Agam

Student Notes

PHYSIOLOGY

1st Edition

Unit 4

Cardio Vascular System

CONTENTS

ESSAY:

SHORT NOTES:

CARDIAC CYCLE

DEFINITION:

Cardiac cycle is defined as the sequence of coordinated events taking place in the heart during each beat. Duration of cardiac cycle is 0.8 seconds.

EVENTS OF CARDIAC CYCLE:

Two events in cardiac cycle

1. Atrial events
2. Ventricular events

ATRIAL EVENTS:

Atrial events are divided into two divisions

1. Atrial systole (0.1s)
2. Atrial diastole (0.7s)

Atrial systole:

- Atria contracts and small amount of blood enters the ventricles.
- This period coincides with **last rapid filling phase of ventricular diastole**
- Atrial systole causes increase in intra-atrial pressure by 4-6 mmHg in right atrium & 7-8 mmHg in left atrium. This pressure rise in right atrium is reflected into the veins and is recorded as **a-wave** from the **jugular vein**
- Contraction of atrial musculature causes the production of fourth heart sound.

Atrial diastole:

- After atrial systole, atrial diastole starts.
- This period coincides with **ventricular systole and most of the ventricular diastole**
- Atrial filling takes place.

VENTRICULAR EVENTS:

Ventricular events are divided into two divisions

1. Ventricular systole (0.3s)
2. Ventricular diastole (0.5s)

The ventricular systole is divided into two subdivisions:

- isometric contraction period
- ejection period.

The ventricular diastole is subdivided into five subdivisions:

- protodiastole
- isometric relaxation
- rapid filling
- slow filling
- last rapid filling (coincides with atrial systole)

Isovolumic (Isometric) contraction period:

- It is characterized by increase in tension without any change in the length of muscle fiber.
- With the beginning of ventricular contraction, the ventricular pressure exceed the atrial pressure very rapidly causing the closure of AV valves
- Closure of AV valves at the beginning of this phase produces first heart sound.
- Since the AV valves have closed and the semilunar valves have not opened, the ventricles contract as a **closed chamber** & pressure inside ventricles rises rapidly to a high level
- Despite the ventricular contraction, the volume of blood in ventricles does not change. Thus, this called as ***isovolumic contraction***
- Rise in ventricular pressure causes opening of semilunar valves.

Ejection period:

- After the opening of semilunar valves, blood is ejected out of the ventricles.
- Ventricles are not completely emptied at the end of ejection period. The amount of blood remaining in ventricles at the end of ejection period is called end systolic volume (ESV). It is 60-80 ml per ventricle.
- Ejection fraction: fraction of end diastolic volume that is ejected out by each ventricle per beat.
- Ejection period is of two phases:
    1. rapid ejection period: during this phase large amount of blood (about $2/3^{rd}$ of stroke volume) is rapidly ejected from both ventricles.
    2. Slow ejection period: the blood is ejected slowly with much less force.

Protodiastole:

- Interventricular pressure becomes less than the pressure in the aorta and pulmonary artery.
- Semilunar valves close which produces second heart sound.

Isovolumic (Isometric) relaxation period:

- It is characterized by decrease in tension without any change in the length of muscle fiber.
- Since semilunar valves have closed & AV valves have not opened yet, the ventricles continue to relax as closed chambers in this phase. The volume of blood in ventricles remains constant, hence it is called the ***isovolumic relaxation phase.***
- Ventricles undergo isometric relaxation and pressure in the ventricles falls than the atrial pressure.

- Thus, atrioventricular valves open at the end of this phase.

Rapid filling phase:

- As atrioventricular valves are opened there is sudden rush of blood into ventricles.
- 70% ventricular filling takes place during this phase.
- Rushing of blood into ventricles produces third heart sound which is not commonly audible in adults but may be heard in children.

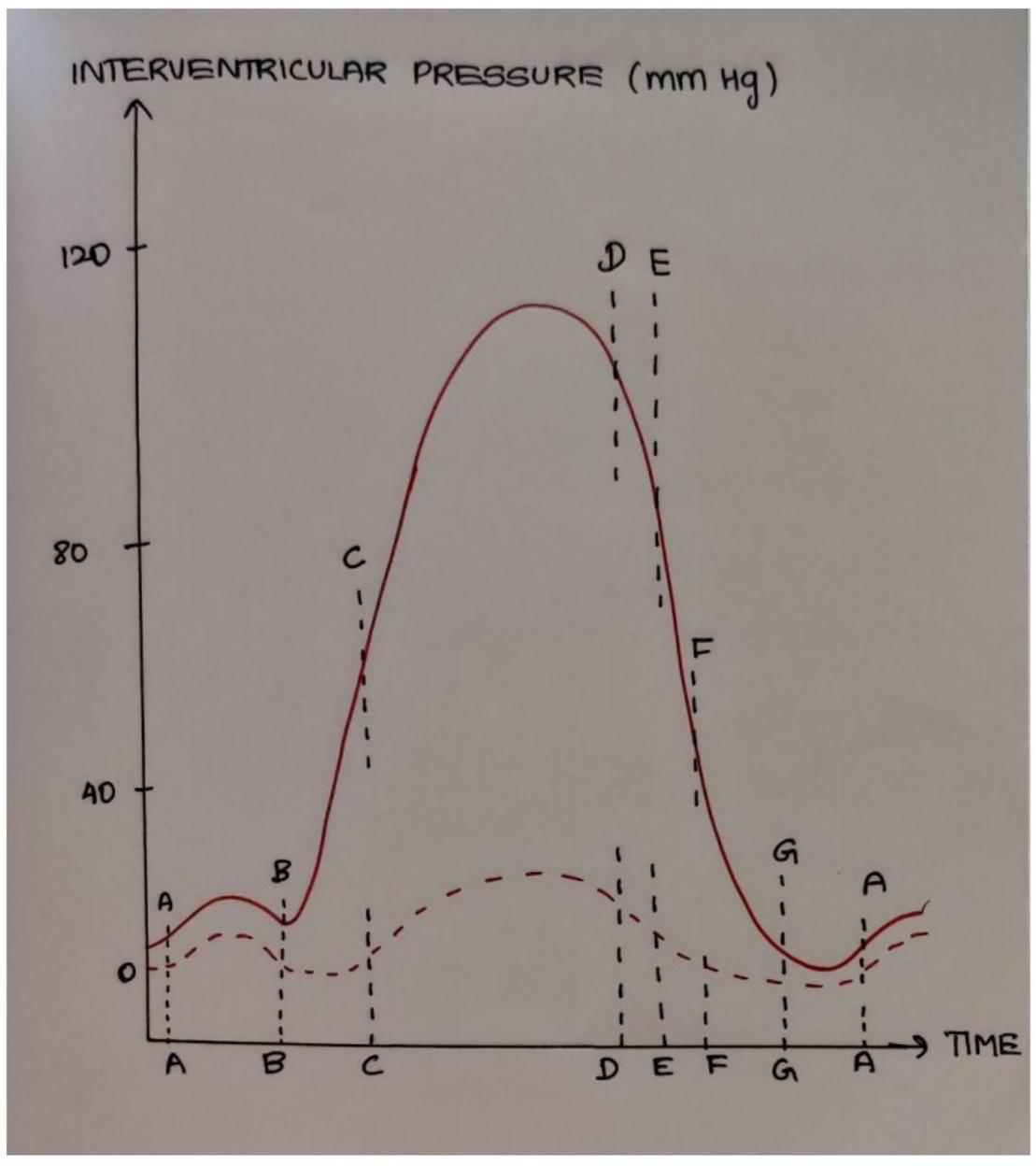
Slow filling phase:

