through the purkinje fibers and spread all over the ventricular muscles.

In human, depolarization of the ventricular muscle starts at the left side of the interventricular septum and moves first to the right across the midportion of the septum. The wave of depolarization then spreads down the septum to the apex of the heart. It returns along the ventricular walls to the AV grooves, proceeding from the endocardial to the epicardial surface. The last part of the heart to be depolarized are the posterobasal portion of the left ventricle, pulmonary conus, and the upper most portion of the septum. *(Pl follow the fig. 6.5)*

(Ref. Guyton 11th ed & Ganong 22th ed; Page-547)

Schematic representation of pathway of impulse in the heart :

SA node

↓

Right atrial muscle | Anterior, middle and posterior internodal pathways | Bachmann's bundle

↓ | ↓ | ↓

AV node | *Left atrial muscle*

↓

His bundle and its branches

↓

Purkinjee fibers

↓

Ventricular muscle.

Excitability & Contractility

Heart muscles responds to a stimulus of adequate strength and duration. The nature of stimulus may be electrical, mechanical or thermal The responds are also electrical, mechanical or thermal.

Excitability : Heart muscle shows excitability, that is, when subjected to a stimulus, it develops a transmembrane action potential (AP). Normally, the SAN regularly generates stimulus → the stimulus is conducted (via conducting tissues) to every single myocardial cell → all such myocardial cells, (because they are excitable), responds by AP.

The resting membrane potential of individual mammalian cardiac muscle cells is about -90 mV (interior negative to exterior). Stimulation produces a propagated action potential that is responsible for initiating contraction.

Depolarization proceeds rapidly, and an overshoot is present, as in skeletal muscle and nerve. But this is followed by a *plateau* before the membrane potential returns to the baseline. In mammalian hearts, depolarization lasts about 2 ms, but the plateau phase and repolarization last 200 ms or more. Repolarization is therefore not complete until the contraction is half over. With extracellular recording, the electrical events include a spike and a later wave that resemble the QRS complex and T wave of the ECG.

As in other excitable tissues, changes in the external K^+ concentration affect the resting membrane potential of cardiac muscle, whereas changes in the external Na^+ concentration affect the magnitude of the action potential.

i. The initial rapid *depolarization* and the overshoot *(phase 0)* are due to opening of voltage-gated Na^+ channels similar to that occurring in nerve and skeletal muscle.

ii. The initial rapid *repolarization (phase 1)* is due to closure of Na^+ channels.

iii. The *subsequent prolonged plateatm (phase 2)* is due to a slower but prolonged opening of voltage-gated Ca^{2+} channels.

iv. Final repolarization *(phase 3)* to the resting membrane potential *(phase 4)* is due to closure of the Ca^{2+} channels and K^+ efflux through various types of K^+ channels.

The voltage-gated Na^+ channel in cardiac muscle has two gates an outer gate that opens at the start of depolarization, at a membrane potential of -70 to -80 mV; and an inner gate that then closes and precludes further influx until the action potential is over (Na^+ channel inactivation).

The slow Ca^{2+} channel is activated at a membrane potential of -30 to -40 mV.

There are three types of K^+ channels that produce repolarization. The *first* produces a transient, early outward

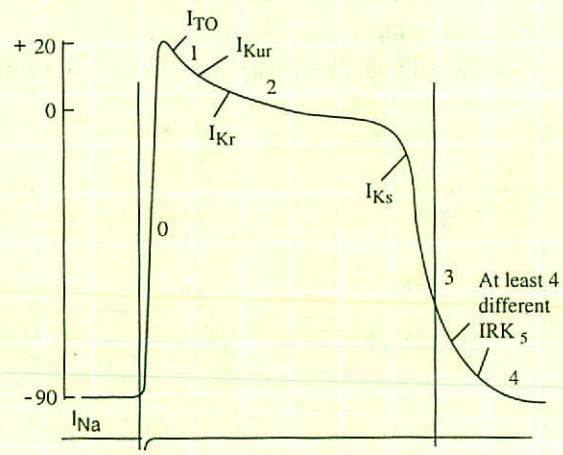


Fig: 6-3. *Phases of the action potential of a cardiac muscle fiber. 0, depolarization due to rapid influx of Na^+ ions; 1, initial rapid repolarization due to closure of Na channels; 2, plateauphase due to slow influx of Na^+ and Ca^{++} through the slow Na^+-Ca^{++} channels; 3, late rapid repolarizaion due to eflux of K^+ ions; 4, base line.*

current (I_{TO}) that produces an early incomplete repolarization. The *second* is inwardly rectifying ie, at plateau potentials it allows K^+ influx but resists K^+ efflux, and only at lower membrane potentials does it permit K^+ efflux. The current it produces is called I_{Kr}. The *third* type is a slowly activating (delayed rectifying) type that produces a current called I_{Ks}. The sum of I_{Kr} and I_{Ks} is a small net outward current that increases with time and *produces repolarization.*

N.B. In cardiac muscle, the repolarization time decreases as the cardiac rate increases.

(Ref. Ganong 22th Edition; Page-78, 80)

The agent which increases the excitability of heart muscle are known as Bathmotropic agent The excitability depends on -

i. Nutrition
ii. O_2 supply
iii. Nerve supply
iv. Optimum concentration of electrolytes.

Contractility : Heart responds the excitation by contraction. This phenomenon occurs due to chemical and mechanical changes in the contractile element- *actin & myosin.* The agent which increases the contractility is called ionotropic agent.The basic mechanical property of the heart muscles is that it can contract. Contraction may or may not cause, shortening. Neverthless, all contractions generate force and produce squeeze on the blood in ventricular or atrial cavities → which in turn, ultimately speaking, causes ejection of blood from these cavities.

When free Ca^{++} ions are available in sufficient concentration within the WMC (or SMC), the Ca^{++} ions bind with the troponin C.

This results in some physiochemical change in the (resting) actin tropomyosin complex, so that tropomyosin loosens → nextly the tropomyosin moves away from the actin → the myosin head binding sites of actin are uncovered.

Nextly, the globular head of myosin attaches with the actin, and this constitutes the cross bridge → swivelling action → movement of actin → shortening.

In relaxed state, the free (Ca^{++}) in the ICF is so low that the tropomyosin continues to cover the active sites of the thin filament.

After the end of a contraction, the ICF free Ca^{++} concentration falls again (relaxation develops).

a. ***All or none law*** : If an adequate stimulus is applied to heart muscle, the heart muscle responds to its maximum, but if the stimulus is not adequate it does not responds at all.

In 1871, Bowditch discovered this law, which, in short, is : apply a thrsehold stimulus (= a stimulus which is just sufficient, neither excess nor less), to the heart, the heart contracts. Now, apply a stronger stimulus. Heart contracts but force of this contraction is not more than that created by the threshold stimulus. Conclusion is, by increasing the strength of stimulus, the force of contraction cannot be increased. So far as the strength of stimulus is concerned heart therefore shows, all or none law, i.e., it contracts with requisite force (when the stimulus is adequate) or does not contract at all (when it is below adequate). (Force of contraction, as stated above, can be changed only by via Frank Starling's law or by changing its inotropic state).

b. ***Frank Starling law*** : Within the physiological limit the greater the length of cardiac muscle fibre the greater will be the force of contraction.

Within physiological limit, greater the end diastolic fibre length of the myocardium (ie greater the end diastolic volume), greater is the force of contraction.

Importance of Frank-Starling's law :

1. *This law ensures that output per stroke of one ventricle is the same in the other ventricle. Thus it can readily be seen that it is a life saving device* : Suppose, the right ventricular output per stroke is slightly greater than that of the left. This will cause accumulation of blood in the left ventricle. But because of the presence of the Frank-Starlingís law, as the blood accumulates, the Frank-Starlingís law begins to operate → more complete evacuation of the left ventricle → the two ventricles now will have the same output again. Lack of presence of this law, then, would have produced a catastroke (accumulation of blood in the pulmonary circulation → pulmonary edema).
2. *Frank-Starling's law is a life saving device in cardiac failure* : Suppose, there is left ventricular failure (LVF) due to insufficiency of contraction of the left ventricle → accumulatiom of blood within the left ventricle and lack of blood supply to the vital organs like brain (which can kill the patient) → accumulation of blood in the ventricle leads to operation of the Frank Starling's law → greater cardiac output → satisfactory supply to the vital organs. In LVF, therefore, the contractility of the left ventricular muscle is poor, but this is compensated by the operation of Frank Starling's law.

Q. 04. **Briefly explain the causation of plateau.**

Ans. *Causation of plateau* :

i. The prolonged plateau *(phase 2)* is due to a slower but prolonged opening of voltage-gated Ca^{++} channels. The slow Ca^{++} channel is activated at a membrane potential of -30 to -40 mV. Ca^{++} channels allow diffusion of mainly of Ca^{++} but of some Na^+ ions as well.
ii. A second factor sometimes partly responsible for the plateau

is that the voltage-gated K^+ channels are even slower than usual to open, often not opening greatly until the very end of the plateau. This delays the return of the membrane potential toward the resting value.

(Ref. Guyton 10th Edition; Page-66)

Factors influencing myocardial contractility

Ans. Factors influencing myocardial contractility are :

I. *Positively inotropic effect exerted by* :
   i. *Sympathetic nerves* : The whole length-tension curve shifts upward and to the left
   ii. *Catecholamines* exert their inotropic effect : *Norepinephrine and epincphrine* : The positively inotropic effect of the norepinephrine liberated at the nerve endings is augmented by circulating norepinephrine, and epincphrine has a similar effect.
   iii. *Xanthines* such as *caffeine* and *theophylline* that inhibit the breakdown of cAMP are positively inotropic.
   iv. *Digitalis and related drugs*
   vi. *Glucagon* which increases the formation of cAMP, is positively inotropic, and it has been recommended for use in the treatment of some heart diseases
   vi. *Changes in cardiac rate and rhythm* also affect myocardial contractility : myocardial contractility increases as the heart rate increases
   viii. *Intracellular* Ca^{++} : Next succeeding contraction is stronger than the preceding normal contraction.

II. *Negatively inotropic effect exerted by* :
   i. Parasympathetic nerves : There is a negatively inotropic effect of *vagal stimulation* on the atrial muscle and a small negatively inotropic effect on the ventricular muscle.
   ii. Hypercapnia, hypoxia, acidosis, drugs such as quinidine, procainamide, and barbiturates depress myocardial contractility.

(Ref. Ganong 22th ed; Page-575, 576)

Q. 00. **What is ionotropic effect?**

a. *Definition* : Alteration of the force of contraction of the heart is termed as ionotropic effect.

b. *Type* :
   i. Positive inotropic effect
   ii. Negative inotropic effect.

Positively inotropic effect exerted by :
   i. Sympathetic nerves
   ii. Catecholamines i.e orepinephrine and epincphrine
   iii. Xanthines such as caffeine and theophylline
   iv. Digitalis and related drugs
   vi. Glucagon
   vi. Changes in cardiac rate and rhythm
   vii.. Intracellular Ca^{++}.

Negatively inotropic effect exerted by :
   i. Parasympathetic nerves
   ii. Hypercapnia
   iii. Hypoxia
   iv. Acidosis
   v. Drugs such as quinidine, procainamide, and barbiturates.

Q. 00. **What is chronotropic effect?**

a. *Definition* : Any alteration of the heart rate by nervous stimulation or drugs is termed as chrono- tropic effect.

b. *Type* :
   I. *Positive chronotropic action* : Means an increase in the heart rate.
   Cause :
      i. Sympathetic stimulation
      ii. Adrenergic drugs i.e adrenaline, noradre- naline
      iii. Anticholenergic drugs i.e atropine.
   II. *Negative chronotropic action.*: Means a decrease in the heart rate.
   Cause :
      i. Parasympathetic stimulation
      ii. Drugs : adrenergic antagonist i.e reserpine, ergotamine etc.

Role of Ca^{++} ions in cardiac action potential

i. Ca^{++} is the current carrying ion in pacemaker tissues- thus produces depolarization in pacemaker tissues.
ii. Ca^{++} is responsible for cardiac action potential (in skeletal muscle and axon Ca^{++} is not involved in action potential).
iii. The involvment of Ca^{++} conductance in cardiac muscle is responsible for the long duration of the cardiac action potential (about 250 ms) copared with only a few milliseconds in skeletal muscle and axon.
iv. The force of myocardial contraction (ionotropic effects) is regulated by influx of Ca^{++} into myocytes.

Refractory period

It is the period during which heart muscle is nonresponsive to external stimuli or restimuli. Refractory period of heart is 0.30 sec. Refractory period is of two types :

i. Absolute refractory period
ii. Relative refractory period.

1. ***Absolute refractory period :*** It is the period of conduction during which heart muscle is refractory to a restimulus.
   The whole depolarization and first one third of repolarization is absolute refractory period. It is about 0.25 second.

2. ***Relative refractory period :*** It is the period of conduction during which heart muscle responds a slight to a very

strong stimulus. The last two third of the repolariztion is relative refractory period. It is about 0.05 sec.

Significance :

i. Heart muscle can not be tetanized.
ii. Heart muscle can not be fatigued.
iii. Ensures enough time for recovery of cardic muscle by getting nutrition and O_2 supply.

During this refractive period heart muscle ensure enough time for recovery by getting nutrition and O_2 supply. For this reason heart muscle is not tetanized or fatigued.

Tonicity

It is state of partial contraction of the heart muscle over the contained blood.

Q. ***What is "pace maker" ? Why SA node is called so?***

Ans. *Pace maker* :

Definition : The general meaning of pace maker is that the rider or runner who sets the pace of the race.

SA node is called the pace maker. It produces 70-80 impulse/minute, which coincides with heart rate.

SA node is called pace maker as :

i. It originates the impulses at first.
ii. It maintains the normal cardiac rhythm.
iii. The rate and rhythm originated by SA node is higher than that of all.

Q. ***Prove that SA node is the pace maker of heart ?***

Ans : Evidences -

1. Its destruction or removal causes cessation of atrial contraction.
2. In high temparature, the SA node is heated so, the rate of heart is increased, selective heating or cooling of this part hasten's or slow's the rate of heart.
3. SA node is first part of heart to show electronegativity.
4. In tissue culture, the pace maker tissues are first to show activity.

Q. ***Why does the SA node control the heart's rhythmicity rather than AV node or Purkinje fibre ?***

Ans. The discharge rate of SA node is much more greater than either AV node or Purkinje fibers. Each time the sinus node discharges, its impulse is conducted into both the AV node & purkinje fibers, discharging their excitable membrane. But the sinus node loses it hyperpolarization much more rapidly than does either of the other two and emits a new impulse before either one of them can reach its own thresold for self excitation. The new impulse again discharges both the AV node and purkinje fibres. This process continue on and on. Thus the SA node controls the beat of heart.

(Ref. Guyton & Hall-11th edition)

Q. ***Describe the velocity of conduction and the rate of impulse generation of heart muscles.***

Ans.

Tissue	*Volocity of conduction. (Meter/second)*	*Rate of impulse generation (Impulse/minute)*
SA node	0.05	70 - 80
AV node	0.05	40 - 60
Bundleof His	*1*	*30 - 36*
Purkinje fiber system	*4*	*15 - 40*
Atrial pathway	*1*	*60*
Atrial muscle	0.3	-
Ventricular muscle	1	20 - 40
	(Ref. Ganong 22th edition)	*(Ref. Guyton 11th edition)*

Q. ***Describe the rate and time taken by impulses to conduct from SA node to heart muscles.***

Ans.

1. From SA node to atrial muscle	: 0.03 second.
2. From SA node to AV node through internodal pathway	: 0.03 second.
3. AV node itself before enters to penetrating portion of AV bundle	: 0.09 second.
4. Penetrating portion of AV bundle	: 0.04 second.
**SA node up to distal portion of AV bundle i.e. from atria to ventricle (.03 +.09 +.04 sec)*	*: 0.16* second.
5. Distal portion of AV bundle to termination of purkinje fiber (i.e up to one third of ventricular muscle)	: 0.03 second.
6. From endocardial surface to epicardial surface of the ventricle	: 0.03 second.
** Initial bundle branches to the last of the ventricular fibers (0.03 + 0.03)*	*: 0.06 second.*

Note: Purkinje fiber leads from AV node through AV bundle into the one third of ventricular muscles.

(Ref. Guyton & Hall-11th ed; Page 116, 117, 118)

Reserve Pace Maker

When the SA node fails to generate impulse then the AV node become the pace maker and produces impulse at its own rate of 40-60 impulse/min. So it is called reserve pace maker. Then the heart contracts at a rate of 40-60/minute.

(Ref. Guyton 11th ed; Page-120)

Ectopic Pace Maker

A pace maker elsewhere than SA node is called ectopic pace maker. Obviously, an ectopic pace maker causes an abnormal sequence of contraction of different part of heart.This often occurs inthe AV node or in purkinje fibers. Under rare conditions a point in the atrial or ventricular muscle develops excessive excitability and becomes the pace-maker.

(Ref. Guyton 11th ed; Page-120)

AV nodal delay

AV node is responsible for the delay in transmission of impulse generated in the SA node. This delay in impulse transmission is called AV nodal delay. It is about 0.09 sec.

Causes of AV nodal delay :

i. Junctional fibres of the AV node are very small in size.
ii. Resting membrane potetials of these fibres are much less negative than the normal resting membrane potential of other cardiac muscle.
iii. Very few gap junctions connect the successive fibres in the pathway, so that there is great resitance to the conduction of excitatory ions from one fibre to the next.
iv. Prolonged refractory period of AV node.

Importance of AV nodal delay : AV nodal delay allows time for the atria to empty their content into the ventricles before the ventricular contraction begins.

(Ref. Guyton 11th edition; page 118)

Idioventricular rhythm of cell

If impulses generated in SA node fails to reach AV node, the AV node generates impulse for the rest of the heart at a rate of about 40 -60 impulse/min. This is called idioventricular rhythm of cell.

Stokes Adams syndrome

When AV block occurs that is, when the cardiac impulse fails to pass from the atria into the ventricles through the AV nodal and bundle system - the atria continue to beat at the normal rate of rhythm of the sinus node, while a new pacemaker develops in the purkinje system of the ventricles and drives the ventricular muscle at a new rate somewhere between 15 and 40 beats per minute. After a sudden block, the Purkinje system does not begin to emit its rhythmical impulses until 5 to 30 seconds later because, before the blockage, the Purkinje fibers had been "overdriven" by the rapid sinus impulses and, consequently, are in a suppressed state. During these 5 to 30 seconds, the ventricles fail to pump blood, and the person faints after the first 4 to 5 seconds because of lack of blood flow to the brain. This delayed pickup of the heart beat is called Stokes Adams syndrome. If the delay period is too long it can lead to death.

(Ref. Guyton 11th ed; Page-121)

Ventricular escape

Stimulation of the vagus nerve decreases the rate and rhythm of SA node and also decreases the excitability of AV junctional fibres there by slowing transmission of cardiac impulse into ventricle.

Very strong stimulation of the vagi can completely stop the rhythmic contraction of SA node or completely block transmission of cardiac impulse through the AV junction. As a result, the ventricles stop beating for 4 to 10 sec. But then some point in purkinje fibers, usually in the ventricular septal porton of the AV bundle develops rhythm of it's own and causes ventricular contraction at a rate of 15-40 beats per minute. This is called ventricular escape.

(Ref. Guyton& Hall-11th edition; Page-121)

Cardiac Cycle

Definition : The cardiac events that occur from the begining of one heart beat to the begining of the next are called the cardiac cycle. ***or***

(Ref. Guyton & Hall-11th edition; Page 106)

The sequence of events that occur in heart in each beat with contraction and relaxation is called cardiac cycle. ***or***

The cyclic repeatation of the various changes in heart from beat to beat, is called cardiac cycle.

Cardiac cycle time

The time taken for one complete cardiac cycle to occur with a normal heart rate.

If the normal heart rate = 75

The cardiac cycle time will be $= \frac{60}{75}$ second.

$= 0.8$ second.

The cardiac cyclle time will be inversely proportional to the heart rate.

Law of cardiac cycle

1. Ventricular systole and diastole does not overlaped by each other.
2. Similarly atrial systole and diastole does not overlaped by each other.
3. Atrial systole is overlaped by the ventricular diastole.
4. Atrial systole coincides with last rapid filling phase.
5. Cardiac cycle time is inversely proportional to heart rate.

Systole

The period of contraction of heart is called cardiac systole or systole.

(Ref. Guyton & Hall-11th Edition; page 106)

Diastole

The period of relaxation of heart is called cardiac diastole or diastole.

The cardiac cycle consist of a systole and diastole.

(Ref. Guyton & Hall-11th Edition; page 106)

Events of cardiac cycle

In Atria :

1. *Atrial systole* : 0.1 second.
2. *Atrial diastole* : 0.7 second.

In Ventricle :

1. *Ventricular systole* : 0.3 second.
   a. Isovolumetric (Isometric) contraction phase : 0.02 to 0.03 second (0.05 second, *Ganong 21th Edition*).
   b. Rapid ejection phase
   c. Reduced ejection phase
2. *Ventricular diastole* : 0.5 second.
   a. Protodiastolic phase : 0.04 second.
   b. Isometric (volumetric) relaxation phase : 0.03 to 0.06 second.
   c. First rapid filling phase
   d. Slow filling phase — 0.4 sec
   e. Last rapid filling phase.

Atrial Systole : It initiates the cardiac cycle as the pace maker (SA node) is in the atria. Contraction of atria propels some additional blood into the ventricles (about 30%). Contraction of the atrial muscle that surrounds the orifices of the superior and inferior vena cava and pulmonary veins narrows their orifices and the inertia of the blood moving towards the heart tends to keep blood in it. However, there is some regurgitation of blood into the veins during atrial systole. About 30% of ventricular filling occurs actively during atrial diastole.

(Ref. Ganong 22th edition; Page 565)

Atrial Diastole : At the end of atrial systole atrial diastole occurs and during this period blood enters into atria from great veins. Its duration is about 0.7 sec. About 70% of ventricular filling occurs passively during atrial diastole.

(Ref. Ganong 22th edition; Page 565)

Ventricular Systole : It starts at the end of atrial systole and lasts for 0.3 second. At the onset of ventricular systole A-V valve closes and produces 1st heart sound. It has following phases-

a. ***Isovolumic or isometric contraction phase*** *:* (Iso= same, metric= lenght) In this phase muscle does not shorten (this is not strictly true because there is apex-to-base shorting and circumferetial elongation.) but the tension increases which is called isometric contraction. At the begining of the ventricular systole (there is a brife period during which) both valves (AV and semilunar) remain closed and the ventricle contracts as a closed cavity. It is called *isometric contraction phase*. This period of isovolumetric contraction lasts about 0.02 to 0.03 second (0.05 second, *Ganong 20th ed*), until the pressures in the left and right ventricles exceed the pressure in the aorta (80 mm of Hg) and pulmonary artery (10 mm of Hg) and the aortic and pulmonary valves open. During isovolumetric contraction, the AV valves bulge into the atria, causing a small but sharp rise in atrial pressure. In this period no blood passes out. It is marked at the onset by the closure of AV valve and its termination by the opening of semilunar valve.

(Ref. Guyton 11th ed; P-108 and Ganong 22th ed; P-565)

b. ***Ejection phase*** *:* During isometric contraction phase the pressure within the ventricle sharply rises and semilunar valve opens. The intraventricular pressure rises to a maximum and then declines somewhat before ventricular systole ends. Peak left ventricular pressure is about 120 mm of Hg, and peak right ventricular pressure is about 25 mm of Hg or less . It has two parts-

   i. *Rapid ejection phase :* It is 1st third of ejection phase during which out flow is about 70% .

   ii. *Slow ejection phase :* It is the last third of ejection phase during which out flow is minimum about 30%.

(Ref. Guyton 11th ed; P-108 and Ganong 22th ed; P-565)

Ventricular Diastole : It starts at the end of ventricular systole and lasts for 0.5 second. Its has following phases -

a. *Protodiastolic phase :* It is the interval between the onset of diastole and closure of semilunar valve. As the diastole starts the pressure in the ventricles falls and after a short interval semilunar valve is closed by the high pressure in aorta. *It produces 2nd heart sound. Duration* : 0.04 second.

b. *Isometric relaxation phase* : It is the interval between the closure of semilunar valve and opening of A-V valve. In this phase both A.V and semilunar valve remain close & the ventricle relaxes at a closed cavity. No blood enters into the ventricle in this phase and there is sharp fall of intraventricular pressure. *Duration :* 0.06 second (0.03-0.06 second).

c. *Filling phase* : At the end of isometric relaxation phase the ventricular pressure falls and AV valve open due to high pressure in atria. Then blood enters into ventricle and ventricular filling phase begins at a ventricular volume of about 45 ml and a diastolic pressure near 0 mm Hg and with the volume increasing to 115 ml and the diastolic pressure rising to about 5 mm Hg.

(Ref. Guyton & Hall-11th edition; page 108)

It has following phase-

   i. *1st rapid filling phase :* It is the 1st part of filling phase. At this phase the intraventricular pressure marked fall and inflow of blood become more intense. So, blood rushes from atria to ventricle and 3rd heart sound will be produced. *Duration :* 1st third of diastole.

   ii. *Slow filling phase :* The amount of filling in this period is minimum.

The filling is slow as :

a. Ventricle already filled with large extent
b. The ventricular pressure slowly rises
c. The valve remains flattering condition.

Duration : Middle third of diastole.

iii. *Last rapid filling phase :* Here ventricular diastole coincides with atrial systole. Due to atrial contraction blood rushes into the ventricle in this phase and produce 4th heart sound. *Duration :* Last third of diastole.

Total filling time : 0.4 second.

(Ref. Guyton 11th ed; P-108 and Ganong 22th ed; P-565 and others)

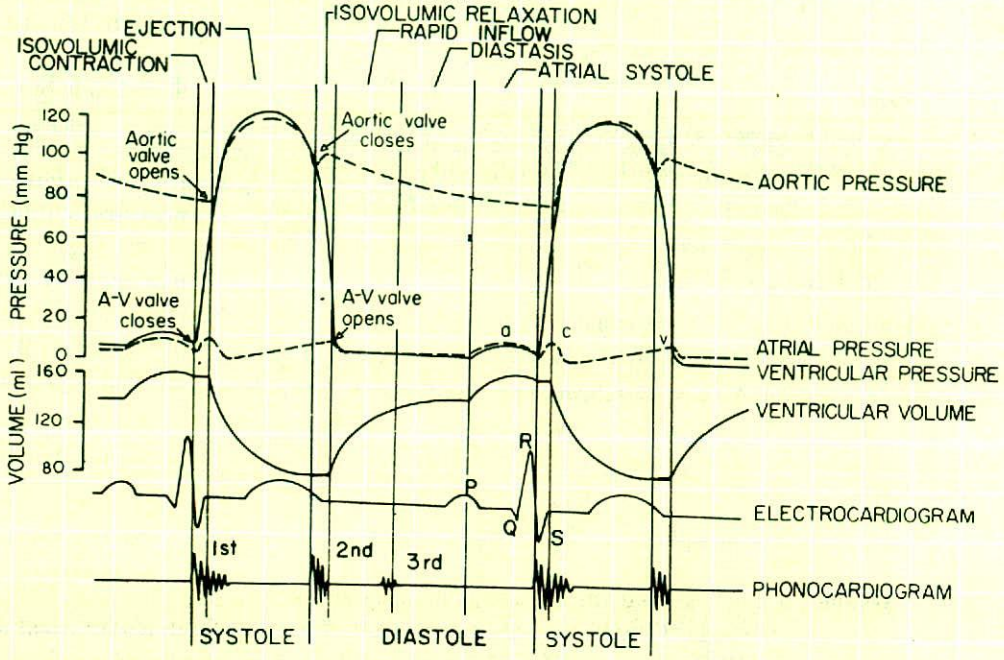


Fig. 6-4. *Events of the cardiac cycle for left ventricular function, showing changes in left atrial pressure, left ventricular pressure, aortic pressure, ventricular volume, the electrocardiogram, and the phonocardiogram.*

Pressure changes during cardiac cycle

Prssure in aorta and right & left ventricles in systole & diastole :

	Aorta (mm Hg)	*Left ventricle (mm Hg)*	*Right ventricle (mm Hg)*
Systole	*120*	*120*	*25*
Diastole	*80*	*0 - 5*	*0*

(Ref. Ganong-22th edition; Guyton 11th edition)

Prssure changes during cardiac cycle :

1. Right atrium : 0-8 mm of Hg
2. Left atrium : 4-12 mm of Hg
3. Right ventricle :
   a. During systole : 15-30 mm of Hg
   b. During diastole : 0-5 mm of Hg
4. Left ventricle :
   a. During systole : 90-140 mm of Hg
   b. During diastole : 04-12 mm of Hg
5. Pulmonary artery :
   a. During systole : 15-30 mm of Hg
   b. During diastole : 05-15 mm of Hg
6. Aorta :
   a. During systole : 90-140 mm of Hg
   b. During diastole : 60-90 mm of Hg

(Ref. Davidson's Medicine)

Relationship of the electrocardiogram to the cardiac cycle

The electrocardiogram shows the P, Q, R, S, and T waves, which are discussed later. They are electrical voltages generated by the heart and recorded by the electrocardiograph from the surface of the body.

The *P wave* is caused by spread of depolarization through the atria, and this *is followed by atrial contraction*, which causes a slight rise in the atrial pressure curve immediately after the P wave. About 0.16 second after the onset of the P wave, the *QRS waves* appear as a result of depolarization of the ventricles, which *initiates contraction of the ventricles* and causes the ventricular pressure to begin rising. Therefore, the QRS complex begins slightly before the onset of ventricular systole.

Finally, one observes the ventricular T wave in the electrocardiogram. This represents the stage of repolarization of the ventricles, at which time the ventricular muscle fibers begin to relax. Therefore, the *T wave* occurs *slightly before the end of ventricular contraction.*

(Ref. Guyton & Hall-11th edition; Page-107)

Changes takes place during cardiac cycle

The changes associated with cardiac cycle are as follows :

1. *Changes in pressure* :
   i. Intraventricular pressure change.
   ii. Intra atrial pressure change.

iii. Pressure change within aorta.
iv. Pressure changes within pulmonary artery.

2. Changes in volume :
   i. Atrial volume change
   ii. Ventricular volume change
3. *Production of heart sound and apex beat.*
4. *Production of pulse and appearance of pulse wave.*
5. Electrical change.
6. *Electro-cardiogram (ECG) changes.*
7. *Systemic, Pulmonary and coronary circulation changes.*

Discussion

A. ***Intra venricular pressuse changes***
   a. *During ventricular systole :*
      1. In isometric contraction period, intraven-tricular pressure sharply rises as ventricule contracts in a closed cavity due to closure of both AV and SL valve.
      2. In maximum ejection phase, since blood passes out pressure should fall in ventricle. But the force of contraction is stronger than out flow, so intraventricular pressure rises.
      3. In reduced ejection phase, force of contrac-tion is less than out flow so intraventricular pressure gradually fall.
   b. *During ventricular diastole :*
      1. In protodiastolic phase pressure continue to fall.
      2. In isometric relaxation phase pressure sharply fall until AV valve open because ventricle relax in a closed cavity.
      3. In first rapid filling phase, though blood rushes into ventricle but pressure slowly falls because rate of relaxation is stronger than filling.
      4. In last rapid filling phase, corresponds with atrial systole, blood pumps into ventricle and pressure suddenly rises.

B. ***Intra atrial pressure changes*** :
   a. During atrial systole, pressure sharply rises and causes first positive curve or a wave.
   b. During atrial diastole, pressure should fall in atria. But instead that, pressure sharply rises due to bulging of AV valve and backward flow of blood towards atria caused by isometric contraction phase. It causes c wave.
   c. Then pressure sharply fall because atria continue to relax and AV valve regained its position.
   d. At the later part of ventricular systole, intra atrial pressure rise and causes V wave.

C. ***Pressure change in aorta***
   1. In isometric contraction phase, slight aortic pressure rises due to bulging of SL valve towards aorta.
   2. When SL valve opens, blood enters into aorta and aortic pressure smoothly rises and falls running parallel with ventricular pressure.
   3. During diastole, pressure fall due to backward flow of aortic blood towards ventrcle.
   4. Then pressure slowly fall because blood passes to the periphery.

D. **Volume changes** :

End diastolic ventricular volume	: About 130 ml
End systolic ventricular volume	: About 50 ml

(Ref. Ganong 22th edition; Page-565)

End diastolic ventricular volume	: 110-120 ml
End systolic ventricular volume	: 40-50 ml

(Ref. Guyton Hall-11th edition; Page-108)

E. **Production of heart sound** :
   a. *1st heart sound :* Sudden closure of AV valve at the onset of ventricular systole.
   b. *2nd heart sound :* Closure of semilunar valves (aortic and pulmonary valves) just after the end of ventricular systole).
   c. *3rd heart sound :* Due to vibrations set up by the inrush of blood from atria to ventricle during the 1st rapid filling phase of ventricular diastole.
   d. *4th heart sound :* Due to inrush of blood from atria to ventricle during last rapid filling phase of ventricular diastole by atrial contraction.

Q. ***Why minimum pressure exist in arterial system during diastole?***

Ans. During systole the propulsive force of heart cause the systolic pressure but during diastole such propulsive force is absent. For this reason minimum pressure exist in the arterial system during diastole.

