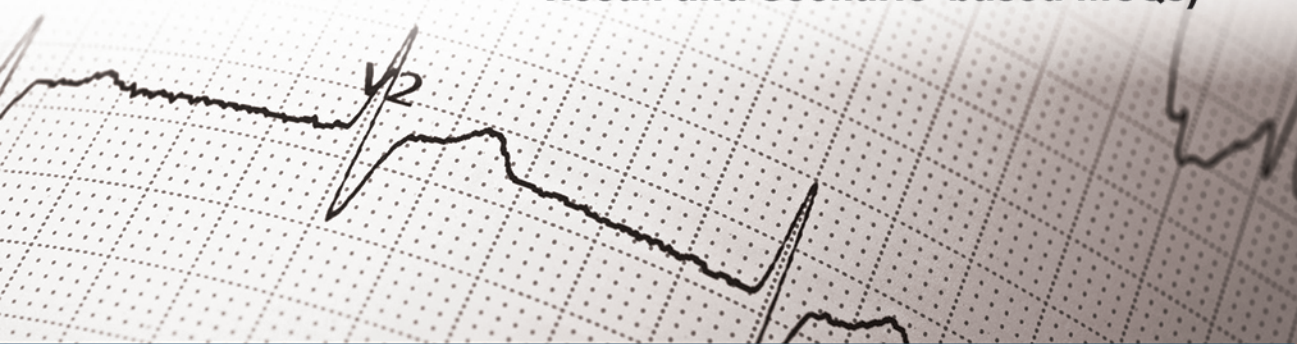
2nd Edition

Nagalakshmi's Solved Question Bank in Physiology

(Essay Questions, Reasoning Questions, Case-based Questions, Integration Modules, Recall and Scenario-based MCQs)



As per the Revised Competency-based Medical Education Curriculum (NMC)

Nagalakshmi V

JAYPEE

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CHAPTER 5

Cardiovascular Physiology

Chapter Outline

PY5.1 Describe the functional anatomy of the heart including chambers and coronary circulation.

PY5.2 Describe the properties of cardiac muscle including its morphology, electrical, mechanical and metabolic functions.

PY5.3 Describe generation, conduction of cardiac impulse along with conduction pathway (including pacemaker potential).

PY5.4 Discuss the physiological events occurring during cardiac cycle, concurrent pressure—volume changes, generation of heart sounds and murmur.

PY5.5 Describe the physiology of electrocardiogram (ECG), and the cardiac axis and its application.

PY5.6 Describe physiological variations in ECG waveforms, abnormal waveforms and intervals, arrhythmias, heart block and myocardial infarction. Explained in applied section.

PY5.7 Describe and discuss hemodynamic of circulatory system.

PY5.8 Describe and discuss local and systemic cardiovascular regulatory mechanisms.

PY5.9 Describe heart rate, factors affecting heart rate, and its regulation.

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PY5.13 Describe the pathophysiology of shock, syncope and heart failure.

PY5.1 Describe the functional anatomy of the heart including chambers and coronary circulation.

Reasoning question **Why SA node is the pacemaker tissues of heart? Name other tissues.**

Sinoatrial node	Discharge rate 70–80 times/min
Atrioventricular node	Discharge rate 40–60 times/min
Bundle of His	Discharge rate 15–40 times/min
Purkinje fibers	Discharge rate 15–40 times/min

Electrical system of the heart

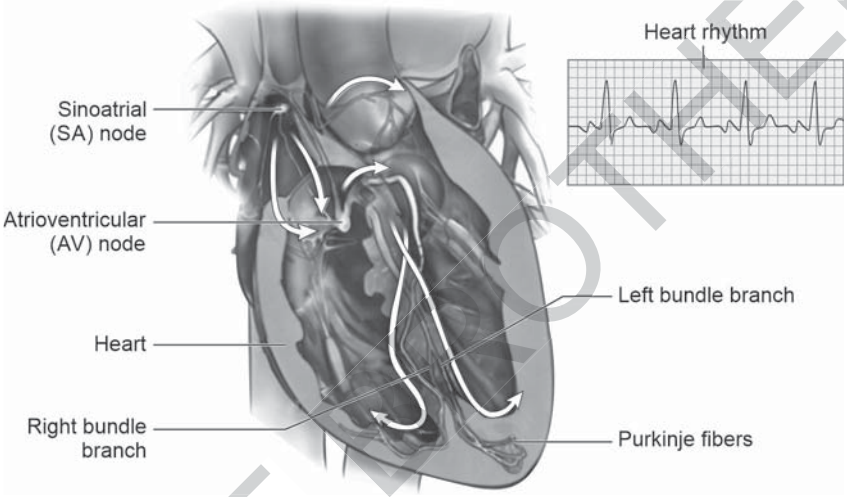


Fig. 5.1: Pacemaker tissues of heart.

Amongst all pacemakers present in heart SA node acts mainly as pacemaker tissue of heart and control the heart rate because rate of intrinsic discharge of SA node is highest when compared to other pacemaker tissues.

Applied Physiology

Ectopic pacemaker: When any other tissues in heart like AV node or bundle of His becomes the pacemaker other than SA node it is called as ectopic pacemaker. This results in abnormal contraction of various parts of heart making it an ineffective pump.

Long essay **Describe features, regulation and measurement of coronary circulation.**

Reasoning question **Why coronary tissue receives less blood during systole?**

Reasoning question **Why endocardium of left ventricle is more prone for myocardial infarction?**

Features of coronary circulation

Heart receives its blood supply from coronary arteries. Right coronary artery supplies right ventricle and posterior portion of left ventricle. Left coronary artery supplies anterior part and lateral part of left ventricle.

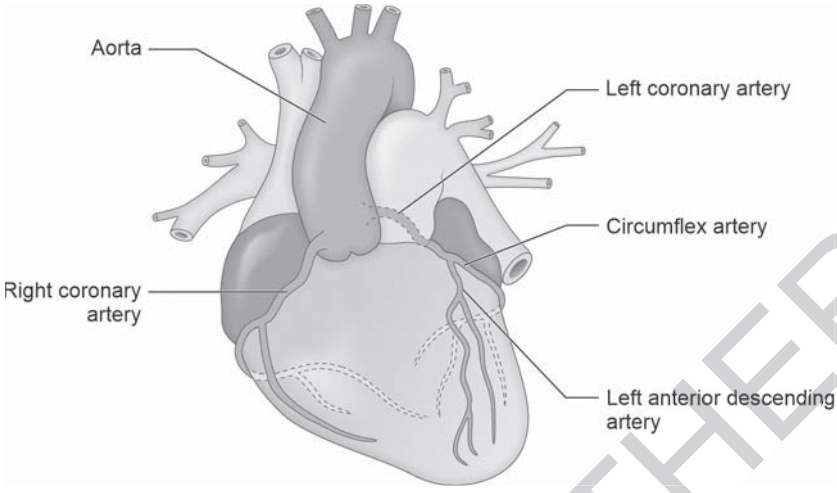


Fig. 5.2: Coronary circulation.

Characteristic features of coronary circulation:

- Coronary blood flow –5% of cardiac output –250 mL/min OR 70 mL/100 g/minute which can increase up to 3 to 4 fold increase during exercise.
- Dominant right or left coronary arteries.
- Coronary arteries are end arteries but have collaterals.
- **Maximum O_2 consumption:** Arterial blood contains 19 mL of O_2/100 mL of blood : 13 mL of O_2 consumed per 100 mL of blood (70–80%). Venous blood contains 6 mL of O_2/100 mL of blood
- Coronary blood flow is mainly controlled by metabolic mechanism than neural.
- Cardiac muscle uses more fatty acids than glucose as source of energy. During hypoxic conditions it produces more lactic acid which explains pain during myocardial infarction.
- Coronary blood flow shows autoregulation.
- **Phasic coronary blood flow:** Coronary tissues receives more blood during diastole and less blood flow during systole.
  - Pressure in aorta = 120/80 mm Hg
  - Pressure in left ventricle = 121/0 mm Hg
  - Pressure in right ventricle = 25/0 mm Hg
- Pressure gradient in left ventricle during systole = –1 mm Hg
- Pressure gradient in left ventricle during diastole = 80 mm Hg
- Pressure gradient in right ventricle during systole = 95 mm Hg
- Pressure gradient in right ventricle during diastole = 80 mm Hg

Therefore, blood flows into left ventricle only during diastole and less during systole where as blood flows into right ventricle during both systole and diastole.