- Also known as diastasis.
- 20% filling occurs during this period.

Last rapid filling phase:

- This period coincides with **atrial systole**.
- 10% of ventricular filling takes place.
- End diastolic volume: amount of blood remaining in the ventricles after the end of diastole. It is 130-150 ml per ventricle.

PRESSURE CHANGES IN CARDIAC CYCLE:



A-B Segment:

- Appears during atrial systole.

- Pressure increases during this period.
- B denotes closure of atrioventricular valves.

B-C Segment:

- Appears during isometric contraction period.
- Interventricular pressure increases.
- C denotes opening of semilunar valves.

C-D Segment:

- Appears during ejection period.
- Because of the thick wall of left ventricle, the pressure rise in left ventricle is about 4-5 times higher than the pressure rise in right ventricle.

D-E Segment:

- Appears during protodiastole.
- Interventricular pressure decreases.
- E denotes the closure of semilunar valves.

E-F Segment:

- Appears during isometric relaxation period.
- Interventricular pressure decreases.
- F denotes the opening of atrioventricular valves.

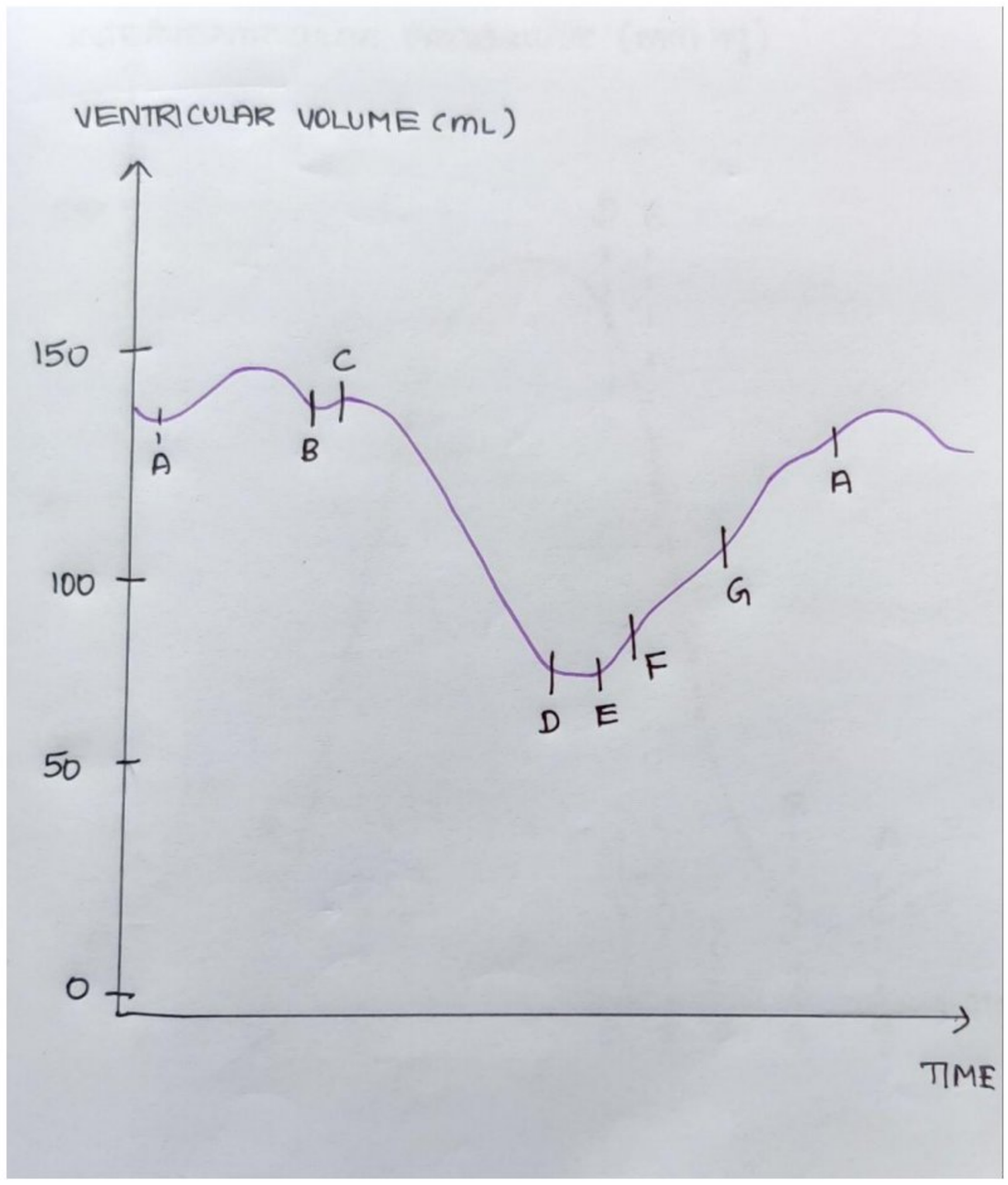
F-G Segment:

- Appears during rapid filling phase.
- Pressure decreases.