Actually the pressure would fall to a very low level but such does not happen due to the elastic recoil of the arteries.

Heart Sound

Definition : The vibratory motion of heart produced during the different events of cardiac cycle conducts through the structure surrounding the heart and produces special audible sound called heart sound.

Causes of heart sound

i. Vibration of the taut valves immediately after closure.
ii. Vibration of the adjacent blood.
iii. Vibration of the walls of the heart and major vessels around the heart.

(Ref. Guyton& Hall-11th edition; Page-269)

Classification of heart sound

Heart sound is classified into four in number :

i. 1st heart sound.

ii. Second heart sound.

iii. 3rd heart sound.

iv. 4th heart sound.

1st and 2nd heart sound is audible by stethoscope, 3rd and 4th heart sound is detected by phonocardiograph.

First heart sound

It is a low, slightly prolonged 'lub' (*first sound*), caused by vibrations set up by the sudden closure of the mitral and tricuspid valves at the start of ventricular systole.

Duration : About 0.15 second.

Fequency : 25-45 Hz.

It is soft when the heart rate is low, because the ventricles are well filled with blood and the leaflets of the AV valves float together before systole.

(Ref. Ganong 22th edition; Page 569)

Procedure of causes of first heart sound : In generating the first heart sound, contraction of the ventricles first causes sudden backflow of blood against the A-V valves (the tricuspid and mitral valves), causing them to close and bulge toward the atria until the chordae tendineae abrupty stop the backbulging. The elastic tautness of the chordae tendineae and of the valves then causes the backsurging blood to bounce forward again into each respective ventricle. This sets the blood and the ventricular walls as well as the taut valves into vibration and causes vibrating turbulence in the blood. The vibrations travel through the adjacent tissues to the chest wall, where they can be heard as sound by the stethoscope.

(Ref. Guyton 11th ed; Page 269)

Causes of loudness of 1st heart sound :

1. *Vigor of ventricular contraction :* Increased Sympathetic supply increases the myocardial contractility, the rate of change in ventricular pressure is increases, and a louder Ist heart sound may heard.
2. *The stiffness of ventricle and valve structures :* With fibrosis, the AV valves become stiffer, leading to a sound that is louder and some what higher in frequency then normal.
3. *The position of mitral valves leaflets at the begining of ventricular systole :* At the time of ventricular systole the mitral valve is widely open, by the time the valve closes, a significant amount of blood moving towards the valve and producing a noisy sound.
4. *P-R. interval :* It is the time between the atrial and ventricular exitation If P-R interval is shorter than normal the AV valve will be open at the onset of ventricular systole, So that when they finally are closed, the sound is louder than normal.

(Ref. Selkurt 5th page - 232)

Second heart sound

It is a shorter, high-pitched 'dup' (*second sound*), caused by vibrations associated with closure of the aortic and pulmonary valves just after the end of ventricular systole.

Duration : About 0.12 second.

Frequency : 50 Hz.

It is loud and sharp when the diastolic pressure in the aorta or pulmonary artery is elevated, causing the respective valves to shut briskly at the end of systole.

The interval between aortic and pulmonary valve closure during inspiration is frequently long enough for the second sound to be reduplicated (*physiologic splitting of the second sound*). Splitting also occurs in various diseases.

(Ref. Ganong 22th edition; Page-569)

Procedure of causes of second heart sound : The second heart sound results from sudden closure of the semilunar valves. When the semilunar valves close, they bulge backward toward the ventricles, and their elastic stretch recoils the blood back into the arteries, which causes a short period of reverberation of blood back and forth between the walls of the arteries and the semilunar valves, as well as between these valves and the ventricular walls. The vibrations occurring in the arterial walls are then transmitted mainly along the arteries. When the vibrations of the vessels or ventricles come into contact with a 'sounding board,' such as the chest wall, they create sound that can be heard.

(Ref. Guyton 11th edition; Page 269)

Third heart sound

A soft, low-pitched *third sound* is heard about one-third of the way through diastole in many normal young individuals. It coincides with the period of rapid ventricular filling and is probably due to vibrations set up by the inrush of blood.

Duration : The third sound has a duration of 0.1 second.

(Ref. Ganong 22th ed; Page 569)

Fourth heart sound

It can sometimes be heard immediately before the first sound when atrial pressure is high or the ventricle is stiff in conditions such as ventricular hypertrophy. It is due to ventricular filling and is rarely heard in normal adults.

(Ref. Ganong 22th ed; Page 569)

Q. ***Why 2nd heart sound has shorter duration than the 1st sound?***

Ans. *First sound* is caused by vibrations set up by the sudden closure of the mitral and tricuspid valves at the start of ventricular systole and the *second sound* is caused by vibrations associated with closure of the aortic and pulmonary valves just after the end of ventricular systole.

The reason for the shorter second sound is that the semilunar

valves are more taut than the A-V valves, so that they vibrate for a shorter period than do the A-V valves.

(Ref. Guyton & Hall-11th edition; Page 269)

Q. ***Why 1st heart sound has longer duration than the 2nd sound?***

Ans. *First sound* is caused by vibrations set up by the sudden closure of the mitral and tricuspid valves at the start of ventricular systole and the *second sound* is caused by vibrations associated with closure of the aortic and pulmonary valves just after the end of ventricular systole.

Semilunar valves are more taut than the A-V valves. The A-V valves vibrate for a longer period than do the semilunar valves. So the 1st heart sound has longer duration than the 2nd sound.

(Ref. Guyton & Hall-11th edition; Page 269)

Q. ***Why 2nd heart sound has a higher frequency than 1st sound?***

Ans. The second heart sound normaly has a higher frequency than the first heart sound for two reasons :

1. Because of the tautness of semilunar valves in comparison with much less taught AV valve.
2. Because of the greater elasticity coefficient of the arteries that provide the principal vibratring chambers for the second heart sound in comparison with the much losser vetricular chambers that provide the vibrating system for the 1st heart sound .

(Ref. Guyton & Hall-11th edition; Page 269)

Q. **Name the sites, where the heart sounds are best heard.**

Ans. There are four different areas on the chest wall where the heart sounds are best heard are given below :

i. *Mitral area :* On the apex beat, in the left 5th intercostal space, 9 cm from the midsternal line.

ii. *Tricuspid area :* Over the left border of the sternum in the left 4th intercostal space.

iii. *Aortic area :* At the right border of the sternum in the right 2nd intercostal space.

iv. *Pulmonary area :* At the left 2nd inter costal space, at the margin of the sternal border.

Clinical importance of heart sounds

The 1st and 2nd heart sound is normally heard through a stethoscope. The quality, character, and timing of these sounds may be altered in various cardiac diseases. So, 1st and 2nd heart sounds are important in :

i. Diagnosis of valvular heart diseases :
   a. Mitral stenosis : Loud 1st heart sound
   b. Calcified mitral valve : Soft 1st heart sound
   c. Aortic stenosis : Reversed splitting 2nd heart sound.

ii. Diagnosis of cardiodynamics status :
   a. Hyper dynamic condition : Loud 1st and 2nd heart sound

iii. Diagnosis of conduction defect :
   a. Bundle branch block : Wide physiological splitting of 2nd heart sound.

iv. Diagnosis of congenital heart disease.

v. Differentiating the murmur whether it is systolic or diastolic in origin.

Q. 04. **Name the clinically audible *(abnormal)* heart sounds with their significance.**

Ans. *Abnormal heart sounds are* : In addition to normal heart sounds, abnormal sounds may be produced during cardiac cycle due to abnormalities in cardiac
valves or abnormal blood flow and pressure gradients. Some abnormal sounds are :

i. Murmurs
ii. Gallop rhythm
iii. Pericardial friction rub
iv. Ejection click
v. Bruits.

i. Murmurs : Are produced by turbulent blood flow through an abnormal valve or high blood flow through a normal valve.
   Character : Murmurs are high frequency sounds and are often described as blowing in quality.
   Significance : Valvular stenosis, valvular incompetence.

ii. Gallop rhythm : The presence of a third or fourth heart sound produces a triple rhythm that, when associated with sinus tachycardia, sounds like a galloping horse- a *gallop rhythm*. The cadence of a gallop rhythm due to third heart sound has been lined to 'Kentucky' whilst that due to a fourth heart sound resembles 'Tennessee'.

ii. Pericardial friction rub : A pericardial friction rub is a scratching or crunching noise produced by the movement of inflamed pericardium. Since it is high frequency, it is best heard with the diaphragm of stethoscope. This is an extra cardiac sound.

iii. Ejection click : An ejection click sound occurs immediately following the first heart sound. The sudden opening of a deformed but mobile aortic or pulmonary valve produces the ejection click.

iv. Bruits : Are murmurs arising from a peripheral artery due to arterial stenosis.

Murmurs

Murmurs, or ***bruits,*** are abnormal sounds heard in various parts of the vascular system. The two terms are used interchangeably, though *murmur* is more commonly used to denote noise heard over the heart than over blood vessels.

Blood flow is laminar and nonturbulent up to a critical velocity; above this velocity, and beyond an obstruction, blood flow is turbulent. Laminar flow is silent, but turbulent flow creates sounds. Blood flow speeds up when an artery or a heart valve is narrowed.

Examples of vascular sounds outside the heart are the *bruit* heard over a large, highly vascular goiter, the bruit heard over a carotid artery when its lumen is narrowed and distorted by atherosclerosis, and the murmurs heard over an aneurysmal dilation of one of the large arteries, an arteriovenous (A-V) fistula, or a patent ductus arteriosus.

The major, but certainly not the only, *cause of cardiac murmurs* is disease of the heart valves. When the orifice of a valve is narrowed (***stenosis***), blood flow through it in the normal direction is accelerated and turbulent. When a valve is incompetent, blood flows backward through it (***regurgitation*** or ***insufficiency***), again through a narrow orifice that accelerates flow.

The timing (systolic or diastolic) of a murmur due to stenosis or insufficiency of any particular valve (*Table*) can be predicted from a knowledge of the mechanical events of the cardiac cycle. Murmurs due to disease of a particular valve can generally be heard best when the stethoscope is over that particular valve; thus-

i. Murmurs due to disorders of the aortic and pulmnonic valves are usually heard best at the base of the heart
ii. Murmurs due to mitral disease are usually heard best at the apex.

Table. *Heart murmurs.*

Valve	*Abnormality*	*Timing of Murmur*
Aortic or pulmonary	Stenosis	Systolic
	Insufficiency	Diastolic
Mitral or tricuspid	Stenosis	Diastolic
	Insufficiency	Systolic

There are other aspects of the duration, character, accentuation, and transmission of the sound that help to locate its origin in one valve or the other. One of the loudest murmurs is that produced when blood flows backward in diastole through a hole in a cusp of the aortic valve. Most murmurs can be heard only with the aid of the stethoscope, but this high-pitched musical diastolic murmur is sometimes audible to the unaided ear several feet from the patient.

In patients with *congenital interventricular septal defects*, flow from the left to the right ventricle causes a *systolic murmur.*

Soft murmurs may also be heard in patients with interatrial septal defects, although they are not a constant finding.

Soft systolic murmurs are common in individuals, especially children, who have no cardiac disease.

Systolic murmurs are also common in anemic patients as a result of the low viscosity of the blood and the rapid flow.

(Ref. Ganong 22th edition; Page 569)

Auscultation

Process of listening for sounds within the body, usually the sounds of thoracic or abdominal viscera by stethoscope inorder to detect some abnormallity.

(Ref. Taber's)

Apparatus for auscultating heart sounds : Heart sound can be detected by :

i. Clinical stethoscope
ii. Haemo-cardiography,
iii. Microphone with oscillography, and also recorded by the phonocardiograph.

Apex beat

Definition : Apex beat is the lowest and outer most point of definite cardiac pulsation.

(Ref. Hutchison's)

Location :

In the left 5th intercostal space, 9 cm from the midsternal line just medial to the left nipple.

Importance :

1. Measurement of hear rate.
2. Position of heart- whether dextrocardia or not.
3. Different heart disease diagnosis
4. Displacement of mediastinum due to pneumothorax, pleural effusion, left ventricular hypertrophy etc.

Pulse

Definition : Pulse is the rhythmic dilatation and elongation of arterial wall as a result of pressure changes created by the intermitten ejection of blood from heart to the already full aorta feeding the arterial system, transmit as a wave to the periphery.

Normal pulse rate :

i. Range : 60-90 /minute
ii. Average : 72 /minute.

Normal pulse

(Description of a pulse) : Normal pulse tracing is called catacrotic pulse. Pulse has a upstroke and down stroke wave, The upstroke wave is ***P*** and have no secon-dary wave in it. It is also called percussion wave. Near the middle of the downstroke there is a sharp depression called dicrotic notch ***D*** which is followed by a small wave called dicrotic

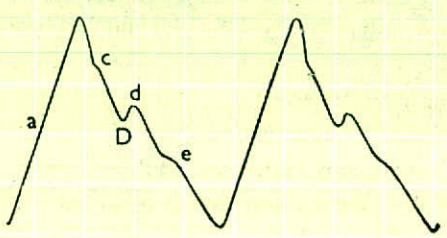


Fig. 6-5. *Normal pulse tracing.*

wave ***d***. Some time secondary oscillation may present above and below the dicrotic notch, These are called pre and post diorotic notch respectively.

P or percussion wave : It is the wave from the starting of the wave upto dicrotic notich, It coincides with ventricular systole.

Dicrotic notch : It is due to sharp fall of arterial pressure. At the end of the systole, aortic valve closes then the column of blood rolling towards left ventricle.

Dicrotic wave : It is due to refluxion of blood on the semilunar valve and produces this wave, It coincides with ventricular diastole.

Classification of pulse

On the basis of site :

A. *Arterial pulse :*
   i. Normal or catacrotic
   ii. Abnormal Pulse-
      a. Anacrotic pulse
      b. Dicrotic pulse
      c. Collapsing pulse
      d. Pulsus paradoxas
      e. Pulsus atterna's
      f. Pulsus bisferiense
      g. Pulsus deficit.

B. *Venous pulse.*

Arterial pulse depends upon :

i. Intermittent discharge of blood from the contracting left ventricles into the arterial system (aorta).
ii. Resistance to the blood in its passage from arterioles in to the capillaries.
iii. The elasticity of vessel wall.

Q. ***What are the points you will remember while examining a pulse ? or***
 # What do you understand by feeling a pulse?

Ans. During examining a pulse we remember the following points :

1. *Rate :* It is the number of pulse per minute. Normally it coincides with heart rate.
2. *Rhythm :* It indicates wheather the beats are equidistant or not that is either regular or irregularly regular or irregularly irregular.
3. *Volume :* It indicates the rise of pulse wave above the diastolic level. It varies with stroke volume. The greater the stroke volume the greater the volume of pulse.
4. *Condition of vessel wall :* By feeling the pulse we can know the condition of the blood vessels-
   a. Soft and easily compressable pulse indicates low cardiac output.
   b. Hard and not compressable pulse indicates atherosclerosis.
5. *Tension :* It is the approximate measure of systolic pressure.
6. *Character :* Wheather normal or abnormal.
7. Radio-femoral delay.

Site of feeling a pulse :

A. *Arterial pulse :*
   1. Radial artery
   2. Brachial artery
   3. Axillary artery
   4. Carotid artery
   5. Facial artery
   6. Femoral artery
   7. Popliteal
   8. Posterior tibial artery.
   9. Arteria dorsallis pedis.

B. *Venous pulse :* Jugular vein.

Procedure of feeling pulse :

Pulse are felt by placing three fingers side by side on the radial artery. Fingers are- index, middle and ring finger.

Q. ***What indicates three fingers?***

Ans.

a. *Index or proximal finger :* Adjust the pressure.
b. *Middle finger :* Remains stationary and feels the appearence and disapperarence of pulse wave.
c. *Ring or distal finger :* Applies a constant maximum pressure to stop retrograde ulnar colateral pulsation.

Significance of pulse :

1. *A Large primary wave indicates :*
   a. Large stroke volume
   b. Low peripheral resistance
   c. Slow heart rate.
2. *A small primary wave indicates :*
   a. Small output
   b. Rapid heart rate.
   c. High peripheral resistance
   d. Stiffness of vessel wall.

Abnormal pulses

1. *Anacrotic pulse :* When a secondary wave is found in the upstroke of pulse traching then it is called anacrotic pulse.
2. *Dicrotic pulse :* When the dicrotic wave become prominent and felt with finger then it is called dicrotic pulse.
3. *Sinus arrythmia :* When the frequency of pulse is

increased during inspiration and falls during expiration called sinus arrythmia. It is normally found in children but less commonly in adult, It occurs due to alternation of vagal tone during inspiration.

4. *Collapsing pulse (Water hammer pulse)* : It is characterised by rapid upstroke and decend of pulse wave without di crotic wave or notch. *Cause :* Aortic incompetence, patent ductus arteriosus, arteriovenous communication.
5. *Pulsus Bisferiens :* It is the combination of slow rising and collapsing pulse. *Cause :* Aortic stenosis and aortic regurgitation.
6. *Pulsus paradoxus :* Occationally the strength of pulse become strong, then weak, then strong, then weak and goes synchrony.
   Cause : pericardial effusion.
7. *Pulsus alternates :* The amplitude of pulse become alternately large and small. *Cause :* Hypertension, left vantricular forces (LVF).

(Ref. Guyton & Hall 11th edition and others)

Velocity of pulse wave

The speed at which the pulse wave travels. It is much more rapid (about 6 times) than the velocity of blood flow.

It depends on two factors :

i. Elasticity of vessel wall.
ii. Inertia of blood.

The velocity of pulse wave increases with age, as the co-efficient of elastic recoil diminishes.

Elasticity of vessel wall

The term is meant by the percentage increase in the volume of the artery with each mm of Hg rise in blood pressure.

Inter relation ship of velocity of pulse wave with elasticity and age-

Age	*Velocity*	*Elasticity*
5	3.6	0.47
20	5.2	0.33
40	7.1	0.24
70	9.5	0.18

Comment : With increasing age, velocity of pluse wave increases but elasticity of vessel wall decrease.

Jugular venous pulse (JVP)

'The internal jugular vein is in direct continuity with the right atrium. Observation of the column of blood in the internal jugular system is therefore a good measure of right atrial pressure.

Measurement : The jugular venous pressure is the vertical height between the manubriosternal angle and the top of the venous wave. The normal jugular venous pressure is usually 3-4 cm H_2O (the patient is positioned at about 45° to the horizontal while measuring JVP.

Jugular venous pulse wave : The jugular venous pressure wave is consists of the three peaks and two troughs. The peaks are described as a, c, and v waves and the troughs are known as x and y descents.

i. The a wave is produced by atrial systole
ii. The c wave is produced by early atrial relaxation
iii. The x descent occurs by atrial relaxation
iv. The v wave is produced by venous return fills the right atrium during continued ventricular systole
v. The y descent follows the v wave when the tricuspid value opens (fall in pressure).

Cardic Output

Definition : The amount of blood that ejected by each ventricle per minute is called cardiac output.

Or Cardiac out put is the quantity of blood pumped into the aorta each minute by the heart.

(Ref. Guyton & Hall-11th Edition; Page 232)

The Cardiac output can also be stated as a product of stroke volume and heart rate i.e.

CO = Stroke volume x heart rate
= 70 x 72 (*Here, Stroke volume = 70 ml/beat Heart rate = 72 beats/minute*)
= 5042 ml
= 5.04 liter/minute.
= About 5 liters/minute. *(Ref Ganong 22th edt page 570)*

Normal values of cardiac output :

i. For young, healthy men, resting cardiac output averages about : 5.6 liters/ minute.
ii. For woman, this volume is 10 to 20 percent less.
iii. The average cardiac output for the resting adult, is often stated to be almost exactly : 5 liters /minute.

(Ref. Guyton & Hall-11th Edition; Page 232)

Cardiac index

The cardiac output per minute per squire meter of body surface area is called cardiac index. *(3.2 L/min/Sq.m - Ganong)*

The average value is = 3.5 liter/min/sq.m of body surface area.

(Ref. Selkert 5th page -273)

Minute volume

It is the volume of blood ejected by each ventricle per minute. It

can be calculated by multiplying the rate of heart per minute by stroke volume.

Minute volume = Heart rate per minute x Stroke volume
= 76 x 70
= 5,250 ml.

Distribution of cardiac output

Cardiac output is distributed as follows :

Organs	*Cardiac output*		*% of total CO*
Liver	1450	ml/ minute	29 % of total CO
Kidney	1200	ml/ minute	21 % of total CO
Brain	750	ml/minute	15 % of total CO
Muscle	600-900	ml/minute	12-18 % of total CO
Coronary	225	ml/minute	4-5 % of total CO
Skin	400	ml/minute	8 % of total CO

(Ref. Guyton & Hall-11th edition)

Cordiac output & regional blood flow in a sedentary man

	Quite standing ml/min	*Exerise ml/min*
Cordiac outpur	*5900*	*24,000*
Blood flow to :		
Heart	*250*	*1000*
Brain	*750*	*750*
Active skeletul mucle	*650*	*20850*
Inactive skeletal muscle	*650*	*300*
Skin-	*500*	*500*
Kidney, liver, gastro intestinal tract etc.	*3100*	*60*

Factors affecting the cardiac output

The factors influence or affecting the cardiac output are :

A. *Physiological :*
1. *Age :* Cardiac output increases with age.
2. *Sex :* Cardiac output is 10% to 20% less in female than male due to less body wt. and surface area etc.
3. *Surface area :* More surface area more will be cardiac output.
4. *Posture :* Cardiac output is greater in sitting and lying than errect posture due to less venous return in errect posture
5. *Exercise :* Cardiac output markedly increases in severe exercise and small amount in moderate exercise due to excess muscular activity and venous return.
6. *Emotion :* Increases cardiac output.
7. *Temperature :* It increase heart rate thence cardiac output.

B. *Pathological :*
1. *Hyperthyroidism :* It increases CO due to increase heart rate and increase body metabolism.
2. *Anaemia :* In anaemia O_2 carrying capacity decreased to compensate this CO increases.
3. *Fever :* CO increases in fever due to increase metabolism and temperature.
4. Fibrilation & Flutter.
5. *Other condition :* Paget's disease, arterio-venous fistula.

Cardiac output decreases in :

a. Hypothyroidism
b. Haemorrhage
c. Congestive cardiac failure
d. Shock
e. Oligaemia.

Effect of various conditions on cardiac output

1. *No change :*
   a. Sleep
   b. Moderate changes in environmental temperature.
2. *Increase :*
   a. Anxiety and excitement (50-100%)
   b. Eating (30%)
   c. Exercise (up to 700%)
   d. High environmental temperature
   e. Pregnancy
   f. Epinephrine.
3. *Decrease :*
   a. Sitting or standing from lying position (20-30%)
   b. Rapid arrhythmias
   c. Heart disease.

(Ref Ganong 22th edition; page-572)

Factors Regulating Cardiac Output

1. *Venous return :* Any thing that increases or diminishes the venous return will alter the CO accordingly. Increased venous return increases end diastolic volume ' increases stroke volume, that increases CO.

   It depends on :
   a. Muscular activity.
   b. Pumping action of heart.
   c. Pressure gradient in vessels.
   d. Respiratory pump.

e. Gravity.

f. Vasomotor tone

2. *Force of contraction of heart :* When force of contraction of heart increases cardiac output also increases.

   It depends on :

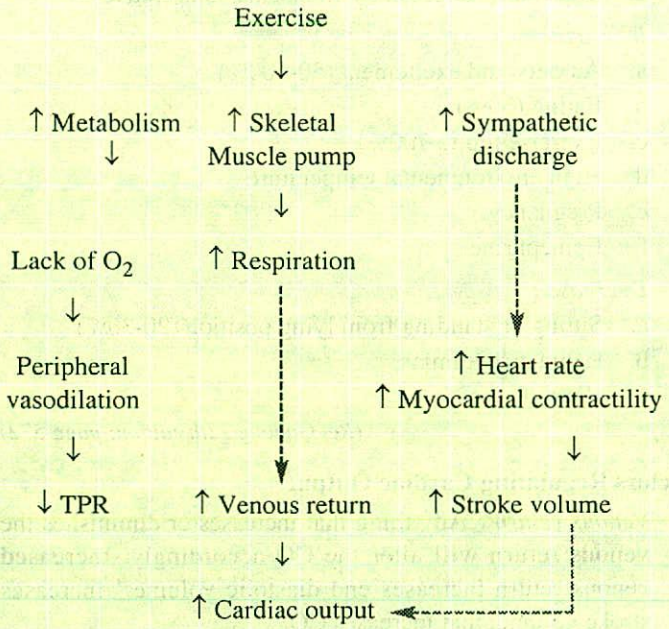
   a. Initial length of muscle fibre.
   b. Nutrition and O_2 supply of heart
   c. Diastolic phase.
   d. Sympathetic stimulation
   e. Circulating hormone level
   f. Ca^{++} level in blood.

3. *Frequency of heart beat :* Cardiac output is the product of stroke volume and heart rate. That is cardiac output = stroke vloume x heart rate. So, if stroke volume remains constant, then increased heart rate increases the cardiac out put that is cardiac out put is directly proportional to heart rate.

4. *Ejection fraction :* The fraction of end diastolic volume that is ejected is called the ejection fraction- usualy equal to about 60% of end diastolic volume.

5. *Peripheral resistance :* Increased peripheral registance → decreases venous return → decreased end diastolic volume → decreased stroke volume → decreased cardiac output.

Q. ***How Exercise increase cardiac output ?***



Methods of cardic output measurement

Cardiac output can be measured by the following way -

A. *Direct methods* (In animals).

B. *Indirect methods* (In human beings) : It is done by the help of following methods :

   a. Fick principle method by using O_2 or CO_2.
   b. Dye methods or indicator dilution technique
   c. Ballisto-cardiography.
   d. Heart lungs preparation.

Fick Principle Methhod

Fick principle : Fick principle states that the amount of a substance taken up by an organ (or by the whole body) per unit of time is equal to the arterial level of the substance minus the venous level (A-V difference) times the blood flow.

Consumption of a substance (ml/min) =

Arterio-venous difference (AO_2 - VO_2) of that substance
Blood flow (cardiac output).

This principle can be applied, of course, only in situations in which the arterial blood is the sole source of the substance taken up. The principle can be used to determine cardiac output by measuring the amount of O_2 consumed by the body in a given period and dividing this value by the A-V difference across the lungs. Because systemic arterial blood has the same O_2 content in all parts of the body, the arterial O_2 content can be measured in a sample obtained from any convenient artery.

A sample of venous blood in the pulmonary artery is obtained by means of a cardiac catheter. Right atrial blood has been used in the past, but mixing of this blood may be incomplete, so that the sample is not representative of the whole body. An example of the calculation of cardiac output using a typical set of values is as follows :

$$CO = \frac{O_2 \text{ consumption (ml/min)}}{\text{Arterio venous difference. } (AO_2 - VO_2)}$$

About 250 ml of O_2 are absorbed by the pulmonary blood from lungs per minute.

The arterial blood O_2 level is 190 ml/liter of blood and venous level of O_2 is 140 ml/liter of blood. So the arterio-venous difference is 50 ml/liter.

$$= \frac{250 \text{ ml/minute}}{\text{190 ml/L arterial blood - 140 ml/L venous blood in pulmonary artery.}}$$

$$= \frac{250 \text{ ml/minute}}{(190\text{-}140) \text{ ml /liter}}$$

$$= \frac{250}{50} \text{ liter/minute}$$

$$= 5 \text{ liter/minute.}$$

So , cardiac output is about 5,000 ml/minute or 5 liter/minute.

It has now become commonplace to insert a long catheter through a forearm vein and to guide its tip into the heart with the aid of a fluoroscope. Catheters can be inserted not only into

the right atrium but also through the atrium and the right ventricle into the small branches of the pulmonary artery.

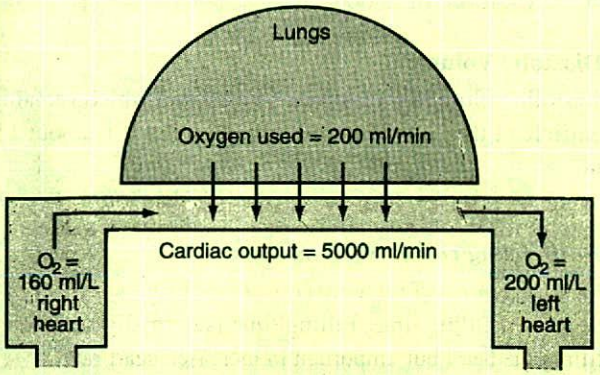


Figure. 6-6. *Fick principle for determining cardiac output.*

Catheters can also be inserted in peripheral arteries and guided in a retrograde direction to the heart and into coronary or other arteries.

(Ref. Ganong 22th Edition; Page 570)

Indicator dilution technique or dye method :

Principle : In measuring the cardiac output by the so-called indicator dilution mithod, a small amount of indicator, such as a dye, is injected into a large vein or, preferably, into the right atrium. This passes rapidly through the right side of the heart, the lungs, an the left side of the heart and, finally, into the arterial system. Then one records the concentration of the dye as it passes through one of the peripheral arteries, giving a *curve* such as one of the two curves shown in figure.

Procedure : In each of these instances, 5 milligrams of Cardio-Green dye was injected at zero time. In the top recording, none of the dye passed into the arterial tree until about 3 seconds after the injection, but then the arterial concentration of the dye rose rapidly to a maximum in about 6 to 7 seconds. After that, the concentration fell rapidly. Before the concentration reached the zero point, some of the dye had already circulated all the way through some of the peripheral vessels and returned through the heart for a second time. Consequently, the dye concentration in the artery began to rise again. For the purpose of calculation, it is necessary to extrapolate the early down-slope of the curve to the zero point, as shown by the dashed portion of the curve. In this way, the extrapolated time-concentration curve of the dye in an artery, without recirculation, can be measured in its first portion and estimated reasonably accurately in its latter portion.

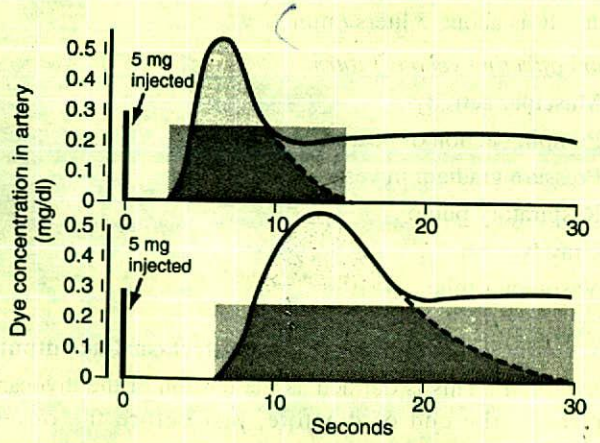


Figure. 6-7. *Extrapolated dye concentration curves used to calculate two separate cardiac output levels by the dilution method. (The rectangular areas are the calculated average concentrations of dye in the arterial blood for the durations of the respective extrapolated curves.)*

Once the extrapolated time-concentration curve has been determined, one can then calculate the mean concentration of dye in the arterial blood for the duration of the curve. In the top example of *figure*, this was done by measuring the area under the entire extrapolated curve and then averaging the concentration of dye for the duration of the curve; one can see from the shaded rectangle straddling the upper curve of the figure that the average concentration of dye was about 0.25 mg/dl of blood and that the duration of this average value was 12 seconds. A total of 5 milligrams of dye was injected at the beginning of the experiment. For blood carrying only 0.25 milligram of dye in each deciliter to carry the entire 5 milligrams of dye through the heart and lungs in 12 seconds, a total of 20 1-deciliter portions of blood would need to pass through the heart during this 12 seconds time, which would be the same as a cardiac output of 2 L/12 second or 10 L/minute.

To summarize, the cardiac output can be determined using the following formula :

$$\text{Cardiac output (ml/minute)} = \frac{\text{Milligrams of dye injected} \times 60}{\text{Average concentration of dye in each milliliter of blood for the duration of the curve} \times \textit{Duration of the curve in seconds}}$$

(Ref. Guyton& Hall-11th edition; Page 244)

Q. ***What are the purpose of cardiac output measurement?***

1. For investigation of congenitial & acute heart disease.
2. For investigation of cardiac septal defect.

Stroke volume

Definition : The amount of blood pumped out by each ventricle in each beat is called stroke volume . It is about 70 ml.

Stroke volume = (End diastolic volume - End systolic volume)

(Ref. Guyton & Hall-11th Edition, Page-108)

Stroke volume index

Stroke volume per square meter of body surface area is called

stroke volume index. It is about 47 ml.

$$= \frac{\text{Stroke volume}}{\text{Surface area}}$$

$$= \frac{80}{1.7}$$

= 47 ml.

Relation between cardiac output, stroke volume and heart rate

Cardiac output = Stroke volume x heart rate.

So, when cardiac out put remain constant, stroke volume and heart rate bears an inverse proportional to each other. That is -

i. Increase heart rate, decreases stroke volume.
ii. Increases stroke volume, decreases heart rate.

Factors affecting stroke volume :

1. Venous return : Stroke volume $\propto$ Venous return.
2. Heart rate ; SV $\frac{1}{\propto}$ Heart rate.
3. Force of contraction : SV $\propto$ Force of contraction.
4. Total peripheral resistance : SV $\frac{1}{\propto}$ TPR

End systolic volume

The end systolic volume is that volume of blood which remains in each ventricle at the end of the ventricular systole. It is about 40-50 ml.