Applied Physiology

Endocardial surface is more prone for myocardial infarction in left ventricle as it receives very less blood during systole.

Regulation of coronary blood flow

- **Autoregulation mechanism:** In spite of wide variation in mean arterial pressure of range between 75 and 170 mm Hg the coronary blood flow remains constant which is known as autoregulation of blood flow.

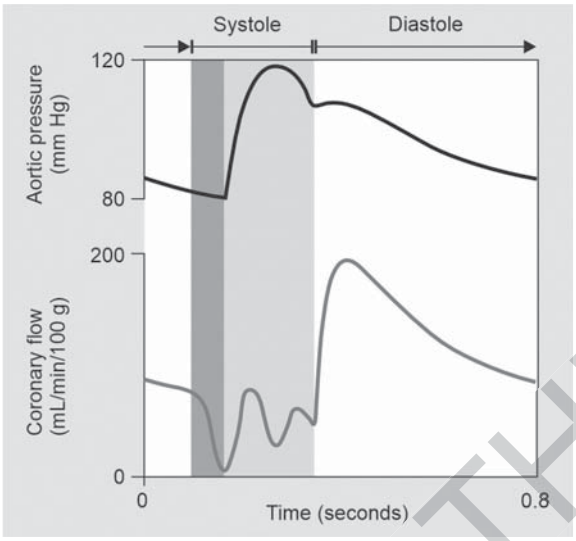


Fig. 5.3: Characteristic features of coronary circulation.

- **Metabolites mechanism:** Coronary blood flow is controlled mainly by metabolites. Whenever work on heart increases, the coronary blood flow also increases proportionately. It is due to effect of some metabolites like: Reduced pO_2, adenosine compound, carbon dioxide, hydrogen ions, potassium, prostaglandins, nitric oxide.
- **Nervous control mechanism:** Coronary blood flow is not mainly controlled by neural mechanisms.

Sympathetic control mechanism

- **Direct effect:** Sympathetic nerve releases norepinephrine which act on alpha receptors causes vasoconstriction and reduces coronary blood flow
- **Indirect effect:** Sympathetic nerve increases heart rate and force of contraction, there by increases coronary work load leading to increased coronary metabolism which releases various metabolites mentioned above which are potent vasodilators leads to increased coronary blood flow.

Parasympathetic control mechanism

- **Direct effect:** Parasympathetic nerve releases acetylcholine causes vasodilatation and increases coronary blood flow.
- **Indirect effect:** Parasympathetic nerve reduces heart rate, there by reduces coronary work load leading to decreased coronary metabolism, leads to reduced coronary blood flow.

Methods of measurement of coronary blood flow.

- Using Fick's principle (nitrous oxide method/Kety method): This method is based on the principle that:
  Amount of substance taken by an organ = blood flow × (A-V) difference of substance.
  In this subject is made to inhale 15% of N_2O and arteriovenous difference of N_2 is measured.
  $$\text{Coronary blood flow} = \frac{N_2O \text{ taken up/min}}{\text{A-V}}$$
- Coronary angiography
- Radionuclide utilization technique
- Electromagnetic flow meter technique

PY5.2 Describe the properties of cardiac muscle including its morphology, electrical, mechanical and metabolic functions.

Long essay **Describe in detail morphological, electrical, mechanical and metabolic property of cardiac muscle.**

Reasoning question **Why cardiac muscle cannot be tetanized?**

Reasoning question **Why cardiac muscle does not get fatigue?**

The properties of cardiac muscle are broadly divided into four types:

1. **Morphological property**
2. **Electrical property**
   - **Excitability (bathmotropism)**
   - **Refractory period**
   - **Rhythmicity (chronotropism)**
   - **Conductivity (dromotropism)**
3. **Mechanical property**
   - **Contractility (ionotropism)**
   - **All or none law**
   - **Staircase phenomenon**
   - **Effect of preload**
   - **Effect of afterload**
   - **Summation**
4. **Metabolic property**

Morphological property

- Cardiac muscle is an involuntary, striated muscle.
- Each muscle fiber is branched, recombining and spreading again.
- At the point of contact of each muscle fiber, there is extensive intercalated disc which are made of gap junctions which allows easy current flow from one muscle fiber to another thus the entire unit act like a functional syncytium.
- There are two syncytia—atria and ventricle.

Electrical properties

Excitability (bathmotropism): Cardiac muscle is an excitable tissue, which responds to threshold stimulus in the form of an action potential. The factors which increase excitability of cardiac muscle are called as positive bathmotropic factors. The factors which decreases excitability of cardiac muscle are called as negative bathmotropic factors.

RMP of cardiac muscle fiber is –90 mV.

- **Phase 0—Depolarization phase:** It is due to opening of voltage-gated sodium channels and calcium channels which makes inside cardiac muscle fiber to become positive up to + 20 mV.
- **Phase 1—Initial repolarization:** It is due to closure of voltage-gated sodium channels and opening of voltage-gated potassium channels.
- **Phase 2—Plateau phase:** It is due to opening of long-lasting type of calcium channels causing calcium influx resulting in plateau phase.
- **Phase 3—Rapid repolarization:** It is due to closure of long-lasting calcium channels and opening of voltage-gated potassium channels causing potassium efflux lead to repolarization.

- **Phase 4—Resting membrane potential:** RMP in cardiac muscle fiber is –90 mV which is due to sodium potassium pump.

The duration of action potential in cardiac muscle is 250 ms which is around 15 times more than skeletal muscle action potential.

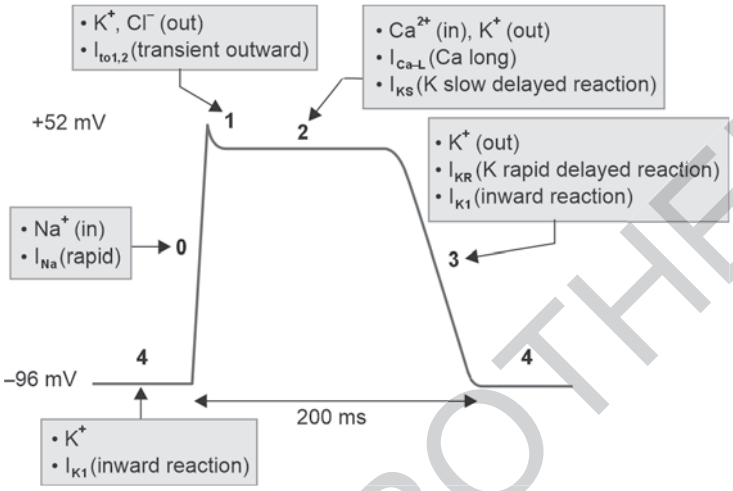


Fig. 5.4: Cardiac muscle action potential.

Autorhythmicity: Explained in pacemaker potential in next question.

- **Refractory period:** It is one of the important property of cardiac muscle action potential. "It is the duration in which cardiac muscle does not respond to the second stimulus. It is around 250–300 ms in cardiac muscle. It is of two types—absolute and relative refractory period.
  - *Absolute refractory period:* It is the period in which cardiac muscle does not respond at all. It extends from phase 0 till phase 3 of action potential. Its duration is around 200–250 ms.

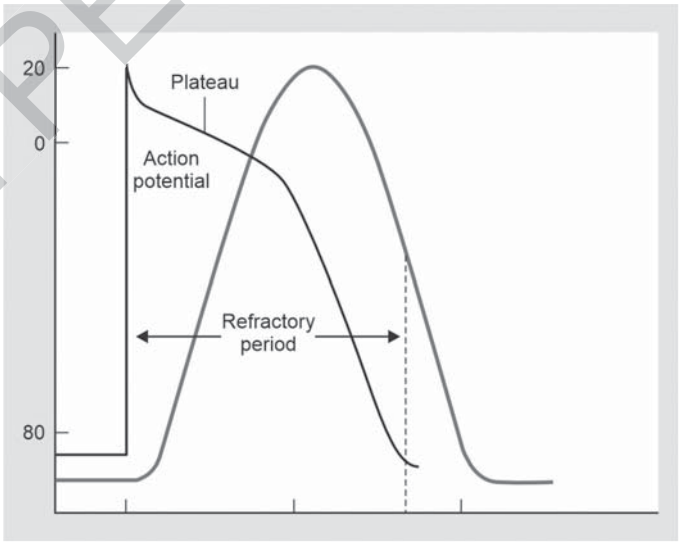


Fig. 5.5: Refractory period in cardiac muscle.