G-A Segment:

- Appears during slow filling phase.
- Pressure decreases further.

VOLUME CHANGES IN CARDIAC CYCLE:



A-B Segment:

- Appears during atrial systole.
- Ventricular volume increases slightly.
- B denotes closure of atrioventricular valves.

B-C Segment:

- Appears during isometric contraction period.
- The ventricular volume is not altered during this period. However, the slight upward deflection seen is an artifact.
- C denotes the opening of semilunar valves.

C-D Segment:

- Appears during ejection period.
- Ventricular volume decreases.

D-E Segment:

- Appears during protodiastole period.
- There is no change in the ventricular volume.
- E denotes closure of semilunar valves.

E-F Segment:

- Appears during isometric relaxation period.
- Volume remains unaltered but the slight upward deflection in the curve is due to artifact.
- F denotes the opening of atrioventricular valves.

F-G Segment:

- Appears during rapid filling phase.
- There is rise in the ventricular volume.

G-A Segment:

- Appears during slow filling phase.
- Ventricular volume increases.

CLINICAL APPLICATIONS:

1.Myocardial infarction:

- Necrosis of myocardium causes by insufficient blood flow due to thrombus, embolus or vascular spasm.
- Ejection fraction is decreased in myocardial infarction.

2.Left ventricular hypertrophy:

- Enlargement and thickening of left ventricle.

ARTERIAL BLOOD PRESSURE

DEFINITION:

- The lateral pressure exerted by the flowing blood against any unit area of the arterial vessel wall.
- Measured in mm Hg.
- The maximum arterial pressure during the systole is called Systolic BP.
- The minimum arterial pressure during diastole is called Diastolic BP.

FACTORS AFFECTING BP:

1. Age: Systolic and diastolic BP both increase with age but after 50-60yr due to poor distensibility of an artery, only systolic BP increases.

2. Gender: Before menopause Males have higher BP after menopause, Systolic BP increases 4-5mmhg higher in Females than in Males.

3. Built of a person: An obese individual has high reading because of increased tissue between the cuff and artery.

4. Climate: Exposure to cold causes vasoconstriction which increases BP.

5.Diurnal variation: Diurnal variation of 5-10mmhg common in Systolic BP.

6. Exercise: BP comes back to normal after 5min of exercise.

7. Emotions: Excitement, and Fear increase systolic BP.

8. Hereditary

9. Sleep: Systolic BP falls during sleep.

10. Posture: Diastolic BP changes with posture (sudden standing increases Diastolic BP).

FACTORS DETERMINING BP:

1. Cardiac output: CO=HR X stroke volume. Systolic BP depends on stroke volume

2. Peripheral resistance: Diastolic bp depends on this.

It varies according to:-

a)*Viscosity of blood:* BP is directly proportional to the viscosity of blood.
b)*Velocity of blood flow:* When a vessel is narrowed. velocity increases and distending pressure of the blood vessel decreases(according to the Bernoulli principle).

c)*Radius of the vessel:* Peripheral resistance is inversely proportional to the fourth power of the radius.
d)*Elasticity of vessel:* BP is inversely related to the elasticity of the vessel.

REGULATION:

1. SHORT TERM REGULATION(NERVOUS SYSTEM CONTROL):

a) Baroreceptors

b) Chemoreceptors

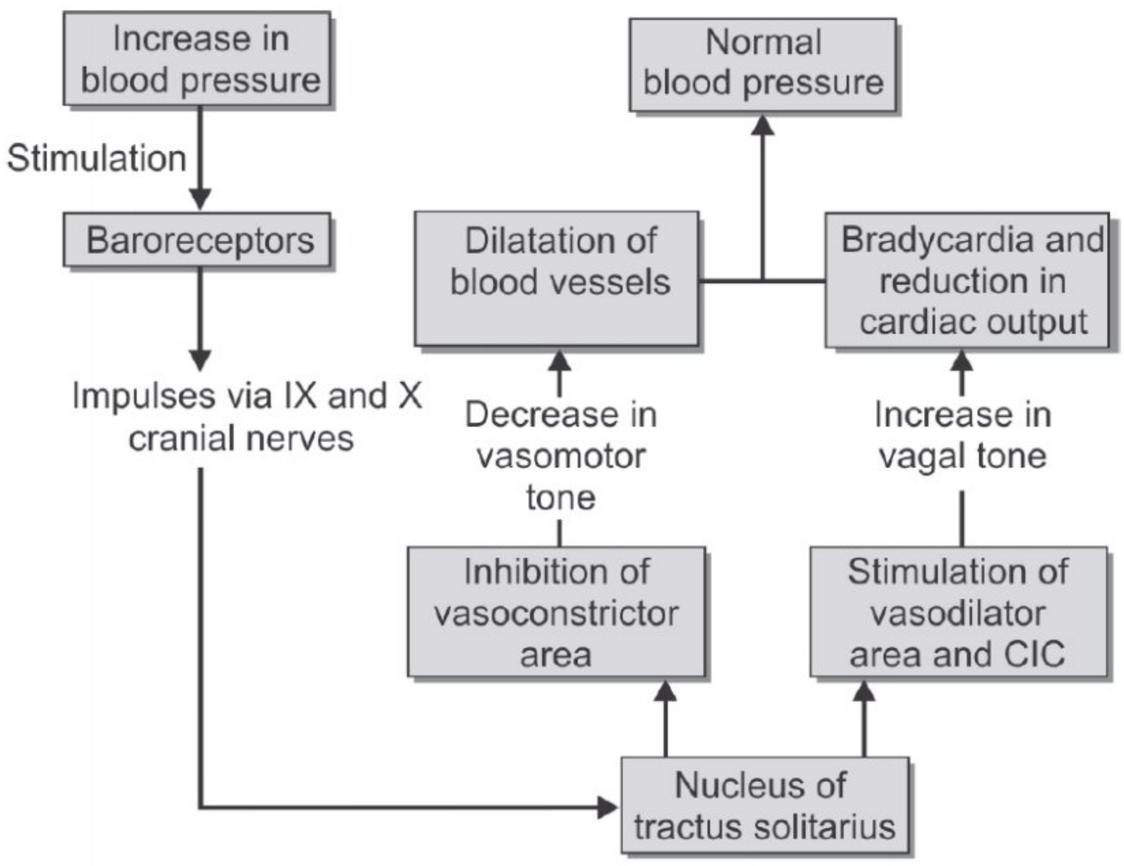
c)CNS ischemic response (Cushing reflex)

