Unit 1

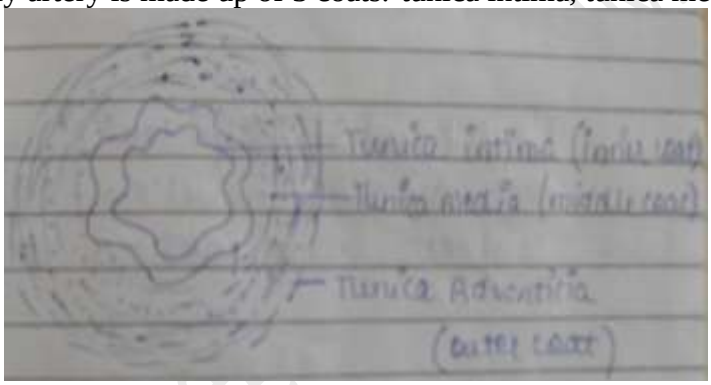
CARDIO VASCULAR SYSTEM:-
It includes the following:
Heart
Blood vessels:-

1. Artries
2. veins
3. capillaries

Arteries:-carry blood away from heart carry oxygenated blood (red color).
Exception:-pulmonary artery (de-oxygenated blood blue color).

- Smaller branches of artery are**:- arterioles.**
- Arteries is also known as:- **distributing channels**.
- Walls of arteries are thick.
- Opening of the artery is called:-lumen.
- Lumen of artery is small.
- Arteries have no valves.

Microscopically artery is made up of 3 coats:-tunica intima, tunica media, tunica adventitia

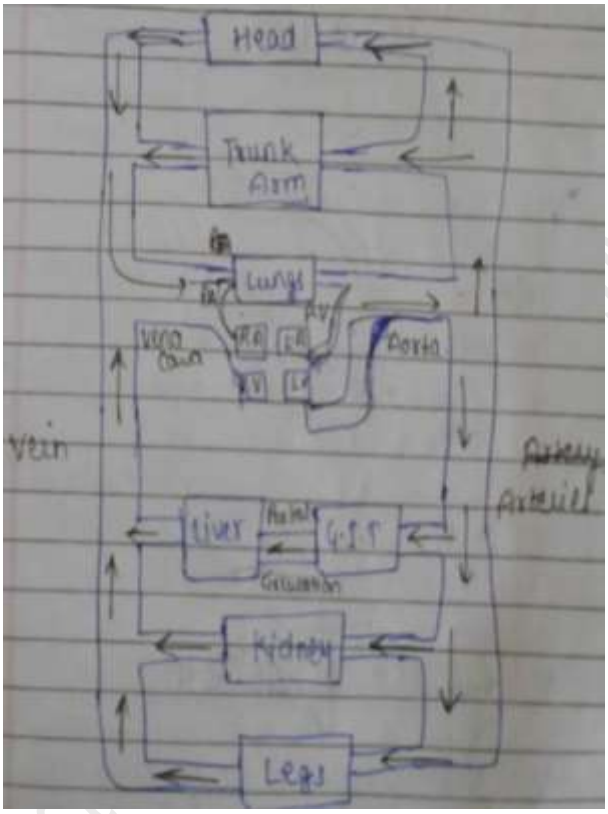


Microscopic structure of artery:-
Nerve supply of artery:- nerve vascularis, nutrient vessels of artery are called vasa vasorum.
Vein:-

- Veins are **collecting channels**
- veins carry blood to the heart.
- Veins carry **deoxygenated blood.**
- Exception: pulmonary veins
- Veins have **thin walls.**
- Veins have valves.
- Smaller branches of vein-**vennules.**
- Lumen of vein is large as compare to artery.
- Microscopically vein is also made up of 3-ill defined coats.
- Nutrient vessels of veins are called vasa vasorum.

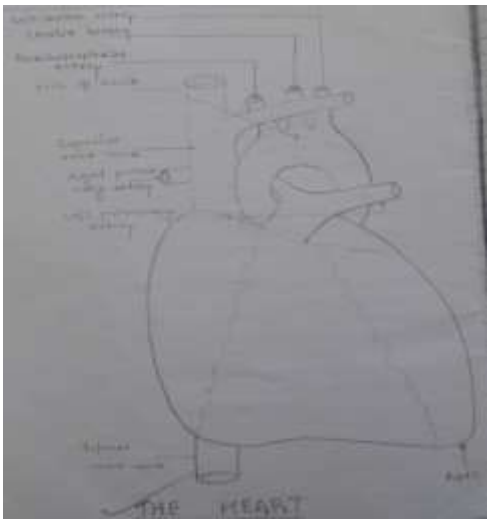
Capillaries:-capillaries are networks of microscopic vessels which connect arterioles to the vennules.

Diagram showing artery, capillaries and vein connection



Schematic representation of Blood flow in the body

Major blood vessels



Introduction:-the heart is a **conical hollow muscular organ** situated in the **middle mediastinum.** It contains two atria, two ventricles, aorta, Superior and Inferior vena cava, pulmonary artery and vein.

Aorta:-The aorta is the great arterial trunk which **receives oxygenated** blood **from the left ventricle** and **distributes it to all parts of the body**. It is situated in thorax in the following three parts:-

1. Ascending aorta.
2. Arch of the aorta.
3. Descending thoracic aorta.

Ascending aorta:-the ascending aorta arises from the upper end of the left ventricle.