- *Relative refractory period:* It is the period in which cardiac muscle might respond to second stimulus if its strength is maximum. It extends from phase 3 till phase 4 of action potential. Its duration is around 50 ms.

- **Clinical significance of refractory period in cardiac muscle:** Due to long refractory period in cardiac muscle, cardiac muscle *cannot be tetanized*. As tetanized heart will be an ineffective pump.

II. Conductivity (Dromotropism)—Explained in PY5.3

Mechanical property

I. Contractility (Ionotropism): It is the ability of cardiac muscle to shorten and generate force to pump the blood. Cardiac muscle begins to contract after depolarization and lasts 1.5 times more than duration of action potential.

Various factors affecting myocardial contractility:

- **All or none law:** When a stimulus is applied either cardiac muscle will not contract at all (None response) or contract (All response) this is due to syncytial arrangement of atrial and ventricular cardiac muscle fibers.

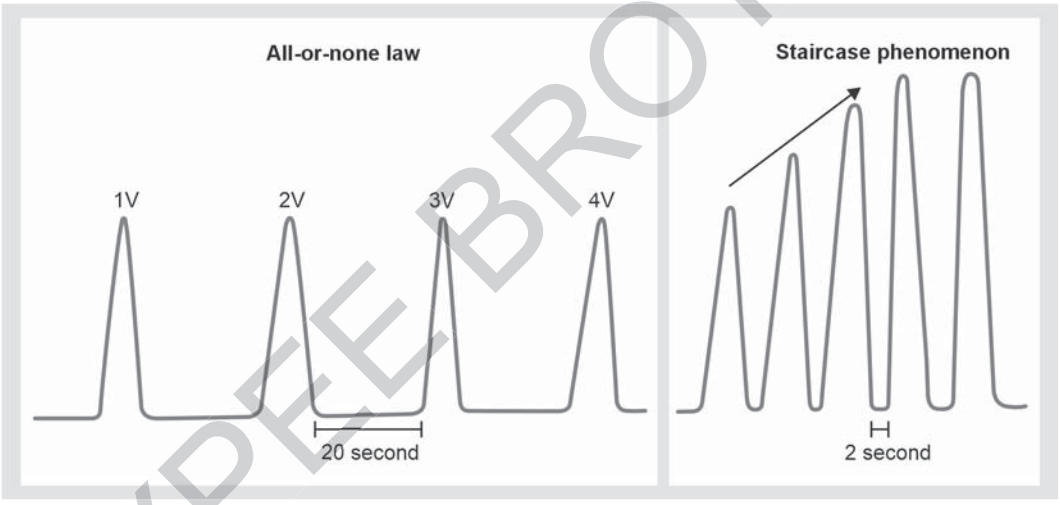


Fig. 5.6: All or none and staircase effect seen in cardiac muscle.

- **Staircase phenomenon/treppe:** In a quiescent heart, when stimulus is given there will be increase in force of contraction in initial 4–5 contractions. This is due to accumulation of sodium which increases calcium inside cardiac muscle cells, there by increases force of contraction.
- **Summation of subminimal stimuli:** In a quiescent heart, when subthreshold stimulus is given within interval of half second, after 15–20 stimuli cardiac muscle contracts due to summation of subminimal stimuli.
- **Effect of preload:** Preload is the load which act on cardiac muscle before it starts contracting. In cardiac muscle the end diastolic volume forms preload. As per Frank–Starling's law as EDV increases there is proportionate increase in force of contraction.
- **Effect of afterload:** Afterload is the load which acts on cardiac muscle after it starts contracting. In cardiac muscle the peripheral resistance forms afterload. As peripheral resistance increases force of contraction reduces.

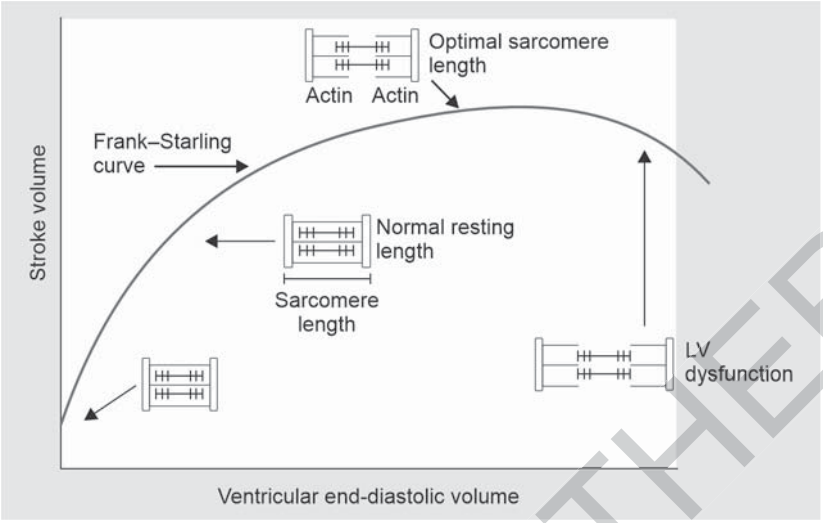


Fig. 5.7: Frank–Starling's curve.

Metabolic property

- Cardiac muscle use only fatty acids as source of metabolism
- Cardiac muscle depend only on aerobic metabolism for energy
- It is rich in capillary density and mitochondria
- The above factors responsible for *cardiac muscle do not get fatigue*

PY5.3 Describe generation, conduction of cardiac impulse along with conduction pathway (including pacemaker potential).

Short essay **Describe the conducting tissues of heart with the help of a neat labeled diagram.**

- **Sinoatrial (S-A node) node:** It is a specialized type of cardiac muscle which is found beneath and lateral to opening of superior vena cava. It has the property of autorhythmicity, i.e., generation of cardiac impulse due to presence of pacemaker (P) cells. It is called as pacemaker of heart as its discharge rate is highest compared to others. Conduction velocity is 0.05 m/sec.
- **Interatrial pathways:** Impulse generated at SA node pass through anterior, middle and posterior interatrial pathways to reach to AV node. The conduction velocity is 0.3 m/sec
- **Atrial muscle:** The impulse generated at SA node reaches along anterior interatrial band to reach to left atrium. The conduction velocity is 0.3 m/sec
- **Atrioventricular (AV) node:** AV node is located in posterior wall of right atrium below tricuspid valve. Once the impulse reaches to AV node there occurs AV nodal delay of 0.1 sec, which help in complete emptying of blood from atria before ventricle begin to contract. The delay is due to reduced number of gap junctions between cells in conduction pathway.
- **A-V bundle/Bundle of His:** Impulse will be conducted forward in AV bundle from atria to ventricles. It divides to right and left bundle branches which run along interventricular septum.
- **Purkinje fibers:** A-V bundle divides to smaller branches forming Purkinje fibers which run below the endocardium in ventricles towards base. The conduction velocity is 4 m/sec due to large amount of gap junctions between the cells.