(Ref. Guyton & Hall-11th Edition, Page-108)

Factors affecting end systolic volume :

a. *Pressure load :* It is the pressure within the aorta . If the aortic pressure is within physiological limit the heart can eject all blood . But if the aortic pressure is not within physiological limit, unless the tension of myocardium is increased, the heart can not eject all blood.

b. *Myocardial contractility :* Heart responds the excitation by contraction. This phenomenon occurs due to chemical and mechanical changes in the contractile elements - actin and myosin.

Normally, heart muscle remain relaxed. Actin, myosin remains dissociated and Ca^{++} is in the sarcoplasmic reticulum When an impulse reaches myofibriles, the Ca^{++} stimulated and the enzyme ATPase which break down the ATP to release energy that helps formation of actin-myosin complex, which causes gliding of actin and myosin . The agent, which increases the contractility is called ionotropic.

The force of contraction of heart depends upon :

i. Initial length of muscle fibres
ii. Nutrition and O_2 supply
iii. Sympathetic stimulation.
iv. Diastolic period.
v. Homometric and heterometric autoregulation
vi. Circulating hormone.
vii. Ca^{++} level of blood.

End Diastolic Volume

End diastolic volume is that volume of blood which remains in the ventricle at the end of ventricular diastole. It is about 110 - 120 ml.

(Ref. Guyton & Hall-11th Edition, Page-108)

Factors affecting end diastolic volume :

a. *Filling time :* The ventricular filling is affected by decreased filling time. Filling time is normally adequate for filling the heart but important in increased heart rate .

b. *Effective filling pressure :* This is the gradient between the inside and out side of the ventricle. It depends upon-
   i. Degree of positivity of intra abdominal pressure.
   ii. Degree of negativity of intrathoracic pressure.

c. *Distensibility of the ventricle :* Heart muscle is distended during diastole to receive blood from periphery. It depends on :
   i. Condition of pericardium.
   ii. Condition of myocardium.

d. *Atrial contraction :* It contributes 30% of blood, when atria contract, and pushed the blood into the ventricle. It is important during increased heart rate .

e. *Venous return :* Increased venous return increases the ventricular filling that increases end diastolic volume.

f. *Ejection fraction :* The fraction of end diastolic volume that is ejected is called the ejection fraction.

Venous return

Definition : It is the amount of blood that come from periphery to right atria of heart in each minute. It is equal to cardiac output . It is about 5 liters / min.

Factors affecting venous return

i. Muscular activity .
ii. Pumping action of heart .
iii. Pressure gradient in vessels.
iv. Respiratory pump .
v. Gravity .
vi. Vasomotor tone .

Q. 06. **Define preload. How preload affect cardiac output?**

i. *Definition* : This is defined as the tension of the myocardial fibres at the end of diastole, just before the onset of ventricular contraction. Preload is therefore related to the degree of stretch of the myocardial fibres.

ii. *Factors affecting pre-load* : In the intact heart the preload

depends on :

a. Ventricular end-diastolic volume
b. Venous return
c. Venous tone
d. Body position
e. Intrathoracic and intrapericardial pressure
f. Atrial contraction.

iii. *Preload affect cardiac output* : Pre-load is one of the major factors that control the force of contraction. An increase in pre-load improves myocardial oxygen consumption and thus increases myocardial force of contraction and so stroke volume and cardiac output.

Q. 07. **Define afterload. How afterload affect cardiac output.**

i. *Definition* : This is the load or resistance against which the ventricle contracts (or this is defined as the myocardial wall tension developed during systolic ejection).

ii. *It is formed by* :

a. Pulmonary and systemic resistance
b. Physical characteristics of the vessel walls
c. Volume of blood that is ejected.

iii. Right ventricular afterload is normally negligible because the resistance of the pulmonary circulation is very low.

iv. *Determinants of afterload* : In the case of the left ventricle the determinants of afterload are :

1. Resistance imposed by the aortic value
2. Peripheral vascular resistance
3. Elasticity of the major blood vessels.

Mean systemic filling pressure

It is the average effective pressure of blood in peripheral circulation, which tends to push the blood to the right atrium.

MSF pressure = 7mm of Hg.

Factors affecting mean systemic filling pressure

A. *Factors increasing mean systemic filling pressure :*

a. Increased blood volume causes increase *mean systemic filling pressure :*
   i. Acute increase15% *Double increase*
   ii. Chronic increase 30% *MSF pressure*
b. Maximum sympathetic innervation.
c. Skeletal muscle contraction.

B. *Factors decreasing mean systemic filling pressure :*

a. Absence of sympathetic innervation.
b. Decrease blood volume.

C. *Homometric autoregulation* : Homo means no change and metric means length. It is a process in which power is added to the cardiac output of the heart to eject the optimum amount of blood without change in the length of muscle fibres, hormonal or nervous influence.

D. *Heterometric autoregulation* : Hetero means change and metric means length. Heart muscle can eject optimum amount of blood without hormonal or nervous influence, but by simple increasing the length and thereby increasing its force of contraction.

E. *Vasomotor tone* : Under normal condition the vasoconstriction area of the vasomotor center transmit signals continually to the sympathetic vasoconstrictor nerve fibers, causing continuous slow fireing of these fibre.This continuous slow fireing is called sympathetic vasoconstrictor tones. This impulse maintain a partial state of contraction in the blood vessels is called vasomotor tone.

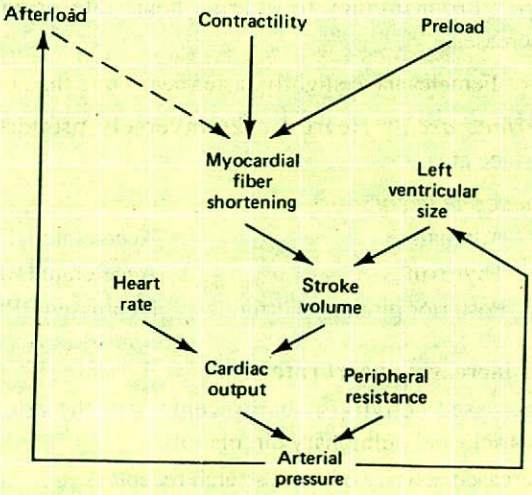
Peripheral resistance

It is the resistance in which blood has to over come while passing through the periphery.

Factors affecting peripheral resistance : Factors on which the peripheral resistance depends-

i. Velocity of blood
ii. Viscosity of blood
iii. Elasticity of arterial walls
iv. State of lumen of blood vessels.

Table. **Interactions between the components that regulate cardiac output and arterial pressure. Solid lines indicate increases and the dashed line indicates a decrease.**



Heart rate

Definition : The number of heart beat per minute is called heart rate.

Normal heart rate :

1. Adult (Range) : 60-90 /minute

	Average	: 72	/minute
2.	In foetus	: 140-150	/minute
3.	In Newborn	: 130-140	/minute
4.	In children	: 80-120	/minute
5.	In old age	: 75-80	/minute

Factors affecting heart rate

1. *Higher Centre :* Stimulation of posterior group of hypothalamic nucleus increases heart rate, while middle group decreases heart rate, stimulation of area 13 of frontal lobe, causes tachycardia.
2. *Respiration :* Heart rate increases during inspiration particularly in children and decreases during expiration.
3. *Cardio-vascular reflexes :*
   a. Baro-receptor reflex -Stimulation of baro receptor decreases the heart rate.
   b. Bain - Bridge reflex - Stimulation of bain Bridge increases heart rate.
4. *Temperature :* Increase body temparature, increases heart rate by directly stimulating the SA node.
5. *Intra cranial pressure :* Increase intracranial pressure stimulates cardio-inhibitory centre and causes slowing heart rate.
6. *Muscular exercise :* It increases heart rate by -
   i. Decreasing O_2, increasing CO_2 & H^+ tension.
   ii. Increasing body temparature.
   iii. Increasing venous return.
   iv. Increasing secretion of adrenalin.
7. *Age :* From infancy to old age heart rate progressively decreases.
8. *Sex :* Females have slightly faster heart rate than male.
9. *Surface area :* Heart rate is inversely proportional to surface area.
10. *Endocrine factors :*
    a. Adrenaline : Accelerate HR.
    b. Thyroxin : Accelerate HR.
    c. Posterior pituitary hormone : Depresses HR.

Factors increasing heart rate

1. Decreased activity of baroreceptors in the arteries, left ventricle and pulmonary circulation.
2. Increased activity of atrial stretch receptors
3. Inspiration
4. Excitement.
5. Anger
6. Most pain stimuli.
7. Hypoxia
8. Exercise.
9. Fever.
10. Epinephrine and norepineprine.
11. Thyroid hormone.
12. Bainbridge reflex.

(Ref Ganong 22th edition; Page 610)

Factors decreasing heart rate

1. Increased activity of baroreceptors in the arteies, left ventricle and pulmonary circulation.
2. Expiration.
3. Fear
4. Grife
5. Increased intra cranial pressure
6. Stimulation of pain fibres in trigeminal nerve.
7. Athlets .

(Ref Ganong 22th edition; Page 610)

Q. 00. **What is sinus arrhythmia**?

Ans. Cardiac irregularity characterised by an increa- sed heart rate during inspiration and decrease in heart rate on expiration. This arrythmia has no clinical significance except in older patients, in whom it may occur in coronary artery diseases.

(Ref. Tabers)

Cause : It may result from any one of many circulatory reflexes that altered the sympathetic and parasympathetic nerve signals to the heart sinus node.

(Ref. Guyton & Hall-10th Edition; Page 148)

Tachycardia

The trem trachycardia means fast heart rate. Increased heart rate above the upper normal physiological limit, usually above 100 beats/ min, is called tachycardia.

(Ref. Guyton & Hall-10th Edition; Page 147)

Cause of tachycardia :

i. *Increased body temperature* : The rate of the heart increases about 10 beats per minute for each degree Fahrenheit (18 beats per degree Celsius) increase in body temperature upto a body temperature of 105°F (40°C); beyond this, the heart rate may decrease because of progressive weakening of the heart muscle as a result of fever.

   Fever causes tachycardia because increased temperature increases the rate of metabolism of the sinus node, which in turn directly increases its excitability and rate of rhythm.

ii. Stimulation of heart by sympathetic nerve : Many factors may caues the sympathetic nervous system to excite the herat. For instant, when a patient loses blood and passes into a state of shock or semishock, sympathetic reflex stimulation of the heart can increase the heart rate to 150 to 180 beats per minute.

iii. Toxic conditions of the heart : Simple weakening of the myocardium usually increases the heart rate because the

weakened heart does not pump blood into the arterial tree to a normal extent, and this elicits sympathetic reflexes to increase the heart rate.

(Ref. Guyton & Hall-10th Edition; Page 147)

Q. 00. **What is sinus tachycardia?**

Ans. A sinus rate of more than 100/minute is called sinus tachycardia.

Causes of sinus tachycardia are :

i. *Physiological* :
   a. An increase in sympathetic activity associated with exercise, emotion.
   b. Pregnancy

ii. *Pathological* :
   a. Anxiety
   b. Fever
   c. Anaemia
   d. Heart failure
   e. Thyrotoxicosis
   f. Phaeochromocytoma
   g. Drugs, e.g. beta-adrenoceptor agonists (bronchodilators)

Bradycardia

The term bradycardia means a slow heart rate. Decreased heart rate below the lower normal physiological limit, usually below 60 beats/ min, is called bradycardia.

(Ref. Guyton & Hall-10th Edition; Page 147)

Cause of bradycardia :

i. Athletes : The athlete's heart is considerably stronger than that of a normal person, a fact that allows the athlete's heart to pump a large stroke volume output per beat even during periods of rest. The excessive quantities of blood pumped into the arterial tree with each beat initiate feedback circulatory reflexes or other effects to cause bradycardia when the athlete's is at rest.

ii. Vagal stimulation : Any circulatory reflex that stimulates the vagus nerve can cause the heart rate to decrease considerably i.e carotid sinus syndrome.

iii. Shock.

(Ref. Guyton & Hall-10th Edition; Page 147, 148)

Q. 00. **What is sinus bradycardia?**

Ans. A sinus rate of less than 60/minute is called sinus bradycardia.

Causes of sinus bradycardia are :

i. *Physiological* :
   a. In normal people during sleep
   b. In athletes.

ii. *Pathological* :
   a. Myocardial infarction
   b. Sinus node disease (sick sinus syndrome)
   c. Hypothermia
   d. Cholestatic jaundice
   e. Raised intracranial pressure
   f. Drugs, e.g. beta-adrenoceptor antagonist, digoxin, verapamil.

Q. 00. **What is the relationship between heart rate and respiration?**

Ans. Heart rate increases during inspiration particu- larly in children and decreases during expiration.

Electrocardiogram (ECG)

Lead

Definition : It denotes the connection of galvanometer with wires to electrods. It is used to traching of ECG.

Types :

It is mainly two types -

1. Bipolar or standard limb leads.
2. Unipolar limb leads.

In addition to these, there is another lead called chest lead.

Bipolar lead

It has two electrodes - One negative another positive. several standard locations are selected, so they are called standard limb leads.

The commonly used bi-polar leads are :

Lead	*Negative terminal*	*Positive terminal*
Lead - I	Right arm	Left arm
Lead - II	Right arm	Left leg
Lead - III	Left arm	Left leg

Lead-I : It records the difference of potentials between the right and left arm electrodes.

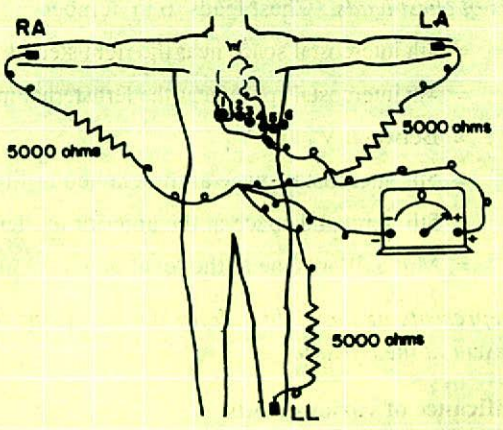


Fig. 6-9. *Leads.*

Lead-II : It records the difference of potentials between the right arm and left leg electrodes

Lead-III : It records the difference of potentials between the left arm and left leg electrodes.

Advantage of Bipolar lead :

i. They are advantageous in demonstrating disturbances of rhythm of heart.
ii. They also suggest genaralised lesions of heart like cardiomyopathies.

Disadvantage of Bipolar lead :

i. As they record the difference of potentials between the two electrodes it is difficult to denote a particular change in relation to single electrode.
ii. It is equally impossible to localize a change in potential in relation to site of lesion restricted to a specific region of the heart.

Unipolar lead

Though called unipolar it actually consists of two connecting leads-

a. *Exploring electrode :* One single electrode is placed at a selected position over the surface of the body.
b. *Indifferent or reference electrode :* Formed by the central terminal consisting of junctions of three different electrodes, placed at right arm, left arm and left leg.

The different unipolar lead are :

Lead	*Exploring electrode*
Lead - aVR	Right arm
Lead - aVL	Left arm
Lead - aVF	Left leg

Chest lead

Defferent point of the chest opposite the heart it gives the actual electrical changes or potential excitability of the heart.

Types of chest leads : Chest leads 6 in number-

V_1 = 4th intercostal space near the right sternal margin,
V_2 = 4th intercostal space near the left sternal margin.
V_3 = Between V_2 and V_4
V_4 = 5th intercostal space at midclavicular line.
V_5 = 5th itercostal space at the anterior axillary line.
V_6 = Mid axillary line at the level of V_4.

All represents at the right side of the heart but the V_5 and V_6 represent at the left side.

Significance of various leads

A. *Bipolar or standard limb lead :* Valuable for diagnosis of-
   i. Arrhythmia
   ii. Preliminary studies of the functional abnormalities of the heart.
B. *Unipolar limb leads :* Most valuable for-
   i. Determining the position of heart.
   ii. Confirming the significance of Q and T waves in the standard limbs.
   iii. Confirming the evidence of ventricular damage or hypertrophy.
C. *Chest leads :* Important for diagnosis or for-
   i. Localization of the recent or old ventricular damage.
   ii. Bundle branch block.
   iii. Detection of ventricular hypertrophy.

Einthoven's Law

Einthoven's law states that if the electrical potentials of any two of the three standard electrographic leads are known at any given instant. There one can be determined mathmati-cally from the first two by simply summing the first two. i. e. the sum total voltage in leads 1. and iii equals to the voltage in lead ii.

Einthoven's Triangle

It is an equilateral triangle drown arbitrarily around the area of the heart . The two apices at the upper part of the tringle

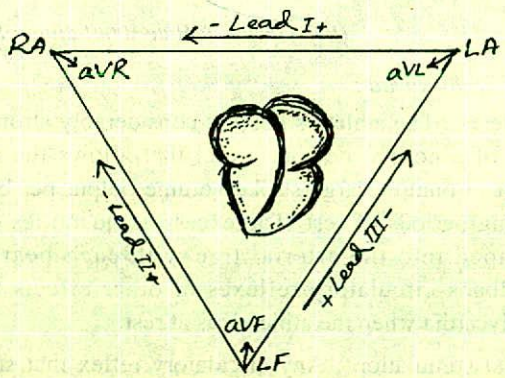


Fig.6-10. Einthoven's Triangle for recording the ECG form limb leads.

represents the points at which the two arm connects electrically with fluid around the heart and lower apex is that of the left leg.

Electrocardiogram (ECG)

Electrocardiogram is the recording from the surface of the body of the electrical changes of the heart in each cardiac cycle.

Description of a normal ECG : A normal ECG shows five consecutive waves PQRST. Of these there are three Positive waves; P, R & T and two negative waves, Q &S and a complex QRS.

1. ***P Wave*** : It is the first upward deflection. It is small, constant wave having a rounded or pointed top.

It represents atrial depolarization.

Duration : 0.10 second (< 0.125)

Voltage : 25 mv.

It indicates :

i. The impulse is originating at the SA node.
ii. It spreads over the atria in the usual direction
iii. There is no defect in conduction.
iv. The strength of contraction, the mass of atrial musculature and its nutrition are normal.

2. ***Q Wave*** : It is the downward deflexion. It is small & often inconspicuous.
   1. It represents septal depolarization.
   2. It is caused by the septal activity.
   3. Prominent Q wave indicates old infarction.

3. ***R Wave*** : It is upward deflection; most constant and conspicuous wave having the tallest amplitude
   It follows immediately upon Q wave.
   It represents apical left ventricular depolarization.

4. ***S Wave*** : It is the downward deflection just next to R wave.
   It represents posterior basal left ventricular depolarization.

5. ***T Wave*** : It is the repolarization wave of ventricles

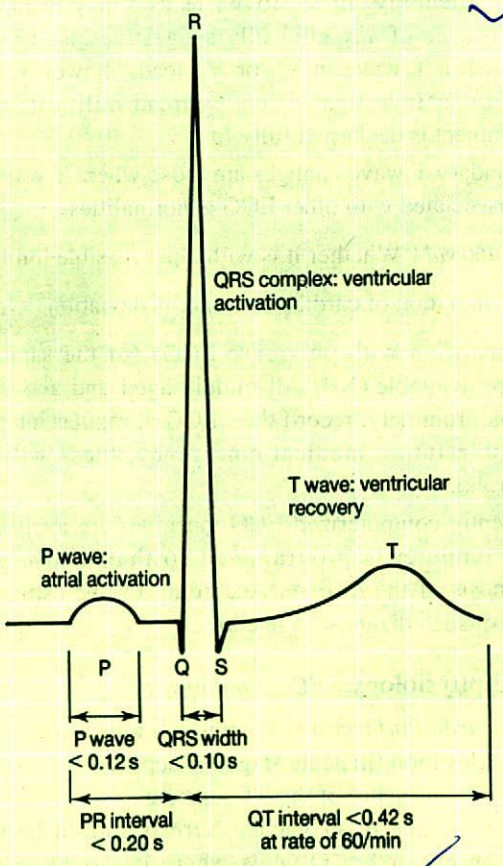


Fig. 6-11. *Tracing of the normal ECG.*

showing upward deflection. It represents ventricular repolarization.

Duration : 0.13second

Voltage : 0.2 to 0.4 mv.

6. ***ECG Complexes*** :
   1. *QRS complex :* It is the ventricular depolarization and atrial repolarization.
      Duration : 0.08-0.10 second.
   2. *QRST(QT) complex :* These four waves are caused by ventricular activity, are collectively known as ventricular complex i.e ventricular depolarization & ventricular repolarization.
      Duration : 0.40-0.43 second.

(Ref. Ganong 22th Edition; Page-551)

7. ***ECG intervals*** :
   i. ***P-R interval*** : It is the time or interval between the peak of P and R wave or between the start of P & Q wave.It is the atrial depolarization and conduction through AV node.
      Duration : 0.18 second (Range : 0.12-0.20 second).
      Importance :
      a. Increased P-R interval indicates that the conduction time from atria to ventricle is increased.
      b. 0.4 Sec P-R interval indicates that the complete heart block may occur at any time.

   ii. ***R-R interval*** : It is the interval between two successive R waves.
      Importance : Same R-R interval in next successive stages indicate rhythmic ventricular depolarization.

   iii. ***P-P interval :*** It is the interval between two successive P waves.
      Importance : Equal intervals in next succes-sive stages indicates rhythmical depolarization of the atrium.

   iv. ***T-P interval :*** Alteration of this interval indicates alteration of heart rate.

   v. ***ST interval*** : QT minus QRS i.e. ventricular repolarization. *Duration* : 0.32 second.

(Ref. Ganong 22th Edition; Page-551)

Importance of ECG

A. *Physiological :*
   i. Normal heart rate is counted
   ii. Condition of heart can be detected.

B. *Clinical :*
   i. Atrial and ventricular hypertrophy.
   ii. Myocardial infarction.

iii. Pericarditis.
iv. Systemic disease that effect the heart.
v. Arrythmia, fibrillation, flutter, abnormal pulse
vi. Effects of drugs -
   a. Digitalis
   b. Quinine.

Some important tips about ECG

1. In the bipolar leads, lead II is maximally +ve, R is tallest in lead II ; also in lead II, Q is absent and T is upright.
2. In the unipolar limb lead, aVR is mostly -ve. Here P and T are inverted, R is very small.
3. In the unipolar chest leads the R wave gradually becomes taller and taller from V_1 to V_5 and S becomes smaller and smaller from V_1 to V_5. Q is present in V_5 and V_6 but absent in V_1.
4. In all leads P wave, PR intervals, QRS complex have normal values and ST segment is isoelectric.

How to read ECG

Finally, some tips are given below, about how to read ECG records of a given subject :

1. ***Heart rate :*** The heart rate is counted as follows : Locate the peaks of any two successive R waves ; count the number of small divisions in between them (for example, say 20 small divisions). [This interval, from R to R peaks represents one cardiac cycle and in this example, has taken 20 small divisions or 0.4 sec]. The heart rate is 1500 divided by the number of divisions between the R to R peaks ; for example, in the example cited above, the heart rate is 1500 divided 20 = 75/min. The figure 1500 is obtained as follows : each small division = 0.04 sec. Therefore in 60 seconds there are 60 divided 0.04 = 1500 such divisions. In short, 1500 small divisions represent one minute.
2. ***Rhythm :*** See whether the rhythm is sinus or not. Sinus rhythm means where the pace maker is SAN. In sinus rhythm, the normal sequence of P,Q,R,S,T are maintained and p waves are upright where they should be upright.

   In a normal ECG record, the heart rate (HR) fluctuates. For example at the beginning of a given ECG record the HR may be 70/ min, but within a few seconds, as the ECG recording continues, the HR may be 75/min. This is normal and is due to (usually) respiration and is called normal sinus arrhythmia. The report, in this case should read, heart rate, varying between 70 to 75/min, sinus arrhythmia.
3. ***P wave :*** Look for, i. whether there is any evidence of atrial hypertrophy (big p wave), inversion of p wave where it should normally be upright (nodal rhythm), absence of p wave and so on.
4. ***QRS complex :***
   i. Look for Q wave abnormality. That is, whether Q is present where it should not be present, or Q is unusually big and so on.
   ii. Look for duration of QRS complex (ie, whether it is within 0.8 sec or at best within 0.1 sec), whether its shape is altered (eg. in left bundle branch block).
   iii. Whether RSR pattern is present.
   iv. Tallness of R wave and depth of S wave - whether they exceed permissible limit.
   v. Whether R becomes progressively taller from V_1 onwards, and so on.
5. ***ST Sgment :*** Whether it is on isoelectric line or elevated or depressed.
6. ***T Wave :*** The interpretation of T wave abnormality is most difficult. T wave may be inverted in leads where it should normally be upright, but that dose not automatically mean there is abnormality . Inversion of T wave where there is no other abnormality in ECG, called , primary T wave changes can be of 2 kinds :
   i. Where the inverted T wave is symmetrical and its apex pointed ; this is pathological and may mean myocardial infarction.
   ii. Where the inverted T wave has round apex, this (particularly in V_1 to V_3 leads) may be, specially in absence of any clinically suggestive history, harmless, such a T wave in V_4 or V_5 leads, however, should be further investigated (e.g.by tread mill test) before the subject is declarned fully fit.

   Secondary T wave changes are those where T wave changes are associated with other ECG abnormalities.
7. ***PR interval :*** Whether it is within permissible limit.
8. Determination of cardiac axis and its deviation.
9. Comparition with previouse ECGs (of the same subject) where available.(NB. All middle aged and above persons, should, routinely, record their ECG at regular intervals. In a possible future medical emergency, these will serve as controls).

   Currently computerized ECG machines are available where the computer is programmed so that is also gives the diagnosis of the disease. According to one estimate, about 85% of such diagnoses are correct.

Applied physiology *(ECG Findings) :*

1. ***Myocardial infarction :*** Common findings are-
   i. Elevation (in acute stage) or depression (in subacute or chronic state) of the ST segment.
   ii. Presence of Q wave where it should be normally absent or big Q wave where it should be normally present.

iii. Inversion of T wave.

iv. Normally the height of R wave progresses from V_1 to V_5 but in myocardial infarction this progressive increase in height does not occur.

2. ***Myocardial ischemia :*** Common finding is depression of ST segment which may or may not be associated with other signs. Presence of a normal ECG, however, does not rule out cardiac ischemia.

3. ***Hypertrophy :*** Left ventricular hypertrophy is usually associated with larger R in V_5 and V_6, deeper S in V_1, V_2 and left axis deviation. These changes may or may not be associated with inversion of T wave and depression of ST.

4. ***Heart Block :*** In AV block the PR interval is more than 0.2 sec. In bundle branch block the duration of QRS is more than 0.11 see. Presence of RSR (in V_1) pattern denotes right bundle branch block. Whereas a slurred and broad QRS in V_5 and V_6 denote left bundle branch block.

5. ***Hyperkalemia :*** In erarly stages of *hyperkalemia*, T wave becomes taller. As the hyperkalemia progresses, AV block and atrial paralysis develop, so that prolonged PR interval and absence of P are found in ECG record. QRS becomes prolonged due to delayed conductivity in the ventricular conducting system.

Q. 05. **What ECG changes you expected to find in hyperkalaemia.**

Ans. Common ECG findings in hyperkalaemia are :

i. *In erarly stages of hyperkalemia* : T wave becomes taller.

ii. *As the hyperkalemia progresses* AV block and atrial paralysis develop, so that-

a. Prolonged PR interval and absence of P are found in ECG record.

b. QRS complexbecomes prolonged due to delayed conductivity in the ventricular conducting system.

Q. 06. **What ECG changes you expected to find in myocardial infraction.**

Ans. Common ECG findings in myocardial infraction are :

i. Elevation (in acute stage) or depression (in subacute or chronic state) of the ST segment.

ii. Presence of Q wave where it should be normally absent or big Q wave where it should be normally present.

iii. Inversion of T wave.

iv. Normally the height of R wave progresses from V_1 to V_5 but in myocardial infarction this progre ssive increase in height does not occur.

Q. 07. **What ECG changes you expected to find in hypokalaemia.**

Ans. Common ECG findings in hypokalaemia are-

i. Flattening of T wave.

ii. Prolongation of QT interval

iii. Appearance of U wave on the end of the T wave.

Q. 07. **ECG changes in hypocalcaemia and hypercalcaemia.**

Ans.

i. In *hypocalcaemia* : Common ECG findings in hypocalcaemia is prolongation of the QT interval.

ii. In *hypercalcaemia* : Common ECG findings in hypercalcaemia is shortening of the QT interval.

Heart block

Q. 01. ***What is heart block? Classify heart block.**

I. *Definition :* Occationally transmission of the impulse throgh the heart is blocked at a critical point in the conduetive system is called heart block.

Sites of heart block : Most common points of heart block are

i. Between the atria and ventricle.

ii. One of the bundle branches of purkinje system

iii. Rarely between the SA node and the atrial musculature.

(Ref. Guyton 10th edition)

II. *Classification or types of heart block :* There are three types of heart block-

i. Sino-atrial heart block.

ii. Atrio-ventricular (A-V) heart block :

Degrees of A-V heart block :

a. *First degree heart block* : All the atrial impulses reaches the ventricle but the P-R interval is abnormally long.

b. *2nd degree heart block* : Not all atrial impulses are conducted to ventricles. There may be for exapmle, a ventricular beat following every second or every third atrial beat (2 :1 block, 3 : 1 block)

c. *3rd degree block or complete heart block* : When no impulse passes from atria to ventricle. It causes the *droped beat*. Here the atrial rate is faster than the ventricle, for this reason, ventricle escape from the control of atria.

(Ref. Ganong 22th edition)

iii. Bundle branch block and hemiblock.

*(Q. **Short notes on- Heart block. Ans. Pl follow the above).*

Q. 01. **Describe A-V type of heart block.**

i. *Definition :* When conduction of impulse between atria and ventricle is blocked.

ii. *Cause :*

a. Localized damage or depression of AV node.

b. Excessive stimulation of vagus nerve.

c. Localized destruction of AV bundle as a result of coronary infract.

d. Pressure on the AV bundle by arteriosclerotic paques.

e. Depression caused by various drug.

(Ref. Guyton 11th edition)

+ *Degrees of A-V heart block* : Pl follow the above. + ECG changes - From below)

Degrees of AV heart block :

1. *First degree heart block :* All the atrial impulses reaches the ventricle but the P-R interval is abnormally long.
2. *2nd degree heart block :* Not all atrial impulses are conducted to ventricles. There may be, for exapmle, a ventricular beat following every second or every third atrial beat (2 :1 block, 3 : 1 block)
3. *3rd degree block or complete heart block :* When no impulse passes from atria to ventricle. It causes the "droped beat". Here the atrial rate is faster than the ventricle, for this reason, ventricle escape from the control of atria.

(Ref. Ganong 22th edition)

Q. 01. **What changes take place in ECG in heart block..**

I. ***In AV block*** : The PR interval is more than 0.2 second.

Degrees of A-V heart block :

i. *First degree heart block* : AV conduction is delayed.
   a. One P wave per QRS complex
   b. P-R interval more than 0.22 second.

ii. *2nd degree heart block* : Some P wave conducts and other fails to go to the ventricles.
   a. Mobitz I block :
      * Progressive lengthening of PR interval
      * One non-conducted beat (P wave fails to conduct)
      * The PR interval before the blocked P wave is much longer than the PR interval after the blocked P wave.
   b. Mobitz II block :
      * PR interval of the conducted beats is constant
      * P wave is not followed by QRS complex.
   c. 2:1, 3:1 block :
      * 2 or 3 P waves per QRS complex
      * Normal and constant PR interval in the conducted beats.

iii. *3rd degree block or complete heart block* : No P wave conducts to the ventricle.
   * Variable P wave rate and QRS complexs
   * Abnormally shaped QRS complexes.

Q.Write short notes on-Third degree heart block)

II. ***In bundle branch block*** : The duration of QRS is more than 0.11 second.

a. Presence of RSR (in V_1) pattern denotes right bundle branch block.

b. Whereas a slurred and broad QRS in V_5 and V_6 denote left bundle branch block.

Bundle of His block

Here the conduction of impulse from AV node to Bundle of His is impaired. It may be right or left bundle block.

SA Heart block

Here SA node fails to generate or fail to transmit the impulse from SA node to the atrial muscle.

Cause :

i. Over vagal stimulation
ii. Vasovagal syndrom.
iii. Over dose of digitalis.

Cardiac Arest

Definition : It is a condition in which the rhythmic contraction of heart occationally stop due to greatly disturbed cardiac metabolism.

Causes :

1. Hypoxia of heart during anesthsia or when the coronary blood flow to the SA node is blocked.
2. Severe myocardial diseases.

Flutter

Flutter means extremely rapid heart beat with reasonably coordinated cotraction of cardiac muscle. The rate of flutter is usually 200-250 times per minute. The flutter occurs frequently in the atria and rarely in ventricle. It occurs when atria become greatly dialated due to valvular heart disease.

Fibrilation : Fibrilation means a very high frequency of heart beat with incordinated contraction of cardiac muscle.