- It is about 5cm long and is enclosed in the pericardium.
- At the root of the aorta there are three dilations of the vessel wall called the aortic sinuses.
- The sinuses are anterior, left posterior and right posterior.

Arch of the aorta:-it is the continuation of the ascending aorta.

- It is situated in the superior mediastinum behind the lower half of the manubrium sterni.

Course:-

1. It begins behind the upper border of the second right sternochondral joints.
2. It runs upwards, backwards and to the left across the left side of the bifurcation of trachea.
3. Then it passes downwards behind the left fourth thoracic vertebra. It thus arches over the root of the left lung.
4. It ends at the lower border of the body of the fourth thoracic vertebra by becoming continuous with descending aorta.

Descending thoracic aorta:-descending thoracic aorta is the continuation of the arch of the aorta.

- It lies in the posterior mediastinum.

Course:-

1. It begins on the left side of the lower border of the body of the fourth thoracic vertebra.
2. It descends with an inclination to the right of the twelfth thoracic vertebra.

Vena cava:- two parts of vena cava:-

1. Superior vena cava.

2. Inferior vena cava.

Superior vena cava:- it brings **deoxygenated blood** from the head and neck, upper limbs and thorax to the heart.

It is form by the union of the right and left branchiocephalic behind the lower border of the sternal end of the first right costal cartilage, pericardium opposite the costal cartilage and terminates by opening into the upper part of the right atrium behind the third right costal cartilage.

Interior vena cava:-this is formed at the level of the 5^{th} lumbar vertebra by common iliac veins and ascends through the abdomen lying close against the vertebral column and parallel to the just to the right of the descending abdominal aorta.

It passes through the tendons portion of the diaphragm into the thorax at the level of the 8^{th} thoracic vertebra. As the inferior vena cava ascends through the abdomen veins draining pelvic & abdominal organ empty into it.

Pulmonary artery :-pulmonary artery carrying deoxygenated blood, leaves the upper part of the right ventricle of the heart.

It passes upwards and divides into left and right pulmonary arteries at the levels of the 5^{th} thoracic vertebra.

Pulmonary artery:-

1. Left pulmonary artery.
2. Right pulmonary artery.

Left pulmonary artery:- it runs to the roots of the left lung where it is divided into two branches and passing into each lobe.

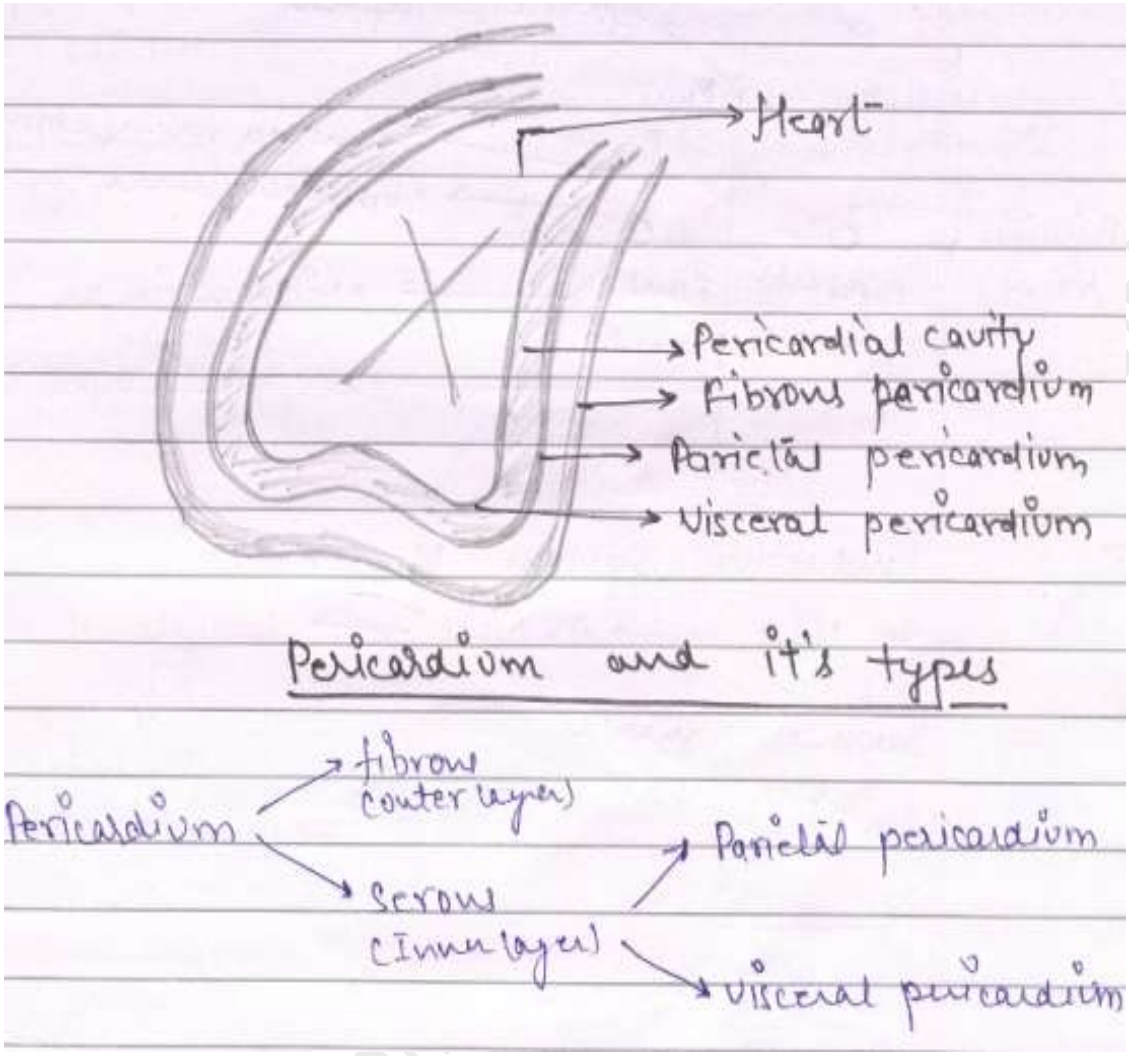
Right pulmonary artery :- it passes to the root of the right lungs and divides into two branches.