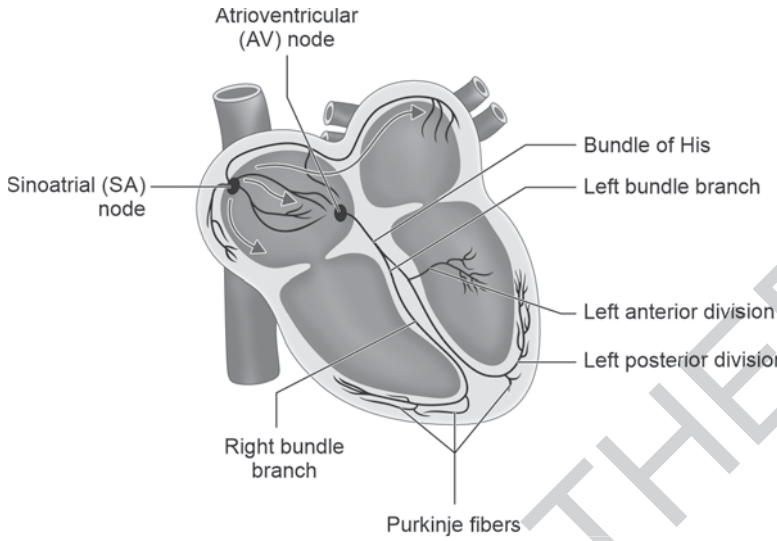


Fig. 5.8: Conducting tissues of heart.

- **Ventricular muscle:** Once the impulse reaches to ventricular muscle it transmit at rate of 0.5 m/sec impulse transmit in form of spirals towards the surface of heart.

Summary of conduction of impulse in heart:

Conducting tissue	*Conduction velocity (m/sec)*	*Time required for transmission of impulse (sec)*
SA node	0.05	Beginning of impulse
Atrial muscle	0.3	0.03
Interatrial tracts	1	0.03
AV node	0.05	0.13
Bundle of His	1	0.16
Purkinje fibers	4	0.19
Ventricular muscle	0.3	0.22

Applied Physiology

Effect of parasympathetic and sympathetic discharge on conduction of impulse:

- **Effect of vagal stimulation: Negative chronotropic and dromotropic**
  - Vagus innervates mainly SA node and AV node. Whenever vagus is stimulated it releases acetylcholine, which reduces rhythm as well as slows down conduction of impulses.
  - *Ventricular escape:* When vagus stimulation is very severe, it causes failure of transmission of impulses from SA node to AV node. This cause AV node to slowly begin its own discharge which might take 5–10 seconds. During those 10 seconds ventricle will not receive any impulse and heart fails to pump blood. This is called as ventricular escape.
- **Effect of sympathetic stimulation: Positive chronotropic, dromotropic, bathmotropic and inotropic**

  Sympathetic nerve innervates SA node, AV node, atrial muscles and mainly to ventricular muscles. When sympathetic nerve is stimulated it releases epinephrine which increases rate of discharge (positive chronotropic), increases excitability (positive bathmotropic), increases

conduction of impulse (positive dromotropic) and increases force of contraction (positive inotropic).

Short answer **What is pacemaker potential? Write its ionic basis.**

Autorhythmicity: Heart has the ability to generate its own beat due to the presence of pacemaker tissues.

The pacemaker tissues present in heart are:
- Sinoatrial node (SAN)
- Atrioventricular node (AVN)
- Bundle of His
- Purkinje fiber

Pacemaker tissues exhibits pacemaker potential/prepotential/slow potential in which RMP will not be stable.

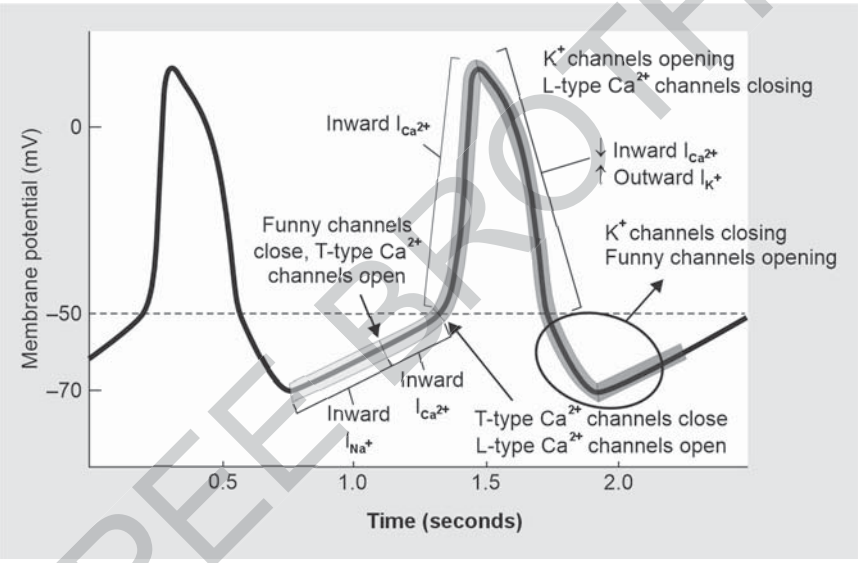


Fig. 5.9: Electrical activity of pacemaker cells.

Causes for prepotential/pacemaker potential: It is due to opening of hyper polarization induced opening of sodium channels (Funny currents) which causes sodium influx which opens whenever potential inside is –60 mV. Later half of prepotential is due to opening of transient calcium channels causing calcium influx.
- **Depolarization phase:** As soon as membrane potential reaches –40 mV, there is opening of long-lasting calcium channels (L-Type ca channel), due to entry of positive charged calcium inside, it makes inside positive up to + 10 mV.
- **Repolarization phase:** It is due to closure of long-lasting calcium channels and opening of voltage-gated potassium channels. This causes potassium efflux resulting in inside becoming negative up to –60 mV.

Applied Physiology

Acetylcholine decreases heart rate by reducing slope of prepotential. Ach bind to muscarinic receptors at SAN act via G protein cause opening of potassium channels and increases potassium

efflux by 3 folds lead to hyper polarization inside SAN. This reduces slope of prepotential which lead to decrease in heart rate **(Negative chronotropic effect)**.

Epinephrine increases heart rate by increasing slope of prepotential. Epinephrine binds to β1 adrenergic receptors at SAN and increases calcium influx and reduces potassium efflux. This decreases slope of prepotential and thereby increases heart rate **(Positive chronotropic effect)**.

PY5.4 Discuss the physiological events occurring during cardiac cycle, concurrent pressure—volume changes, generation of heart sounds and murmur.

Long essay **Describe various phases of cardiac cycle. Explain left ventricular pressure volume changes with the help of Wigger's diagram.**

OR

Short essay **Describe the pressure—volume change in left ventricle occurring during cardiac cycle.**

Definition: The electrical and mechanical cardiac events which occur from one heartbeat to next is called as cardiac cycle. If heartbeat is around 72 beats/min then duration of one cardiac cycle is 60/72 = 0.8 seconds.

Phases of cardiac cycle:

Atrial events:

- Atrial systole—0.1 seconds
- Atrial diastole—0.7 seconds

Ventricular events:

- Ventricular systole—0.3 seconds
  - Isovolumetric contraction—0.05 seconds
  - Rapid ejection phase—0.10 seconds
  - Slow ejection phase—0.15 seconds
  - Total duration—0.3 seconds
- Ventricular diastole– 0.5 seconds
  - Protodiastole—0.04 seconds
  - Isovolumetric relaxation—0.06 seconds
  - Rapid filling phase—0.10 seconds
  - Slow filling phase—0.20 seconds
  - Last rapid filling phase—0.10 seconds
  - Total duration—0.5 seconds

Description of each event of cardiac cycle:

- **Atrial systole:**
  - **Duration:** 0.1 second
  - **Mechanical changes:** During atrial systole, atria contracts which pushes additional 25% of blood from atria to ventricles. This corresponds to last phase of ventricular diastole.
- **Atrial diastole:**
  - **Duration:** 0.7 seconds
  - **Mechanical changes:** During atrial diastole atria relax which causes pressure in atria to fall. Blood flow from great veins to atrium. Due to gradual filling of atria pressure builds

which causes opening of atrioventricular valves and blood flow continuously from atria to ventricles. During atrial diastole 75% of blood flows from atrium to ventricles.
Ventricular events:

- **Ventricular systole:**
  - **Isovolumetric contraction:**
    - **Duration:** 0.05 seconds
    - **Mechanical changes:**

With the beginning of ventricular contraction
↓
Pressure in ventricle increases up to 80 mm Hg
↓
Result in closure of atrioventricular valve (production of 1st heart sound)
↓
Additional 0.02–0.03 seconds required for opening of aortic and pulmonary valves against pressure gradient
↓
During this time all valves are closed and ventricle are still contracting
↓
So that tension inside ventricles increases without any change in volume of blood. So this phase is called as isovolumetric contraction