Angina pectoris

It is the cardiac pain that begins to appear whenever the load on the heart becomes to great in relation to the coronary blood flow, due to development of progressive constriction of the coronary arteries. It is usually felt beneath the upper sternum and is often also transferred to surface areas of the body, most often to the Lt. shoulder but also frequently to the neck and even to the side of the face or to the right arm and shoulder.

Treatment of angina pectoris :

1. Administration of vasodilator drugs.
   a. Nitro glycerine.
   b. Amyl nitrite gives immediate relief from the pain in acute anginal attack.
2. Blockage of the sympathetic nervous stimulation of the heart by propranolol.

Echocardiography

Wall movement and other aspects of cardiac function can be evaluated by echocardiography, a noninvasive technique that does not involve injections or insertion of a catheter. In echocardiography, pulses of ultrasonic waves, commonly at a frequency of 2.25 MHz, are emitted from a transducer that also

functions as a receiver to detect waves reflected back from various parts of the heart. Reflections occur wherever acoustic impedance changes, and a recording of the echoes displayed against time on an oscilloscope provides a record of the movements of the ventricular wall, septum, and valves during the cardiac cycle. When combined with Doppler techniques, echocardiography can be used to measure velocity and volume of flow through valves. It has considerable clinical usefulness, particularly in evaluating and planning therapy in patients with valvular lesions.

(Ref. Ganong 21th ed; Page-570)

Blood Pressure

Definition : It is the lateral pressure exerted by the moving colum of blood on the vessel wall per unit area (sq. mm) by its contained blood while flowing through it.

Blood pressure = Cardiac output x Peripheral resistance.

Importance of blood pressure

1. It is essential for the flow of blood through the circulatory tree.
2. It provides motive force for filtration at the capillary bed which is essential for -
   a. Tissue nutrition.
   b. Formation of urine
   c. Formation of lymph
   d. For venous return.

Lateral pressure

It is that pressure when force is exerted at right angles to the direction of flow at any point within a tube filled with a circulating fluid.

(Resistance is opposition to force)

Basal blood pressure

It is the lowest pressure necessary in main-taining blood flow sufficient for needs of the body.

Criteria for recording basal blood pressure :

i. The subject is in reclining state.
ii. At least 5-6 hours after last meal.
iii. After resting for at least 30-40 minutes in a comfortably warm room.
iv. A mind at possible ease.

Causal blood pressure

Any pressure that is recorded under ordinary circumstances of life is known as the causal blood pressure.

Types of blood pressure

1. *Systolic pressure :* It is the maximum pressure during systole.
   It is about : 100 - 140 mm of Hg,
   Average : 120 mm of Hg.
2. *Diastolic pressure :* It is the minimum pressure during diastole.
   It is about : 60 - 90 mm of Hg,
   Average : 80 mm of Hg.
3. *Pulse pressure :* It is the difference between systole and diastolic pressure.
   It is ablut 30 - 40 mm of Hg.
4. *Mean pressure :* It is the average pressure persists in the circulation. It is the diastolic pressure plus one third (1/3) of pulse pressue.
   It is about : 78-98 mm of Hg.
   Average : 96 mm of Hg.

Significance of different types of BP

1. *Systolic pressure :* It indicates-
   a. The extent of work done by heart.
   b. The force with which the heart is working.
   c. The degree of pressure which the arterial walls have to withstand.
   d. It increases during excitement, exercise, meals etc.
   e. It decreases while sleep, rest etc.
2. *Diastolic pressure*
   a. It indicates the constant load against which the heart works.
   b. Increased diastolic pressure indicates that heart is approching to failure
   c. It is the index of peripheral resistance.
3. *Pulse pressure :* It indicates the cardiac output.
4. *Mean pressure :* Mean pressure is the driving force for blood flow. It depends on cardiac output and peripheral resistance.

Physiological variation of blood pressure

1. *Age :*

Infant	: 60/30	mm of Hg
1 year	: 80/40	mm of Hg
3 years	: 100/60	mm of Hg
20 Years	: 120/80	mm of Hg
45 years	: 145/90	mm of Hg
70 years	: 170/95	mm of Hg

2. *Sex :* In female, the blood pressure is slightly lower (5 mm of Hg), cause is unknown. After menopause, it reaches male level.
3. *Build :* The systolic pressure is usually high in an obese person.
4. *Exercise :* In strenuous exercise, the systolic pressure rises,

even up to 180 mm of Hg. In moderate exercise, it slighly rises. The diastolic pressure is usually lower.

5. *Posture :* During standing, diastolic pressure is slightly higher; systolic pressure lower. In recumbent position, this condition is reversed.
6. *Diurnal variation :* During day time, pressure rises upto 2-O'clock and then there is a slight fall. In case of night workers, the blood pressure rises during morning.
7. *During sleep :* During deep sleep, there is fall of blood pressure by 15-20 mm of Hg.
8. *After meal :* Blood pressure is raised during digestion due to increased cardiac output up to 20 mm of Hg.
9. *Emotion & Excitement :* Raises systolic pressure considerably.
10. *Respiration :* There are fluctuations of blood pressure due to variations in stroke volume and peripheral resistance.

    At a normal respiratory rate, the blood pressure falls during most part of inspiration. At a slower respiratory rate, the blood pressure is slightly higher during inspiration.

N.B.

1. In adult, diastolic blood pressure and systolic blood pressure, should not be (persistently) over 90 and 140 mm of Hg (excepting in old persons).
2. Acute fall (shock) and rise (hypertension) of BP are dangerous. Blood pressure homeostasis is maintained by
   i. Neural : Neural control starts immediately (when ever there is destabilising of blood pressure homeostasis) and consists of reflexs (mainly baroreceptor) readjustments.
   ii. Fluid volume control mechanisms : Whereas fluid volume control takes a few hours to establish fully and consists of renin-angiotensin-aldosterone and ANP mechanisms.
3. Clinically sphygmomanometer is the instrument which is used to measure the blood pressure but the instrument is not wholly dependable.
4. In hypertension, drugs are aimed to restore the blood pressure homeostasis, influencing, eithre-
   i. The neural mechanism, or
   ii. The fluid volume readjustment.

Factors controlling blood pressure

The factors that maintain blood pressure may conveniently be discussed under two main headings -

A. *Cardiac output* : Cardiac output depends upon-
   i. Blood volume
   ii. Venous return
   iii. Force of contraction of heart.
   iv. Frequency of heart beat.

B. *Peripheral resistance* : Peripheral resistance depends upon-
   i. Elasticity of arterial wall
   ii. Velocity of blood
   iii. Viscocity of blood
   iv. State of lumen of blood vessels.

Factors influencing blood pressure

Factors influencing blood pressure are as follows :

1. *Peripheral registance :* Increased peripheral registance increases blood pressure. It depends on
   a. Elasticity of arterial wall
   b. Velocity of blood
   c. Viscosity of blood
   d. State of vessel lumen.
2. *Cardiac output :* It increases blood pressure. It depends on heart rate and stroke vloume. storke volume again depends on-
   i. *End systolic volume :*
      a. Force of cotraction
      b. Peripheral resistance
   ii. *End diastolic volume :*
      a. Venous return
      b. Diastolic period.
      c. Contraction of heart.
3. *Age :* Blood pressure rises with age.

Infant	:	60 / 30 mm of Hg.
Children	:	100 /60 mm of Hg
Adult	:	120 / 80 mm of Hg.
Old	:	145 / 90 mm of Hg.

4. *Sex :* 10 % less in female.
5. *Postures :* In recumbent position blood pressure is lower than in standing or sitting posture.
6. *Exercise :* It increases blood pressure.

Q. 00 **What is venous pressure?**

i. *Definition* : It is the pressure in the venous system.

ii. *Pressure at different sites* :
   1. In the venules : 12 -18 mm of Hg.
   2. Larger veins : 5.5 mm of Hg. (about)
   3. *Certral venous pressure* : Certral pressure is the pressure of the great veins at their entrance into the right atrium. It is about 4.6 mm of Hg. But flactuates with respiration & heart action.

      Gravitational effect :

      It increased by 0.77 mm Hg for each cm below the right atrium and also decreased by 0.77 mm of Hg for each cm above the right atrium.

4. The mean pressure at the anticubitul vein = 7.1 mm of Hg.

Measurement of blood pressure

Blood pressure is measured by -

1. *Direct method :* The artery is exposed and an arterial cannula of which one end is inserted directly into the lumen of the exposed vessel and the other end is connected to the U-shaped mercury manometer that show the actual blood pressure in mm of Hg.
2. *Indirect method :*
   i. Oscillatory
   ii. Palpatory : Can measure only sytolic pressure
   iii. Auscultatory : Can measure both systolic and diastolic.

Auscultatory method

1. *Site* : Brachial artery (Arm)
2. *Instrument :* Sphygmomanometer
   The instrument consists of an inflatable rubber bag covered by nondistensable envelope of cotton fabric, called the cuff. The cavity of the bag is connected with a mercury manometer and a hand pump. A small valve connected with a screw will allow the air to escape from the air bag.
3. *Principle :* The principle is to balance the air pressure against the brachial artery pressure. The air pressure is applied from a rubber bag connected with a hand pump and a mercury manometer to measure the air pressure.
4. *Method :* The instrument is kept at the level of the heart and the cuff is tied round the upper arm. The chest piece of a stethoscope is placed on the brachial artery, a little below the cuff. Pressure is raised to 200 mm of Hg and then gradually released. Variations of sounds are heard-
   i. *1st phase :* Sudden appearance of a *clear taping sound*. This indicates systolic pressure. It persists while the pressure falls through 15 mm of Hg.
   ii. *2nd Phase :* The tap sound is replaced by a *murmur* persisting for another 15 mm of Hg.
   iii. *3rd phase* : The murmur is replaced by a *clear loud* going sound lasting for the next 20 mm of Hg.
   iv. *4th phase* : The loud sound suddenly becomes *muffled* and rapidly begins to fade. This point indicates diastolic pressure.
   v. *5th Phase :* Disappearance of all sounds.

Korotkoff sound

Blood flow through the smooth blood vessel, normally provides no sound, but if the brachial artery is partially occluded by inflated cuff then blood pours out in jet into the open vessel beyond the cuff, and gives rise to terbulance of flow which sets up vibration and produces sound called Korotkoff sound. It has four different stages.

1. *Tapping sound :* At the point at which systolic pressure in the artery just exceeds the cuff pressure, a spurt of blood passes through with each heartbeat and, synchronously with each beat, a tapping sound is heard below the cuff. The cuff pressure at which the sounds are first heard is the systolic pressure.
2. *Loud sound :* On decreasing the pressue (10 mm of. Hg) the sound become loud.
3. *Dull sound :* Further decreasing the pressure (15 mm of Hg.) the sound become dull.
4. *Muffle sound :* Further decreasing the pressure (15 mm of Hg) the sound become muffled and gradually disappears. The point at which the muffled sound start is diastolic pressure (British) or disapper's is diastolic pressure (American).

The diastolic pressure in resting adults correlates best with the pressure at which the sound disappears. However in adults after exercise and in children, the diastolic pressure correlates best with the pressure at which the sounds become muffled. This is also true in diseases such as hyperthyroidism and aortic insufficiency.

(Ref. Ganong 22th edition; page-589)

Turbulence

Turbulent flow means that the blood flows cross wise in the vessel as well as along the vessel, usually forming whorls in the blood called eddy currents.

When the rate of blood flow becomes too great or when it passes by an obstruction in a vessel or when it passes over a rough surface, the flow may then become turbulent rather than streamline.

(Ref. Guyton 11th edition)

Laminar flow

When blood flows through a smooth vessel each layer of blood maintains same distance from the wall. The central portion of blood remains in the centre of the vessel. This type of flow is called laminar flow or streamline flow.

Q. ***Why blood pressure increases in old age, exercise and pregnancy?***

1. ***In old age :*** Blood pressure increases due to -
   a. Athero-sclerotic change in the artery and its wall become thick.
   b. Artery become stiff & its elasticity decreases.
2. ***In exercise :*** Blood pressure rises due to -
   a. Increased venous return.
   b. Increased metabolic activity & much energy & O_2 is required.
   c. Increased heart rate & cardiac out put due to increased venous return.

3. ***In pregnancy :*** Blood volume both plasma & corpuscles increase which increases cardiac out put, thus blood pressure is raised.

Q. ***Which is more important, the systolic or diastolic pressure ?***

Ans. Diastolic pressure is the level at which the heart is pumping blood. If the diastolic pressure is raised, heart is doing more work, Therefore it may be hypertrophied, leading to heart failure. So, diastolic pressure is more important than systolic pressure.

Hypertension

Definition : Hypertension is a clinical condition characterized by persistance rise of blood pressure above the normal range in respect of age and sex. Hypertension occurs when diastolic pressure is greater than 90 mm of Hg and systolic pressure is above 150 mm of Hg.

Type :

Hypertension is of two types -
1. Primary or essential hypertension.
2. Secondary hypertension.

Essential hypertension : When arterial blood pressure persistently exceeds 150/90 or 160/100 mm of Hg. Its cause is unknown. It is of two types -

a. *Benign form* : In the early stages the hypertension is moderate, e.g. 210/110 mm of Hg. The blood pressure, specially the systolic, fluctuates considerably; during sleep or emotional and physical rest, the pressure is normal; in states of stress the pressure rises to excessive levels. Later the hypertenstion becomes fixed in the abnormal range and can not be reduced to normal by rest or sedatives. After a period which may vary from a few years to 20 years : death occurs from heart failure, vascular accedents (haemorrhage or thrombosis), or renal failure.

b. *Malignant form* : The condition is so named because death occurs within months to 2 years of its first recognition. The BP is much higher than in the benign form e.g 260/150 of Hg.

Secondary hypertension : It is due to other diseases, as renal diseases, phaeochromo- cytoma, by excess secretion of glucocorticoids or aldosterone, by coarctation of aorta.

(Ref. Wrights 13th page 152-153)

Vloume loading hypertension : Hypertension occurs due to excess extracellular fluid volume. It is due to excess salt intake or salt retension from kidney.

Vasoconstrictor hypertension : It is due to continuous infusion of vasoconstrictor agent into blood or by excessive secretion of vasoconstrictor from endocrine gland. The vasoconstrictors are-

i. Angiotensin-II
ii. Norepinephrine
iii. Epinephrine.

Regulation of Blood Pressure

Two major types of arterial pressure control system in the body:
1. Nervous mchanism
2. Humoral mechanism.

1. **Nervous mechanism** : Has two different types-
   i. ***Short terms regulation :*** Occurs within seconds to minites.
      A. *Mechanism occurs within seconds :*
         a. Baroreceptor feed back mechanism,
         b. Chemoreceptor feed back mechanism.
         c. Central nervous system ischemic mechanism.
      B. *Mechanism occurs within minutes :*
         a. Renin angiotensin vasoconstrictor mechenism.
         b. Capillary fluid shift mechanism.
         c. Stress relaxation changes in vasculature.
   ii. ***Longterm regulation :***
      a. Renal body fluid mechanism.
      b. Renin angiotensin mechanism.

2. **Humoral regulation**
   a. Epinephrin- norepinephrin mechanism
   b. Vasopressin vasoconstrictor mechanism (ADH)
   c. Renin angiotensin aldoterone mechanism.

(Ref. Guyton & Hall-11th edittion)

Comparison between short-term and long-term regulation of blood pressure :

Short term regulation	*Long term regulation*
i. Rapid onset (seconds to minutes)	i. Slow onset (hours to days)
ii. Controls blood pressure for short duration	ii. Controls blood pressure for long duration
iii. It cannot bring the blood pressure all the way back to normal.	iii. It brings the BP all the way back to normal.

Reflexes & control mechenisms in cardiovascular system

Cardiovascular reflexes are the major instruments by which
i. Heart rate
ii. Blood pressure
iii. Blood flow are maintained and readjusted to the needs of the body.

Each reflex, officially should have-

i. Afferent limb
ii. Center (eg.VMC, cardiac center etc), and
iii. Efferent limb.

However, owing to extensive interconnections between them it is no longer very fashionable to call them individual cardiac centers as they do not work independently. Most reflexes operate principally via the sympathetic system al though the vagus has considerable influence on the heart (but not on the blood vessels in general). Both sympathetic and para-sympathetic discharge tonically. Most efferent sympathetics are vasoconstrictors and withdrawal of these tonic vasocontrictor activity of the sympathetic leads to vasodilation (and also bradycardia). However, sympathetic vasodila tors also exist. The NT for vasoconstrictors are noradrenalin but the NT for sympathetic vasodilators are Ach.

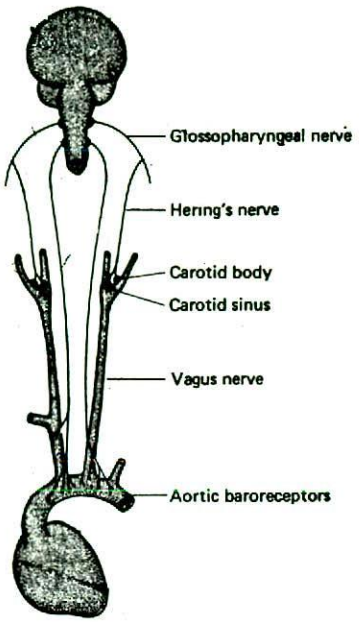


Fig. 6-12. *The baroreceptor system.*

Baroreceptors mechanism

Baroreceptors : The baroreceptors are stretch spray-type nerve endings (receptors) in the walls of the arteries; they are stimulated when stretched.

Sites/locations of baroreceptors :

1. Baroreceptors are extremely abundant-
   i. In the wall of each internal carotid artery slightly above the carotid bifurcation, an area known as the *carotid sinus.*
   ii. In the wall of the aortic arch.
2. A few baroreceptors are located in the wall of every large artery of the thoracic and neck regions.

Response of the baroreceptors to pressure :

1. *Carotid sinus receptors* : They are not stimulated at all by pressures between 0 and 50 to 60 mm of Hg, but above these levels, they respond progressively more rapidly and reach a maximum at about 180 mm of Hg.
2. *Aortic baroreceptors* : The responses of these are similar to those of the carotid receptors except that they operate, in general, at pressure levels about 30 mm Hg higher.

In the normal operating range of arterial pressure, around 100 mm Hg, even a slight change in pressure causes a strong change in baroreflex signals to readjust the arterial pressure back toward normal. Furthermore, the baroreceptors *respond much more to a rapidly changing pressure* than to a stationary pressure.

Baroreceptor control system of blood pressure :

1. Baroreceptors are stimulated when they are stretched due to increase arterial pressure. These signals are transmitted from each carotid sinus through the very small *Hering's nerve* to the glossopharyngeal nerve and then to the *tractus solitarius* in the medullary area of the brain stem. Signals from the arch of the aorta are transmitted through the vagus nerves also into the same area of the medulla.
2. After the baroreceptor signals have entered the tractus solitarius of the medulla, secondary signals *inhibit the vasoconstrictor center.* The net effects are-
   i. *Vasodilatation* of the veins and arterioles throughout the peripheral circulatory system.
   ii. *Decreased heart rate* and *strength of heart contraction.*

   Therefore, excitation of the baroreceptors by pressure in the arteries reflexly *causes the arterial pressure to decrease* because of both a decrease in peripheral resistance and a decrease in cardiac output.

   Conversely, *low pressure has opposite effects*, reflexly causing the pressure to rise back toward normal.

(Ref. Guyton & Hall-11th edition: Page 209)

Baroreceptor

Increase arterial pressure
↓
Arterial baroreceptors stimulation
↓

↓	↓
Vagus nerve	Glossopharyngeal nerve
↓	↓
Cardioinhibitory centre stimulated	Vasomotor centre inhibited
↓	↓
Increase parasympathetic discharge to SA and AV node and ventricular muscle	Venodilatation Vasodilatation
↓	
Decrease heart rate Decrease myocardial contractility	↓

↓
→ Decrease cardiac output
Decrease total peripheral resistance
↓
Decrease arterial pressure.

Pressure buffer system

Nerves from the baroreceptors are called buffer nerves. Baroreceptor system opposes an increase and a decrease in the arterial pressure; so, it is often called a pressure buffer system.

Merits and demerit of baroreceptor regulation :

i. Merits :
   a. Rapidly acting blood pressure control mechanism
   b. A diurnal variation of blood pressure is corrected by baroreceptor mechanism.

ii. *Demerit* :
   a. Very short period of action
   b. Acts only in response to changing pressure, not to static.

Carotid Sinus & Aortic Arch

The carotid sinus is a small dilation of the internal carotid artery just above the bifurcation of the common carotid into external and internal carotid branches. Baroreceptors are located in this dilation. They are also found in the wall of the arch of the aorta. The receptors are located in the adventitia of the vessels. They are extensively branched, knobby, coiled, and intertwined ends of myelinated nerve fibers that resemble Golgi tendon organs. Similar receptors have been found in various other parts of the large arteries of the thorax and neck in some species. The afferent nerve fibers from the carotid sinus and carotid body form a distinct branch of the glossopharyngeal nerve, the carotid sinus nerve , but the fibers from the aortic arch form a separate distinct branch of the vagus only in the rabbit. The carotid sinus nerves and vagal fibers from the aortic arch are commonly called the buffer nerves.

(Ref. Guyton & Hall-11th edition; Page 209)

Control of Arterial Prssure by the Carotid and Aortic Chemoreceptors- Effect of Oxygen Lack on Arterial Pressure : Closely associated with the baroreceptor pressure control system is a chemoreceptor reflex that operates in much the same way as the baroreceptor reflex except that chemoreceptors, instead of stretch receptors, initiate the response.

The chemoreceptors are chemosensitive cells sensitive to oxygen lack, carbon dioxide excess, or hydrogen ion excess. They are located in several small organs 1 to 2 millimeters in size : two carotid bodies, one of which lies in the bifurcation of each common carotid artery, and several aortic bodies lies adjacent to the aorta. The chemoreceptors excite nerve fibers that, along with the baroreceptor fibers, pass through Hering's nerves and the vagus nerves into the vasomotor center.

Each carotid or aortic body is supplied with an abundant blood flow through a small nutrient artery, so that the chemoreceptors are always in close contact with the arterial blood. Whenever the arterial pressure falls below a critical level, the chemoreceptors become stimulated because of diminished blood flow to the bodies and therefore diminished availability of oxygen as well as excess buildup of carbon dioxide and hydrogen ions that are not removed by the slow flow of blood.

The signals transmitted from the chemoreceptors into the vasomotor center excite the vasomotor center, and this elevates the arterial pressure. This reflex helps to return the arterial pressure back toward the normal level whenever it falls too low.

The chemoreceptor reflex is not a powerful arterial pressure controller in the normal arterial pressure range because the chemoreceptors themselves are not stimulated strongly by pressure changes until the arterial pressure falls below 80 mm Hg. Therefore, it is at the lower pressures that this reflex becomes especially important to help prevent still further fall in pressure.

(Ref. Guyton & Hall-11th edition; Page 211)

Schemetic representation of the chemoreceptors mechenism of blood presssure control :

Decreased arterial pressure
↓
Decreased blood supply to chemoreceptors
↓
Hypoxia, excess CO_2 (local) and excess H^+ (local)
↓
Chemoreceptors are stimulated
↓
Signals go to vasomotor area and causes excitation of the vasomotor area
↓
Vasoconstriction (i.e increased total peripheral resistance)
Increased myocardial contractility
↓
Elevation of arterial pressure.

Central nervous system ischemic response. Control of arterial pressure by the brain's vasomotor center in response to diminished brain blood flow :Most nervous control of blood pressure is achieved by reflexes that originate in the baroreceptors, the chemoreceptors, and the low-pressure receptors, all of which are located in the peripheral circulation outside the brain. When blood flow to the vasomotor center in the lower brain stem becomes decreased enough to cause nutritional deficiency, that is, to cause cerebral ischemia, the neurons in the vasomotor center itself respond directly to the ischemia and become strongly excited. When this occurs, the systemic arterial pressure often rises to a level as high as the heart can possibly pump. This effect is believed to be caused by failure of the slowly flowing blood to carry carbon dioxide away from the vasomotor center, the local concentration of carbon dioxide then increases greatly and has an extremely potent effect in stimulating the sympathetic nervous control areas in the brain's medulla. It is possible that other factors such as the build up of lactic acid and other acidic substances, also contribute to the marked stimulation of the vasomotor center and to the elevation in pressure. This arterial pressure elevation

in response to cerebral ischemia is known as the central nervous system ischemic response, or simply CNS ischemic response.

The magnitude of the ischemic effect on vasomotor activity is tremendous : it can elevate the mean arterial pressure for as long as 10 minutes sometimes to as high as 250 mm Hg. The degree of sympathetic vasoconstriction caused by intense cerebral ischemia is often so great that some of the peripheral vessels become totally or almost totally occluded. The kidneys, for instance, often entirely cease their production of urine because of arteriolar constriction in response to the sympathetic discharge. Therefore, the CNS ischemic response is one of the most powerful of all the activators of the sympathetic vasoconstrictor system.

(Ref. Guyton & Hall-11th edition; Page 212)

Schematic representation of the central nervous system ischemic mechanism of blood pressure control :

Decreased arterial pressure
↓
Decreased cerebral blood flow
↓
Cerebral ischemia
↓
Decreased blood flow to vasomotor center
↓
Accumulation of CO_2, lactate and other metabolites in the vasomotor center
↓
Direct stimulation of vasomotor center (Sympathetic nervous control area)
↓
Vasoconstriction (i.e increased total peripheral resistance)
↓
Increased arterial pressure
↓
Increased cerebral blood flow.

Vasopressin mechenism

Decrease circulatory volume i.e hypovolaemia → Decrease blood pressure ↓

Osmoreceptor in the hypothalamus are stimulated → **Stimulate vasopressin sectretion from the posterior pituitary** ↓

Increased plasma osmolality ↑

Anti-diuresis (i.e. decrease urine volume) ↓

Vasoconstiction ↓

↓
Increase blood volume ↓ — Increase total peripheral resistance ↓
Increase cardiac output ↓ — ↓
↓
Increase arterial pressure.

Norepinephrine vasoconstriction mechanism

Decreased arterial pressure
↓
Sympathetic stimulation
↓
↓
Stimulation of adrenal medulla
↓
Catecholamine secretion
↓ — ↓
Vasoconstriction — Increase total peripheral resistance
↓ — ↓
Increase arterial pressure.

Renal body fluid mechanism

When blood pressure rises, causes kidney out put of water and salt increases. This inturn decreases extracellular fluid volume and decreases blood volume. The decreased blood vloume decreases pumping action of heart which return arterial blood pressure back to normal. Conversely, when blood pressure low, the output of water and salt by kidney decreases that increases ECV and ultimately blood volume. The increased blood volume increases mean circulating filling pressure that increases venous return then cardiac out put. This increased cardiac output then increases blood pressure.

↑ B.P
↓
↑ Output by Kidney
↓
↓ E.C.F. volume
↓
↓ Blood volume
↓
↓ Pumping action of heart
↓
↓ B.P

Renin-Angiotensin system

Its role in pressure control and hypertension : Aside from the capability to control pressure through changes in extracellular fluid volume, the kidneys have another powerful mechanism for cotrolling pressure. It is the renin-angiotensin system.

Renin is a small protein enzyme released by the kidneys when the arterial pressure falls too low. In turn, it raises the arterial pressure in several ways, thus helping to correct the initial fall in pressure.

Components of the renin-angiotensin system: Renin is synthesized and stored in an inactive form called prorenin in the juxtaglomerular cells (JG cells) of the kidneys. When the arterial pressure falls, intrinsic reactions in the kidneys themselves cause many of the prorenin molecules in the JG cells to split and release renin. Most of the renin enters the blood and then passes out of the kidneys to circulate throughout the entire body. However, small amount of renin do remain in the local fluids of the kidney and initiates several intrarenal functions.

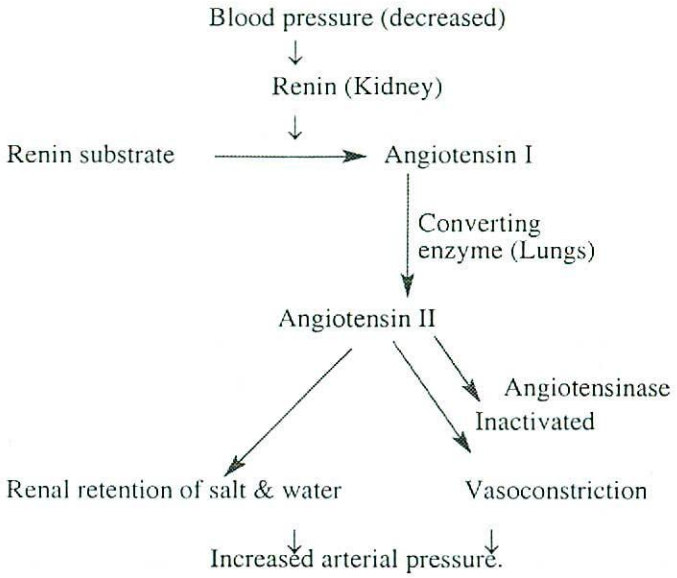


Diagram. Angiotensin Vasoconstriction mechanism for arterial pressure control.

Renin itself is an enzyme not a vasoactive substance. It acts enzymatically on another plasma protein, a globulin called renin substrate (or angiotensinogen) to release a 10 amino acid peptide, angiotensin I. Angiotensin I has mild vasoconstrictor properties but not enough to cause significant functional changes in circulatory function. The renin persists in the blood for 30 minutes to 1 hour and continue to cause formation of angiotensin I during this entire time.

Within a few seconds after formation of the angiotension I, two additional amino acids are split from it to form the 8 amino acid peptide angiotensin II. This conversion occurs almost entirely during the few seeonds while the blood flows through the small vessels of the lungs, catalyzed by the enzyme *converting enzyme* that is present in the endothelium of the lung vessels.

Angiotensin II is an extremely powerful vasoconstrictor, and it has other effects as well that affect the circulation. It persists in the blood only for 1 or 2 minutes because it is rapidly inactivated by multiple blood and tissue enzymes collectively called angiotensinase.

During its persistence in the blood, angiotensin II has two principal effects that can elevate arterial pressure. The first of these, vasoconstriction, occurs rapidly. Vasoconstriction occurs intensely in the arterioles and to considerably less extent in the veins. Constriction of the arterioles increases the peripheral resistance, thereby raising the arterial pressure. Also, the mild constriction of the veins promotes increased venous return of blood to the heart, thereby helping the heart pump against the increasing pressure.

Angiotensin causes the kidneys to retain both salt and water in two ways-

1. Angiotensin acts directly on the kidneys to cause salt and water retention.
2. Angiotensin cause the adrenal glands to secrete aldosterone, and the aldosterone inturn increases salt and water reabsorption by the kidney tubules (Cortical collecting tubules).

1. ***Mechanisms of the direct renal effects of angiotensin to cause renal retention of salt and water :*** Angiotensin has several intrarenal effects that make the kidneys retain salt and water. Probably the most important is to constrict the renal blood vesels, thereby diminishing blood flow through the kidneys. As a result, less fluid filters through the glomeruli into the tubules. Also the slow flow of blood in the peritubular capillaries reduces their pressure, which allows rapid osmotic reabsorption of fluid from the tubules. Thus, for both of these reasons, less urine is excreted. In addition, angiotensin has a moderate effect on the tubular cells themselves to increase tubular reabsorption of sodium and water. The total result of all these effects is significant, sometimes decreasing urine output fourfold to sixfold.

2. ***Stimulation of aldosterone secretion of angiotensin and the effect of aldosterone in increasing salt and water retention in the kidneys.***

   Angiotensin is also one of the most powerful controllers of aldosterone secretion. Therefore, when the renin-angiotensin system becomes activated, the rate of aldosterone secretion usually increases at the same time. One of the most important functions of aldosterone is to cause marked increase in sodium reabsorption by the kidney tubules (cortical collecting tubule), thus increasing the extracelluar fluid sodium. This then causes water retention, thus increasing the extracellular fluid volume and leading secondarily to still more long term elevation of the arterial pressure.

   Both the direct effect of angiotensin on the kidney and its effect acting through aldosterone are important in long-term arterial pressure control. However, research in our own laboratory has suggested that the direct effect of angiotensin on the kidneys is perhaps three or more times as potent as the indirect effect acting through aldosterone even though the indirect effect is the one most widely known.

(Ref. Guyton & Hall-11th edition; Page 223, 224)

Bain Bridge reflex

Rise of pressure in the ritght atrium stimulates the baroreceptors present in its wall. The impulse passes through vagus nerve and reflexly stimulates the H. R & strengh of conraction.

(Ref. Guyton & Hall-11th edition)

Summary of

The integrated, multifaceted system for arterial pressure regulation

Arterial pressure is regulated not by a single pressure controlling system but instead by several interrelated systems each of which performs a specific function. For instance, when a person bleeds severely so that the pressure falls suddenly, two problems confront the pressue control system. The *first* is the survival, that is, to return arterial pressure immediately to a high enough level that the person can live through the acute episode. The *second* is to return the blood volume eventually to its normal level so that the circulatory system can re-establish full normally, including return of the arterial pressure all the back to its normal value, not merely back to a pressure level required for survival.