The larger branches carrier blood to the middle and lower lobes and the smaller branches to the upper lobe.

Pulmonary vein : It leave each lung, returning oxygenated blood to the left atrium of the heart during atrial systole .

PERICARDIUM

Pericardium membrane is the muscular membrane which surrounded the heart.



Pericardial fluid filled between parietal & visceral pericardial.
Pericardial effusion:-amount of pericardial fluid increases in the heart diseases.

The Heart

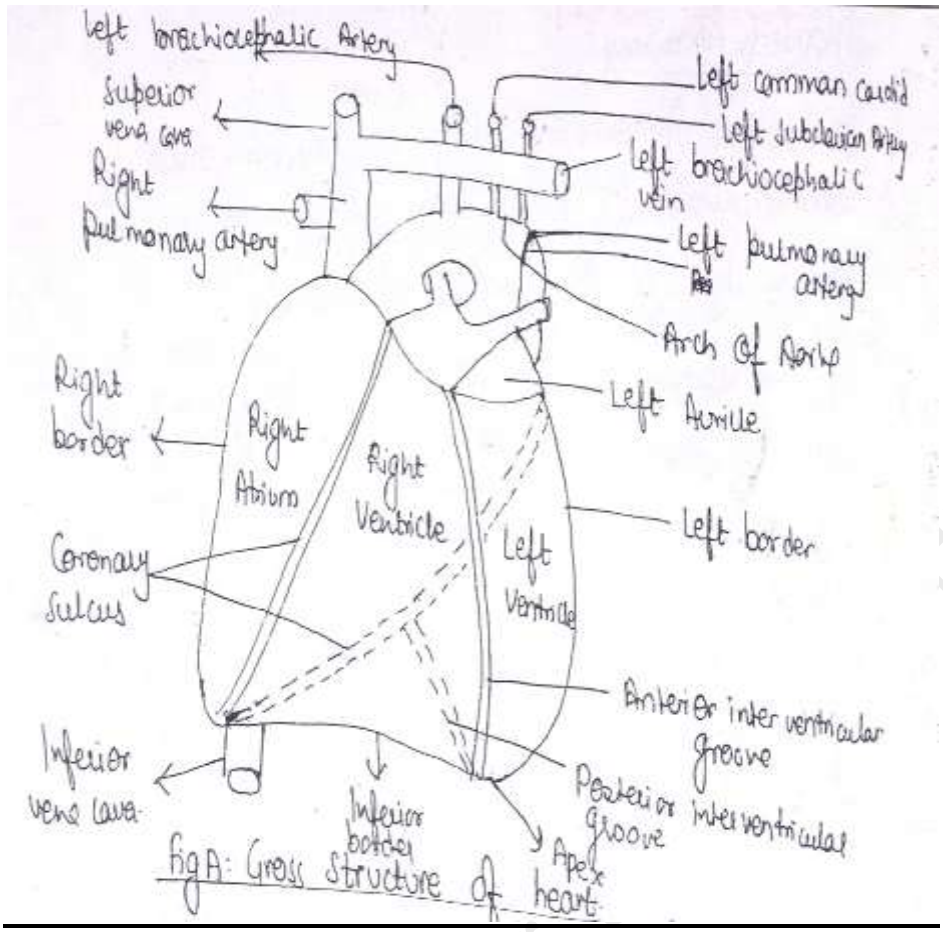


fig A: Gross Structure of heart

- Heart is a **muscular pump** enclosed in pericardium.
- Heart is conical, muscular hollow organ situated in the mediastinum.
- The heart is placed obliquely behind the sternum in such a way that about 1/3rd of the heart lies on the right side of the medial plane and 2/3rd lies on the left side of the median plane.
- Size of heart is about 12*9cm.
- Weight in males about 300gm, in females about 250gm.

External feature:-

- Human heart is 4 chambered organ.
- It has 2 atria--→ Right artrium.
  Left artrium.
  2 ventricles---→Right ventricle
  Left ventricles.
- Auricle is an appendage of atrium.
- The atria are separated from ventricles by atrio-ventricular groove.
- The ventricles are separated from each other by inter ventricular septum.
- The heart has an apex directed downwards and forward formed by left ventricle.

Borders:- heart has 4 borders.