- **Rapid and slow ejection phase:**
  - **Duration:** 0.10 seconds and 0.15 seconds
  - **Mechanical changes:**

When the pressure in ventricles increases up to 80 mm Hg in left ventricle
↓
This lead to opening of semilunar valves (aortic and pulmonary)
↓
Rapid ejection of blood from ventricles into aorta or pulmonary artery with increases in pressure up to 120 mm Hg in left ventricle
↓
Around 60% of end diastolic volume will be ejected during ejection phase (ejection fraction) 70% of ejection fraction will be ejected during rapid ejection phase and 30% of ejection fraction will be ejected during slow ejection phase

- **Ventricular diastole:**
  - Protodiastole:
    - **Duration:** 0.04 seconds
    - **Mechanical changes:**

At the end of ventricular systole, ventricle muscle starts relaxing
↓
Pressure in ventricles start decreasing up to 80 mm Hg in left ventricle than pressure in aorta
↓
This result in backflow of blood from aorta to left ventricle
↓
This result in closure of semilunar valves (aortic and pulmonary valves) responsible for production of 2nd heart sound

- **Isovolumetric relaxation**:
  - **Duration:** 0.06 seconds
  - **Mechanical changes:**

For around 0.06 seconds after the ventricles start relaxing, all valves are closed and ventricle still relaxing

↓

As a result pressure in ventricles drastically reduces up to 2–3 mm Hg in left ventricle without any change in its volume of blood, due to this it is called as isovolumetric relaxation phase

  - **Filling phase (Rapid, slow and last rapid filling phase/atrial systole)**
    - **Duration:** 0.10, 0.20 and 0.10 seconds
    - **Mechanical changes:**

At the end of isovolumetric relaxation, pressure in ventricles reduces up to 2–3 mm Hg

↓

This results in opening of atrioventricular valves

↓

This causes rapid flow of blood from atria to ventricles

↓

- In the initial 0.1 seconds filling occurs rapidly (rapid filling phase)
- In the next 0.20 seconds filling occurs slow (slow filling phase/diastasis)
- In both rapid and slow filling phase around 75% of blood flow form atria to ventricles
- The last rapid filling phase/atrial systole for 0.1 seconds cause additional 25% of blood flow from atria to ventricles

Pressure and volume changes during cardiac cycle

Name of phase	*Pressure change in right ventricle*	*Pressure change in left ventricle*	*Volume changes in left ventricle*
Ventricular systole			
• Isovolumetric contraction	8 mm Hg	80 mm Hg	No change in volume
• Rapid ejection phase			
• Slow ejection phase	25 mm Hg 15 mm Hg	120 mm Hg 100 mm Hg	80 mL of blood eject out (stroke volume)
Ventricular diastole			
• Protodiastole	8 mm Hg	80 mm Hg	No change in volume
• Isovolumetric relaxation	1–2 mm Hg	2–3 mm Hg	
• Rapid filling phase			
• Slow filling phase			
• Last filling phase	0 mm Hg 0 mm Hg 6–7 mm Hg	0 mm Hg 0 mm Hg 7–8 mm Hg	Volume increases up to 130 mL (End diastolic volume)

Wigger's Diagram

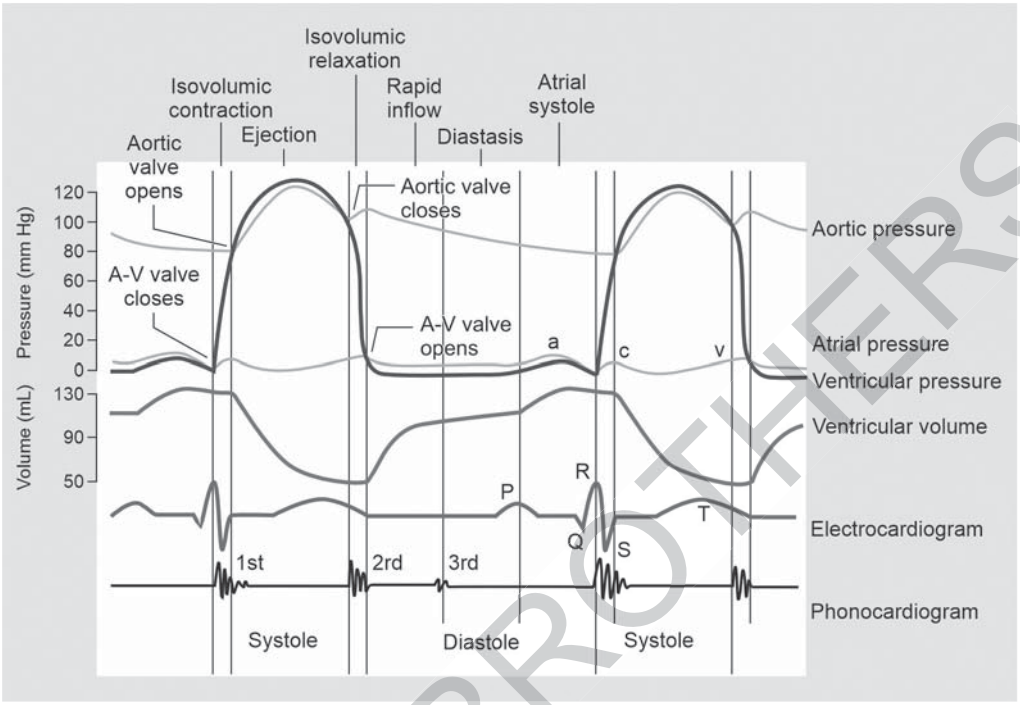


Fig. 5.10: Mechanical and electrical events at cardiac cycle.

Pressure–volume loop:
It is an alternate method of representing cardiac cycle. Pressure volume loop is obtained by plotting ventricular pressure along Y-axis and ventricular volume along X-axis.

	Name of phase	*Volume changes*	*Pressure changes*
a	Filling phase of ventricles	130 mL increase in volume	6–7 mm Hg
b	Isovolumetric contraction	No change in volume	Increase from 8 to 80 mm Hg
c	Ejection phase	Around 80 mL of blood eject out	Pressure increase up to 120 mm Hg
d	Protodiastole and IVR	No change in volume	Pressure reduces from 120 to 2–3 mm Hg

Electrical events during cardiac cycle:
The electrical events occurring during cardiac cycle can be recorded using ECG. P wave recorded in ECG occurs before atrial systole. QRS wave recorded in ECG before ventricular systole. T wave produced due to ventricular repolarization recorded in ECG occurs before ventricular diastole.