A) BARORECEPTORS (pressure buffer system):

TABLE 43-1 Working of the Baroreflex

Components of a Typical Reflex	Components of the Baroreflex
Stimulus	Increased arterial blood pressure
Receptor	Baroreceptors: carotid sinus, aortic arch baroreceptors
Afferent nerve	Hering nerve (IX cranial nerve) from carotid sinus Depressor nerve (X cranial nerve) from arch of the aorta
Integrating center	Nucleus tractus solitarius—vasomotor center
Efferent nerve	Vagus: increased activity Sympathetic: decreased activity
Effector organs	Heart, blood vessels
Physiological effects	Decreased heart rate, decreased cardiac contractility Vasodilation resulting in decreased cardiac output and decreased resistance

Flow chart 4.1: Regulation of blood pressure by baroreceptor mechanism



- Small fluctuations in mean BP activate baroreceptors.
- Important for maintaining postural change in BP.
- Baroreceptors are reset in chronic hypertension(they don't get stimulated even if BP is high).

B)CHEMORECEPTOR:

Receptor: Carotid and Aortic body.

Stimulus: Oxygen, ph of blood, CO_2.

- These are stimulated when mean BP falls below 80 mm Hg.
- Increased accumulation of H^+ due to decreased oxygen stimulates the chemoreceptors and increases BP.

C)CNS ISCHEMIC RESPONSE:

- Activated when mean BP<40 mmHg.
- When intracranial pressure is increased, the blood supply to the vasomotor area is decreased. This causes local hypoxia and hypercapnia. The vasomotor area is excited to increase BP which can increase blood supply to the vasomotor area.
- The increased BP decrease the heart rate(the only exception is when BP increases heart rate increases).

2. LONG TERM REGULATION OF BP:

A) RENIN-ANGIOTENSIN SYSTEM:

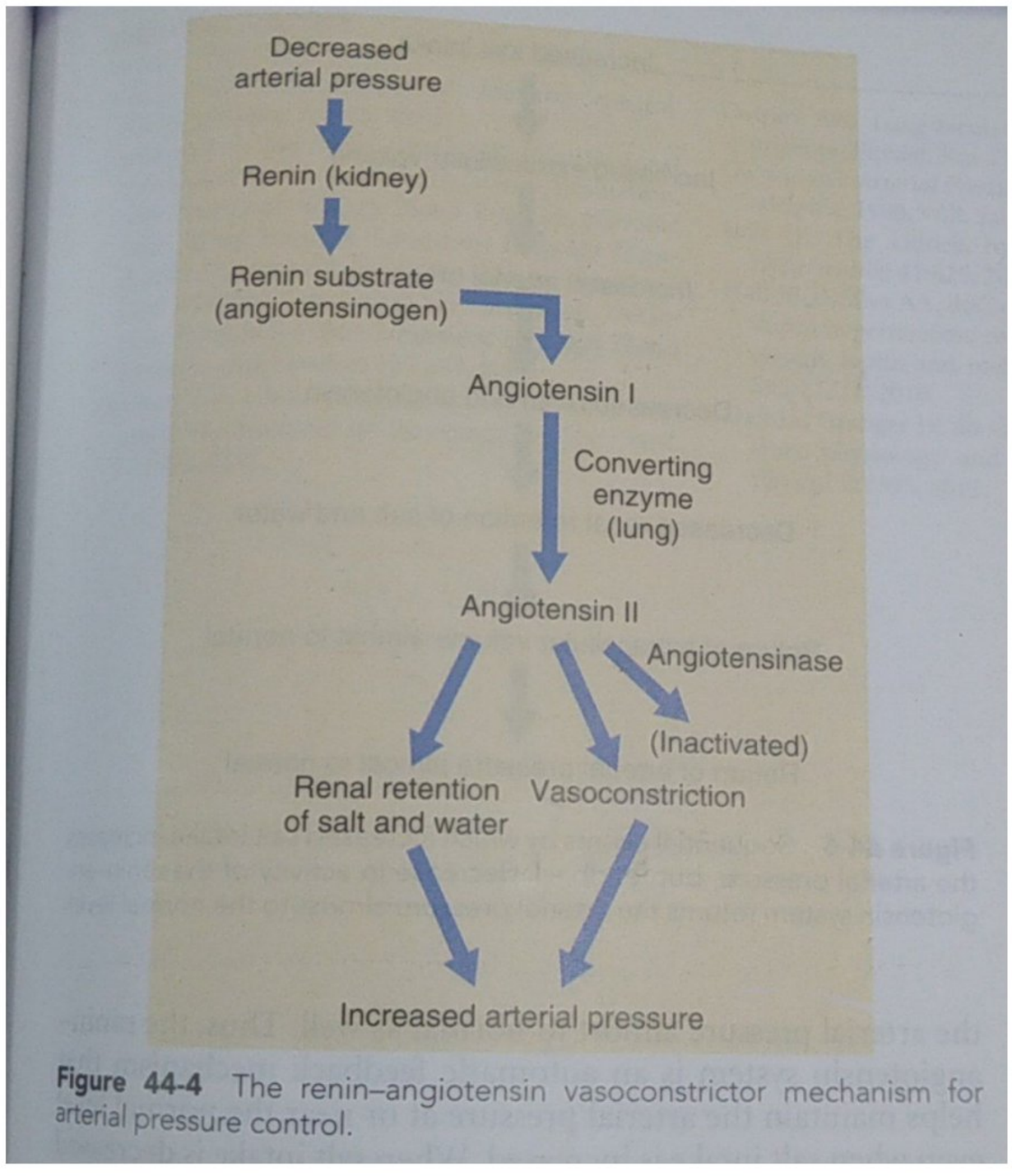


Figure 44-4 The renin–angiotensin vasoconstrictor mechanism for arterial pressure control.

The function of Angiotensin II:

- Cause sodium and water retention by acting directly on the kidney.
- Release of aldosterone from adrenal glands causes salt and water reabsorption from the kidney.

B)HORMONAL:

Hormones that help in the long term regulation of BP other than angiotensin are:

- Aldosterone= Causes an increase in BP.
- ADH =Increases BP.
- Catecholamines=epinephrine, norepinephrine.
- Atrial natriuretic peptide =Causes vasodilation and decreases BP

3.OTHER MECHANISMS FOR REGULATION OF BP ARE:

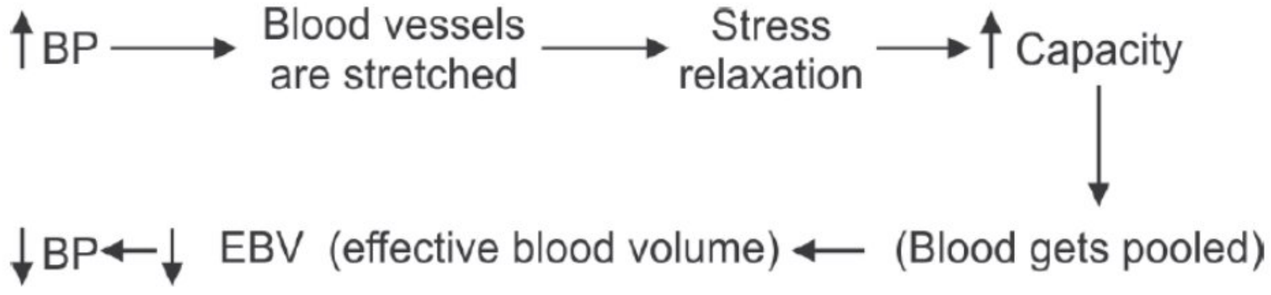
A)Capillary fluid shift:

When increase in BP more fluid is filtered through the capillary wall into the interstitial space. So blood volume decreases, resulting in decreased BP. Reverse changes take place when BP falls.

B)Stress relaxation of blood vessels:

Stress Relaxation of Vasculature

Whenever the smooth muscle is stretched for prolonged time it goes into relaxation.



Reverse changes takes place when BP falls.

ELECTROCARDIOGRAM

It is defined as the summated electrical activity of the heart fibres developed during each heart beat recorded from the surface of the body.

Electrocardiography:

- It is the technique by which an electrocardiogram (ECG) is recorded and analysed .

Electrocardiograph :

- It is the instrument used for recording ECG.

Normal ECG:

- Each beat of the heart will result in a complex of waves called **PQRST** complex.
- The baseline is called isoelectric or iso potential line.

Positive waves	Negative waves
P,R,T waves	Q,S waves
Occasionally	
(U wave)	