These mechenisms can be divided into three groups :

1. Those that react rapidly, within seconds or minutes. They are-
   i. Baroreceptor feedback mechenism
   ii. Central nervous system ischemic mechenism
   iii. Chemoreceptor mechenism.
2. Those that respond over an intermediate time period, minutes or hours.
   i. Renin angiotensin vasoconstrictor mechenism
   ii. Stress relaxation of the vasculature
   iii. Shift of fluid through the tissue capillary walls in and out of the circulation to readjust the blood volume as needed.
3. Those that provide long-term arterial pressure regulation, days, months, years.
   i. Renal-blood volume pressure control mechenism (which is the same as the renal-body fluid pressure control mechenism.

(Ref. Guyton & Hall-11th edition; Page 230)

Rapidly acting pressure control mechenism (acting within seconds or minutes) : The rapidly acting pressure control mechenisms are almost entirely acute nervous reflexes or other nervous responses. After any acute fall in pressure, as might be caused by severe haemorrhage, the nervous mechenisms combine-

1. To cause constriction of the veins and provide transfer of blood to the heart.
2. To cause increased heart rate and contractility of the heart and provide greater pumping capacity by the heart.
3. To cause constriction of the peripheral arterioles to impede the flow of the blood out of the arteries.

All these effects occur instantly to rise the arterial pressure back into a survival range.

When the pressure rises suddenly too high, as might occur in response to a drug or extreme, rapid overadministration of a blood transfusion, the same mechenisms operate in the reverse direction, again returning the pressure back toward the normal range.

(Ref. Guyton & Hall-11th edition; Page 230)

Pressure control mechenisms that act after many minures : These mechenisms become mostly activated within 30 minutes to several hours.Their effects can last for long periods, days if necessary. During this time, nervous mechenisms usually fatigue and become less and less effecive, which explains the importance of these non-nervous intermediate time pressure control measures.

i. ***The stress-relaxation mechenism*** is demonstrated by the following example : When the pressure in the blood vessels becomes too high, they become stretched keep on stretching more and more for minutes or hours; as a result, the pressure in the vessels falls toward normal. This continuing stretch of the vessels, called stress-relaxation, can serve as an intermediate-term pressure 'buffer'.

ii. The ***capillary fluid shift mechenism*** means simply that any time the capillary pressure falls too law, fluid is absorbed by capillary osmosis from the tissues into the circulation, thus building up the blood volume and increasing the pressure in the circulation. Conversely, when the capillary pressure rises too high, fluid is lost out of the circulation into the tissues, thus reducing the blood volume as well as all the pressures throughout the circulation.

(Ref. Guyton & Hall-11th edition; Page 230)

Long term mechenisms for arterial pressure regulation : It takes a few hours to show sigficant response. It eventually develops a feedback gain for control of arterial pressure equal to infinity. This means that this mechenism can eventually return the arterial pressure all the way back, not merely partway back, to that pressure level that provides normal output of salt and water by the kidneys.

Many factors can affect the pressure-regulating level of the renal-body fluid mechenism. One of these is aldosterone. A decrease in arterial pressure leads within minutes to an increase in aldosterone secretion, and over the next hour or days, this plays an importasnt role in modifying the pressure control characteristics of the renal-body fluid mechenism. Especially important is the interaction of the renin-angiotensin system with the aldosterone and renal fluid mechenisms.

Thus the arterial pressure control begins with the saving measures of the nervous pressure controls, then continues with

the sustaining characteristics of the intermediate pressure controls, and finally, is stabilized at the long-term pressure level by the renal-body fluid mechenism. This long- term mechenism in turn has multiple interactions with the renin-angiotensin-aldosterone system, the nervous system, and several other factors that provide special presssure control capabilities for special purposes.

(Ref. Guyton & Hall-11th edition; Page 231)

Applied :

Carotid sinus syndrome : Strong pressure on neck over the bifurcation of carotid artery excites the baroreceptors and causes fall of blood pressure upto 20 mm of Hg in normal person. In old person particularly after the formation of calcified arteriosclerotic plaques in the carotid artery, neck pressure (even light collars) over the carotid sinus produces marked fall in arterial pressure causing fainting of the person, such effect is called carotid sinus syndrome.

(Ref. Guyton & Hall-11th edition; Page 148)

Stokes Adams syndrome : When AV block occurs that is, when the cardiac impulse fails to pass from the atria into the ventricles through the AV nodal and bundle system - the atria continue to beat at the normal rate of rhythm of the sinus node, while a new pacemaker develops in the purkinje system of the ventricles and drives the ventricular muscle at a new rate somewhere between 15-40 beats per minute. After a sudden block, the Purkinje system does not begin to emit its rhythmical impulses until 5 to 30 seconds later because, before the blockage, the Purkinje fibers had been "overdriven" by the rapid sinus impulses and, consequently, are in a suppressed state. During these 5 to 30 seconds, the ventricles fail to pump blood, and the person faints after the first 4 to 5 seconds because of lack of blood flow to the brain. This delayed pickup of the heartbeat is called Stokes Adams syndrome. If the delay period is too long it can lead to death.

(Ref. Guyton & Hall-11th edition; Page 121)

Extrasystole or premature beat or ectopic beat

Definition : A premature contraction is a contraction of the heart prior to the time that normal contraction would have been expected. This condition is frequently called extrasystole or premature beat.

Causes : Most premature contractions results from ectopic foci in heart; and the probable causes of ectopic foci are-

i. Local areas of ischemia.
ii. Small calcified plaques at different points in heart, which press against the adjacent cardiac muscle, so that some of the fibres are irritated.
iii. Toxic irritation of the AV node, purkinje system or myocardium caused by drugs, nicotine or caffine.
iv. Mechanical irritation during cardiac catheterization.

(Ref. Guyton & Hall-11th edition; Page 150)

*In **CVS shock***, the cardiac output is not sufficient to meet the demands. Nature's compensatory mechanisms consist mainly of neural (baroreceptor reflex etc), fluid shift and renin angiotensin mechanism.

Heart failure may be seen as pump failure and the cardiac output is insufficient to the need. Compensatory mechanisms include a. Frank starling's law b. Sympathetic overactivity (due to baroreceptor and other reflexes). Pump failure is aggravated because of the operation of Laplace's law.

The ***murmurs*** of valvular disease,logically, can occur even if the critical value of Reynold's number is not exceeded , although exercising the patient increases the propensity of turbulence and murmur.

Common sites of ***heart blocks*** are AVN, bundle branch (right/left). Sick sinus syndrome affects the SAN.

Atrial fibrillation is a chronic condition whereas ventricular fibrillation usually kills instan taneously.

Vascular system

Definition : The vascular system is a closed system of conduits that carry blood from the heart to the tissues and back to the heart.

The forward motion of blood is due to :

i. Pumping action of the heart.
ii. Elastic recoil of the vessels during diastole
iii. Compression of the veins by skeletal muscle during exercise
iv. Negative pressure in the thorax during inspiration.

Classification of blood vessels

Blood vessels can be classified in two ways :

1. Anatomically.
2. Functionally.

1. ***Anatomical classification :***

   A. *Artery :* Vessels which carries oxygenated blood (except pulmonary artery) is called artery. These are -
      i. Large arteries : Aorta and its branches.
      ii. Medium sized arteries : Brachial, Radial, Popleteal etc.
      iii. Small arteries : Arterioles, Terminal arterioles.
      iv. Capillaries or sinusoids.

   B. *Veins:* Vessels which carries deoxygenated blood (except pulmonary veins) is called veins. These are :
      i. Large veins : Superior and Inferior vena-cava and their large tributeries.
      ii. Medium sized vein : Brachial, Tibial veins etc.
      iii. Small veins, Venules etc.

2. **Functional classification of vessels**

a. ***Windkesel (distribution vessels)*** : The large vessels which arises from heart and distributes blood to the whole body, e.g arteries upto the arteriolar level. Percentage of elastic fibers high; elastic recoil ++.

Function : Windkessel effect; vis a tergo.

Applied physiology : Arteriosclerosis →loss of Windkessel.

Effect : Systolic blood pressure increase but diastolic blood pressure falls.

b. ***Resistance vessels*** : The vessels which control blood flow by exerting resistance on the vessel wall, e.g arterioles, terminal arterioles.

Elastic fibers almost nil; percentage of smooth muscles (++).

Function : Seat of peripheral resistance. Smooth muscles are strongly influenced by sympathetic nerves, noradrenalin, adrenalin.

Appllied physiology : Essential hypertension → narrowing of lumen → Diastolic blood pressure increased .

Distribution of vascular resistance :

1. Small arteries & arterioles : 47 %
2. Capillaries : 27 %
3. Arteries : 19 %
4. Veins : 7 %.

(Ref. Colour Atlas of physiology)

c. ***Exchange vessels*** : The vessels which are concerned with the exchange of gases, nutrients, vitamins and hormones between the blood and the body fluid, e.g capillaries. Single layer of endothelium.

Function : Exchange of nutrients, gases and waste products.

d. ***Capacitance vessels*** : The vessels which contains a large volume of blood e.g veins and venacva.

Wall is thicker than venules but much thinner than aorta.

Function : Holds extra fluid (acts as a reservior) without BP elevation.

Venules : Wall is only slightly thick.

General structure of the vascular tree

The aorta and big arteries has three coats :

i. Tunica adventitia
ii. Tunica media
iii. Tunica intima. (i.e tunic = coat).

i. Tunica *intima* : Tunica intima is the innermost coat. Internally it has a lining of single layer of epithelium called the (vascular) endothelium. This layer is very smooth and silky in health. This layer, therefore, is in contact with the blood. External to the endothelial layer, lies, mainly some elastic fibers and this layer of elastic fibers is called, the *lamina propria.*

The lamina propria is externally bounded by a layer of elastic fibers called the fenestrated membrane.

Therefore tunica intima = the endothelial layer + lamina propria +fenestrated membrane.The region of lamina propria is also called the subendothelial region.

In the subintimal layer, cholesterol and other lipid materials are first deposited in the disease called atherosclerosis.

ii. *Tunica media* : External to the fenestrated membrane lies the *tunica media.* In tunica media, there are smooth muscles as well as good deal of elastic fibers. The layer is fairly thick .

iii. *Tunica adventitia :* External to the tunica media, lies the layer called *tunica adventitia.* It consists of collagenous fibers mainly.

The above mentioned histological structure is typical of the Windkessel vessels (aorta and the big arteries). In smaller arteries the picture changes to some extent.

Differences from large arteries or windkessel vessel

1. ***Smaller arteries*** : The amount of elastic fibers, both in the tunica intima and tunica media are much less and the proportion of smooth muscular tissue in the tunica media increases.
2. ***Arterioles*** : The elastic fibers have disappeared from the media and the tunica media consists only of smooth mucles.
3. ***Capillaries*** : There is no adventitia or media. The intima consists only a layer of endothelium standing on a basement membrane.
4. ***Veins***
   i. Walls thinner than arteries.
   ii. Elasticity is very low compared to the arteries.
   iii. Smooth muscle content is also very low.

Vascular smooth muscle

Characteristics :

i. *Position* : Smooth muscles of media are placed circularly round the vesscls. Therefore, when they contract, the diameter of the vesscls is reduced.

ii. *Nerve supply* :
Sympathetic fibers (nor adrenalin)
Receptor : Alpha receptor.

Sympathetic stimulation → release of noradrenalin → combine with alpha receptor → contraction (Sympathetic fibers exert a tonic effect even at rest).

N.B. Alpha receptor can also combine with adrenalin (epinephrine) and phenyl epinephrine; which are also, therefore powerful vasoconstrictors.

*Phenoxybenzamine and dibenamine produce alpha blocking and therfore cause powerful vasodilatation and fall of arterial blood pressure.

3. Sympathetic fibers exert a tonic effect even at rest.
4. Some arterioles (skeletal muscles) contain β_2 receptors → causes vasodialtation. Similarly there is histaminergic receptors.
5. *Cholenergic receptors* : Presents in some smooth muscles which can combine with acetylcholine (Ach) → vasodilatation → fall of blood pressure (these cholenergic receptors, strengly enough are not attached with any nerve fibers).
6. *Others* :
   a. Smooth muscles can be stimulated or inhibited by number of agents :
      i. local O_2 tension
      ii. Lactic acid
      iii. Adenosine
      iv. Histamine
      v. Bradykinin
      vi. Prostaglandins
      vii. Anti-diuretic hormone (ADH) and others.
   b. *Vasomotor tone* : Indicates the degree of tone of the vascular smooth muscle. Thus when the vasomotor tone is high, the vessels are narrowed and vice versa.
   c. *Vasoactive agents* : Substances which can act on vascular smooth muscles.
   d. *Vasoactive amines* : Vasoactive agents which are amine in chemical nature (e.g histamine, 5 hydroxytryptamine).
   e. *Vasopressor agent* : Is a substance that causes contraction of vascular smooth muscle e.g Nor-adrenalin.

Effect of Sympathetic nerve on vascular smooth muscles :

1. Arterioles of some region constricts → e.g Splanchnic and cutaneous.
2. Arterioles of some region dilates → Coronary muscles, skeletul muscles.
3. Arterioles of some other resion are not affected → e.g brain.

* Sympathetic nerve (Stimulation) thus causes redistribution of blood supply.

Capillaries

A. *Diameter* :
   i. At the arterial end : 5mm
   ii. At the venous end : 9 mm

B. *Total area of all the capillary walls in the body* :
Exceeds 6300 square meter (in adalt).

C. ***Structure*** :
Varies from organ to organ

A. *Types of endothelium* :
   i. Continious : This type endothelium present in brain (excluding circumventricular organs), skin, skeletal muscle, lungs, heart.
   ii. Fenestrated : This type endothelium present in gastro-intestinal tract (intestinal mucosu), glomerulus in kidney.

B. *Structure of endothelium in various organ* :
   i. *Muscles (skeletal, cardial & smooth muscle)* : The junction between the endothelial cells permit the passage of molecules up to 10 nm in diameter (vesicular trnsport).
   ii. *Brain* : Tight junction present. Non fenestrated. They permit the passage of small molecules.
   iii. *Endocrine glands (most), intestinal villi and parts of the kidneys* : Fenestration (20-100 mm in diameter) present that permit the passage of relatively large molecules and make the capillaries porous.

iv. *Liver* : Endothelium is discontinious. There are large gaps between endothelial cells that are not closed by membranes (600-3000 nm in diameter).

N.B. Capillaries & post capillary venules have pericytes outside the endothelial cells. They are contractile & release a wide variety of vasoactive agents. They also synthesize and release constituents of the basement membrane & extra cellular matrix.

Function : Regulation of flow through the junctions between endothelial cells, particulary in the presence of inflammation.

Characteristics of various types of blood vesels in humans :

Vessel diameter	*Lumen thickness*	*Wall*	*All Vessels of Each Type*	
			Approximate total cross sectional area (cm^2)	*Percentage of blood volume contained)*
Aorta	*2.5 cm*	*2 mm*	*4.5*	2
Artery	0.4 cm	1 mm	20	
Arteriole	30 μm	20 μm	400	1

Vessel diameter	Lumen thickness	Wall	All Vessels of Each Type: Approximate total cross sectional area (cm^2)	All Vessels of Each Type: Percentage of blood volume contained)
Capillary	5 μm	1 μm	4500	5
Venule	20 μm	2 μm	4000	
Vein	0.5 cm	0.5 mm	40	54
Vena cave	3 cm	1.5 mm	18	

In systemic vessels there is an additional 12% in the heart and 18% in the pulmonary circulation.

Microcirculation

Microcirculation includes the precapillary sphincter region, capillaries and the smallest venules. The name (i.e. microcirculation) owes to its origin to the fact that they can be seen only under the microscope.

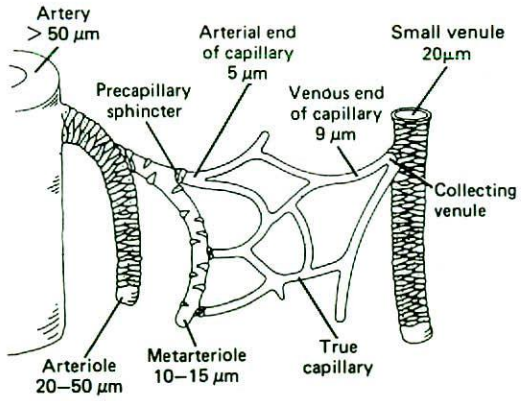


Fig. 6-13. *Microcirculation.*

Arterioles give rise to meta-arterioles which give rise to capillaries. The capillaries drain via a short collecting venules to the venules. The walls of the arteries, arterioles, and small venules contains relatively large amounts of smooth muscles. There are scattered smooth muscle cells in the walls of the meta-arterioles, and the opening of the capillaries are guarded by muscular preapillary spincters.

Factors affecting the caliber of the arterioles

Constriction	Dilation
Local factors	
Decreased local temperature	Increased CO_2 and decreased O_2
Autoregulation	Increased K^+,adenosine, lactate, etc.
	Decreased local pH
	Increased local temperature
Local hormones	No
Endothelin-1	Kinins
Locally released platelet serotonin	
Circulating hormones	
Epinephrine (except in skeletal muscle and liver	Epinephrine in skeletal muscle and liver
Norepinephrine	CGRP-alpha
AVP	Substance P
Angiotensin II	Histamine
Circulating Na^+- K^+ ATPase inhibitor	ANP
Neuropeptide Y	VIP
Neural factors	
Increased discharge of noradrenergic vasomotor nerves.	Decreased discharge of noradrenergic vasomotor nerves.
	Activation of cholinergic dilator fibers to skeletal. muscle

Circulation

Definition : It is the process of blood and lymph flow through a close system of vessels, called circulation.

Types of circulation

There are three types of blood circulation :

1. *Systemic circulation or greater circulation* : Passage of blood from left vetricle to the tissue and from the tissue to right atrium is called systemic circualation.
2. *Pulmonary or lesser circulation* : Passage of blood from right ventricle to the lungs & from the lungs to the left atrium is called pulmonary circulation.
3. *Portal circulation* : Blood from heart generally passes through one set of capillaries before draining into the heart. But in certain organ where the blood passes through two sets of capillaries or sinusoids before draining into the heart. The resulting system is known as portal system.

   It has two types :

   a. Arterial portal system e. g, renal circulation.
   b. Venous portal system e. g.
      i. Hepatic portal system.
      ii. Hypophyseal portal system.

Schemetic representation of the circulation of blood

Heart left ventricle	*Left atrium*
↓	↑
↓	**Pulmonary vein**
↓	↑
↓	Lungs
↓	↑
Large arteries	Pulmonary arteries
↓	↑
Medium arteries	Right ventricle
↓	↑
Small arteries	Right atrium
↓	↑
↓	Large veins
↓	↑

Arteries → ***Microcirculation*** → *Venules*

Importance of circulation

1. To supply the O_2, nutrition, vitamins to the tissue.
2. To carry away different metabolic waste product and CO_2 from tissue for elemination.
3. To prevent intravascular coagulation of blood.
4. Helps to maintain thermal balance through out the body.
5. Maintain optimum environment for cellular function.

Factors maintaining circulation

1. *Pumping action of heart :* This is the main motive force of circulation.
2. *Elastic recoil of arteries :* During ventricular systole certain amount of blood is being led directly into the aorta and thus vessel is stretched (Potential energy is gained). But due to elasticity of vessels wall and the aorta, recoils during ventricular diastole and thus blood moves forword (due to change of potential energy into kinetic energy for flow) towards the periphery.
3. *Pressure gradient :* Blood pressure gradually falls from the left to right side of heart, that is; in big arteries average pressure 120 mm of Hg, in the arterioles 50-60 mm of Hg, in the capillaries 15 mm of Hg and in the veins the pressure falls further, while the pressure near the heart is 0 mm of Hg. Due to this pressure gradient blood passes from higher to the lower pressure.
4. *Respiration :* During inspiration intra thoracic pressure falls and intra-abdominal pressure rises. Hence, with each inspiration, venous blood from abdomen is sucked up by the thorax, helps in venous return and acts as a great force in maintaining circulation.
5. *Muscular exercise :* When muscles contract, they squeeze the capillaries and veins, thus helps venous return and help in maintaining circulation.
6. *Effect of gravity :* Above the level of heart, it helps venous ruturn. but below the level of heart, it works aganist it.

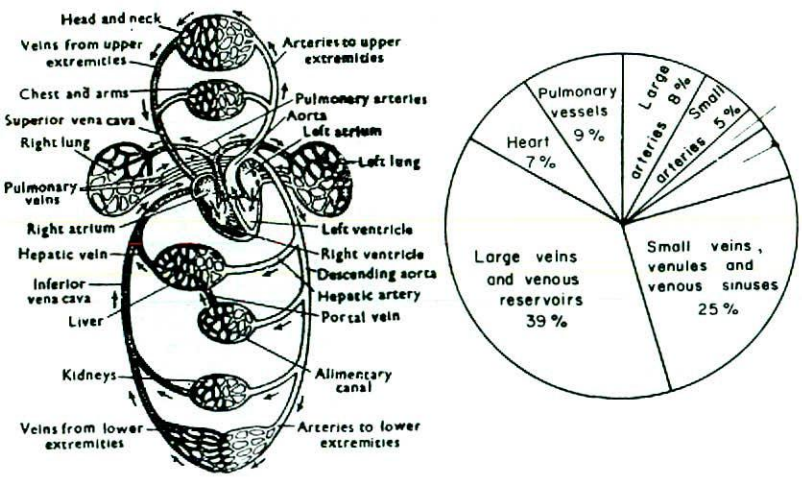


Fig. 6.14 *The different parts of circulatory system.*

Difference between systemic & pulmonary circulation

Systemic	*Pulmonary*
1. Passage of blood from left ventricle to tissues and from the tissues to right atrium.	1. Passage of blood from right ventricle to the lungs and from the lungs to the left atrium.
2. It begins in the left ventricle & via most of all part of body ends in the right atrium.	2. It begins in the right ventricle & via lungs ends in the left atrium.
3. Here artery carries oxygenated blood and veins carries deoxygenated blood.	3. Here pulmonary artery carries deoxygenated blood & pulmonary vein carries oxygendated blood.
4. It supplies nutrition to body tissue.	4. It carries blood to lungs for oxygenation.
5. Here BP is high.	5. Here BP is relatively low.

Q. 00. **What do you mean by high and low pressure system.**

i. High pressure system : The arterial system including the left ventricle.

ii. Low pressure system : The systemic veins, pulmonary circulation and heart chambers other than left ventricle. *(Ref. Ganong 21th Edition; page-589)*

Distribution of blood

Approximately 84 percent of the total blood volume of the body

is in the systemic circulation, with 64 % in the vein, 15 % in the arteries and 5 % in the capillaries. The heart contains 7 % of blood and the pulmonary vessels 9 %.

Q. 00. **State about the humoral regulation of circulation.**

Ans. *Humoral regulation of circulation* : Hormones regulate the circulation by constrict or dilate of the caliber of the arterioles :

i. *Hormones that constrict the arterioles* :
   a. *Local hormones* :
      1. Endothelin-1
      2. Locally released platelet serotonin
   b. *Circulating hormones* :
      1. Epinephrine (except in skeletal muscle and liver)
      2. Norepinephrine
      3. AVP
      4. Angiotensin II
      5. Circulating Na^+-K^+ ATPase inhibitor
      6. Neuropeptide Y.

ii. *Hormones that dilate the arterioles :*
   a. *Local hormones* :
      1. NO (nitrious oxide)
      2. Kinins
   b. *Circulating hormones* :
      1. Epinephrine in skeletal muscle and liver
      2. CGRP-alpha
      3. Substance P
      4. Histamine
      5. ANP
      6. VIP.

(Ref. Ganong 21th Edition; page-603)

(Q. Name the vasoactive substances affecting cardiovascular system.
Q. Name the factors affecting the caliber of the arterioles)

Q. 00. **Name the vasodilator and vasoconstirctor agents.**

Vasoconstrictor agent	*Vasodilator agent*
Local factors :	
Decreased local temperature	Increased CO_2 and decreased O_2
Autoregulation	Increased K^+,adenosine, lactate, etc.
	Decreased local pH
	Increased local temperature.
Endothelial products :	
Endothelin-1	NO
Locally released platelet serotonin	Kinins
Thromboxane A_2	Prostacycline
Circulating hormones :	
Epinephrine (except in skeletal muscle and liver)	Epinephrine in skeletal muscle and liver
Norepinephrine	CGRP-alpha
AVP	Substance P
Angiotensin II	Histamine
Circulating Na^+-K^+ ATPase inhibitor	ANP
Neuropeptide Y	VIP
Neural factors :	
Increased discharge of noradrenergic vasomotor nerves.	Decreased discharge of noradrenergic vasomotor nerves.
	Activation of cholinergic dilator fibers to skeletal muscle

(Ref. Ganong 22th Edition; page-603)

Blood Flow

Q. 00. ***Why blood flows through the vessels.***

Ans. Blood flows through the vessels because-

1. Forward motion imparted to it by the pumping of the heart.
2. Diastolic recoil of the walls of the arteries.
3. Compression of the veins by the skeletal mucles during exercise.
4. Negative pressure in the thorax during inspiration.

Resistance to flow depends on

1. Mostly upon the diameter of the vessels principally the arterioles.
2. Minor degree upon the viscosity of the blood.

Blood flow to the each tissue is regulated by :

1. Local chemical mechenisms.
2. General neural and hormonal mechenisms : That dilate or constrict the vessels of the tissue.

Biophysical consideration (*Arterial*)

i. ***Flow*** : In portion of the vascular system is equal to effective perfusion pressure in that portion divided by the resistance.

$$\text{Flow (F)} = \frac{\text{Pressure (p)}}{\text{Resistance (R)}}$$

ii. ***Effective perfusion pressure*** : It is the mean intraluminal pressure at the arterial end minus the mean pressure at the venous end.

iii. ***Velocity and flow of blood*** :
   1. Aorta (Proxiomal portion) : 40 cm/sec (Range : 120 - 0 cm/sec).
   2. Capillaries : About 0.07 cm/sec (Transient tissue from

the arteriole to the venular end of an average sized capillary is 1 - 2 seconds.)

3. Great veins : About : 10 cm/sec.

iv. ***Laminar flow*** : The flow of blood in the vesscls, like the flow of liquids in narrow rigid tubes, is normally laminar (Stream line).

1. Whithin the blood vessels, an infinitely thin layer of blood in contact with the wall of the vessel does not move.
2. The next layer within the vessels has a low velocity.
3. The next a higher velocity and so forth.
4. Velocity being greatest in the center of the stream.

Turbulence :

Laminar flow occurs at velocities up to a certain critical velocity. At or above this velocity, flow is turbulent. Turbulent flow creats sounds. The probability of turbulent is related to the-

i. Diameter of the vessels
ii. Viscosity of the blood.

$$Re =' \frac{PDV}{r}$$

Here, Re = Renolds number, named for the men who described the relationship.

P = Density of the fluid.

D = Diameter of the tube.

V› = Velocity of flow

n*= Viscosity of the fluid.

Interpretation :

1. Not turbulent : When Re is less than 2000
2. Turbulent : When Re is more then 3000.

Q. 00. **What is Reynold's number?**

Ans.

$$Re = \frac{PDV}{n}$$

Re = ***Renolds number***, named for the men who described the relationship.

P = Density of the fluid

D = Diameter of the tube

V = Velocity of flow

n*= Viscosity of the fluid.

Interpretation :

1. Not turbulent : When Re is less than 2000
2. Turbulent : When Re is more then 3000.

Viscosity :

1. Plasma : 1.8 times as viscus as water.
2. Whole blood : 3 - 4 times as viscus as water.

Thus viscosity depends most part on the hematocrit.

a. In polycythemia : viscosity increase.
b. In anaemia : viscosity decrease.
c. Condition (diseases) in which plasma protein /immunoglobins increase.
d. Diseases in which red blood cells are abnormaly rigid e.g hereditary spherocytosis.

Plasma Skimming

In the vessels, red cells tend to accumulate in the centre of the flowing stream. Consequently, the blood along the side of the vessels has a low hematocrit, and branches leaving a large vessel at right angles may receive a disproportionate amount of red -cell -poor blood. This phenomenon, which has been called plasma skimming.

Effect : Plasma skimming is the reason why the haematocrit of capillary blood is regularly about 25% lower than the whole body hematocrit.

Peripheral resistance

It is the resistance in which blood has to over come while passing through the periphery.

Factors affecting peripheral resistance :

Factors on which the peripheral resistance depends-

i. Velocity of blood
ii. Viscosity of Blood
iii. Elasticity of arterial walls
iv. State of lumen of blood vesesl.

Resistance in cardiovascular system is sometimes expressed in R units. Which are obtained by dividing pressure in mm Hg by flow in mL/Sec.

Example :

When the mean aortic pressure = 90 mm of Hg and left ventricular output = 90 mL/Sec.

Then,

$$\text{The total peripheral resistanc} = \frac{90 \text{ mm Hg}}{90 \text{ mL/Sec}}$$

$$= 1 \text{ R unit.}$$

Distribution of the circulating blood volume at rest :

1.	Systemic veins	: 50%
2.	Heart cavities	: 12%
3.	Pulmonary circulation	: 18%
4.	Aorta	: 2%
5.	Arteries	: 8%
6.	Arterioles	: 1%
7.	Capillaries	: 5%

(Blood in arteries = 16%)

When extra blood is administered by trunsfusion, less then 1% of it is distributed in the arterial system and all rest is

Region (Kg)	% of total CO	Blood flow		Consuption		Percentage of total O_2 consuption
		ml/min	*ml/100g/min*	*ml/min*	*ml/100g/min*	
1. Liver (2.6)	27.8	1500	57.7	51	2.0	20.4
2. Kidneys (0.3)	23.3	1260	420	18	6.0	7.2
3. Brain (1.4)	13.9	750	54	46	3.3	18.4
4. Skin (3.6)	8.6	462	12.8	12	0.3	4.8
5. Skeletal muscle (31)	15.6	840	2.7	50	0.2	20.0
6. Heart muscle (0.3)	4.7	250	84	29	9.7	11.6
7. Rest of the body (23.8)	6.2	336	1.4	44	0.2	17.6
Whole body (63)	*100*	*5400*	*8.6*	*250*	*0.4*	*100*

found in the systemic veins, pulmonery circulation and heart chambers other than the left ventricle.

*High pressure system : Arterial system +Lt ventricle.

*Low pressure system : all systemic veins, pulmonary circulation, heart chambers (right & left atrium +right ventricle).

Fluid exchange through the capillaries

(Equilibration with interstitial fluid) :

In capillary passages of substances are :

1. Substance passes through the junctions between endothelial cells and through fenestration (when they are present).
2. Some also pass through the cells by vesicular transport.
3. In case of lipid soluble substences, throguh the cytoplasm.

The factors other than vesicular transport that are responsible for transport across the capillary wall are -

a. Diffusion : e.g Nutrients, waste materials, O2, glucose, Co2 etc.

b. Filtration : Depends upon (across the capillary wall) :
   a. Hydrostatic pressure gradient
   b. Osmotic pressure gradient.

   (These two are also called starling forces.)

Fluid movement :

Thus, Fluid movement

$$= K[(P_c + p_i) - (P_i + p_c)]$$

Here,

K= Capillary filtration co-efficient (It takes into account and is proportionate to, the permeability of the capillary wall & the area available for filtration).

P_c= Capillary Hydrostatic pressure

P_i= Interstitial hydrostatic pressure. (It varies one organ to another, and there is considerable evidence that it is sub-atmospheric about (-) 2 mm Hg in subcutaneous tissue. It is positive in the liver and kidneys and is as high as 6 mm Hg in the brain.

p_c = Capillary colloid osmotic pressure

p_i = Interstitial colloid osmotic pressure.

p_i is usually negligible, so the osmotic pressure gradient (p_c - p_i) usually equals the oncotic pressure (i.e Capillary colliodal osmotic pressure).

So,

i. Fluid move into the interstitial space at the aroteriolar end of the capillary, where the filtration pressure across its wall exceeds the oncotic pressure.

ii. Fluid move into the capillary at the venular end, where the oncotic pressure exceeds the filtration pressure.

Filtration pressure :

a. Filtration pressure *(outward)* at the arteriolar end : (37-25) = 12 mm of Hg.

b. Filtration pressure (*inward*) at the venular end = (25-17) = 8 mm of Hg.

2. *In other capillaries* :
   i. Renal glomeruli : Fluid moves out of almost the entire length of the capillaries.
   ii. Intestines : Fluid moves into the capillaries through almost their entire length.

Total amount of filtration through the capillaries per day

= 24 Litre

1. This is about 0.3% of the cordiac out put.
2. About 85% of the filtered fluid is reabsorbed into the capillaries and the remainder returns to the circulation via the lymphatics.

Force causing reabsorption of fluid at the venous end of the capillary :

The low pressure at the venous end of the capillary changes the

balance of forces infavour of absorption as follows :

A. *Forces tending to move fluid inward :*
   i. Plasma colloidal osmotic pressure 28 mm Hg.
   Total inward force = 28 mm of Hg.

B. *Forces tending to move fluid outward :*
   i. Capillary pressure : 10 mm of Hg.
   ii. Negative interstitial free fluid pressure :(-) 3 mm of Hg (i.e outward force is 3 mm of Hg).
   iii. Interstital fluid colloid osmotic pressure : 8 mm Hg.

   Total outward force : 21 mm of Hg (10+3+8 = 21 mm Hg)

Summation of forces :

Inward force : 28 mm Hg.

Outward force : 21 mm Hg.

Net inward force = 7 mm of Hg.