1. **Upper border**:-formed by 2 atria, mainly by left atrium.
2. **Right border**:-formed by right atrium.

3. **Left border**:-formed by left ventricles and small part of the left atrium.
4. **Inferior border**:-formed by right ventricle.

Surfaces:-heart has 3 surfaces.

1. **Anterior surface (sterno costal surface**):-formed mainly by right atrium and right ventricle and partly by left ventricle and left atrium.
2. **Inferior (diaphragmatic surface**):-left 2/3rd is formed by left ventricle and right 1/3rd by right ventricle.

Left surface:-formed mainly by left ventricle & at the upper end by left autricle.

Apex of heart: It is situated in the left 5th inter costal space 9cm lateral to midsternal line. The pulsation may be felt and is known as apex beat.

- The base of heart posterior surface of heart is formed mainly by left atrium and small part of right atrium.

Chambers of heart:-

1. **Right chamber**:-
    - It is the right upper chamber of heart.
    - It receives venous supply deoxygenated blood from whole body by superior and inferior vena cava.

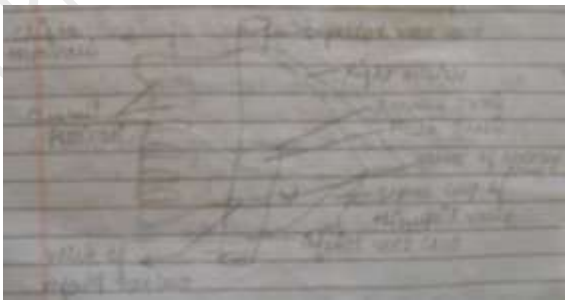
External features of right atrium:-

- This chamber receives superior vena cava at the upper end and inferior vena cava at lower end.
- The upper end is prolonged to left to from right auricle.
- Along the right border is a shallow vertical groove called at **sulcus terminalis** which is internal muscular ridge called as **crista terminalis.**
- The upper part of sulcus terminalis contains **SA node** (sina atrium node) also known as **pacemaker of the heart.**

Right antrio ventricular orifice:- it is guarded by **tricuspid valve** which maintains unidirectional how of blood from right atrium to right ventricle.

Internal features of right atrium:-

The interior of right atrium is divided into 3 parts.



- Smooth posterior part.
- **Rough anterior part** :-it is present a series of transverse muscular ridges called as **musculii pectinati** which has comb like appearance these arises from internal muscular ridges known as **crista terminalis.**
- **Septal wall inter atrial septum**:-these is a presence of shallow depression called as **fossa ovalis** in lower part. The margin of **fossa ovalis** is prominent.

The remains of **foramen ovale** which is normally closed at birth may be present.

Right ventricle:- it receives blood from right atrium.

External features:-it has 2 surfaces.

1. Anterior (sternocostal surface).
2. Interior (diaphragmatic surface).

Internal features:-interior of right ventricle has 2 parts.

Inflowing part:-it is rough due to presence of muscular ridges called as trabeculae carnae. There are three types

a. Ridges (fixed elevations).
b. Bridges.
c. Pillars (papillary muscles) : one end attached to ventricular wall and other end is connected to the cusps of tricuspid values by chordanae tendinae.

Out flowing part (infundibulum):-interior also show 2 orifices.

a. Right atrioventricular orifice guarded by **tricuspid valve.**
b. Pulmonary orifice guarded by **pulmonary valve.**

Left atrium:-

- Its appendage is known as left auricle.
- It receives oxygenated blood from lungs through set of 4 pulmonary veins.
- It pumps blood to left ventricle through left atriventricular orifice biscuspid/mitral orifice guarded **biscuspid/mitral valve**.
- Internally septal wall shows **fossa lunata.**

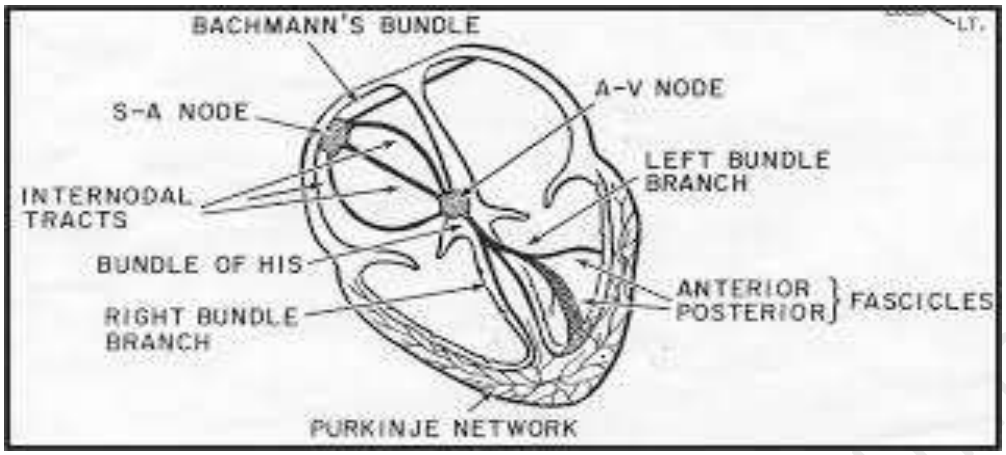
Left ventricles:-

- Left ventricle receives oxygenated blood from left atrium and pumps it into aorta.
- Externally left ventricle has 3 surfaces.
  a. Anterior (sternocostal surface).
  b. Interior (diaphragmatic) surface.
  c. Left surface.
- Interior of left ventricle is divided into 2 part.
  a. Lower rough part.
  b. Upper smooth part.
- Interior also show 2 orifice-
  a. Left atrioventricular orifice guarded by **bicuspid valve.**
  b. Aortic orifice guarded by **aortic valve.**
- There are 2 well developed **papillary muscles**. The chordanae tendinae from both these papillary muscles connect to the cusps of mitral value.
- The wall of the left ventricle are 3 times thicker than walls of right ventricle.(**physiological hypertrophy)**.