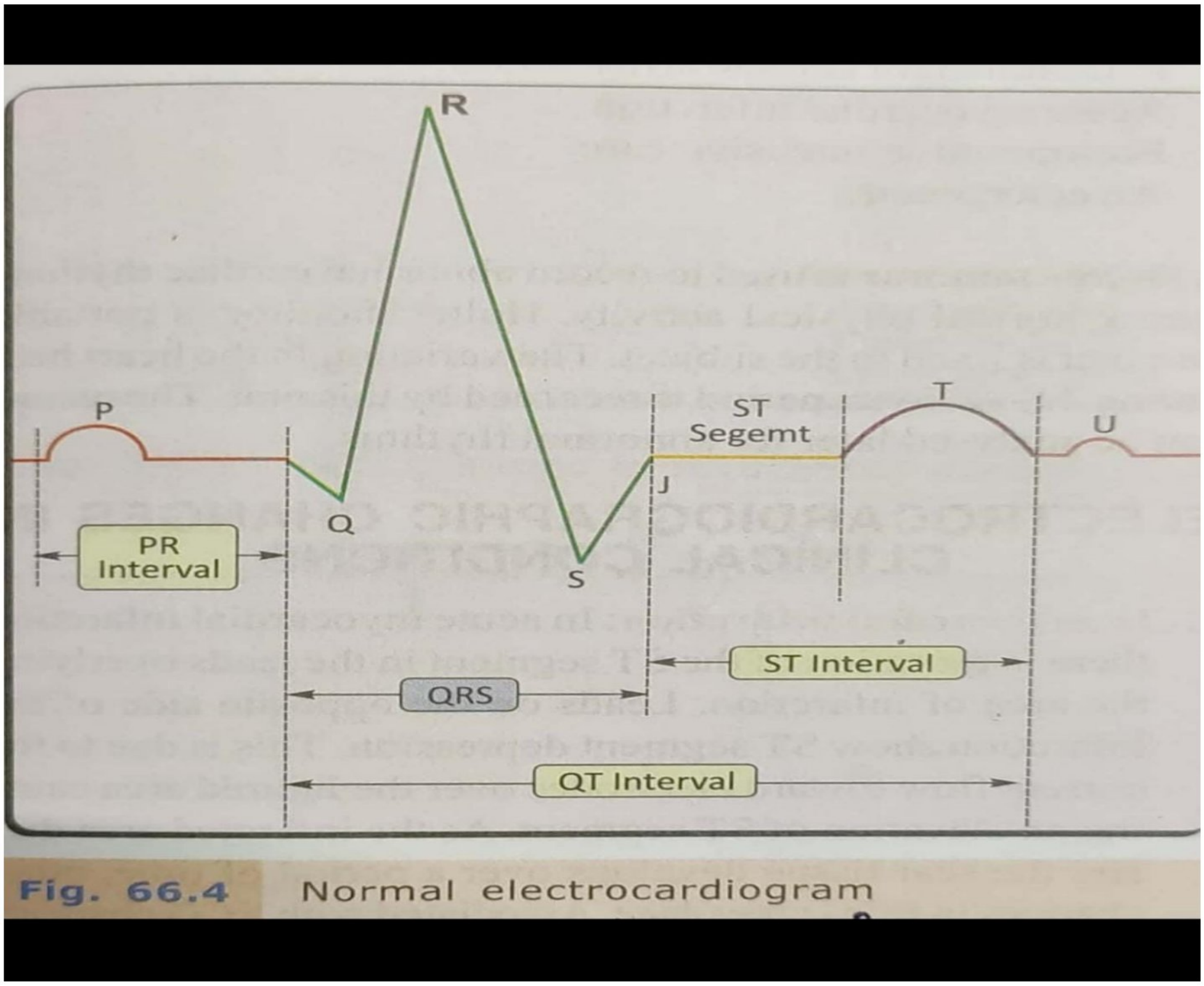


Fig. 66.4 Normal electrocardiogram

1. P-Wave:

- It represents atrial depolarisation.
- Normal duration is 0.08-0.12 sec.
- Normal voltage is 0.1-0.3 mV P.
- Wave represents normalcy of the atrial myocardium.

It under- goes changes with the diseases of the atria.

a)Atrial hypertrophy-'P'wave becomes larger or bifid.

b) Atrial flutter-Abnormal 'P' waves called 'F' waves.

c) Atrial fibrillation-Abnormal 'P' waves called 'f'waves.

d) Atrial ectopic (or) Atrial tachycardia-Inverted 'P' wave.

2. QRS complex:

- This represents ventricular depolarisation, i.e, the normalcy and integrity of the ventricular myocardium.
- Normal duration is 0.05-0.12 sec.
- Normal voltage 1-3 mV.
- ECG from the left ventricle will have more amplitude than that from the right ventricular ECG

3.Q wave:

- This is a small negative wave.
- Represents the depolarization of the mid portion of the interventricular septum.
- This is absent in right ventricular leads.
- This is present in all the bipolar leads as both electrodes are active.

4. 'R' wave:

- It is a prominent positive wave.
- Amptitude may reach upto 25 mV.
- It is due to the potential travelling to the apex of the ventricle and activating major portion of both the ventricles.
- Peak of 'R' wave is the safest period as the ventricle is in absolute refractory period at this time and so does not respond to any external stimulus. Hence cardio version is done by applying DC shock during this period.

5. S wave:

- It is a negative wave and is due to the potential travelling to the base of the ventricles.
- RS complex is prominent in right ventricular leads.

Abnormalities:

a) Ventricular hypertrophy-QRS amplitude increases.

b) Myocardial infarction

c) Complete AV block.

d) Bundle branch block

e) Ventricular ectopics

f) Constrictive pericarditis

6. T wave:

- It is a rounded positive wave and represents ventricular repolarization.
- Normal duration is 0.27 sec.

- Volt age varies from 0.2 to 0.5 mV. It is normally a +ve wave except in LIII, a VR and V, where it is -ve.
- Peak of T wave is the vulnerable period or dangerous period as any stimulus during this period would cause arrhythmias. During this period ventricular repolarization is in different stages.

7. U wave: It is a small positive wave caused by delayed repolarization of the papillary muscle, mostly seen in V2 to V4 leads.

Intervals:

8. PR interval:

- It refers to the time interval between the beginning of 'P' wave to the beginning of Q' or 'R' wave.
- This represents AV conduction time, (time taken by the impulse to travel from SA node to the ventricle).
- Normal duration is 0.12-0.21 sec.
- It is inversely proportional to the heart rate.
- **PR interval is prolonged in:**
  a) 1°H block, e.g., digitalis toxicity. Simple prolongation of PR interval.
  b) 2° H block, e.g., Wenkebach's phenomenon.
  In both these conditions, there is a partial AV block.
- In the Wenkebach phenomenon, the PR interval increases with the successive beats and at one stage only P wave is obtained without QRST. It is followed by an other normal PQRST complex.

- **PR interval is shortened in:**

  a)Wolf-Parkinson-White syndrome (WPW syndrome)

  b) Lown-Ganong-Levine syndrome (LGL syndrome)

9. ST interval:

- Denotes the interval between the end of 'S' to the end of 'T' wave;
- Normal up to 0.32 sec.
- It represents the time taken for ventricular repolarization.

10. QT interval:

- Denotes the interval between the beginning of Q wave to the end of T wave.
- The normal is 0.4 sec.
- It denotes the time taken by ventricular depolarization and repolarization. Normally, the beginning of ventricular systole coincides with the summit of R wave and ends with the completion of T wave.

- QT is shortened in hypercalcaemia and QT prolonged in hypocalcaemia.
- **RT interval** denotes the duration of ventricular systole.

Segements

11. PR segment:

The region between the end of P wave and the beginning of 'Q' wave. This denotes the time interval between completion of atrial depolarization and beginning of ventricular depolarization.

12. J point:

It indicates the end of 'S' wave. It is useful in assessing 'S,T' elevation or depression.

CARDIAC OUTPUT

DEFINITION:

- The amount of blood ejected by each ventricle per min
- CO=Stroke volume×Heart rate

FACTORS AFFECTING CARDIAC OUTPUT:

Factors that affect stroke volume and heart rate affect cardiac output.