Thus the force that causes fluid to move into the capillary, 28 mm of Hg, is greater than that opposing reabsorption, 21 mm Hg. The difference, 7 mm of Hg is the reabsorption pressure. This reabsorption pressure. (7 mm Hg) draws the fluid from the tissue space into the venous end of the capillary.

(Ref. Guyton & Hall11th Edition, Page 189)

Factors responsible for fluid exchange between the blood capillaries and intestitial space

The exchanges of fluid between plasma and the tissue spaces (interstitial spaces), both ways is a continuous process. The exchange has been considered to be due to the operation of the following factors :

1. *Capillary membrane and its permeability :*
   a. Lipid soluble materials through the cells
   b Lipid insoluble materials through the pores.
   c. Large molecular substances like protein dextran etc. through pinocytosis.
2. Diffusion
3. Filtration
4. Absorption
5. Metabolic activity of the tissue.

Forces causing filtration at the arterial end of the capillary

The approximate average forces operating at the arterial end of the capillary that cause movement through the capillary membrane are :

A. *Forces tending to move fluid outward :*
   1. Capillary hydrostatic pressure 30 mm of Hg.
   2. Negative interstitial free fluid pressure- 3mm Hg (i.e *Outward force 3 mm of Hg*).
   3. Interstitial fluid colloidal osmotic pressure- 8 mm Hg.

   Total outward force = 41 mm Hg(30+3+8 =41 mmHg).

B. *Forces tending to move fluid inward :*
   1. Plasma colloidal osmotic pressure- 28 mm Hg.
   Total inward force = 28 mm Hg.

Summation of forces :

Out ward forces : 41 mm Hg

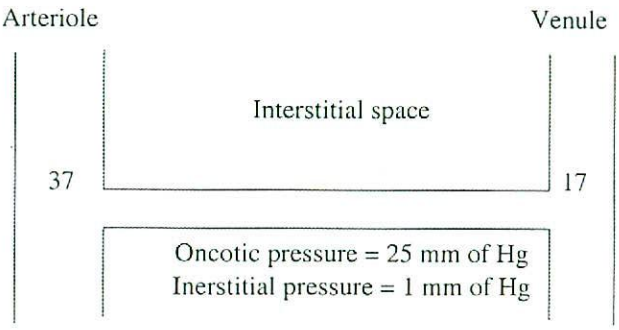
In ward forces : 28 mm Hg.

Net out ward force = 13 mm Hg (41-28 mm of Hg).

This 13 mm Hg filtration pressure causes on the average about 0.5 percent of the plasma to filter out of the arterial end of the capillaries into the interstitial spaces.

(Ref. Guyton & Hall-11th Edition, Page 189)

1. ***In Skeletal muscle :***



Lymphatic circulation

24 hours lymph flow = 2 - 4 litres.

Lymphatic vessels

1. *Initial lymphatics* (Lack of valve and smooth mucles in their wall) : They drain into the collceting lymphatics.
2. *Collecting lymphatics* (Have valves & smooth muscles in their walls).

Causes of flow :

a. Contraction of smooth mucles in peristaltic fashion (major factor).
b. Movement of skeletal muscles.
c. Negative intrathoracic pressure during inspiration.
d. Suction effect of high velocity flow of blood in the veins in which the lymphatics terminates.

Functions :

1. Fluid efflux normally exceeds influx across the capillary walls, but the extra fluids enters the lymphatics and drain through them back into the blood.
2. Proteins are returned to the blood stream via lymphatics (is equal to 25-50% of total circulating plasma protein /day) mainly from liver & intestine and smaller quantities from other tissues.

3. The transport of absorbed long chain fatty acids & cholesterol from the intestine via the lymphatics.

Capillary circulation :

At any one time, only 5% of the circulating blood is in the capillaries, but this 5% is in a sense the most important part of the blood volume because it is across the systemic capillary walls that O_2 and nutrients enter the interstitial fluid and Co_2 and waste products enter the blood stream. The exchange across the capillary walls is essential to the survival of the tissues.

Lymphatic circulation

Fluid efflux normally exceeds influx across the capillary walls, but the extra fluid enters the lymphatics and drains through them back into the blood. This keeps the intestinal fluid pressure from rising and promotes the turnover of the tissue fluid . The normal 24 hours lymph flow is 2 - 4 litre.

Functional anatomy :The lymphatic channels are present in every tissue, excepting the central nervous system and cornea. The lymph channels start as blind capillaries lined by single layer of endothelium but no basement membrane. In these blind capillaries tissue fluid and some protein molecules enter from the tissue. But these fluid and protein molecules, once they enter these channels, cannot leave them. These vessels then drain into bigger vessels which are fewer in number, which in turn, open into still bigger (and still fewer in number) vessels. Ultimately all lymphatic fluid opens into right and left subclavian veins respectively. Thus the fluid and the protein of the tissue fluid are returned to the blood.

On its way the lymphatic channels are interrupted by lymph nodes. On approaching a lymph node, a lymph channel breaks up into several afferent channels, all of which enter the lymph nodes. From the lymph node one (or sometimes more) single channel, called efferent channel, draining the lymph node, emerges and continues onward. The lymph node adds lymphocytes and antibodies to the flowing lymph. Therefore, these lymphocytes and antibodies eventually enter the blood in the subclavian vein. The lymphatic channels contain valves which ensure a unidirectional flow.

Functions of the lymph :

1. Plasma proteins which somehow excape into the tissue fluid are returned by the lymph flow. It has been calculated that about 200 gms of protein are returned to the blood through the lymphatics per day. In addition, the lymphatic circulation also carries hormones to some extent.
2. The lymphocytes and the antibodies enter the blood via the lymph.
3. The lymph nodes destroy any incoming bacteria. Thus if there be a bacterial infection, the afferent channels contain bacteria, but as the bacteria enter the lymph node, they are destroyed, so the efferent channel is sterile (bacteria free). Before entering blood, the lymph thus passes through about eight lymph nodes.
4. From the intestine, big lipid molecules (plus a little proteins) enter the lymph channels within the villi and thus enter the blood. Such big fatty molecules are called chylomicrons. After a heavy fat meal, the serum is often milky because of heavy concentration of chylomicron in it.

Interstitial fluid volume :

Interstitial fluid volume = 15% of total body weight (or 75% of extracelluar fluid component).

Interstitial fluid depends upon :

1. Capillary pressure
2. Interstitial fluid pressure
3. The oncotic pressure
4. The capillary filtration co-efficient
5. The numbers of the active capillaries
6. The lymph flow
7. The total ECF volume : The ratio of pre-capillary to post-capillary venular resistance is also important.

N.B.
Edema : is the accomulation of interstitial fluid in abnormally large amount.

Causes of increase interstitial fluid volume and edema :

1. Increased filtration pressure :
   a. Arteriolar dilatation
   b. Venular constriction
   c. Increased venous pressure
      i. Heart failure
      ii. Incompetent valve
      iii. Venous obstruction
      iv. Increased total ECF volume
      v. Effect of gravity etc.
2. Decreased osmotic pressure gradient across the capillary :
   a. Decreased plasma protein level.
   b. Accumulation of osmotically active substances in interstitial space
3. Increased capillary permeability
   a. Substance P
   b. Histamine & related substance
   c. Kinins, etc.
4. Inadequate lymph flow.

(Ref. Ganong 22th Edition)

Cerebral Circulation

a. Vessels :
   1. Two internal carotid arteries.

2. Two vertebral arteries.

The vertebral arteries unite to from the basilar artery and the basilar artery and the carotids form the circle of Willis below the hypothalamus.

The circle of Willis is the orgin of the six large vessels supplying the cerebral cortex.

N.B. A relatively small fraction of the total arterial flow is carried by the vertbral orteries.

There is no crossing over, probably because the pressure is equal on both sides i.e one carotid artery is distributed almost exclusively to the cerebral hemisphere on that side.

b. ***Anastomoses*** : There are precapillary anastomoses between the cerebral arterioles but flow through these channels is generally insufficient to maintain the circulation and prevent infarction when a cerebral artery is occluded.

c. ***Venous drainage*** :

1. Deep veins
2. Dural sinuses.

These veins empties principally into internal jagular veins. Although a small amount of blood drains through the ophthalmic & pterygoid plexus -through emissary veins to the scalp and down the system of paravertebral veins in the spinal canal.

d. ***Anatomical features of brain capillaries*** :

1. *Non fenestrated.*
2. *There are tight junctions between the endothelial cells* : That limits the passages of substances through the junctions.
3. *There are relatively few vesicles in the endothelial cytoplasm* : So there is little vesicular transport (i.e endocytocytosis & exocytosis).
4. *The brain capillaries are surrounded by the end feet of astrocytes* : These endfeet are closely applied to the basal lamina of the capillaries, but they do not cover the entire capillary wall and there are gaps of about 20 nm between endfeet. However the endfeet induce the tight junctions in the capillaries.

e. ***Nerve Innervation*** :

3 Systems of nerve innervate the crebral blood vessels :

1. Postganglinic sympathetic neurons.
2. Cholenergic neuron.
3. Postganglionic cholenergic neuron.

Q. 04. ***Name the factors that regulate cerebral circulation.**

Ans. *Factors affecting overall cerebral blod flow are:*

i. Intracranial pressure
ii. Local constriction and dilation of cerebral arteries.
iii. Mean arterial pressure at brain level
iv. Viscosity of blood
v. Mean venous pressure at brain level.

(Ref. Ganong 22th Edition; page-617)

Coronary circulation

The right atrium receives blood through the superior venacava (blood is collected from upper part of body), the inferior venacava (blood from lower part of body), the coronary sinus and other smaller veins draining the wall of heart (venae cordis minimie and anterior cardiac vein).

From right atrium blood passes into right ventricle through right atrio-ventricular opening. From right atrium blood passes into the lungs through pulmonary trunk.

The left atrium receives the blood from the lungs through four pulmonary veins. The blood from left atrium passes into the left ventricle via left atrioventricular orifice.

From the left ventricle blood enters into the ascending aorta via aortic opening and passes into systemic circulation.

Q. 05. ***Give clinical importance of coronary circulation.**

Ans. When flow through a coronary artery is reduced to the point that the myocardium it supplies becomes hypoxic, 'P factor' accumulates and ***angina pectories*** develops. If the myocardial ischemia is severe and prolonged, irreversible changes occur in the muscle, and the result is ***myocardial infarction***. The most common cause of myocardial infraction is rupture of an atheroslerotic plaque, or hemorrhage into it, which triggers the formation of a coronary occluding clot at the site of the plaque.

(Ref. Ganong 22th Edition; page 622)

Q. 01. ***Name the factors that regulate coronary circulation.**

Ans. *Variations in coronary flow* : At rest, the heart extracts 70-80% of the O_2 from each unit of blood delivered to it. O_2 consumption can be increased significantly only by increasing blood flow. Therefore, it is not surprising that blood flow increases when the metabolism of the myocardium is increased. The caliber of the coronary vessels, and consequently the rate of coronary blood flow, is influenced not only by pressure changes in the aorta but also by chemical and neural factors. The coronary circulation shows considerable autoregulation.

i. ***Chemical factors*** : The close relationship between coronary blood flow and myocardial O_2 consumption indicates that one or more of the products of metabolism cause coronary vasodilation. Factors suspected of playing this role include O_2 lack and increased local concentrations of CO_2, H^+, K^+, lactate, prostaglandins, adenine nucleotides, and adenosine. More than one of these vasodilator metabolites could be involved. Asphyxia, hypoxia, and intracoronary injections of cyanide all increase coronary blood flow 200-300% in denervated as well as intact hearts, and the feature common to these three stimuli is hypoxia of the myocardial fibers. A

similar increase in flow is produced in the area supplied by a coronary artery if the artery is occluded and then released. This ***reactive hyperemia*** is similar to that seen in the skin. There is evidence that in the heart it is due to release of adenosine. The adenosine appears in addition to ameliorate the reperfusion-induced injury that occurs when blood flow is reestablished.

(Ref. Ganong 22th Edition; page 622)

ii. ***Neural factor*** : The coronary arteries contain a- adrenergic receptors, which mediate vasoconstriction, and b- adrenergic receptors, which mediate vasodilatation.

(Ref. Ganong 22th Edition; page 622)

Pulmonary circulation

1. ***Vascular bed*** : Pulmonary vascular bed resembles the systemic circulation except -
   i. Thickness : Walls of the pulmonary artery and its large branches are about 30% as thick as the wall of the aorta and small arterial vessels.
   ii. Arterioles : are endothelial tubes with relatively little muscle in their walls.
   iii. Post capillary vessel wall : There is also some smooth pulmonary capillaries : are large, and there are multiple anastomoses, so each alveolus sits in a capillary of basket.
2. ***Pressure*** : The entire vascular system is a distensible low pressure system.
   i. Systolic blood pressure : 24 mm of Hg.
   i. Diastolic blood pressure : 9 mm of Hg.
   ii. Mean pressure : 15mm of Hg.
   iii. Blood pressure in left atrium: 8 mm of Hg

   So pressure gradient = 15 - 8 mm of Hg = 7 mm of Hg.

   N.B. Pressure fall from the pulmonary artey to the capillaries is relatively small.

   Capillary blood pressure =10 mm of Hg (about).
3. ***Volume*** :

   At any one time is about 1 litre.

   In capillaries less than 100 ml.
4. ***Velocity*** : Root of the pulmonary artery : 40 cm/sec (about) (as that in aorta).

   It falls of rapidly then rises slightly again in the larger pulmonary veins.

   A red cell gets time to traverse the pulmonary capillaries-
   a. At rest : 0.75 second
   b. During exercise : 0.3 second or less.
5. ***Eflect of gravity on blood flow*** :

   There is linear increase in pulmonary blood flow from the apices to the bases of the lungs.
   1. The pressure in the capillaries at the top of the lungs : is close to the atmospheric pressure in the alveoli and is just sufficient to maintain perfusion.
   2. In the middle portion of the lungs : The pulmonary arterial and capillary pressure exceeds alveolar pressure.
   3. In the lower portion of the lungs : Alveolar pressure is lower than the pressure in all parts of the pulmonary circulation and blood flow is determined by the arterial-venous pressure difference.

   N.B For details of pulmonary pressure, pl follow the *Respiratory system.*

Peripheral pooling of blood

Results when an individual rises from a supine to an erect position. Standing causes approximately 500 ml of blood to shift from the pulmonary circulation to the dependent veins of the legs. In the absence of reflex compensations, there is a reduction in venous return.

Venous circulation

Blood flows through the blood vessels, including the veins primarily because of the pumping action of the heart, although venous flow is aided by the heart beat, the increase in the negative intrathoracic pressure during each inspiration, and contractions of skeletal muscles that compress the veins (muscle pump).

Venous Pressure & Flow : The pressure in the venules is 12-18 mm Hg. It falls steadily in the larger veins to about 5.5 mm Hg in the great veins outside the thorax. The pressure in the great veins all their entrance into the right atrium (Cental venous pressure) averages 4.6 mm Hg but fluctuates with respiration and heart action.

Peripheral venous pressure, like arterial pressure, is affected by gravity. It is increased by 0.77 mm Hg for each cm below the right atrium and decreased by a like amount for each 1cm above the right atrium the pressure is measured.

When blood flows from the venules to the large veins, its average velocity increases as the total cross sectional area of the vessels decreases. In the great veins, the velocity of blood is about one-fourth that in the aorta, averaging about 10 cm/second.

Circulation through the special regions

Resting blood flow & O_2 consumption of various organs in a 63 kg adult man with a mean arterial blood pressure of 90 mm of Hg and an O_2 consumption of 250 ml/min.

Blood Pressure

Arterial pressure :

1. In the aorta and in the brachial and other large arteries : 120/70 mm of Hg.

a. Mean pressure = About 50 mm of Hg.

2. In the large & medium sized arteries : pressure falls slightly, because their resistance to flow is low.
3. In the small arteries & arterioles : Pressure falls rapidly, because these are the main sites of peripheral resistance.
   a. Mean pressure at the end of the arterioles = 30 - 35 mm of Hg.
   b. Pulse pressure at the end of the arterioles = 5 (about) mm of Hg.

Gravitational effect : The pressure in any vessel above the heart level is decreased and that in any vessel, below the heart lvel, increased- is about 0.77 mm Hg/cm.

Example : If pressure at heart level = 100 mm of Hg

i. 50 cm above the heart, the pressure is- 62 mm of Hg (100-0.77x50 = 100-38.5 = 61.5 mm of Hg).
ii. 105 cm below the heart level the pressure is- 180 mm of Hg (100 + 0.77x105 = 100 + 80 = 180 mm of Hg).

Capillary pressure : Capillary pressure vary considerably. Typical values in human nail bed capillaries are-

1. At the arteriolar end : 32 mm of Hg.
2. At the venous end : 15 mm of Hg.

Pulse pressure :

1. At the arteriolar end : about 5 mm of Hg.
2. At the venous end : approximately (0) mm of Hg.

Venous pressure :

1. In the venules : 12 -18 mm of Hg.
2. Larger veins : 5.5 mm of Hg. (about)
3. Cetral venous pressure : 4.6 mm of Hg. But flactuates with respiration & heart action.

   (Cetral pressure is the pressure of the great veins at their entrance into the right atrium).

   Gravitational effect : It increased by 0.77 mm Hg for each cm below the right atrium and also decreased by 0.77 mm of Hg for each cm above the right atrium.
4. The mean pressure at the anticubitul vein = 7.1 mm of Hg.

Shock

Q. 00. ***What is shock?**

i. *Defination :* It is an abnormal physiological condition resulting from inadequate propultion of blood to the aorta thus causes inadequate blood flow perfusing the capillaries of tissues and organs.

ii. *Types of shocks* :
   1. Hypovolumic shock :
      a. Haemorrhagic
      b. Traumatic
      c. Dehydration
      d. Burn
   2. Cardiogenic shock
      a. Myocardial infarction
      b. Congestive cardiac failure
      c. Arrythmias
   3. Normovolemic shock (septic shock, anaphylatic shock)
   4. Neurogenic shock.

   (Q. *Classify shock. *or* name the types of shock.).

Tissue necrosis in severe shock

Not all cells of the body are equally damaged by shock because some tissues have better blood supplies than others. For instance, the cells adjacent to the arterial ends of capillaries receive better nutrition than the cells adjacent to the venous ends of the same capillaries. Therefore, one would expect more nutritive deficiency around the venous ends of capillaries than elsewhere. For instance necrosis in the center of a liver lobule, the portion of the lobule that is last to be bathed by the blood as it passes through the liver sinusoids.

Similar punctate lesions occur in heart muscle, although here a definite repetitive pattern, such as occurs in the liver, cannot be demonstrated. Nevertheless, the cardiac lesions play an important role in leading to the final irreversible stage of shock. Deteriorative lesions also occur in the kidneys, especially in the epithelium of the kidney tubules, leading to kidney failure and occasionally uremic death several days later. Deterioration of the lungs also often leads to respiratory distress and death several days later- called the shock lung syndrome.

(Ref. Guyton& Hall-11th Edition; page 283)

Hemorrhage

It means abnormal internal or external discharge of blood. It may be arterial, venous or capillary. Arterial blood is bright red; flows in spouts. Capillary blood is of a reddish colour; exudes fom tissue. Venous blood is dark red; flow is continuous.

Q. 03. ***What are the compensatory haemodynamic responses that occur to maintain adequate tissue blood flow following acute moderate haemorrhage.**

Ans. *Compensatory reactions activated by hemorrhage*:

i. Vasoconstriction
ii. Tachycardia
iii. Venoconstriction
iv. Tachypnea → increased thoracic pumping
v. Restlessness → increased skeletal muscle pump- ing (in some cases)
vi. Increased movement of interstitial fluid into capillaries
vii. Increased secretion of norepinephrine and epinephrine
viii. Increased secretion of vasopressin
ix. Increased secretion of glucocorticoids
x. Increased secretion of renin and aldosterone

xi. Increased secretion of erythropoietin
xii. Increased plasma protein synthesis.

(Ref. Ganong 22th Edition; Page 637)

Q. 00. **State the effect of-**

i. potassium	v. acetylcholine
ii. calcium	vi. atropine
iii. sodium	vii. temperature

iv. adrenaline - on heart function.

i. *Effect of potassium (K^+) on heart :*

a. *Excess potassium* in the extracellular fluids causes the heart to become extremely dilated, and flaccid and slows the heart rate.

b. *Very large quantities* can also block conduction of the cardiac impulse from the atria to the ventricles through the AV bundle. Elevation of potassium concentration to only 9 to 12 mEq/ liter-two to three times the normal value can cause such weakness of the heart and abnormal rhythm that this can cause cardiac death.

(Ref Guyton & Hall-11th Edition; page 113)

ii. *Effect of calcium (Ca^{++}) on heart* :

a. An excess of calcium ions causing the heart to go into spastic contraction opposite to effect of K^+. This is due to direct effect of Ca^{++} ion in exciting the cardiac contractile process.

b. Conversely, a deficiency of Ca^{++} causes cardiac flaccidity, similar to effect of K^+.

(Ref Guyton & Hall-11th Edition; page 113)

iii. *Effect of sodium (Na^+) on heart* :

a. An excess of sodium ions depresses the cardiac function, that is dilate and flaccid the heart and slows the heart rate as that of K^+ but for an entirely different reason.
Na compete with Ca^{++}, i.e the greater the sodium ion concentration in the extracellular fluid the less the effectiveness of the Ca^{++} in causing contraction when an action potential occurs.

(Ref Guyton & Hall-11th Edition)

iv. *Effects of adrenaline* : It increases the rate and force of contraction. It-
i. Stimulates SA and AV node
ii. Increases conductivity and automaticity.

v. *Effect of acetylcholine* : It decreases the rate and force of contraction of heart.

vi. *Effect of atropine* : At low dose it lowers the heart rate by vagul stimulation. But at high dose it increases heart rate by medullary stimulation.

vii. *Effect of temperature* : Increase body temparature, increases heart rate by directly stimulating the SA node.

Concept of hydrostatic pressure and oncotic pressure

Hydrostatic pressure

Hydrostatic pressure is the pressure which is caused by the effects of gravity on a fluid filled system *i.e* it occurs as a result of the weight of the blood in the vessels.

Types with normal values : :
i. Capillary hydrostatic pressure (Pc) range : 30-45 mm Hg
ii. Interstitial hydrostatic pressure (Pi) range : 1 mm Hg.

Effects :
i. *Capillary hydrostatic pressure* : An increase in capillary hydrostatic pressure results in increase in filtration (i.e. outward flow).
ii. *Interstitial hydrostatic pressure* : An increase in interstitial hydrostatic pressure hinders additional fluid filtration from the capillaries.

Oncotic pressure

Oncotic pressure results from the osmotic pressure of blood proteins.

Types with normal values :
i. Plasma colloid osmotic pressure : 25-27 mm of Hg
ii. Interstitial oncotic pressure : 5-10 mm of Hg.

Importance / significance :
i. *Plasma colloid osmotic pressure* (blood oncotic pressure) provides a force that tends to reabsorb fluid into the capillaries from the interstitial space.
ii. *Interstitial oncotic pressure* tends to pull fluid out the vascular system.

Vascular endothelium

The walls of the blood vessels are made up of a single layer of endothelial cells. Endothelium occupies a strategic interface between blood and body. The endothelial lining varies from organ to organ.

Types of endothelium :
i. *Continuous type of endothelium* : The junctions between endothelial cells permit the passage of molecules up to 10 mm in diameter. They are found in vascular beds of skeletal, cardiac and smooth muscle.
ii. *Fenestrated endothelium* : Large transcellular openings (2000-20,000 nm) exist in the endothelial cells. They are found in most endocrine glands. GIT and parts of the kidney (renal glomeruli).
iii. *Endothelium with tight junctions* : Junctions between endothelial cells are lighter (fused), though physiological evidence suggestive of presence of pores approximately 400 nm in diameter. They are found in most body tissue and brain (no pores).

iv. *Endothelium with gap junctions* : There is large gap between endothelial cells, so endothelium is extremely porous and discontinuous. They are found in vascular bed of bone marrow, liver, and spleen i.e. in sinusoidal capillaries.

Functions of endothelium : The vascular endothelium is a *cardiovascular endocrine organ*. It is an active metabolic tissue and has many regulatory rules :

i. *Vasomotor control* : Enzyme located on the endothelial surface control the level of circulatory compounds such as bradykinin, serotonin, angiotensin and adenine nucleotides.

ii. *Maintenance of an adequate organ blood flow* : Endothelial derived substance *endothelial derived relaxing factor (EDRF)* or *nitric oxide (NO)* evokes relaxation of vascular smooth muscle and have anti-platelet property.

iii. *Pro and anti-thrombotic mechanism* : The normal endothelium is negatively charged (platelet also negatively charge) anti-thrombotic surface, which under physiological conditions does not react with platelets or blood constituents. The endothelial lining of the blood vessels also forms prostacycline that inhibits platelet aggregation.

iv. *Metabolic and immunological functions* :

v. *Regulation of vascular cell growth*

vi. *Modulation of imunoresponses*.

Substances secreted by the endothelium

Endothelial cells synthesize a number of mediators which contribute to the physiological control of circulation, vascular tone, and platelets functions.

These include :

i. *Prostacycline ($PG\text{-}I_2$)* : Prostacycline is synthesized by endothelial cells from arachidonic acid via cyclo-oxygenase pathway. Endothelial cells can produce cyclo-oxygenase enzyme also. Prostacycline promotes vasodilatation and inhibits platelets aggregation i.e. counteracts to thromboxane A_2.

ii. *Endothelial derived relaxing factor (EDRF) or NO* : The endothelial derived relaxing factor (EDRF), has now been identified as nitric oxide (NO). NO is synthesized from arginine by the enzyme *NO synthase*. The release of NO can be triggered by the sheer stress (flow) and by a number of substances including bradykinin, histamine, noradrenaline, substance P, platelet products, serotonin, and thrombin.

iii. *Endothelins (ET_5)* : Endothlein are the families of 21 amino acid polypeptide produced by the endothelial cells. At least three different types of endothelins are recognized.
   a. Endothelin-1 (ET-1) : Brain, kidney and endothelial cells.
   b. Endothelin-2 (ET-2) : Intestine.
   c. Endothelin-3 (ET-3) : Intestine and adrenal glands.

Functions of endothelins :

1. *Cardiovascular effects* :
   a. Long lasting vasoconstriction followed after initial vasodilatation.
   b. Positive chronotropic and ionotropic effects on myocardium.
2. *Neuroendocrine effects* :
   a. Increase plasma level of ANP, renin, aldosterone and catecholamines.
   b. Modulates synaptic transmission.
3. *Gastrointestinal tract* : Regulates gastrointestinal blood flow.
4. *Metabolism* : Enhances gluconeogenesis
5. *Renal effects* :
   a. Renal vasoconstriction, decrease GFR, and renal blood flow
   b. Decrease Na^+ reabsorption through inhibition of Na^+-K^+ ATPase.
6. *Pulmonary effects* : Causes bronchoconstriction.

Hypertension

The level of blood pressure varies with age, sex, race and country. Hypertension is defined arbitrarily at levels above generally accepted *normals*, for example 140/90 mm Hg at the age of 20, 160/95 mm Hg at the age of 50.

WHO criteria includes :

160/95 : definitely hypertensive

140/90 : borderline hypertensive.

Classification of hypertension

Classification	*Range (mm Hg)*
i. ***Diastolic*** :	
a. Normal	: < 85
b. High normal	: 85-89
c. Mild hypertension	: 90-104
d. Moderate hypertension	: 105-114
e. Severe hypertension	: > 115
ii. ***Systolic (when diastolic <90)*** :	
a. Normal	: < 140
b. Borderline systolic hypertension	: 140-159
c. Isolated systolic hypertension	: > 160

Clinical classification : Clinically hypertension is classified as-

i. *Primary or essential hypertension (90-95%)* : In the large majority cases no cause can be identified, and this form of hypertension is know as primary or essential hypertension.

ii. *Secondary hepertension (10%)* : A cause of hypertension can be discovered in less than 10% of patients; such are known as secondary hypertension.

Causes of hypertension

i. *Essential or primary hypertension* : No single factor has been found; many factors are probably responsible :
   a. Genetic and familial
   b. Socio-economic : related to social deprivation
   c. Dietary factors : obesity, high salt intake, high alcohol
   d. Humoral factors : high renin, reduced NO, ANP
   c. Neurotransmitter : acetylcholine, noradrenaline, substance P, serotonin, dopamine.

ii. *Secondary hypertension* :
   a. Renal diseases : Glomerulonephritis, pyelonephritis, polycystic kidney disease.
   b. Endocrine causes : Pheochromocytoma, Cushing syndrome, acromegaly.
   c. Cardiovascular causes : Coarctation of aorta.
   d. Drugs : Estrogen containing oral contraceptives, anabolic steroids, vasopressin etc.

Features of hypertension

i. May be asymptomatic

ii. Patient may present with complications due to hypertension:
   a. Congestive cardiac failure
   b. Hypertensive encephalopathy
   c. Cerebral hemorrhage or infraction (stoke)
   d. Hypertensive retinopathy
   c. Progressive renal failure.

3. Occasionally headache.

***Direction** : Write T for true & F for false against each of the following statement.*

Circulation

Q. 01. **In physiological state**
- T a. capillaries contain 5% blood.
- F b. veins contain 25% blood.
- F c. arterioles contain 10% blood.
- F d. veins contain 5% blood.
- F e. capillaries contain 25% blood.

Q. 02. **Veins differ from the arteries in that**
- T a. they are considered as capacitance vessels
- T b. they show more distensibility.
- T c. their tunica media have lesser quantity of smooth muscles.
- F d. they have more hydrostatic pressure.
- F e. they offer more resistance to blood flow.

Q. 03. **Arterioles**
- T a. provide most of the peripheral resistance.
- T b. play a major role in the regulation of blood pressure
- T c. play a major role in the regulation of local blood flow.
- F d. are known as exchange vessels.
- F e. play a major role in the regulation of heart rate

Q. 04. **Systemic veins**
- T a. contain valves that pmmote venous return during muscular activity.
- T b. act as a capacitance vessels in the circulation.
- T c. constrict in response to sympathetic activation.
- F d. are the main resistance vessels in the circulation.
- F e. have a lower compliance than systemic arteries.

Q. 05. **Vein**
- T a. is made of thin vascular wall.
- T b. contains most of the blood.
- T c. has sympathetic vasoconstrictor innervations.
- F d. receives nutntion from vasa vasornm arising from their lumen.
- F e. is known as resistance vessel.

Q. 06. **The venous system can act as a reservoir for peripheral blood chiefly because of the**
- T a. large volume capacity of the venous system
- F b. low oxygen saturation in the venous system
- F c. low compliance of the venous wall.
- F d. absence of smooth muscle fibers in the venous wall.
- F e. superficial location of the veins.

Q. 07. **Blood flow**
- T a. may be increased locally by endothelial production of nitric oxide.
- T b. is inversely proportional to vascular resistance.
- F c. to the heart is maximum during ventricular systole.
- F d. to the skin is largely under local metabolic control.
- F e. to the brain is largely under autonomic nervous control.

Q. 08. **The blood flow**
- T a. is inversely proportional viscosity of the blood.
- T b. increases when blood volume is increased
- T c. is directly proportional to the vessel diameter
- F d. is directly proportional to viscosity of the blood
- F e. decreases when blood volume is increased

Q. 09. **Blood flow (Velocity) is**
- T a. increased if resistance is decreased.
- T b. inversely proportional to the hematocrit value.
- F c. inversely proportional to the cross sectional area of vessel.
- F d. the greatest in the aorta.
- F e. not a major determinant of blood pressure.

Q. 10. **The velocity of the blood flow is**
- T a. lower in the vein than in the arteries.
- T b. higher in the arteries than in the arterioles.
- F c. higher in the venules than in the arterioles.
- F d. higher in the capillaries than in the arterioles.
- F e. higher in the vein than in the arteries.

Q. 11. **Least change of blood flow during exercise occur in**
- T a. brain.
- F b. intestine.
- F c. heart.
- F d. skin.
- F e. kidney.

Q. 12. **During exercise blood flow**
- T a. remains same in abdominal organs.
- T b. decreases in skin.
- T c. increases in active muscle.

F d. decreases in lungs and heart.
F e. increases in skin.

Q. 12. **Circulatory adjustment occurs during exercise by**
T a. increasing the cardiac output.
T b. increasing the sympathetic activity over heart.
T c. increasing the arterial pressure.
F d. decreasing the total peripheral resistance.
F e. decreasing the myocardial force of contraction

Q. 13. **Positive ionotropic agents**
T a. are mediated by β-adrenergic receptors.
T b. decrease left ventricular end-diastolic pressure.
F c. decrease the ventricular performance.
F d. are mediated by muscarinic receptors.
F e. decrease maximum left ventricular pressure.

Q. 14. **With increasing distance from the heart, arterial**
T a. PO_2 falls.
T b. walls contain more smooth muscle than elastic tissue.
F c. flow has a greater tendency to be turbulent.
F d. walls contain less smooth muscle than elastic tissue.
F e. mean pressure tends to rise slightly.

Q. 15. **Net outward force in the foot will be increased by**
T a. fall in plasma colloid osmotic pressure.
T b. decreased plasma albumin concentration.
T c. increased cpaillary permeability.
F d. increased interstitial fluid pressure.
F e. arteiolar constriction.