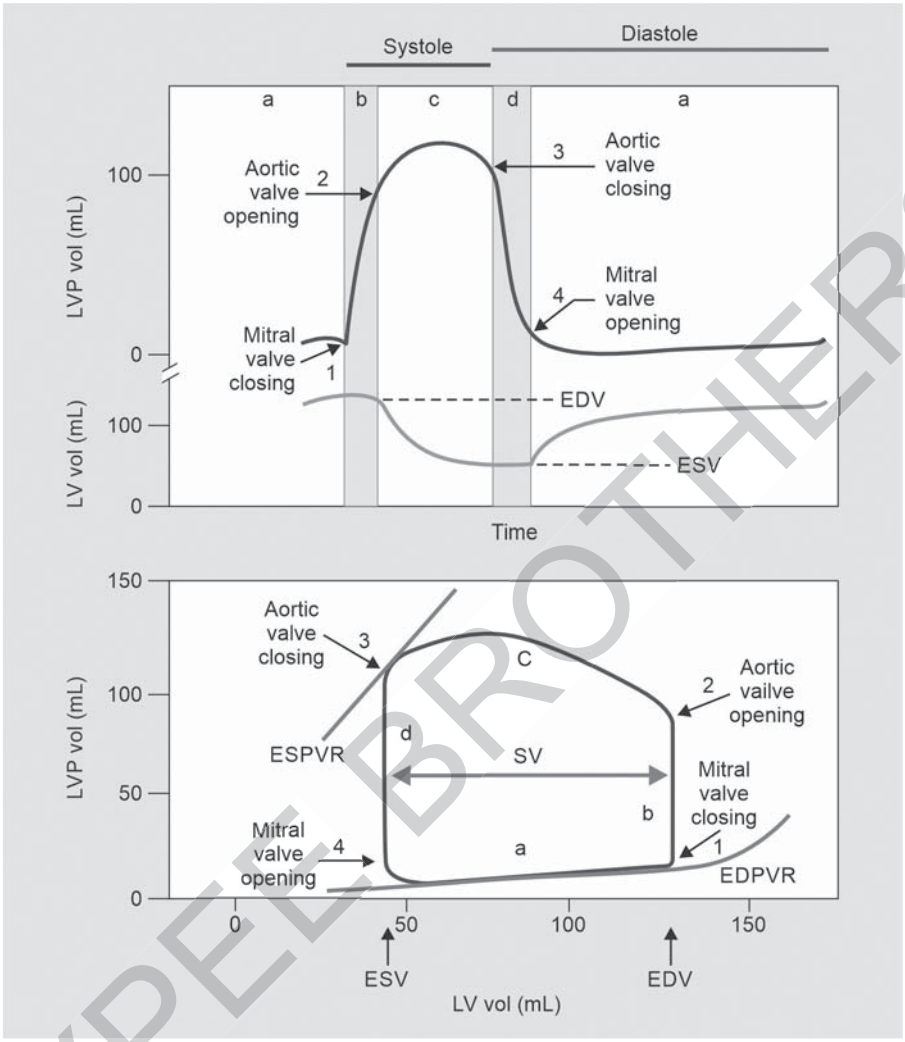


Fig. 5.11: Pressure-volume loop of left ventricle.

Short answer **Differentiate between first and second heart sounds.**

	First heart sound (HS1)	*Second heart sound (HS2)*
Cause	Vibrations set due to closure of AV valves	Vibrations set due to closure of semilunar valves
Relation with cardiac cycle	Occurs at the beginning of ventricular systole during isovolumetric contraction	Occurs at the beginning of ventricular diastole during isovolumetric relaxation
Pitch	Low (25–45 Hz)	High (50 Hz)
Duration	Longer duration (0.15 seconds)	Shorter duration (0.12 seconds)
Sound like	Lubb	Dubb
Correlation with ECG	It coincides with R wave in ECG	Coincides with end of T wave
Sites for auscultation	Better heard in mitral and tricuspid area	Better heard in aortic and pulmonary area

- **Mitral area:** Correspond with apical impulse located at left 5th intercostals space medial to midclavicular line
- **Tricuspid area:** Located at left 5th intercostals space at lower end of sternum
- **Aortic area:** Located at right 2nd intercostals space lateral to sternum
- **Pulmonary area:** Located at left 2nd intercostals space lateral to sternum

Sites of Cardiac Auscultation

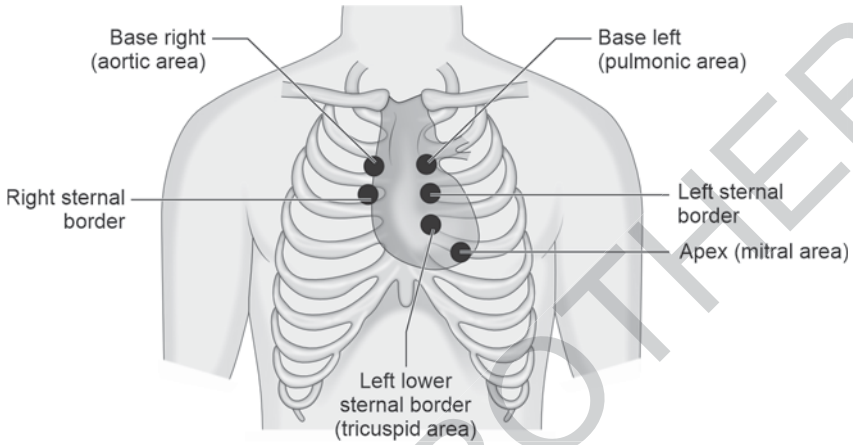


Fig. 5.12: Sites of cardiac auscultation.

Short essay **Describe jugular venous pressure (JVP)**

Jugular venous pressure (JVP) reflects right atrial pressure which can be recorded using intracardiac catheterization. Recorded JVP shows three positive waves (a, c, v) and two negative waves (x and y)

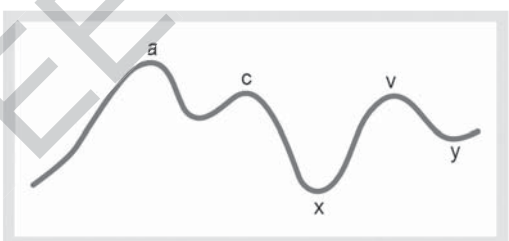


Fig. 5.13: Jugular venous pressure.

- **a wave:** It is a positive wave recorded during atrial systole when intra atrial pressure in right atrium increases up to 4–6 mm Hg.
- **c wave:** It is a positive wave recorded during isovolumetric contraction. When the pressure in the ventricles rises sharply, then there will be bulging of AV valve in to atria rising intra atrial pressure.
- **v wave:** It is a positive recorded during ventricular diastole. As long as AV valve are closed, the atrial pressure gradually increases.
- **x wave:** It is a negative recorded during ventricular systole. When ventricle contracts it pulls AV valve down there by decreasing intra atrial pressure.
- **y wave:** It is a negative wave recorded during rapid filling phase of ventricles, during this AV valve open allowing rapid flow of blood from atria to ventricles.

PY5.5 Describe the physiology of electrocardiogram (ECG), and the cardiac axis and its application.

Long essay **Describe various waves, intervals and segments of electrocardiogram (ECG). Add a note on various leads used in electrocardiography.**

OR

Short essay **Describe the various leads used in ECG recording.**

OR

Short essay **Describe various waves, intervals and segments of electrocardiogram (ECG).**

Electrocardiogram (ECG): "It is the surface recording of electrical activities of heart occurring during cardiac cycle". Because body fluid contains lot of electrolytes it acts as volume conductor, so it act as good conductor of electricity. As a result electrical activity of heart can be recorded by keeping the electrodes on surface of body. The procedure of recording ECG is called as electrocardiography and the apparatus is called as electrocardiograph.

Waves associated with ECG:

- **P wave**
  - **Voltage:** 0.1– 0.3 mV
  - **Duration:** 0.1 sec
  - **Cause:** Atrial depolarization and represents spread of excitation from SA Node to atrial muscles.
- **QRS complex:** It is a complex composed of three waves Q, R and S.
- **Q wave:** It is a negative wave.
  - **Voltage:** Less than 0.2 mV
  - **Duration:** Less than 0.04 sec
  - **Cause:** Depolarization of mid portion of interventricular septum
- **R wave:** It is a positive wave.
  - **Voltage:** 2.0 mV
  - **Duration:** Less than 0.04 sec
  - **Cause:** Depolarization of ventricular muscles.
- **S wave:** It is a negative wave.
  - **Voltage:** Less than 0.2 mV
  - **Duration:** Less than 0.04 sec
  - **Cause:** Depolarization of basal portion of ventricle
- **QRS complex:** extends from beginning of Q wave to end of S wave.
  - **Duration:** 0.08–0.12 sec
  - **Voltage:** 1.5–2 mV
- **T wave:** It is a positive wave.
  - **Voltage:** 2.0–3 .0 mV
  - **Duration:** 0.27–0.30 sec
  - **Cause:** Repolarization of ventricular muscles.
- **U wave:** It is a positive wave due to depolarization of papillary muscles which is not recorded in ECG.

Segments:

- **PR segment:** It is an isoelectric line followed by P wave.
  - **Duration:** 0.04 sec
  - **Cause:** paucity of conduction at AV Node.