Conducting system of Heart:- it includes:

1. SA node (sinuatrial nodes).
2. AV node (atrio-ventricular).
3. Bundles of his :
   Right bundles branch.
   Left bundles branch.
4. Purkinje fibers.

- The system is made up of myocardium which is specialized for initiation and conduction of cardiac impulses. Cardiac muscle fibers are cross striated.



SA node:-situated in upper part of sulcus terminalis.

- It is the pacemaker of the heart.
- It beats at the rate 60-90 beats/minute.
- It generates impulse & initiated heart beat.
- This impulse travels through the atrial wall & reaches AV Node.

AV Node:-present in lower part of inter atrial septum.

- It is able to generate its own impulse at rate of 50-60beats/minute.

Bundle of His:-it is the only muscular connection between atrium & ventricle.

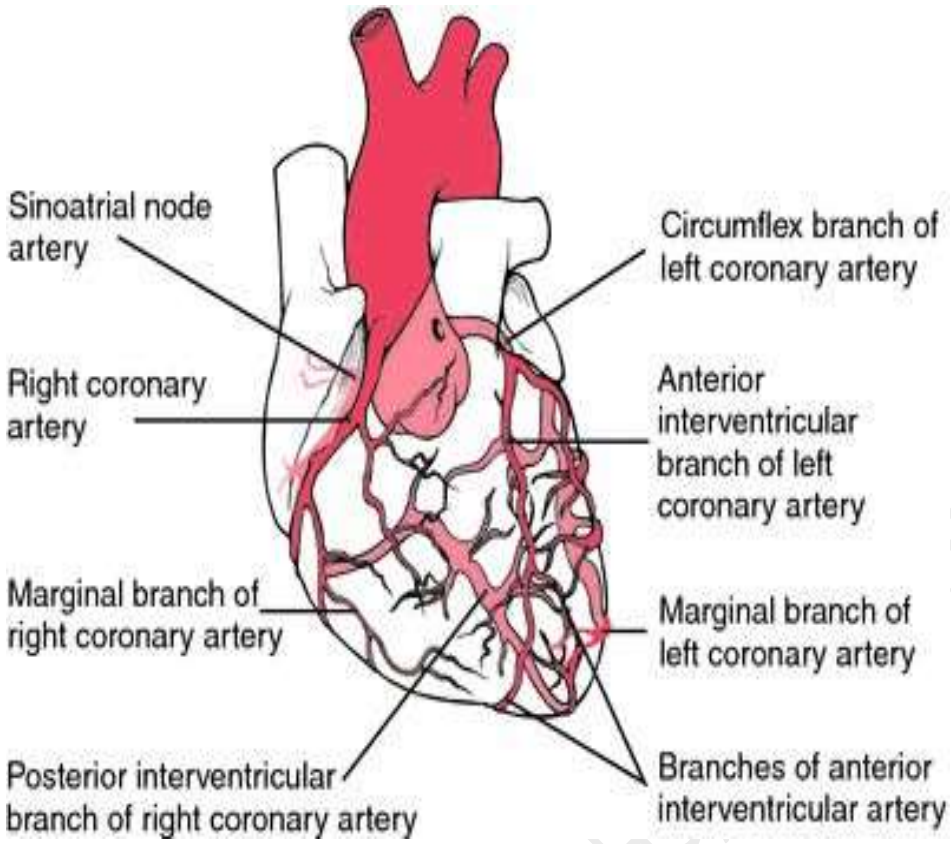
- It begins at AV node and crosses the atrio-ventricular ring and descends along posterior border of the inter-ventricular septum and divides into right and left branches.

a. **Right branches of bundle of His**:-it passes down the right side of inter ventricular septum and reaches the anterior wall of right ventricle where it divides into purkinje fibers.
b. **Left branches of bundle of His**:-it passes down the left side of inter ventricular septum as is distributed to left ventricle after dividing into purkinje fibers.

Purkinje fibers:-they form a subendo-cardial plexus. They are large pale fibers striated only at the margins. They generate beat at the rate less than 40beats per minute

Applied anatomy:-any defect or damage to conducting system result in cardiac arrhythmias (rhythm of heart is lost).

Arterial supply of heart:-



Coronary arteries supply blood to heart.

- There are two coronary arteries:
  a. Right coronary artery.
  b. Left coronary artery.

Right coronary artery:-it arises from ascending aortic sinus of ascending aorta.

Course of right coronary artery:-it passes forwards and to right to emerge on the surface of heart between pulmonary trunk root and right auricle.

It then runs downwards in right anterior coronary sulcus to the junction of right & left interior border of heart.