Q. 16. **Vasovagal fainting or syncope**
T a. is associated with skeletal muscle vasodilation
T b. is more likely to occur in standing than lying position.
T c. is loss of consciousness.
F d. is associated with tachycardia.
F e. is more likely to occur in a cold than hot environment.

Q. 17. **Increased sympathetic drive to the heart**
T a. increases the rate of conduction in purkinje fiber.
T b. increases the rate of impulse generation.
T c. increases the myocardial metabolis
F d. decreases the ejection fraction of the left ventricle.
F e. decreases the coronary blood flow.

Q. 18. **Sympathetic drive to the heart is increased**
T a. in excitement.
T b. in exercise
T c. in hypotension.
F d. during sleep.
F e. when parasympathetic drive is decreased.

Q. 19. **Vasoconstriction occurs due to**
T a. norepinerphrine secretion.
T b. formation of angiotensin II.
T c. increased K^+
T d. release of thromboxane A_2
F e. release of CO_2

Q. 20. **Peripheral resistance increases due to**
T a. dehydration.
T b. vasoconstriction.
T c. increased viscosity of blood.
F d. increased distance of a blood vessel from the heart.
F e. anemia.

Q. 21. **Resistance to blood flow is**
T a. the lowest in capillary.
T b. inversely proportional to diameter.
T c. directly proportional to haematocrit.
F d. directly proportional to diameter.
F e. determined by arterial pressure.

Q. 22. **Distribution of vascular resistance**
T a. Small arteries & arterioles : 47 %
T b. Capillaries : 27 %
T c. Arteries : 19 %
T d. Veins : 7 %.
F e. None

Q. 23. **Maximum oxygen content in fetus is in**
T a. umbilical vein.
F b. pulmonary vein.
F c. thoracic inferior vena cava.
F d. ductus arteriosus.
F e. descending aorta

Q. 24. **Maximum resistance to blood flow occurs in the**
T a. meta-arterioles.
T b. arterioles.
F c. capillaries.
F d. aorta.
F e. vena cava.

Q. 25. **Local metabolic activity is the chief factor for determination the rate of blood flow to**
T a. skeletal muscle.
T b. heart.
F c. kidney.
F d. skin.
F e. lung.

Q. 26. **Coronary blood flow increases during**
T a. stimulation of parasympathetic nerve.
T b. myocardial hypoxia
T c. adenosine infusion
F d. early systole
F e. stimulation of parasympathetic nerve

Q. 27. **Coronary blood flow is increased by**
T a. β-adrenergic blockadge.

F b. a decrease in systemic blood pressure.
F c. vagal stimulation.
F d. a decrease in arterial PO_2
F e. an increase in arterial PCO_2

Q. 28. **Myocardial blood flow to the left ventricles increases during**
T a. stimulation of sympathetic nerves to the heart.
T b. myocardial hypoxia.
T c. adenosine infusions.
F d. arterial hypertension.
F e. early systole.

Q. 29. **Coronary artery blood flow**
T a. is regulated mainly by myocardial O_2 consumption.
T b. accounts for about 5% of the cardiac output.
F c. does not depend upon arterial blood pressure.
F d. is greatest during systole in the left coronary artery.
F e. is greatest during diastole in the right coronary artery.

Q. 30. **Coronary blood flow is**
T a. regulated by autonomic nervous system.
T b. decreased in the reflex response to a fall in the arterial blood pressure.
T c. more during diastole than systole.
F d. stable throughout the cardiac cycle.
F e. is increased during early systole.

Q. 31. **Coronary blood flow increases during**
T a. adenosine infusion
T b. hypertension
T c. myocardial hypoxia
F d. decreased myocardial activity
F e. ventricular systole.

Q. 32. **Forces tending to move fluid outwards across the capillaries are**
T a. negative interstitial free fluid pressure.
T b. interstitial fluid colloid osmotic pressure.
T c. capillary hydorstatic pressure at arterial end.
F d. plasma colloid osmotic pressure.
F e. capillary hydrostatic pressure at venous end.

Q. 33. **Lymph is formed**
T a. 2 -3 liters/ day.
T b. from interstital fluid.
F c. 0.5 -1 liter/day.
F d. from venous blood.
F e. from capillary fluid.

Q.34. **Effects of excess potassium ion in ECF on heart are**
T a. heart may be flaccid.
T b. A-V conduction block may occur.
T c. heart become extremely dilated.
F d. heart rate will be increased.
F e. heart may be spastic.

Q. 35. **Coronary vasodilators released during ischemia of cardiac muscle are**
T a. hydrogen ion.
T b. adenosine.
T c. potassium ion.
F d. bicarbonate ion.
F e. sodium ion.

Q. 36. **Anginal pain due to coronary artery disease**
T a. may be elicited by sudden exposure to cold.
T b. is typically made worse by treatment which lowers the systemic blood pressure.
T c. may be precipitated by anaemia.
F d. is caused by accumulation of metabolites in the myocardium.
F e. is relieved by sympathomymetic drugs.

Q. 37. **Pulmonary circulation differs from systemic circulation in that it**
T a. consists of vessels of larger diameter
T b. is mixed with systemic circulation in the heart
T c. low resistance circuit
F d. consists of thick walled vessels
F e. is considered as greater circulation.

Q. 38. **CapIllaries with tight junctions allowing the passage of only small molecukes are found in**
T a. Brain
F b. Skin
F c. Kidney
F d. Muscle
F e. None.

Q. 39. **Hypoxia causes vasoconstriction in**
T a. Lungs
F b. Muscle
F c. Liver
F d. Spleen
F e. All.

Q. 40. **Blood flow through a vessel varies directly with**
T a. Pressure difference
F b. Resistance
F c. Viscosity
F d. Length of vessel.
F e. None

Q. 41. **Hypovolemic shock is characterised by the following**
T a. Hypotension
T b. Cold and clammy skin
T c. Intense thirst
T d. Tachycardia
F e. Inhibition of respiration

Q. 42. **Maximum surface area is present in**
- T a. Capillary
- F b. Arterioles
- F c. Artery
- F d. Vein.
- F e. All.

Q. 43. **Most permeable capillaries**
- T c. Kidney
- F a. Post pituitary
- F b. Liver
- F d. Small intestine
- F e. Lung

Q. 44. **Pre capillary sphincter relaxation is caused by**
- T a. Local metabolites
- F b. Circulating catecholamines
- F c. Sympathetic activity
- F d. Fall in capillary pressure
- F e. Viscosity.

Q. 45. **Which one of the following is the correct sequence in increasing order of their basal blood supply (ml/min/100 gm of tissue)?**
- T a. Brain, heart, kidney
- F b. Heart, brain, kidney
- F c. Brain, kidney, heart
- F d. Kidney, heart, brain
- F e. All.

Q. 46. **Blood brain barrier is deficient at**
- T a. Area postrema
- F b. Thalamus
- F c. Meta thalamus
- F d. Cerebellum
- F e. Pons.

Q. 47. **The regional arterial resistance of the mesenrty and kidney vessels is reduced by**
- T a. Dopamine
- F b. Dobutamine
- F c. Nor adrenaline
- F d. Isoprenaline
- F e. Leukotrine.

Q. 48. **Adrenaline increases all of the following in heart except**
- T a. Refractory period
- F b. Automaticity
- F c. Conduction velocity
- F d. Contractility
- F e. All.

Q. 49. **Increased blood flow in muscle during exercise is not because of**
- T a. High lactate
- F b. Low pH
- F c. High CO_2
- F d. Low PO_2
- F b. All.

Q. 50. **Splanchnic circulation in vigrous activity is affected by**
- T a. Decrease capacitance with shunting of blood
- F b. Decrease blood supply due to vasoconstriction
- F c. Decrease blood supply due to venoconstriction
- F d. Increase capillary congestion
- F e. All.

Q. 51. **The followings tend to increase in old aget**
- T a. Residual volume
- T b. Systolic BP
- T c. Pulse pressure
- F d. Vital capacity
- F e. All.

Q. 52. **During exercise, blood flow does not decrease in**
- T a. Coronary circulation
- F b. Cutaneous circulation
- F c. Hepatosplanchnic circulation
- F d. Renal circulation.
- F e. Intestinal circulation.

Q. 53. **Blood brain barrier is maximum permeable to**
- T a. CO_2
- F b. Na^+
- F c. K
- F d. Chloride
- F e. Ca.

Q. 54. **Blood brain barrier is made up of**
- T a. Astrocytes
- F b. Oligodendrocytes
- F c. Oligodendroglia
- F d. Microglia
- F e. Macroglia.

Q. 55. **Coronary blood flow stops during**
- T a. Isovolumetric contraction
- F b. Protodiastole
- F c. End of diastole
- F d. Isometric contraction
- F e. Ejection phase.

Q. 56. **Splanchnic vesels and venules contain what perocen tage of blood volume:**
- T a. 20 - 30%
- F b. 10 - 20%
- F c. 40 - 50%
- F d. 60 - 70%
- F e. 12 - 13%

Q. 57. **All of the following ions cause vasodilatation**
- T a. Kinins
- T b. Mg
- T c. K
- F d. Ca
- F e. None

Q. 58. **Major blood reservoirs of the body include the following**
- T a. Liver sinuses
- T b. Major abdominal veins
- T c. Venous plexus of the skin
- T d. Venous sinuses of the spleen
- F e. Throracic vena cava

Q. 59. **Velocity of blood is times that of urine:**
- T a. 5-6 times more that of water
- F b. Same as of ECF
- F c. 10 times of urine
- F d. 5-6 times less that of water
- F e. 3 times more that of water

Q. 60. **Velocity of blood flow is inversely proportional to:**
- T a. Cross-sectional area
- F b. Viscosity
- F c. Flow
- F d. Length
- F e. None

Q. 61. **Turbulant blood flow is produced by**
- T a. Decreased harmatocrit
- F b. Decreased velocity of circulation
- F c. Decreased cardiac output
- F d. All of the above
- F e. None of the above

Q. 62. **Select the statement which best characterises lymph capillaries**
- T a. Have a discontinuous basement membrane
- F b. Have smaller diameter than blood capillaries
- F c. Less permeable than blood capillaries
- F d. Have no endothelial lining
- F e. All.

Q. 63. **What one structural feature do all blood capillaries have in common?**
- T a. A continuous basement membrane
- F b. Absence of intracellular fenestrations in the endothelial cells
- F c. Presence of intracellular fenestrations
- F d. A discontinuous basement membrane
- F e. Remain patent in the healthy subject

Q. 64. **Venoconstriction is exhibited by**
- T a. Carotid artery occlusion
- T b. Valsalva manoeuvre
- T c. Asphyxia
- T d. Haemorrhage
- F e. Lying down.

Q. 65. **What does prevent blood loss after rupture of small blood vessel?**
- T a. Vasuconstriction
- T b. Formation of fibrin threads
- T c. Formation of platelet plug
- T d. Production of haematoma to increase pervascular pressure
- T e. All.

Q. 66. **Vascular distensibility is least for the following vascular segment**
- T a. Systemic artery
- F b. Pulmonary artery
- F c. Systemic vein
- F d. Pulmonary vein
- F e. All.

Q. 67. **Blood flow through left coronary artery**
- T a. Increases when myocardial hypoxia is present
- F b. Regulated by sympathetic vasedilator nerves
- F c. Creates during early systole
- F d. Decreased in reflex response to fall in blood pressure
- F e. None.

Q. 68. **What best characterises the sinusoids?**
- T a. Are not found in skeletal muscle
- F b. Have smaller diameter than lymph capillaries
- F c. Have a continuous endothelial lining
- F d. Have a continuous besement membrane
- F e. None.

Q. 69. **Sympathetic vasoconstrictor tone is diminished in response to increased activity of**
- T a. Carotid sinus pressor receptors
- F b. Carotid body chemoreceptors
- F c. Pain receptors
- F d. Medullary chemoreoeptors
- F e. None.

Q. 70. **Maximum surface area of circulating system is seen in**
- T a. Capillaries
- F b. Arteries
- F c. Veins
- F d. Arterioles
- F e. Vena cava.

Cardiac muscle

Q. 71. **Cardiac muscles**
- T a. have more myoglobin than that of skeletal muscle

T b. have intercalated disc
T c. are inteitonnected as latticework fashion
F d. have many tight junctions
F e. act as single syncytium.

Q. 72. **Cardiac muscle is characterized by having**
T a. the same graphic relation between initial fiber length total tension as skeletal muscle.
T b. a T system located at the Z lines.
T c. intercalated disks connecting muscle fibers.
F d. same action potential as that of skeletal muscle
F e. the same refractoiy period characterstics of skeletal muscle.

Q. 73. **Intercalated disks are found in**
T a. cardiac muscle
F b. skeletal muscle
F c. nervous tissue
F d. smooth muscle
F e. glandular tissue.

Q. 74. **Properties of cardiac muscle are**
T a. refractory period.
T b. conductivity.
T c. autoexcitability.
F d. adaptation.
F e. summation.

Q. 75. **Impulse generating power of junctional tissues are**
T a. purkinje fibres : 15-40 impulses/min.
T b. SA node : 70-80 impulses/min.
F c. A-V node : 30-36 impulses/min.
F d. bundle of His : 15-40 impulses/min.
F e. inter nodal pathway : 20-40 impulses /min.

Q. 76. **The cardiac impulse is**
T a. transmitted through the S-A node at a velocity of 0.05 m/second
T b. transmitted from S-A node within 0.03 seconds.
F c. generated in atrioventricular (A-V) node
F d. transmitted from right atrium to left atrium through inter nodal pathways
F e. rapidly transmitted through atrioventricular (A-V) bundle.

Q. 77. **Sinoatrial node cells are**
T a. innervated by the vagus
F b. unable to generate impulse when completely denervated.
F c. found in both atria
F d. connected to the AV node by fine bundles of purkinje tissue.
F e. stable with regard to their membrane potentials

Q. 78. **Conduction velocity in the SA node is**
T a. 0.05 meter/second
F b. 0.5 meter/second
F c. 0.4 meter/second
F d. 1 meter/second
F e. 4 meter/second

Q.79. **Sino-atrial node cells are**
T a. innervated by the vagus nerves
F b. connected to the A-V node by fine bundles of purkinje
F c. called reserve pace-maker
F d. found in both atria
F e. unable to generate impulses when heart is completely denervated.

Q.80. **SA nodal fibers**
T a. transmit impulses at a higher velocity.
T b. are inherently leaky to sodium ions.
F c. have threshold voltage of about -55 mV.
F d. need external impulse for their stimulation.
F e. originate impulse at a rate of 100 beats/min normally.

Q. 81. **SA node**
T a. is the normal pace marker of heart.
T b. is innervated by vagus.
T c. discharges most rapidly.
T d. controls the beat of the heart.
F e. has contractile element.

Q. 82. **The sinoatrial node is normally the pacemaker of the heart because**
T a. it initiates the cardiac impulse.
T b. of its highest rate of impulse generation.
F c. it is controlled by autonomic nervous system
F d. it is located in the right atrium.
F e. of its proximity to AV node.

Q. 83. **Pacemaker potential of SA node**
T a. results from decreasing K^+ conductance.
T b. depends upon increasing Ca^{++} conductance.
F c. slows with hyperkalemia.
F d. spreads up in vagus stimulation.
F e. results from decreasing Na^+ conductance.

Q. 84. **Prominent pacemaker is seen in**
T a. sinoatrial node.
F b. bundle of His.
F c. purkinje fibers.
F d. atrial muscle cells.
F e. ventricular muscle cells.

Q. 85. **Which of the following normally has the most promi nent pre potential?**
T a. SA node
F b. purkinje fibers
F c. ventricular muscle cell.

F d. artial muscle cell
F e. bundle of His

Q. 86. **A-V nodal delay occurs as**
T a. very few gap junctions between the fibers.
T b. the fibres are smaller than atrial muscle fibers.
T c. resting membrane potentials are much less negative.
F d. A-V node recieves the impulse originating from S.A node
F e. it acts as a reserve pacemaker.

Q. 87. **AV nodal delay**
T a. is shortened by sympathetic stimulation.
T b. is due to diminished number of gap junctions in conducting pathway.
T c. is about o.16 second.
T d. enhances ventricular filling.
F e. is due to excess calcium.

Q. 88. **The Purkinje tissue cells in the heart**
T a. are responsible for the short duration of QRS complex.
T b. are responsible for the configuration of the QRS complex.
T c. conduct impulses faster than some neurous.
T d. are larger than ventricular myocardial cells.
F e. lead to contraction of the base before the apex of the heart.

Q.89. **Purkinje fibers**
T a. have very few myofibrils
T b. transmit cardiac impulse at the highest rate
F c. are thin
F d. contain few amount of glycoprotein
F e. contain less number of gap junctions.

Q. 90. **Increased sympathetic drive to the heart**
T a. increases the rate of conduction in purkinje fiber.
T b. increases the rate of impulse generation.
T c. increases the myocardial metabolis
F d. decreases the ejection fraction of the left ventricle.
F e. decreases the coronary blood flow.

Q. 91. **The portion of the heart with the fast conduction velocity for action potential is the**
T a. purkinje fibers.
F b. Left atrium
F c. bundle of His
F d. Right atrium
F e. the ventricles

Q. 92. **Cardiac impulse are produced due to the opening of**
T a. L Channel of calcium.
T b. slow calcium-sodium channel.
T c. T Channel of calcium.
T d. K Channel.
F e. fast sodium channel.

Q. 93. **Conduction of the wave of depolarization in the heart is slowest in the**
T a. A-V junctional tissue.
F b. atrium.
F c. venticular endocardium.
F d. ventricle.
F e. purkinje fibers.

Q. 94. **The last part of the heart to be depolarized are**
T a. uppermost portion of the septum.
T b. posterobasal portion of the left ventricle.
T c. pulmonary conus.
F d. posterior part of the right atrium.
F e. interatrial septum.

Q. 95. **Ventricular muscle receives impulses directly from the**
T a. purkinje system.
F b. AV node.
F c. bundle of His.
F d. right and left bundle branches.
F e. SA Node.

Q. 96. **Plateau is present in the action potential of**
T a. cardiac muscle.
F b. skeletal muscle.
F c. smooth muscle.
F d. nervous tissue.
F e. connective tissue.

Q. 97. **Plateau in the cardiac muscle**
T a. is due to slow calcium-sodium channel.
T b. causes muscle contruction to last up to 15 times as long as in skeletal muscle.
F c. is due to fast sodium channel.
F d. is due to efflux of K^+
F e. is due to efflux of Na^+.

Q.98. **Contractility of myocardium can be increased by**
T a. moderate increase of body temperature
T b. infusion of fluid containing calcium
T c. increased stimulation of sympathetic nerves
F d. increased stimulation of baroreeeptors
F e. infusion of potassium containing fluid.

Q. 99. **Repolarization phase of action potential of ventri cular muscle fibers is caused by the opening of**
T a. K^+ Channels.
F b. HCO_3 channels.
F c. Na^+ Channels.
F d. Cl^- Channels.
F e. Ca^{++} channels.

Q. 100. **Cardiac glycosides (digitalis) enhances the contrac tile process of cardiac muscle fibers by**
- T a. inhibiting the Na^+-K^+ATP ase of cell membrane
- F b. stimulating cardiac β-adrenergic receptors.
- F c. blocking cardiac β-adrenergic receptors.
- F d. stimulating intracellular production of cAMP.
- F e. decreasing the amount Ca^{++} available to myofibrils.

Q. 101. **Due to Frank-Starling mechanism of heart**
- T a. both the heart rate and force of contraction is increased
- T b. within physiological limit, heart pumps all the blood that comes to it
- T c. their is no damming of blood in peripheral circulatory system
- F d. heart muscle can not be tetanized
- F e. atria can complete its contraction before the contrac tion of the ventricle

Q. 102. **Myocardial contractility is increased by**
- T a. increasing the interaction force between actin and myosin filaments
- T b. increasing the initial length of cardiac muscle within physiological limit
- T c. local application of acetylcholine
- F d. local application of epinephrine
- F e. decreased sympathetic activity.

Q. 103. **Force of contraction of heart**
- T a. is increasesd in aneima
- F b. is not related to nutrition
- F c. is decreasesd by increased ventricular filling
- F d. is increased with parasympathetic stimulation
- F e. is decreased by athletic training.

Q. 104. **Force of contraction of heart muscle depends upon**
- T a. circulatory hormones.
- T b. sympathetic stimulation.
- T c. Ca^{++} level in ECF.
- T d. initial length of muscle fibers.
- F e. parasympathetic stimulation.

Q. 105. **The strength of contraction of verticular muscle**
- T a. is more when peripheral resistance is increased.
- T b. is more during strenuous exercise.
- T c. increases when end diastolic venticular filling pressure rises.
- F d. increases when serum potassium level rises.
- F e. increases when blood calcium level falls.

Q. 106. **Increased vagal tone leads to**
- T a. decreased AVconduction
- F b. increased heart rate.
- F c. increased refractrory period of atria
- F d. increased ventricular contractility
- F e. increased ectopic beats

Q. 107. **Conduction velocity is least in**
- T a. AV node
- F b. Bundle of HIS
- F c. SA node
- F d. Purkinjee fibres
- F e. All.

Q. 108. **Fastest conducting tissue in human heart**
- T a. Purkinje fibres.
- F b. AV node
- F c. SA node
- F d. Bundle of His
- F e. None.

Q. 109. **Velocity of transmissions is fastest through following part of heart.**
- T a. Bundle of His
- F b. AV node
- F c. Atria
- F d. Ventricle
- F e. Internodal pathway

Q. 110. **Adrenaline increases the following in heart**
- T a. Contractility
- T b. Automaticity
- T c. Conduction velocity
- F d. Refractory period
- F e. All.

Q. 111. **Parasympathetic stimulation would decrease the following**
- T a. Atrial
- T b. SA node rhythmicity contractility
- T c. Heart rate
- F d. A-V conduction time
- F e. All.

Q. 112. **Absolute refractory period in heart**
- T a. Lasts till cardiac contraction
- T b. Longer than refractory period in skeletal muscle
- T c. A phase of cardiac cycle in which heart cannot be stimulated by any amount of stimulus
- T d. Corresponds with duration of action potentiai
- T e. All of the above

Q. 113. **Right and left vagus respectively go to :**
- T a. SA node, AV node
- F b. AV node, SA node
- F c. AV node, bundle of His
- F d. SA node, bundle of His
- F e. All.

Q. 114. **Repolarization of ventricular muscle**
- T a. Begins in apical epicardium
- F b. Occurs last at apex
- F c. Begins in septum

F d. Begins at AV node
F e. Begins at SA node.

Q. 115. **For cardiac muscle, V max can be used as a measure of**
T a. Rhythmicity
F b. Excitability
F c. Contractility
F d. Conductivity
F e. All.

Q. 116. **A decrease in the velocity of impulse conduction through the A-V node will usually cause**
T a. The P0 interveal to increase
F b. The P0 interval to decrease
F c. Disappearance of the T-wave
F d. Increased heart rate
F e. Atrial fibrillation

Q. 117. **Fibres of A-V junction**
T a. Modified muscle fibres
F b. Modified nerve fibres
F c. Highly contractile
F d. Conduct impulse rapidly
F e. All.

Q. 118. **Aortic valve incompetence characteristically produce**
T a. Hypertrophied left ventricle
T b. Increased myocardial blood flow
T c. Increased pulse pressure
T d. All of the above
F e. None.

Q. 119. **Absolute refractory period of heart is the gap of time in which**
T a. No action potential from another part of heart will reexcite the heart muscle
F b. Heart is in diastole
F c. Unresponsive to neural stimuli
F d. None of the above
F e. All.

Q. 120. **When pacemaker is in another area of heart, it denotes**
F a. Abnormal conducting tissue
F b. Abnormal S-A node
F c. Abnormal with neural controlling system
F d. All of the above
T e. None of the above

Q. 121. **In wolff parkinson white syndrome, there exists a connection between atria and**
T a. Ventricles
F b. Bundle of His
F c. AV node
F d. Purkinjee fibres
F e. None of the above

Q. 122. **SA Node**
T a. Initiates the impulse at faster rate
F b. Generate the impulse spontaneously
F c. Increases the parasympathetic activity
F d. Increases sympathetic activity
F e. None of the above

Q. 123. **Vagal stimulation can cause**
T a. Delayed A-V conduction
T b. Increased ventricular contraction
T c. Decreased heart rate
F d. Increased atrial contraction
F e. All.

Q. 124. **Myocardial oxygen demand**
T a. Directly proportional to Heart rate
F b. Inversely proportional to heart rate
F c. Increased by digitalis
F d. Not related to heart rate
F e. All.

Q. 125. **Vagus inhibits pacemaker potential by**
T a. Hyperpolarisation
T b. Increased pre potential
T c. Increase k^+ permeability
F d. Stabilising resting membrane potential
F e. All.

Q. 126. **Conduction velocity is maximum in**
T a. Bundle of His
F b. SA node
F c. AV node
F d. Right ventricle
F e. Atria.

Q. 127. **SA node is located**
T a. Subepicardially
F b. Epicardially
F c. Intramyocardial
F d. Endocardially
F e. None.

Cardiac cycle

Q. 128. **Cardiac cycle**
T a. time is inversely proportional to heart rate.
T b. is composed of systole and diastole.
T c. beings with the atrial systole.
F d. begins with the ventricular systole.
F e. time is directly proportional to heart rate.

Q. 129. **During cardiac cycle**
T a. the contracting ventricles shorten from apex to base

T b. the atrioventricular (A-V) valves close at the beginning of systole
F c. systole begins in the left atrium
F d. first heart sound is heard in protodiastole
F e. atrial and ventricular systole occur simultaneously.

Q. 130. **During cardiac cycle**
T a. the cyclical changes are same in each beat of the heart.
T b. contraction begins normally in the right atrium.
F c. impulses spread directly from atria to ventricle.
F d. sometimes 4th heart sound can be auscultated.
F e. murmurs are produced normally.

Q. 131. **Cardiac cycle time is**
T a. inversely proportional to the heart rate.
T b. affected mostly in diastole.
F c. affected mostly in systole.
F d. eight seconds.
F e. directly proportional to the heart rate.

Q. 132. **During isometric contraction phase of ventricle**
T a. both the atrio-ventricular and semilunar valves are closed.
T b. there is production of QRS complex.
T c. intraventricular pressure sharply rises.
F d. there is production of 3rd heart sound.
F e. the pressure in the aorta is rising.

Q. 133. **Regarding isovolumic (isometric) contraction period of ventricular systole**
T a. it continues until the ventricular pressure overcome the pressure in the aorta and the pulmonary artery.
T b. ventricles contract as a closed cavity.
T c. ventricular contraction occurs but there is no emptying of blood.
F d. shortening of the length of the ventricular muscles occurs.
F e. volume of the ventricular contents decrease

Q. 134. **In isovolumetric ventricular contraction phase of the cardiac cycle**
T a. atrial pressure decreases.
T b. ventricular pressure increases rapidly.
F c. there is opening of A-V valves.
F d. there is an increased atrial pressure.
F e. there is rapid rise of aortic pressure.

Q. 135. **During isometric ventricular contraction period**
T a. no change of ventricular blood volume occurs.
T b. both the atrioventricular and semilunar valves are closed.
T c. there is sharp rise of ventricular pressure
F d. the atrioventricular valves are open and semilunar valves are closed.
F e. there is sharp rise of atrial pressure

Q. 136. **In isometric contraction period**
T a. the duration is 0.05 second.
T b. ventricles are in systole.
T c. ventricles are contracting as closed cavities.
F d. there is a shortening of cardiac muscle.
F e. semilunar valves remain open.

Q. 137. **During rapid ejection phase of ventricular systole**
T a. aortic pressure is rising.
T b. mitral valve is closed.
F c. ventricle will be expelled to its minimal volume.
F d. aortic valve is closed.
F e. first heart sound is produced.

Q. 138. **During protodiastolic period there is**
T a. porduction of 2nd heart sound.
F b. closure of semilunar valve.
F c. opening of A-V valve.
F d. isometric relaxation of ventricle.
F e. isotonic contraction of ventricle.

Q. 139. **During ventricular diastole, ventricular filling**
T a. gives rise to third heart sound in children
T b. gives a concept of preload.
T c. is most rapid in the first half of diastole.
F d. is occured about 80% by atrial contraction.
F e. begins during isometric relaxation of ventricle.

Q. 140. **Ventricular filling**
T a. (25%) occurs due to atrial contraction.
T b. causes 2nd and 3rd heart sounds.
T c. occurs due to rapid rush of blood from atria
F d. occurs due to blood coming from inferior vena cava.
F e. causes production of 1st heart sounds.

Q. 141. **Ventricular filling**
T a. depends on filling time.
T b. depends on atrial contraction.
T c. begins during isovolumetric relaxation period
F d. begins during isovolumetric contraction period
F e. can occur only when artial pressure is greater than atmospheric pressure.

Q. 142. **Immediately after closure of semilunar valves, there is**
T a. isotonic relaxation
T b. isometric relaxation phase
F c. isotonic contraction
F d. isometric contraction phase
F e. production of 1st heart sound.

Q. 143. **In isometeric relaxation period**
T a. both the valves remain closed.

T b. ventricles are relaxing as closed cavities.
F c. the blood enters into the ventricle from atria
F d. intraventricular pressure rises.
F e. duration is 0.05 second.

Q. 144. **Semilunar valves open**
T a. when ventricular pressure rises above aortic pressure.
T b. when ventricular pressure rises above 80 mmHg.
F c. when ventricular pressure rises above 120 mmHg.
F d. when atrial pressure rises above ventricular pressure.
F e. when atrial pressure rises above ventricular pressure.

Q. 145. **Cardiac cycle duration in man is**
T a. 0.8 seconds
F b. 0.4 seconds
F c. 1.2 seconds
F d. 1.6 seconds
F e. 8.0 seconds

Q. 146. **What is true for extrasystole in ventricle?**
T a. Fails to produce radial pulse
T c. Associated with abnormal QRS complex
T d. Tendency to be followed by a compensatory pause
F b. Hints at serious heart ailment
F e. All.

Q. 147. **Ventricular filling**
T a. Produces 3rd heart sound in some healthy persons
F b. Depends mainly on contraction of atria
F c. Begins during isometric ventricular relaxation
F d. Will not occur unless atrial pressure is higher than atmospheric pressure
F e. All.

Q. 148. **Which of the following is true about ventricular filling**
T a. Filling pressure is important for cardiac output
F b. Atrial contraction mainly contributes
F c. Maximum during isometric ventricular relaxation
F d. Lnotropic state of myocardium limits the cardiac output.
F e. None.

Q. 149. **The cause of post extrasystolic potentiation is**
T a. Ca^{++}
F b. K^{+}
F c. Na^{+}
F d. Mg^{++}
F e. Cl^{-}.

Cardiac Output

Q. 150. **The cardiac output**
T a. is increased when storke volume is increased
T b. from the right and left ventircle are the same
T c. raises in hot environment.
F d. is not increased when heart rate is increased
F e. usualy rises when a person stands up.

Q. 151. **Cardiac output**
T a. increases when stroke volume rises.
T b. is greater than cardiac index.
T c. usually rises when a person lies down.
F d. does not rise after exercise.
F e. does not depend on venous return.

Q. 152. **Factors regulating cardiac out put are**
T a. heart rate.
T b. peripheral resistance.
T c. venous return.
F d. exercise.
F e. posture.

Q. 153. **Factors affecting cardiac output are**
T a. end diastolic volume (EDV)
T b. heart rate.
T c. storke volume.
F d. total peripheral resistant (TPR)
F e. end systolic volume (ESV)

Q. 154. **Cardiac output is increased by**
T a. increasing heart rate in response to exercise
T b. increasing ventricular end-diastolic volume
T c. increased β-sympathetic nervous system activity.
F d. the administration of digoxin to normal subjects at rest.
F e. increasing ventricular after-load.

Q. 155. **Cardiac output is not affected by**
T a. sleep
F b. pregnancy
F c. high environmental temperature.
F d. exercise
F e. anxiety

Q. 156. **Fick's principle is used for measuring**
T a. Cardiac output
F b. GFR.
F c. Blood pressure
F d. Pulse pressure
F e. Lung volumes

Q. 157. **Cardiac output in liter per minute divided by heart rate is the index**
T a. mean stroke volume.
F b. mean arterial pressure.
F c. cardiac index.
F d. ejection fraction.
F e. cardiac efficiency.

Q. 158. **Organ to have the maximum oxygen consumption after liver is**
- T a. skeletal muscle
- F b. heart
- F c. kidney
- F d. brain
- F e. skin.

Q.159. **Cardiac output increases**
- T a. in lying posture
- F b. with increased heart rate of about 200 beats/minute
- F c. during expiration
- F d. with stimulation of baroreceptors
- F e. with increased systolic pressure

Q. 160. **Cardiac output**
- T a. usually rises when a person lies down.
- T b. rises in hot environment.
- T c. is normally expressed as the output of one ventricle in liters/minutes.
- T d. may not increase when heart rate rises.
- F e. does not increase in exercise following denervation of the heart.

Q. 161. **Ejection fraction of the heart**
- T a. is the portion of end diastolic volume that is ejected by heart.
- T b. is the guideline for conraction of heart.
- F c. is reduced in exercise.
- F d. is normally 10%.
- F e. is increased during sleep.