- **ST segment:** It is an isoelectric line from the end of S wave to beginning of T wave.
  - **Duration:** 0.04–0.08 sec
  - **Cause:** Time from end of ventricular depolarization to beginning of ventricular repolarization.
- **TP segment:** It is an isoelectric line from the end of T wave to the beginning of P wave of ext cardiac cycle. It mainly depends on heart rate.
  - **Duration:** 0.2 sec

Intervals:

- **PR interval:** It is the time from the beginning of P wave to the beginning of Q or R wave.
  - **Duration:** 0.12–0.20 sec
  - **Cause:** It represents atrial depolarization and spread of depolarization to Bundle of His. It is the interval between beginnings of atrial excitation to beginning of ventricular excitation.
- **QT interval:** It is the time from beginning of Q wave to the end of T wave.
  - **Duration:** 0.35 sec
  - **Cause:** It signifies ventricular depolarization and ventricular repolarization
- **J point:** It is the point at the end of S wave to beginning of ST segment. It is a point of no electrical activity of heart. It is an important point for assessment of ST segment elevation or depression.

Leads used in recording of ECG:

There are two types of leads used in ECG recording.

1. **Unipolar limb leads and unipolar chest leads**
2. **Bipolar limb leads**

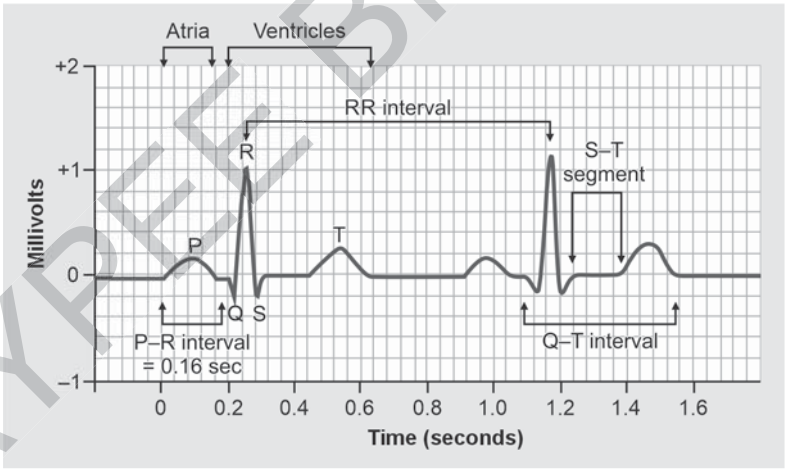


Fig. 5.14: Waves, intervals and segments of ECG.

Bipolar limb leads: In this both the recording electrodes are active and potential differences among them will be recorded. There are three standard bipolar limb leads.

1. **Lead I:** Records the potential difference between electrodes placed on left and right arm. Positive terminal is on left arm and negative terminal on right arm. (LA-RA)
2. **Lead II:** Records the potential difference between electrodes placed on left leg and right arm. Positive terminal is on left leg and negative terminal on right arm. (LF-RA)

3. **Lead III:** Records the potential difference between electrodes placed on left leg and left arm. Positive terminal is on left arm and negative terminal on right arm. (LF-LA)

Einthoven's triangle: It is a triangle drawn around the heart as a current source. Right arm, left arm and left foot forms the apices of triangle.

Einthoven's law: The sum of potential recorded in lead I and III is equal to lead II. Lead II = Lead I + Lead III.

UNIPOLAR LEADS

In unipolar recording only one electrode will be active and the other electrode is indifferent electrode which is kept at zero potential.

- **Unipolar limb leads:** They record the electrical activity of heart in frontal plane.
  - **V_R (right arm):** Reflects the electrical activity of cavity of ventricle
  - **V_L (left arm):** Reflects the electrical activity of left outer side of heart
  - **V_F (left foot):** Reflects the electrical activity of inferior surface of heart
- **Augmented limb leads:** They record the electrical activity of heart in frontal plane. The arrangement has made in such a way that potential recorded in augmented leads are more when compared to non augmented leads.

$$aV_R = \frac{3V_R}{2}$$

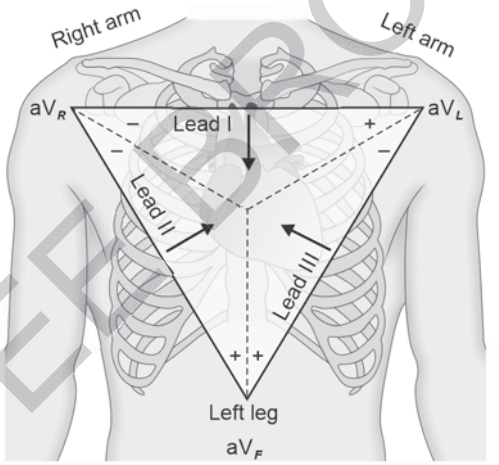


Fig. 5.15: Unipolar and bipolar limb leads.

Unipolar chest leads: They record the electrical activity of heart in horizontal plane.

- **V_1 and V_2:** Reflects electrical activity of right ventricle
- **V_3 and V_4:** Reflects electrical activity of interventricular septum
- **V_5 and V_6:** Reflects electrical activity of left ventricle
- **V_1:** Placed at right 4th intercostals space lateral to sternum
- **V_2:** Placed at left 4th intercostals space lateral to sternum
- **V_3:** Placed in between V_2 and V_4
- **V_4:** Placed at left 5th intercostals space in midclavicular line
- **V_5:** Placed at left 5th intercostals space in anterior axillary line
- **V_6:** Placed at left 5th intercostals space in mid axillary line

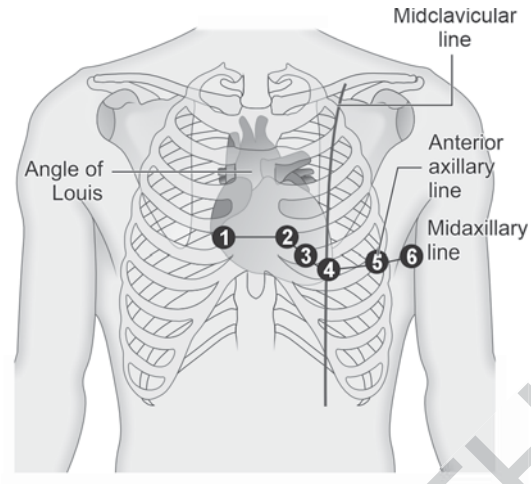


Fig. 5.16: Unipolar chest leads.

ECG leads and their respective view of heart:

Anterior wall	Lead I, aVL, V1–V3
Posterior wall	Lead II, III, aVF
Lateral wall	V4, V5 and V6
Septal wall	V3, V4

Short answer **What is P-R interval? Write its significance.**

Answer given in the previous question.

Reasoning question **Describe the vector analysis of ECG. Write its significance.**

Cardiac vector is an arrow that represents algebraic sum of current flow in heart with head of arrow towards positive direction and length of arrow represents magnitude of potential. Normally during most of ventricular depolarization, the current flow is from base of heart towards apex, with apex positive and base negative, so the resultant mean QRS vector is +59°.

Hexagonal reference system/Hexaxial reference system:

Axis is the direction from negative electrode to positive electrode. Axis of three bipolar and three augmented limb leads are: Lead I: Zero degree; Lead II: +60 degree; Lead III: +120°; aVR: +210°; aVF: +90°; aVL: –30°.

Vector analysis of normal ECG:

- **P wave: Atrial depolarization wave**—during atrial depolarization the impulse begins in SA node and spreads to entire atrial muscles and resultant vector is along the axes of all three standard limb leads. Therefore we record a positive P wave in lead I, II, III, aVL, aVF, V_4, V_5 and V_6, negative P wave in aVR and biphasic P wave in V_1 and V_2.
- **QRS complex: Ventricular depolarization**—during ventricular depolarization, the first part to become depolarized is left endocardial surface of septum which cause vector towards right side which explains small negative q wave in V_4, V_5, V_6 and small positive r wave in V_1 and V_2. Depolarization then involves both sides endocardial septum then enters to endocardial surface of ventricles and current flow towards outside to ventricular muscle. During this

current flow usually from base to apex which explains large positive R wave in V_5, V_6 and large negative S wave in V_1 and V_2. The last part of ventricles to get depolarized is basal part of ventricle in which vector is towards base from apex. This explains small negative S wave in V_5.

- **T wave: Ventricular repolarization**—usually ventricle start repolarizing 0.15 seconds after it depolarizes and completes by 0.35 seconds. Usually the apex of ventricles starts repolarizing earlier than interventricular septum, this cause the vector of repolarization in same direction as depolarization. This leads to positive T wave in V_2, V_3, V_4, V_5, V_6, lead I, II, whereas negative T wave is recorded in V_1 and aVR.