It winds around the inferior border to reach the diaphragmatic surface of heart.

Then it runs backwards & to left in the posterior coronary sulcus to reach to the posterior inter ventricular groove.

It terminates by anastomosing with left coronary artery.

Branches:-

1. Marginal artery.
2. Nodal artery.
3. Posterior inter ventricular artery.
4. Branch to right atrium.
5. Infundibular branch.
6. Terminal branch.

Area of distribution:-

- Right atrium.

- Greater part of right atrium, smaller part of left ventricle.
- Posterior part of inter ventricular septum.
- Whole of conducting system of heart except part of left branches of AV bundle.

Left coronary artery:-it arises from left posterior aortic sinus.

Course of left coronary artery:-the artery runs forwards and to left and emerge between pulmonary trunk and left auricle.

Here it gives branch to anterior inter ventricular groove, further continuation of left coronary artery is called circumflex artery.

The artery runs to the left in left anterior coronary sulcus.

It then winds around the left border of heart and continues in left posterior coronary sulcus.

It terminates by anastomosing with right coronary artery.

Branches:-

1. Anterior inter ventricular artery.
2. Branch to diaphragmatic surface of left ventricle.
3. Left atrium branch.
4. Pulmonary branch.
5. Terminal branch.

Area of distribution:-

- Left atrium.
- Greater part of left ventricle, small part of right ventricle.
- Anterior part of inter ventricular septum.
- Part of left branch of AV bundle.

Applied anatomy:-

- Thrombosis of coronary artery is common cause of sudden death in person in middle age and elderly. This is due to myocardial infection.
- If obstruction is partial then it leads to a condition known as angina pectoris.

Venous drainage of heart:-

Coronary sinus:-largest vein of heart. It is situated in left posterior coronary sulcus. It ends by opening into posterior wall of right atrium.

Nerve supply of heart:-

Parasympathetic ---→through vagus (Cardio inhibitory in function (slow heart rate).

Sympathetic----→supply from upper 2-5 thoracic segment of spinal cord

Cardio acceleratory in function

CARDIAC CYCLE:

systole=contraction diastole= relaxation.

1) Atrial contraction: (duration 0.1 sec)

During ventricular relaxation blood flows from atria to ventricles.

About 75% of blood flows to ventricles before atrial contraction.

When both atria contracts almost simultaneously and pupms remaining 25% of blood flows in respective ventricles (therefore even when if atrial fails to function it is unlikely to be noticed unless a person exercises)

Contraction of atria increses pressure inside atria to 4-6 mmHg in right atrium and abt. 7-8mmHg in left atrium.

The pressure rise in right atrium is reflected into the veins and this wave is recorded as 'a' wave

(recorded from jugular vein with the help of a transducer)
2) Then there is a period of atrial relaxation for rest of caridac cycel abt duration 0.7 sec during which various ventricular events occur in a sequences as follows:
3)Ventricular contration (0.3 sec)
At the termination of atrial contration,the pressure of blood in ventricles rises (normally < 12mm Hg) .
Rising the ventricular pressure now exceeds the atrial pressure.
This causes closure of AV valves(tricuspid & mitral valves) which is a major component responsible for generating 1st heart sound.
Then there are following phases of ventricular contration:

a) Isovolumic contraction phase:

At beginning of this phase AV valve are closed but semilunar valves (pulmonary and aortic valves) are not yet opened.
Thus,ventricular chambers are sealed from both atria and arteries.
The ventricles starts contracting but volume of blood inside both ventricles remains the same hence this phase is called as isovolumic phase of contraction.
This phase lasts for abt.0.05 sec.
During this phase ventricles contract as a closed chamber and pressure inside the ventricles rises rapidly to a high value.
When pressure in left ventricle is slightly above 80 mmHg and right ventricular pressure slightly above 8 mmHg then the ventricular pressures push the semilunar valves open.
This causes ejection of blood from ventricles to the respective arteries in next phases.

b) Rapid ejection phase:

As soon as the semilunar valves open blood is rapidly ejected.
Abt.2/3rd of stroke volume is ejected in this rapid ejection phase.
Stroke volume: Volume of blood that is ejected by each ventricles with each beat.It is 70ml.
The duration of this phase is abt 0.11 sec.
The pressure inside the left ventricle rises to 120mmHg during this phase.
The end of rapid ejection phase occurs at abt the peak of ventricular and atrial contraction pressure.
The right ventricle ejection begins before that of left and continues even after the left ventricular ejection is complete.
As the both ventricles almost eject same volume of blood,the velocity of right ventricular ejection is less than that of left ventrcicle.

c) Reduced ejection phase:

During later 2/3rd of contratction rate of ejection declines.
During this phase of reduced ejection,rest one third stroke volume is ejected.
This phase lasts for abt 0.14 sec.
During the period of slow ejection ventricular pressure fall to a value slightly lower than that in Aorta but still blood continues to empty into Aorta because blood flowing out has built up momentum.
As this momentum decreases,kinetic energy of momentum is converted to pressure in the Aorta.
This causes aortic pressure to rise slightly above that of ventricle.