Q. 162. **Venous return depends upon**
- T a. mean systemic filling pressure.
- T b. pressure defference between capillaries and venules.
- T c. muscular exercise.
- T d. respiration.
- F e. resistance to blood flow.

Q. 163. **Factors affecting venous return are**
- T a. pumping action of heart.
- T b. gravity.
- T c. muscle activity.
- F d. arterial blood pressure.
- F e. heart rate.

Q. 164. **Within physiological limit, increased venous return**
- T a. increases force of contraction of heart.
- T b. increases cardiac output.
- F c. decreases systolic blood pressure.
- F d. decreases intrathoracie pressure.
- F e. decreases heart rate.

Q. 165. **Venous return is enhanced during exercise by**
- T a. decreasing cardiac output.
- T b. venoconstriction.
- T c. skeletal muscle pump.
- T d. increased depth of inspiration.
- F e. decreased blood volume.

Q. 166. **End diastolic volume depends on**
- T a. filling time.
- T b. filling pressure.
- T c. cardiac distensibility.
- T d. artial contraction.
- F e. myocardial contraction.

Q. 167. **If the end diastolic volume increases**
- T a. starling's law is applicable
- T b. stroke volume is increased.
- F c. cardiac output is decreased.
- F d. the force of contraction of heart decreases.
- F e. heart rate is decreased.

Q. 168. **A decrease in storke volume results from**
- T a. decrease in ventricular compliance.
- T b. decrease in ventricular contractility.
- F c. decrease in ventricular afterload.
- F d. decrease in heart rate.
- F e. increase in ventricular preload.

Q. 169. **In the denervated heart, left venticular storke work increases when**
- T a. right ventricular output increases.
- T b. the veins constrict.
- T c. the end diastolic length of the ventircular fibers increases.
- T d. peripheral resistance rises.
- F e. blood volume falls.

Q. 170. **During exercise**
- T a. heart rate is increased
- T b. sympathetic discharge increases
- T c. venous return increases
- F d. O_2 uptake by the exercising muscle decreases
- F e. total peripheral resistance increases

Q. 171. **Which of the following organ extracts the greatest amount of O_2 from the blood that it receives?**
- T a. lung.
- T b. heart.
- F c. skin.
- F d. kidney.
- F e. brain.

Q. 172. **When vasoconstrictor discharge is increased there is**
- T a. increased cardiac output
- T b. decreased blood stores in the venous reservoirs.
- T c. increased arteriotar constriction
- T d. increased blood pressure
- F e. decreased heart rate

Q. 173. **Preload of the heart is determined by**
- T a. End diastolic volume
- F b. Ejection systolic volume
- F c. End systolic volume
- F d. Systolic vascular resistance
- F e. Peripheral resistance.

Q. 174. **Normal hepatic blood flow per minute is**
- T a. 50 cc/100 gm of liver tissue
- F b. 100 cc/100 gm of liver tissue
- F c. 200 cc/100 gm of liver tissue
- F d. 300 cc/100 gm of liver tissue
- F e. 150 cc/100 gm of liver tissue

Q. 175. **Ejection fraction increases with**
- T a. End. diastolic volume
- F b. End systolic volume
- F c. Peripheral vascular resistance
- F d. Venodilation
- F e. None.

Q. 176. **Ejection fraction of the ventricle refers to the ratio of**
- T a. Stroke volume / end diastolic volume
- F b. Amount of blood received / amount of blood ejected
- F c. End-systolic volume / end diastolic volume
- F d. Stroke volume / end systolic volume.
- F e. None.

Q. 177. **Direct Fick method of measuring cardiac output requires estimation of :**
- T a. O_2 content of arterial blood
- T b. O_2 consumption per unit time
- T c. Arteriovenous O_2 difference
- T d. O_2 content of blood from right ventricle
- T e. All.

Q. 178. **The following factors increase cardiac output :**
- T a. Pregnancy in late stages
- T b. Histamine
- T c. Eating
- T d. Medulla is removed
- F e. Sitting from lying position

Q. 179. **Coronary blood flow stops during**
- T a. Isovolumetric contraction
- F b. Protodiastole
- F c. End of diastole
- F d. Isometric contraction
- F. e. None.

Q. 180. **Cardiac index in a normal person is :**
- T a. 3.2
- F b. 2.1
- F c. 4.6
- F d. 5.9
- F e. 2.3

Q. 181. **Blood flow in ml/100 gm/min is maximum in :**
- T a. Kidneys
- F b. Liver
- F c. Heart
- F d. Skin
- F e. All.

Q. 182. **The average coronary blood flow in human being at rest is % of cardiac output :**
- T a. 4 - 5%
- F b. 5 - 10%
- F c. 10 - 15%
- F d. 15 - 20%
- F e. 2 - 4%

Q. 183. **The organ with maximum O_2 consumtion per minute**
- T a. Liver
- F b. Brain
- F c. Skeletal muscle
- F d. Heart
- F e. Skin.

Q. 184. **Liver has the maximum O_2 consumption (51 ml/min), the next organ to have the maximum O_2 (ml/min) is :**
- T a. Skeletal muscle
- F b. Heart
- F c. Brain
- F d. Kidney
- F e. Skin.

Q. 185. **The renal blood flow (in ml/minute) is :**
- T a. 1260
- F b. 250
- F c. 800
- F d. 1500
- T e. 1100

Q. 186. **Under resting condtilions, the cardiac output is about ..... L/minute**
- T a. 5.25
- F b. 2
- F c. 4
- F d. 7.5
- F e. 4.5

Q. 187. **Blood supply of liver in ml per minute is**
- T c. 1500
- F a. 800
- F b. 1200
- F d. 1800
- F e. 900

Q. 188. **Work performed by the left ventricle is geater than that performed by the right ventricle due to differences in**
- T a. Arterial pressures
- F b. Blood velocity
- F c. Stroke volume
- F d. Atrial pressures
- F e. None.

Q. 189. **What increases the stroke work done by left ventricle?**
- T a. Systemic hypertension
- T b. Increased sympathetic tone in heart
- T c. Aortic stenosis
- T d. Aortic insufficiency
- T e. All of the above

Q. 190. **Linear velocity of blood at normal cardiac output in the aorta is**
- T a. 32 cms /second
- F b. 64 cms /second
- F c. 8 cms /second
- F d. 50 cms /second
- F e. 18 cms /second.

Q. 200. **Cardiac output in L/min divided by heart rate equals**
- T c. Mean stroke volume
- F a. Cardiac efficiency
- F b. Cardiac index
- F d. Mean arterial pressure
- F e. None.

Heart Rate

Q. 201. **Heart rate**
- T a. is usually increased in hyperthyroidism.
- T b. rises after a meal.
- T c. is usually low amoung the athlets during resting conditions.
- F d. decreases in hypotension.
- F e. decreases after infusion of adrenaline.

Q. 202. **The variation of heart rate is mainly brought about by**
- T a. vagal tone.
- F b. venous return.
- F c. sympathetic stimulation.
- F d. CO_2 excess.
- F e. O_2 lack.

Q. 203. **Heart rate is accelerated by**
- T a. hypoxia
- T b. fever
- T c. inspiration
- F d. fear.
- F e. expiration

Q. 204. **The dicrotic notch on the aortic pressure curve is caused by**
- T a. closure of the aortic valve
- F b. closure of the pulmonary valve
- F c. rapid filling of the left ventricle.
- F d. closure of the mitral valve
- F e. closure of the tricuspid valve

Q. 205. **During exercise**
- T a. heart rate is increased
- T b. sympathetic discharge increases
- T c. venous return increases
- F d. O_2 uptake by the exercising muscle decreases
- F e. total peripheral resistance increases

Q. 206. **Blood pressure increases and heart rate decreases in response to**
- T a. increased intracranial pressure.
- F b. decreased intracranial pressure
- F c. exercise.
- F d. increased body temperature.
- F e. exposure to high altitude.

Q. 207. **Murmurs (or bruits) may be detected by auscultation**
- T a. over dilatations in arteries
- T b. over constrictions in arteries
- T c. in young adult in severe anaemia.
- T d. over vessels in which there is turbulence
- F e. over large arteries in healthy adults

Q. 208. **Maximum heart rate with exercise**
- T a. 200
- F b. 120
- F c. 140
- F d. 160
- F e. 72

Q. 209. **Occlusion of common carotid artery on both sides leads to**
- T a. Increase in HR & BP
- F b. Increase in BP & decrease in HR
- F c. Decrease in HR & BP
- F d. No effect on BP & HR
- F e. None.

Q. 210. **In athletes bradycardia is because of**
- T a. Increased vagal tone
- F b. Decreased sympathetic tone
- F c. Cardiac output
- F d. Low venous return
- F e. None.

Q. 211. **In an unacclatimised person suddenly exposed to**

cold atmosphere, changes seen are

T a. Tachycardia
F b. Increase in systolic BP
F c. Shift of blood from shell to core
F d. Non-thermogenic shivering
F e. None.

Pulse

Q. 212. **Following points are to be considered during examination of a pulse**

T a. condition of vessel wall.
T b. rate
T c. volume
F d. peripheral resistance
F e. rhythm

Q. 213. **Radial pulse**

T a. if normal then is called catachrotic pulse.
T b. is normally 60 -90 beats/minute.
T c. coincides with the apex beat.
F d. if normal then is called anacrotic pulse.
F e. if normal then is called dicrotic pulse.

Q. 214. **What is not true of 'a' wave of venous pulsations in neck?**

T a. Occurs just after in carotid artery
F b. Exaggeration in tricuspid stenosis
F c. Abolished in atrial fibrillation
F d. Exaggerated in complete heart block when P wave falls between QRS and T waves
F e. All.

Q. 215. **What is not true for jugular venous pulse?**

T a. Commonly not visible with normal heart
F b. Pressure typically raised in right ventricular failure
F c. Pressure typically raised in partial obstruction of SVC
F d. Pulsations exaggerated in tricuspid incompetence
F e. None.

Heart sound

Q. 216. **The first heart sound is**

T a. produced at the beginning of ventricular systole.
T b. caused by closure of atrioventricular valves.
T c. caused by closure of mitral and tricuspid valves.
F d. caused by closure of aortic and pulmonary valves.
F e. produced at the beginning of ventricular diastole.

Q. 217. **Characteristics of 2nd heart sound are**

T a. it is heard when ventricles are relaxing.
T b. it has higher frequecy.
F c. it is mainly related to turbulence set up by closure of semilunar valves.
F d. its duration is longer than the 1st heart sound.
F e. it is best heard in mitral area.

Q. 218. **The second heart sound differs from the first heart sound in that it is**

T a. heard when the ventricles are relaxing
T b. higher in frequency
F c. best heard at mitral area
F d. longer lasting than the first heart sound
F e. louder, when listened by stethoscope.

Q. 219. **The second heart sound**

T a. corrosponds to the QRS complex of the ECG.
T b. is produced due to closure of the aortic valve.
F c. is longer lasting than the first heart sound.
F d. is produced due to closure of the mitral valve.
F e. corrosponds to the P wave of the ECG.

Q. 220. **Splitting of the second heart sound into two compo nent is enhanced by**

T a. delayed closure of the pulmonary valve.
F b. early closure of the pulmonary valve
F c. prolongation of atrial systole
F d. delayed closure of the aortic valve
F e. delayed closure of the mitral valve

Q. 221. **The 4th heart sound is caused by**

T a. last rapid filling phase of the ventricle
T b. first rapid filling phase of ventricle.
F c. closure of aortic valve
F d. closure of mitral valve
F e. rapid ventricular filling

Q. 222. ***Heart sound heard immediately before the first heart sound when atrial pressure is high**

T a. Fourth
F b. First
F c. Second
F d. Third
F e. None.

Q. 223. **First heart sound occurs during the period of**

T a. Isovolumetric contraction
F b. Isotonic relaxation
F c. Isovolumetric relaxation
F d. Isotonic contraction
F e. None.

Q. 224. **The second heart sound differs from firstheart sound in that**

T a. Has higher frequency
F b. Is ocassionally split
F c. Duration greater than first sound
F d. Due partly to turbulence set up by valve closure.
F e. None.

Q. 225. **First component of first heart sound is usually clearly heard on ventral surface of chest at :**
- T a. 5th intercostal space to left of sternum
- F b. 2nd intercostal space to right of sternum
- F c. 2nd intercostal space to left of sternmum
- F d. 5th intercostal space over sternum
- F e. 4th intercostal space over sternum.

Q. 226. **Dicrotic notch in aortic pressure curve is :**
- T a. Coincident with 2nd heart sound
- F b. Magnified by aortic regurgitation
- F c. Absent in arteriosclerosis
- F d. Of no diagnostic value
- T e. Coincident with 1st heart sound.

ECG

Q. 227. **In the electorcardiogram the**
- T a. T wave is due to ventricular depolarization.
- T b. QRS complex follows the onset of ventricular contraction.
- T c. P wave is due to atrial depolarization.
- F d. T wave is due to ventricular repolarization.
- F e. P wave is due to ventricular depolarization.

Q. 228. **P wave**
- T a. duration is 0.l second.
- T b. is absent in SA nodal heart block.
- T c. is due to atrial depolarization.
- F d. duration is 0.01 second.
- F e. is due to ventricular depolarization.

Q. 229. **P-R interval**
- T a. indicates the atrial deoplarization and conduction A-V node.
- T b. becomes short in paroxysmal supraventricular tachycardia
- T c. is 12 to 20 second.
- T d. becomes prolonged in heart block.
- F e. is 0.6 second.

Q. 230. **QRS complex is**
- T a. due to venticular depolarization.
- T b. prolonged and deformed in bundle branch block.
- T c. 0.08 to 0.10 second long in duration.
- F d. due to atrial repolarization.
- F e. due to atrial depolarization.

Q. 231. **QRS complex indicates**
- T a. ventricular depolarization.
- F b. AV node conduction time.
- F c. atrial repolarization.
- F d. venticular depolarization.
- F e. atrial depolarization.

Q. 232. **The T wave of the ECG is releated to**
- T a. ventricular repolariztion.
- F b. buhdle of His conduction.
- F c. ventricular depolarization.
- F d. atrial repolarization.
- F e. atrial depolarization.

Q. 233. **ST segment indicates**
- T a. ventricular repolarization
- F b. ventricular depolarization and ventricular repolariza tion.
- F c. atrial repolarization
- F d. ventricular repolarization
- F e. atrial depolarization

Q. 234. **P-R interval in the ECG corresponds to**
- T a. Time interval between onset of atrial contraction and onset of ventricular contraction.
- F b. Time delay in the AV Node
- F c. S-A nodal conduction time
- F d. Ventricular depolarisation
- F e. None.

Q. 235. **Electrocardiographic leads designated V_1, V_2 etc refer to**
- T a. Unipolar chest leads
- F b. Unipolar limb leads
- F c. Bipolar limb leads
- F d. Bipolar chest leads
- F e. All.

Q. 236. **A person with eyes closed and mind wandering will have the following wave in ECG**
- T a. Alpha waves
- F b. Beta waves
- F c. Delta waves
- F d. Theta waves
- F e. None.

Q. 237. **Normal QRS interval is**
- T a. 0.08 - 0.1 second
- F b. 0.12 - 0.20 second
- F c. 0.24 - 0.32 second
- F d. 0.05 - 0.08 second
- F e. 0.5 - 0.8 second

Q. 238. **Changes in mean electrical axis of the ventricles may be caused by**
- T a. Muscular necrosis
- T b. Bundle branch block
- T c. Change in body position
- T d. Hypertrophy ot one ventricle
- T e. All.

Blood Presure

Q. 239. **Increased sympathetic drive to the heart**

T a. increases the rate of conduction in purkinje fiber.
T b. increases the rate of impulse generation.
T c. increases the myocardial metabolis
F d. decreases the ejection fraction of the left ventricle.
F e. decreases the coronary blood flow.

Q. 240. **Sympathetic drive to the heart is increased**
T a. in excitement.
T b. in hypotension
T c. in exercise.
F d. during a vasovagal attack.
F e. when parasympathetic drive is increased.

Q. 241. **Sympathetic drive to the heart is increased during**
T a. excitement
T b. exercise
F c. vasovagal attack.
F d. grief
F e. hypertension

Q. 242. **Sympathetic stimulation causes**
T a. increased venous capacitance
F b. increased total peripheral resistance
F c. increased blood pressure
F d. increased heart rate
F e. increased cardiac output.

Q. 243. **Sympathetic drive to the heart is increased**
T a. in excitement.
T b. in exercise
T c. in hypotension.
F d. during sleep.
F e. when parasympathetic drive is decreased.

Q. 244. **Vasoconstriction occurs due to**
T a. norepinerphrine secretion.
T b. formation of angiotensin II.
T c. increased K^+
T d. release of thromboxane A_2
F e. release of CO_2

Q. 245. **Vascular resistance is increased**
T a. when vessel is increased in length.
T b. when visosity is increased.
F c. when vascular radius is halved.
F d. when wall thickness of vessel is increased.
F e. from arteriolar to the capillary bed.

Q. 246. **Arterioles offer more resistance to blood flow due to**
T a. a greater pressure drop along their length.
F b. smaller internal diameters.
F c. a smaller total cross-sectional area.
F d. thick fibrous wall.
F e. richer parasympathertic innervation

Q. 247. **Peripheral resistance increases due to**
T a. dehydration.
T b. vasoconstriction.
T c. increased viscosity of blood.
F d. increased distance of a blood vessel from the heart.
F e. anemia.

Q. 248. **Resistance to blood flow is**
T a. the lowest in capillary.
T b. inversely proportional to diameter.
T c. directly proportional to haematocrit.
F d. directly proportional to diameter.
F e. determined by arterial pressure.

Q. 249. **Arteriolar constriction may result from**
T a. locally released scrotonin.
T b. decreased local temperature.
T c. Increased adrenargic discharge.
F d. the axon reflex.
F e. decreased adrenergic discharge.

Q. 250. **When the radius of the resistance vessels is increased, which of the following is increased?**
T a. capillary blood flow.
F b. viscosity of the blood.
F c. hematocrit.
F d. systolic blood pressure.
F e. diastolic blood pressure.

Q. 251. **When the radius of the resistance vessel is increased**
T a. the diastolic blood pressure will be decreased.
T b. the systolic blood pressure will be increased.
F c. both the systolic and diastolic blood pressures will be increased.
F d. the systolic blood pressure will be decreased.
F e. both the systolic and diastolic blood pressures will be decreased.

Q. 252. **Right atrial pressure**
T a. increases after massive transfusion.
T b. is equal to the atmospheric pressure.
T c. may be as low as 3 to 5 mm Hg after severe hemorr hage.
F d. decreases in heart failure.
F e. increases when the heart pumps strongly.

Q. 253. **Pulse pressure**
T a. is the difference between systolic and diastolic blood pressure.
T b. increases during moderate exercise.
F c. is the differece between mean pressure and diastolic blood pressure.
F d. decreases during severe exercise.
F e. increases during mild exercise.

Q. 254. **Pulse pressure increases**

T a. in patent ductus arteriosus.
T b. when there is greater stroke volume.
T c. during old age.
F d. in hypothyroidism.
F e. due to increased compliance of the arterial tree.

Q. 255. **Mean arterial pressure is**
T a. directly proportional to the total peripheral resistance.
T b. diastolic pressure plus one third of pulse pressure
F c. measured by sphygmomanometer.
F d. arithmetical mean of systolic and diastolic pressure.
F e. diastolic pressure plus two third of pulse pressure

Q. 257. **Diastolic pressure**
T a. is the minimum pressure during ventricular diastole.
T b. is more in standing position.
T c. is 60-90 mm of Hg.
F d. depends on cardiac output.
F e. is more in lying condition.

Q. 258. **In adult in standing position, the mean arterial pressure**
T a. at the heart level is approximately 100 mm Hg.
T b. in a large artery in the head is less than 70 mmHg.
F c. in a large artery in the foot is nearly 60 mm Hg.
F d. does not differ by changing posture from lying to standing position.
F e. is unaffected by changes in emotional state.

Q. 259. **Within physiological limit, an increase in right atrial pressure**
T a. increases cardiac output.
F b. increases heart rate.
F c. decreases renal blood flow.
F d. decreases systemic arterial pressure.
F e. decreases intrathoracic pressure.

Q. 260. **The local blood vessels dilate due to increased concentration of**
T a. sodium ions.
T b. potassium ions.
T c. magnesium ions.
F d. calcium ion.
F e. endothelin.

Q. 261. **Blood pressure increases and heart rate decreases in response to**
T a. increased intracranial pressure.
F b. decreased intracranial pressure
F c. exercise.
F d. increased body temperature.
F e. exposure to high altitude.

Q. 262. **Pressure on carotid sinus leads to**
T a. reflex bradycardia
F b. Brain bridge reflex.
F c. tachycardia
F d. increased cardiac output
F e. increased blood pressure

Q. 263. **Increased pressure within the carotid sinus causes**
T a. reflex bradycardia.
T b. vasodilation.
T c. venodilation.
F d. increased heart rate.
F e. a fall in venous pressure.

Q. 264. **Minute by minute variation of arterial pressure is controlled**
T a. baroreceptor feedback control mechanism.
F b. capillary fluid shift mechanism.
F c. chemorecptor mechenism.
F d. central nervous system ischemic response (CNS)
F e. renin-angiotensin vasconstrictor mechanism

Q. 265. **Baroreceptor reflex mechanism is**
T a. effective in negative chronotropic and inotropic effect on heart
T b. silent until the blood pressure is 60 mm Hg
T c. the rapidly controlled blood plasma regulatory mechanism.
F d. maximally stimulated when blood pressure is 120 mm Hg.
F e. the last ditch of regulation of blood pressure.

Q. 266. **The baroreceptor reflex**
T a. operates in the normal range of artertial blood pressure.
T b. has receptors in the carotid sinus and aortic arch.
F c. results in tachycardia when a person stands up.
F d. involves sympathetic vasodilator cholinergic fibers.
F e. has receptor located in the carotid bodies and aortic bodies.

Q. 267. **Angiotensin II exerts physiological effect on the**
T a. afferent and efferent arteriole of glomerulus
T b. adrenal gland
T c. blood vessels
F d. bone marrow
F e. spleen.

Q. 268. **The stretch receptors related to arterial pressure control are**
T a. baroreceptors.
T b. receptors in the wall of the atria.
T c. receptors in the wall of the pulmonary artery.
F d. chemoreceptors
F e. osmoreceptors.

Q. 268. **The important long term regulators of arterial pressure**

T a. is renin-angiotensin aldosteron mechanism
T b. are chemoreceptor.
T c. is blood volume
F d. is renin-angiotensin mechanism
F e. are baroreceptors

Q. 269. **During moderate exercise**
T a. end-diastolic volume increases.
T b. diastolic blood pressure remains unchanged.
F c. venous return decreases.
F d. both systolic and diastolic pressures increase.
F e. systolic blood pressure decreases.

Q. 270. **Circulatory adjustment occurs during exercise by**
T a. increasing the cardiac output.
T b. increasing the sympathetic activity over heart.
T c. increasing the arterial pressure.
F d. decreasing the total peripheral resistance.
F e. decreasing the myocardial force of contraction

Q. 271. **Pulmonary wedge pressure corresponds to**
T a. left atrial pressure
F b. right atrial pressure
F c. palmonary venous pressure.
F d. right ventricular pressure
F e. left ventricular pressure

Q. 272. **Vasomotor center is**
T a. in the medulla.
T b. inhibited by impulses from baroreceptors
F c. stimulated by Frank starling machanism
F d. inthepons.
F e. inhibited by impulses from chemoreceptor.

Q. 273. **Autoregulation is not present in**
T a. skin
F b. kidney
F c. brain
F d. liver
F e. myocardium.

Q. 274. **When vasoconstrictor discharge is increased there is**
T a. increased cardiac output
T b. decreased blood stores in the venous reservoirs.
T c. increased arteriotar constriction
T d. increased blood pressure
F e. decreased heart rate

Q. 275. **The basis for Koratkows sounds is due to:**
T a. Arterial turbulence
F b. AV valve closure
F c. Aortic valve closure
F d. Arterial expansion
F e. None.

Q. 276. **Maximum peripheral resistance is seen in**
T a. Arterioles.
F b. Capillaries
F c. Veins
F d. Aorta
F e. Venules

Q. 277. **Major part of total peripheral resistance is due to:**
T a. Arterioles
F b. Medium and small arteries
F c. Venules
F d. Capillaries
F e. Venules

Q. 279. **Doubling the vessel diameters would change the resistance to:**
T a. 1 PRU
F b. 2 PRU
F c. 8 PRU
F d. 6 PRU
F e. 4 PRU

Q. 280. **The arterial pulse pressure in the femoral artery is normally**
T a. Greater than the pulse pressure in the upper aorta
F b. Less than the pulse pressure in the upper aorta
F c. Less than 20 mm Hg
F d. Equal to the pulse pressure in the upper aorta
F e. None of the above

Q. 281. **Peripheral chemoreceptors is stimulated maximally by**
T a. Cyanide
F b. Anaemia
F c. Hypocapnia
F d. Alkalosis
F e. None.

Q. 282. **Carotid body baroreceptor is most sensitive to**
T a. Systolic blood pressure
F b. Mean blood pressure
F c. Diastolic blood pressure
F d. Pulse pressure
F e. None.

Q. 283. **Angiotensinogen is produced by**
T a. Kidney
F b. Liver
F c. Atrium
F d. Hypothalamus
F e. Lungs.

Q. 284. **Occlusion of common carotid artery on both sides leads to**
T a. Increase in HR & BP
F b. Increase in BP & decrease in HR

F c. Decrease in HR & BP
F d. No effect on BP & HR
F e. All.

Q. 285. **Carotid sinus stimulation would result in**
T a. Decreased sympathetic discharge to heart
F b. Decreased vagal activity
F c. Increased heart rate
F d. Increased vasomotor tone
F e. None.

Q. 286. **Carotid sinus afferents pass through**
T a. X nerve
F b. IX nerve
F c. V nerve
F d. VII nerve
F e. VIII nerve

Q. 287. **Baroreceptors mainly act through the**
T a. Parasympathetic system
F b. Sympathetic system
F c. Cerebral cortex
F d. Blood volume
F e. Pons.

Q. 288. **In brain ischaemia, systemic blood pressure rises, this is called:**
T a. Cushing reflex
F b. Monro-Kellie doctrine
F c. Autoregulation reaction
F d. White reaction
F e. None of the above

Q. 289. **Essential hypertension is generally associated with an early increase in**
T a. Cardiac work
F b. Oxygen use
F c. Coronary flow
F d. Cardiac output
F e. All.

Q. 290. **Pulse pressure in a particular vessel is determined chiefly by**
T a. Distensibility
F b. Distance from heart
F c. Frictional characteristics lumen
F d. Cross sectional area
F e. None.

Q. 291. **During blood pressure measurement if the muffled sound does not disappear with return of mercury to 0, the conclusion is**
T a. Low haematocrit
F b. Aortic stenosis
F c. Zero diastolic pressure
F d. Patent ductus arteriosus
F e. None.

Q. 292. **Upto what systolic pressure is the brain capable of autoregulation**
T a. 65 mmof Hg
F b. 55 mmof Hg
F c. 45 mmof Hg
F d. 75 mmof Hg
F e. 85 mmof Hg

Q. 293. **Renin secretion is stimulated by**
T a. Cardiac failure
T b. Low Na^+ in proximal tubule
T c. Sympathetic stimulation
F d. High Na^+ in proximal tubule
F e. None.

Q. 294. **Heart is most sensitive to**
T a. Epinephrine
F b. Nor Epinephrine
F c. Metanephrine
F d. Ephedrine
F e. Atropine.

Q. 295. **Tachycardia in hypertension is caused by stimulation of**
T a. Beta 1 receptor
F b. Alpha 1 receptor
F c. Alpha 2 receptor
F d. Beta 2 receptor
F e. Beta 3 receptor

Q. 296. **Stimulation of baroreceptor leads to**
T a. Decrease in blood pressure and heart rate
F b. Increase in blood pressure and heart rate
F c. Increased intracranial tension
F d. Decreased intracranial tension
F e. None.

Q. 297. **Most potent vasopressor is**
T a. Cortisol
F b. Angiotensin II
F c. Renin
F d. Aldosterone
F e. All.

Q. 298. **In hypertension, there is**
T a. Sodium retention
F b. Reflex vasoconstriction
F c. Renal afferent arteriolar dilatation
F d. Water retention
T e. Ca retention.

Q. 299. **Which of the following does not have natriuretic actions**
T a. Aldosterone
F b. Natriuretic peptide
F c. Angiotensin

F d. Prednisolone
F e. None.

Applied

Q.300. **Effects of excess potassium ion in ECF on heart are :**
T a. heart may be flaccid.
T b. A-V conduction block may occur.
T c. heart become extremely dilated.
F d. heart rate will be increased.
F e. heart may be spastic.

Q. 301. **Coronary vasodilators released during ischemia of cardiac muscle are**
T a. hydrogen ion.
T b. adenosine.
T c. potassium ion.
F d. bicarbonate ion.
F e. sodium ion.

Q. 302. **Anginal pain due to coronary artery disease**
T a. may be elicited by sudden exposure to cold.
T b. is typically made worse by treatment. which lowers the systemic blood pressure.
T c. may be precipitated by anaemia.
F d. is caused by accumulation of metabolites in the myocardium.
F e. is relieved by sympathomymetic drugs.

Q. 303. **In complete heart block the**
T a. QRS complexes are abnonnal but regular.
T b. atria and ventricles beat independently.
T c. ventricular filling shows beat to beat variability.
F d. atrial beat becomes irregular.
F e. ventricular rate falls below 100 beats/min.

Q. 304. **When the AV bundle is completely interrupted as in complete heart block**
T a. ventricular rate falls below 50/minute.
T b. P-R interval shows beat to beat variability.
T c. ventricular filling shows beat to beat variability
F d. QRS comple shows beat to beat variability.
F e. atrial beat becomes irregular.

Q. 305. **Factors causing hypoeffective heart are**
T a. hypertention.
T b. diphtheritic damage.
T c. cardiac anoxia.
F d. hypertrophy of beau muscle.
F e. nervous stimulation.

Q. 306. **In haemorragic shock, there is a decrease in**
T a. venous return.
T b. coronary flow.
T c. cardiac work.
T d. cardiac output.
F e. plasma CO_2

Q. 307. **During which of the following compensatory reactions does hemorrhage generate?**
T a. vasoconstrictjon
T b. increased secretion of renin.
T c. venoconstrictjon
T d. increased secretion of catecholamjnes
F e. bradycardia

Q. 308. **Stokes-Adams syndrome occurs in**
T a. third degree heart block
F b. right or left bundle branch block.
F c. SA nodal block
F d. first degree heart block
F e. second degree heart block

Q. 309. **Murmurs (or bruits) may be detected by auscultation**
T a. over dilatations in arteries
T b. over constrictions in arteries
T c. in young adult in severe anaemia.
T d. over vessels in which there is turbulence
F e. over large arteries in healthy adults.

Q. 310. **Occlusion of common carotid artery on both sides leads to**
T a. Increase in HR & BP
F b. Increase in BP & decrease in HR
F c. Decrease in HR & BP
F d. No effect on BP & HR
F e. None.

Q. 311. **Hypovolemic shock is characterised by the following**
T a. Hypotension
T b. Cold and clammy skin
T c. Intense thirst
T d. Tachycardia
F e. Inhibition of respiration

Q. 312. **40% loss of blood volume in a patient is best managed by**
T a. Saline infusion
F b. Vasopressor agents
F c. Cardiac stimulants
F d. Intracardiac adrenaline
F e. All.

Q. 313. **All are increased during exercise**
T a. Cardiac output
T b. Venous return
T c. Coronary blood flow
F d. Peripheral vascular resistance
F e. All.

Q. 314. **One of the following is earliest indication of concealed acute bleeding**

T a. Tachycardia
F b. Postural HT
F c. Oliguria
F d. Cold clamy fingers
F e. Sweeting

Q. 315. **Rise of pulmonary arterial pressure is cause by**
T a. Hypoxia
F b. Acidosis
F c. Alkalosis
F d. All of the above
F e. None of the above

Q. 316. **Which is going to best declare the case as that of interatrial septal defect with other cardiac abnormalities?**
T a. Elevated PO_2 in pulmonary artery
F b. Elevated pressure in left atrium
F c. Elevated pressure in right atrium
F d. Systolic murmur
F e. None.

Q. 317. **Ventricular fibrillation**
T a. Follows ventricular tachycardia
T b. Associated with quick fall in cardiac output
T c. Induced by alternating current during vulnerable phase
F d. Induced by alternating current
F e. None

Q. 318. **Left ventricular failure tends to cause**
T c. Presystolic murmur over heart
F a. No breathlessness in lying position
F b. Decrease in ventricular end diastolic pressure
F d. Rise in lung compliance
F e. All of the above.

Q. 319. **What is seen following obstruction in major coronary artery?**
T a. Commonly rise in body temperatrue
T b. Reflex vagal inhibition of heart may further damage the myocardium
T c. Commonly ventricular fibrillation
F d. ST depression commonly in lead II
F e. All of the above.

Q. 320. **The first reactive change to occur after hemorrhage is**
T a. Vasocontriction
F b. Tachycarida
F c. Raised cortisol
F d. Raised adrenaline
F e. None of the above.