Determining axis from standard lead ECG:

- Calculate voltage of QRS in lead I by subtracting voltage of positive wave with negative wave. Plot the voltage on lead I axis.
- Calculate voltage of QRS in lead III by subtracting voltage of positive wave with negative wave. Plot the voltage on lead III axis.
- Draw perpendicular lines from both lead I and III till they intersect
- Draw a line from center to the point of intersect which shows the vector of lead II

Normal cardiac vector is –30° to +90°.

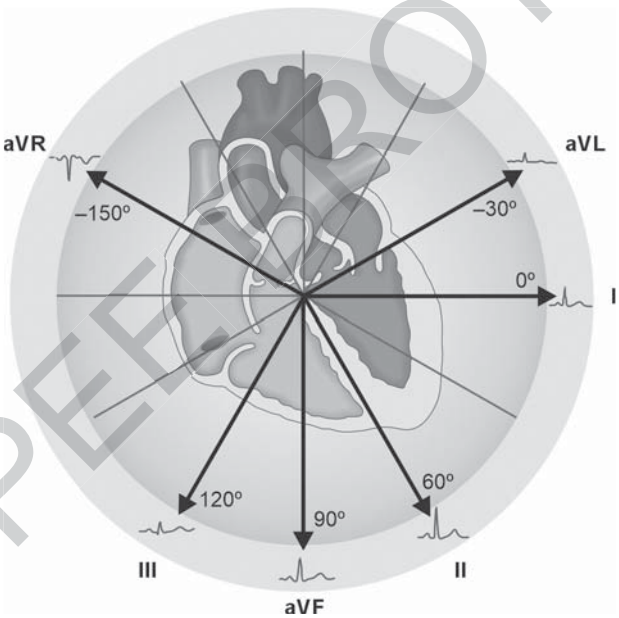


Fig. 5.17: Vectorial analysis of various ECG leads.

- –30° to +30° is normal left axis deviation which represents horizontal position of heart
- +30° to +75° deviation shows oblique position of heart
- +75° to +90° deviation shows vertical position of heart
- Any axis deviation of less than –30 represents abnormal left axis deviation of heart seen in left ventricular hypertrophy as seen in aortic stenosis, aortic regurgitation.
- Any axis deviation of more than +90° represent abnormal right axis deviation of heart seen in right ventricular hypertrophy as seen in interventricular septal defect, Fallot's tetralogy.

PY5.6 Describe physiological variations in ECG waveforms, abnormal waveforms and intervals, arrhythmias, heart block and myocardial infarction. Explained in applied section.

PY5.7 Describe and discuss hemodynamic of circulatory system.

Reasoning question **Describe application of Poiseuille's law in hemodynamics?**

Poiseuille's law:

$$F = \frac{\Delta P \pi r^4}{8 \eta L}$$

It expresses that:

- Flow of blood through blood vessel is directly proportional to fourth the power of radius of vessel
- Flow of blood through blood vessel is directly proportional to pressure gradient at the end of vessel
- Flow of blood through blood vessel is inversely proportional to viscosity of blood
- Flow of blood through blood vessel is inversely proportional to length of vessel

Reasoning question **Why capillaries being so thin, does not rupture?**

Capillaries are exchange vessels are thin and delicate. They have less diameter of around 5–9 μ. They are made of layer of endothelial cells covered by basement membrane.

As per Laplace's law $p = 2T/r$

As radius of capillaries is small, to maintain pressure tension developed in capillary wall is less which is around 16 dynes/cm when compared to aorta which is around 1,70,000 dynes/cm. Due to this capillaries being so thin and delicated does not rupture.

Reasoning question **How blood flow is maintained even during diastole?**

Windkessel is a German word meaning elastic reservoir. This effect is seen in big arteries like aorta. During diastole, the aorta recoils and potential energy help in pushes blood even during diastole creating DBP of around 80 mm Hg called as windkessel effect. This plays important role in maintaining continuous blood flow even during diastole.

PY5.8 Describe and discuss local and systemic cardiovascular regulatory mechanisms.

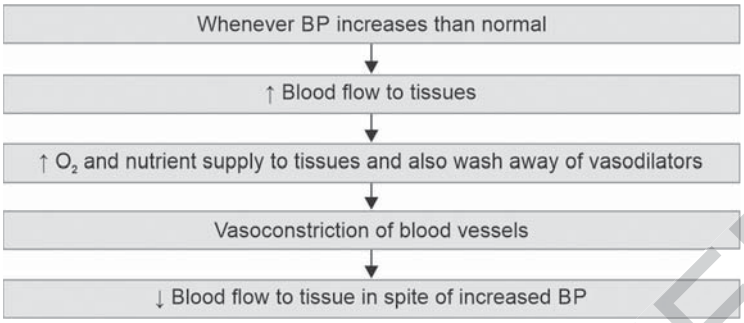
Reasoning question **How is the blood flow to heart is maintained constant in spite of variation in blood pressure? Explain various theories of it.**

In some tissues like kidney and heart, in spite of wide variation of BP between 75 and 170 mm Hg the blood flow to tissue will remain constant which is known as autoregulation of tissue blood flow.

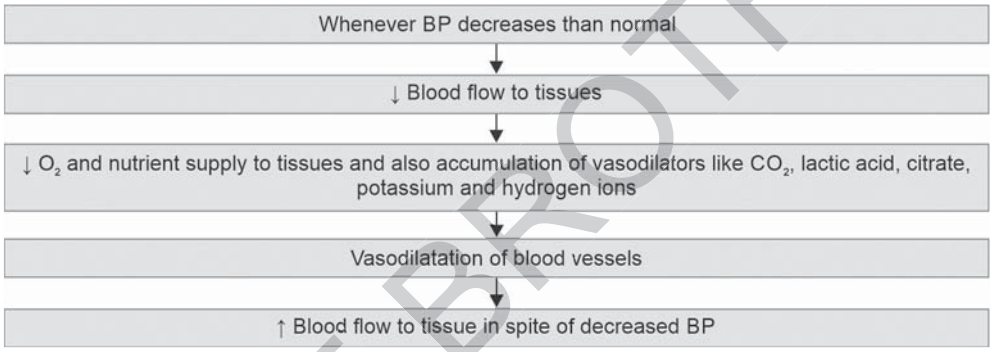
The mechanisms responsible for tissue autoregulation are:

- Metabolic theory
- Myogenic theory

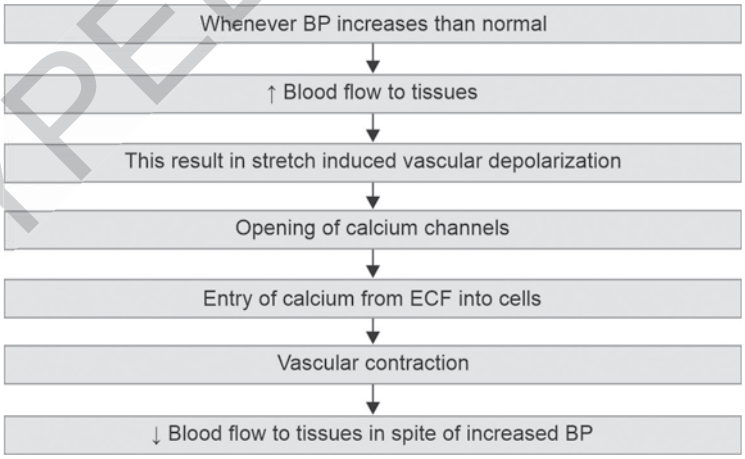
Metabolic theory of autoregulation whenever BP increases:



Metabolic theory of autoregulation whenever BP decreases:



Myogenic theory of autoregulation:



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- For each competency, different types of frequently asked structured long essay questions along with answers are given.
- For each competency, different types of frequently asked short essay questions along with answers are given.
- For each competency, different types of frequently asked short answer questions along with answers are given.
- Reasoning questions with answers are added.
- Integration modules are discussed.
- Answers are mainly in the form of key points, flowcharts, diagrams, illustrations, schematic diagrams which will help students in better understanding and remembering.

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