4) Ventricular relaxation: (0.5 sec)

It occurs in following phase:

a) Protodiastole:

At the end of ventricular systole ventricles start relaxing allowing rapid fall in the intraventricular pressure.
This is the period of protodiastole which lasts for 0.004 sec.
At the end of this phase elevated pressure in distended arteries (Aorta and Pulmonart artery) immediately pushes the blood back toward the ventricles which snaps the aortic and pulmonary semilunar valves closes.
This is the major component in generating 2nd heart sound (closure of semilunar valve).
It also causes dicrotic notch in the down slope of aortic pressure called Incisura.
Incisura indicates end of systole and the onset of diastole

b**) Isovolumic relaxation phase:**

The ventricles continue to relax as closed chambers as semilunar valves are closed and AV valves are not yet open.
This causes rapid fall of pressure inside ventricles (from 80mmHg to abt 2 to 3 mmHg in left ventricle)
This phase lasts for 0.06 sec.
Because the ventricular volume remains constant this phase is called isovolumic phase.
When ventricular pressures fall below atrial pressure the AV valves opens.

c) Rapid filling phase:

During ventricular contraction because AV valve are closed ,large amount of blood accumulates in Atria because Veins continue to empty the blood into them and this causes increase in the pressure inside the Atria.
High atrial pressure causes blood to flow rapid into ventricles.
Then pressures in both the chamber fall as ventricular relaxation continues.

d) Reduced filling phase or diastasis:

After the rapid filling phase,pressure in atria and ventricles rises slowly as blood continues to return to the heart.
This decreases the rate of blood flow from atria to ventricles causing slow filling of ventricles called diastasis.
During rapid filling and diastasis phase abt 75% of blood passes from atria to ventricles.
Then next cycles begins with atrial contraction.

CONTROL OF HEART RATE

Heart rate is normally determined by the pacemaker activity of the sinoatrial node (SA node) located in the posterior wall of the right atrium. The SA node exhibits automaticity that is determined by spontaneous changes in Ca^{++}, Na^{+}, *and* K^{+} *conductances.* This intrinsic automaticity, if left unmodified by neurohumoral factors, exhibits a spontaneous firing. This intrinsic firing rate decreases with age.
Heart rate is decreased below the intrinsic rate primarily by activation of the *vagus nerve* innervating the SA node. Normally, at rest, there is significant vagal tone on the SA node so that the resting heart rate is between 60 and100 beats/min. This vagal influence can be demonstrated by administration of atropine, a muscarinic receptor antagonist, which leads to increase in heart rate depending upon the initial level of vagal tone.

For heart rate to increase above the intrinsic rate, there is both a withdrawal of vagal tone and an activation of *sympathetic nerves* innervating the SA node. This reciprocal change in sympathetic and parasympathetic activity permits heart rate to increase during exercise.

Heart rate is also modified by circulating catecholamines acting via β_1-adrenoceptors located on SA nodal cells. Heart rate is also modified by changes in circulating thyroxin (thyrotoxicosis causes tachycardia) and by changes in body core temperature (hyperthermia increases heart rate).

SA nodal dysfunction can lead to sinus bradycardia, sinus tachycardia, or sick-sinus syndrome.

The maximal heart rate that can be achieved in an individual is estimated by
Maximal Heart Rate = 220 beats/min − age in years

CARDIAC OUTPUT

The **cardiac output** is simply the amount of blood pumped by the heart per minute. Necessarily, the cardiac output is the product of the **heart rate**, which is the number of beats per minute, and the **stroke volume**, which is amount pumped per beat.

CO = HR X SV

The cardiac output is usually expressed in liters/minute.

Regulation of the Heart Rate

Let's first consider the regulation of the heart rate because this is the most straightforward of the two factors. The regulation boils down primarily to **parasympathetic and sympathetic effects**.

In a young person, an SA node without either parasympathetic or sympathetic effects will drive about 100 beats/min. This, of course, is substantially faster than the usual resting heart rate. Thus, at rest there is normally **parasympathetic tone** that keep the heart rate down to around 60-70 beats/min.

In order to increase the heart rate from the resting rate, the parasympathetic tone is withdrawn until the heart rate is about 100 beats/min. Then norepinephrine is released by sympathetic nerves. This increases the slope of the pacemaker potential by, for example, opening a Ca^{++} channel via a G protein. In this way, the pacemaker potential reaches threshold faster and the heart rate is increased.

Because the adrenergic receptors in the heart are all **beta receptors**, the hormone epinephrine has the same effect as norepinephrine released by sympathetic nerves. (But in various other organs, norepinephrine and epinephrine do not produce the same actions.)

Regulation of the Stroke Volume

Sympathetic Effect
Autonomic nerves not only innervate the SA node, but also are found elsewhere in the heart. Norepinephrine released by sympathetic nerves increases the force with which ventricular muscle fibers contract (by increasing the Ca^{++} effect). This tends to be significant mainly at the greatest levels of exercise.

Afterload
The **aortic pressure** influences the stroke volume for a straightforward reason. If the aortic pressure increases, this pressure reduces the volume of blood that flows into the aorta during systole. The aortic pressure is called **afterload** because it is the "load" experienced by the ventricle after it begins contracting.

A drug might reduce the afterload, for example, by dilating arterioles. This allows blood to flow from the arteries more easily, thereby preventing the arterial pressure from increasing as blood is injected into it by the ventricle.

Frank-Starling Mechanism
However, the factor we will be most concerned with is the **Frank-Starling mechanism**. Unfortunately, it is also the one most difficult to get your mind around. The Frank-Starling mechanism leads to changes in the stroke volume as a result of changes in the **end-diastolic volume**.

The end-diastolic volume is the volume of a ventricle at the very end of filling and just before systole begins. This can change because the ventricles are flexible and under different circumstances, the amount of blood flowing in during diastole varies. If less blood flows into the ventricle as it fills, the end-diastolic volume goes down. If more blood flows in, the end-diastolic volume goes up.

The Frank-Starling effect is due to the fact that heart muscle fibers **respond to stretch by contracting more forcefully**. This is not a passive, elastic effect, but rather due to an **increased expenditure of ATP** energy.

Thus, if the end-diastolic volume increases, the muscle fibers are lengthened and the ventricle contracts more forcefully, ejecting a greater stroke volume. The figure to the right shows this Frank-Starling effect.

For the right ventricle, this is the pressure in the right atrium, because this is the pressure that is experienced by the right ventricle as it fills. Since there is no valve at the entrance to the right atrium, the pressure in the right atrium is necessarily the same as the pressure in the veins at the entrance to the right atrium. This pressure in the large veins at the entrance to the right atrium is called the **central venous pressure**.

In other words, the central venous pressure is the same at the right atrial pressure, and this is the pressure that determines the filling of the right ventricle and thus its end-diastolic volume. The

central venous pressure always is only a few mm Hg, but nonetheless it does change enough to significantly affect the stroke volume. In particular, posture changes this pressure and that is the factor with which we are here most concerned.

central veins are very compliant structures, pressure cannot increase again in them until blood flows back into the thorax.

The Effect of Muscle Contraction on Stroke Volume

Lying down, of course, is one factor that would increase the amount of blood in the veins in the thorax and thus the central venous pressure. However, another important factor is muscle contraction. If the standing person begins walking, the contractions of the leg muscles squeeze on the leg veins, thereby forcing blood from those veins up into the thorax. This is called **muscle pumping**.

Thus, as a standing person begins walking, the end-diastolic volume and thus the stroke volume increase.

Muscle pumping works on the veins, but not the arteries, because veins are **large**, **highly compliant** and the larger ones have **valves**. In other words, contracting skeletal muscles serve as auxillary pumps, squeezing blood back into the central veins.

BLOOD PRESSURE

Blood pressure (**BP**) is the Lateral pressure exerted by circulating blood upon the walls of blood vessels.

The endogenous regulation of arterial pressure is not completely understood, but the following mechanisms of regulating arterial pressure have been well-characterized:

- Baroreceptor reflex: Baroreceptors in the high pressure receptor zones detect changes in arterial pressure. These baroreceptors send signals ultimately to the medulla of the brain stem, specifically to the rostral ventrolateral medulla (RVLM). The medulla, by way of the autonomic nervous system, adjusts the mean arterial pressure by altering both the force and speed of the heart's contractions, as well as the total peripheral resistance. The most important arterial baroreceptors are located in the left and right carotid sinuses and in the aortic arch.[41]
- Renin-angiotensin system (RAS): This system is generally known for its long-term adjustment of arterial pressure. This system allows the kidney to compensate for loss in blood volume or drops in arterial pressure by activating an endogenous vasoconstrictor known as angiotensin II.
- Aldosterone release: This steroid hormone is released from the adrenal cortex in response to angiotensin II or high serum potassium levels. Aldosterone stimulates sodium retention and potassium excretion by the kidneys. Since sodium is the main ion that determines the amount of fluid in the blood vessels by osmosis, aldosterone will increase fluid retention, and indirectly, arterial pressure.

- Baroreceptors in low pressure receptor zones (mainly in the venae cavae and the pulmonary veins, and in the atria) result in feedback by regulating the secretion of antidiuretic hormone (ADH/Vasopressin), renin and aldosterone. The resultant increase in blood volume results in an increased cardiac output by the Frank–Starling law of the heart, in turn increasing arterial blood pressure.

These different mechanisms are not necessarily independent of each other, as indicated by the link between the RAS and aldosterone release. When blood pressure falls many physiological cascades commence in order to return the blood pressure to a more appropriate level.

1. The blood pressure fall is detected by a decrease in blood flow and thus a decrease in Glomerular filtration rate (GFR).
2. Decrease in GFR is sensed as a decrease in Na^+ levels by the macula densa.
3. The macula densa cause an increase in Na^+ reabsorption, which causes water to follow in via osmosis and leads to an ultimate increase in plasma volume. Further, the macula densa releases adenosine which causes constriction of the afferent arterioles.
4. At the same time, the juxtaglomerular cells sense the decrease in blood pressure and release renin.
5. Renin converts angiotensinogen (inactive form) to angiotensin I (active form).
6. Angiotensin I flows in the bloodstream until it reaches the capillaries of the lungs where angiotensin converting enzyme (ACE) acts on it to convert it into angiotensin II.
7. Angiotensin II is a vasoconstrictor which will increase bloodflow to the heart and subsequently the preload, ultimately increasing the cardiac output.
8. Angiotensin II also causes an increase in the release of aldosterone from the adrenal glands.
9. Aldosterone further increases the Na^+ and H_2O reabsorption in the distal convoluted tubule of the nephron