Factors affecting Stroke:

1. Preload-end diastolic volume.
2. Myocardial contractility.
3. Afterload-peripheral resistance.

1)PRELOAD:

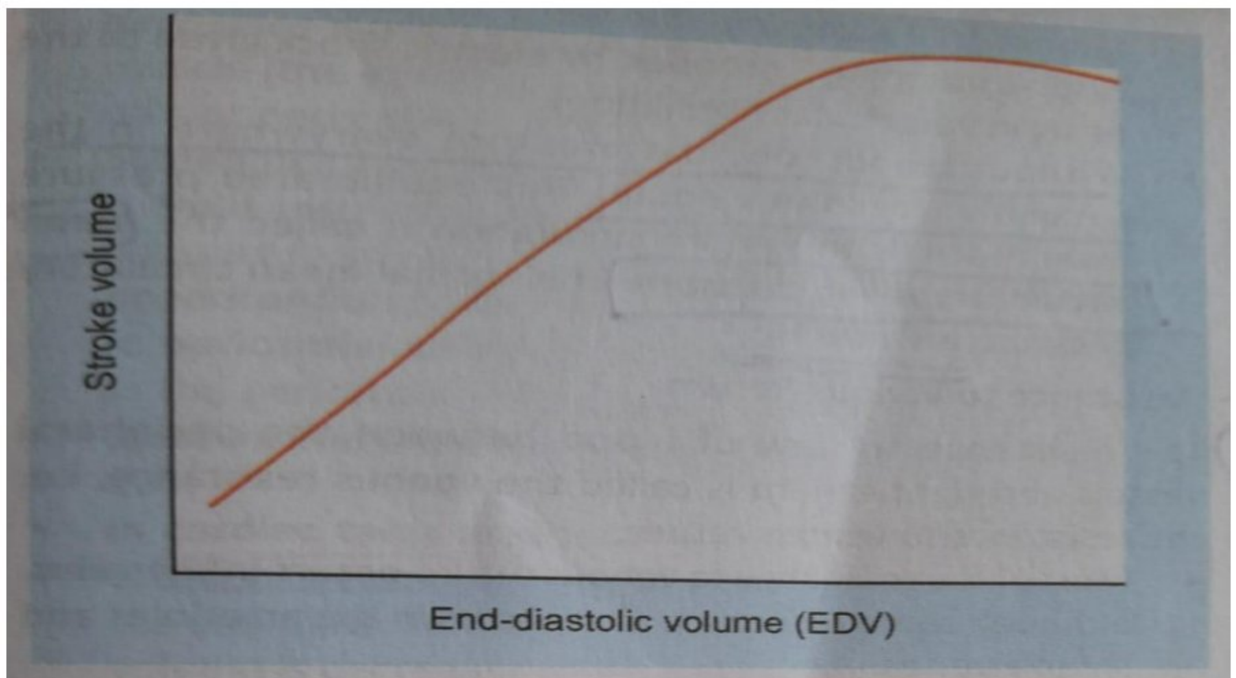


Fig. 48.5: Ventricular function curve (Starling curve). Note, stoke volume increases with increased EDV. EDV represents end-diastolic pressure, which indicates the end-diastolic fiber length. The increase in fiber length increases stroke volume almost linearly till the physiological limit is reached.

FRANK -STARLING LAW:

"Within the physiological limit, the force of contraction is directly proportional to the initial length of muscle Fibre, Increased ventricular filling Increases the Fibre length Increases the force of contraction."

Increased ventricular filling

⇩

Increases the Fibre length

⇩

Increases the force of contraction

Factors affecting preload:

1)Venous return.

2)Atrial pump activity.

3)Ventricular compliance

1)Venous return:

Defined as the amount of blood that returns to the right atrium from systemic venous circulation

-> Depends on

a)Skeletal muscle pump in lower limbs:

Veins are surrounded by skeletal muscle. Contraction of skeletal muscle during walking compresses veins and pushes blood towards the heart.

b)Thoracic pump:

Inspiration

⇩

Intrathoracic pressure becomes more negative. Negative pressure

⇩

Transmitted to the great veins

⇩

Central venous pressure falls

⇩

Facilitates venous return.

c)Abdominal pump:

Inspiration

⇩

Descent of diaphragm Increased abdominal pressure

⇩

Compresses the intra abdominal blood vessel

⇩

Blood is pushed towards the heart Venous return increases

(Blood is not pushed towards leg veins-because of valves)

d)ECF volume:

A- Diarrhea,vomiting

⇩

Decreases ecf volume

⇩

Decreases cardiac output

B-pregnanacy

⇩

Increased ecf volume

⇩

Increases cardiac output

e)Sympathetic activity:

Veins-sympathetic fibres stimulation in exercise

⇩

Vasoconstriction

⇩

Increased venous return

f)Right atrial pressure:

Venous return curve

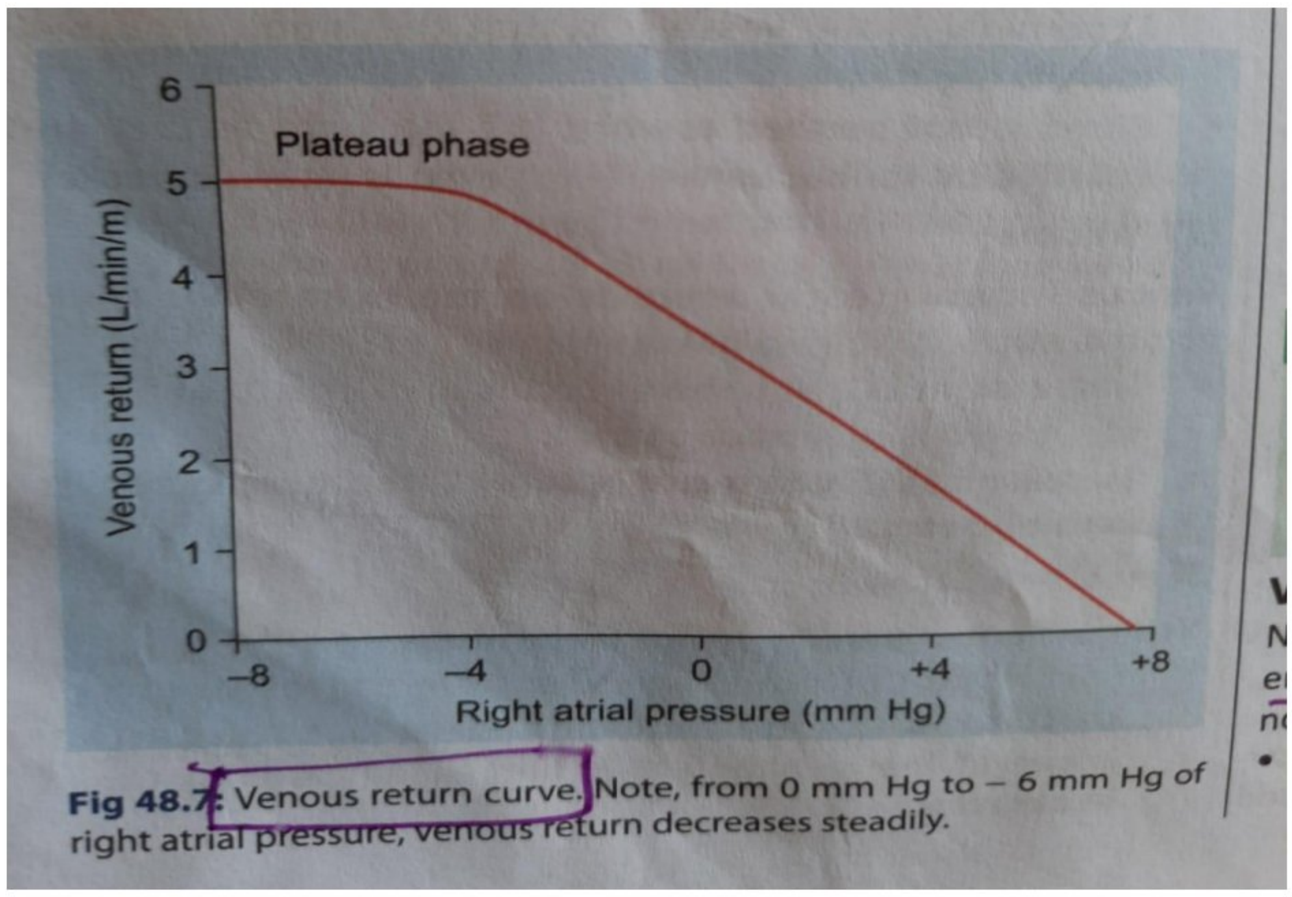


Fig 48.7: Venous return curve. Note, from 0 mm Hg to – 6 mm Hg of right atrial pressure, venous return decreases steadily.

0-6mm hg

⇩

Increase in pressure

⇩

Decreased venous return

[Above 7 mm Hg venous return =0]

- ***<u>When right atrial pressure falls below -2mmHg</u>***

No increase in venous return.

⇩

Plateau phase

⇩

Bc2 of collapse of veins

⇩

negative pressure in right atrium

⇩

Sucks the walls of vein chest Vein collapse

⇩

No increase in venous return.

g)Degree of filling by systemic circulation:

Effective volume of blood-in the entire circulation determines the venous return.

Temporarily stopping the heart to pump blood

⇩

Without blood flow,the pressure everywhere in the circulation becomes equal.

⇩

This equilibrated pressure levels that fills the entire circulation called mean circulatory filling pressure

h)Resistance to venous return:

Venous obstruction

⇩

Venous resistance increases

⇩

Blood begins to be dammed up

⇩

But due to distensible character of the vein ,rise in venous pressure to overcome resistance occurs little

⇩

Hence blood flow into right atrium Decreases. -

⇩

Resistances in arterioles and small arteries Blood accumulates in the arteries

⇩

Arteriole -less capacitance

⇩

Hence rises the pressure gently It overcomes the increased resistance

MOST OF THE RESISTANCE TO VENOUS RETURN OCCUR IN THE VEINS

2)Atrial pump activity:

- Ventricular filling occurs almost passively.
- Only 15-20 percent of ventricular filling at rest occurs due to the atrial pump. Hence normally the atrial pump does not contribute significantly to stroke volume.

3)Ventricular compliance:

- Ventricular muscle is compliant(stretchable).
- In cardiac myopathies, infiltrative disease of the heart

⇩

Decreased compliance of Ventricular muscle

⇩

Ventricular filling Decreases

⇩

CO Decreases.

- **Cardiac tamponade**: Hemopericardium, Massive pericardial effusion. EDV decreased ,CO decreases

2)MYOCARDIAL CONTRACTILITY:

Depends on

- **Ventricular muscle mass -**

Pathology : Muscle atrophy in cardiomyopathy or myocardial infarction.

⇩

Decreases muscle mass

⇩

Decreases cardiac output.

Normal: Athlete- Physiological hypertrophy

⇩

Increases CO mainly by increasing stroke volume, rather than the heart rate.

- **Autonomic activity:**
  Ventricles are supplied by sympathetic fibres.

Sympathetic stimulation

⇩

Increases contractility

Vagal stimulation

⇩

Decreases heart rate Decreases cardiac output.

- **Hormonal Factors:**

Catecholamines

⇩

Increases contractility.

⇩

Acetylcholine Decreases the contractility Glucagon and insulin are inotropic.

⇩

Thyroxine causes tachycardia since it Increases the beta1 receptors on nodal tissues.

- **Chemical factors:**

Digitalis Inhibits Na-K ATPase activity increases intracellular Na Decreases sodium gradient

⇩

Decreases the activity of Na-Ca exchanger Intracellular accumulation of CA
Increased myocardial contractility

3)AFTERLOAD:

- PR is inversely proportional to cardiac output.
- Here CO is altered without a change in Ventricular muscle length-hence chemometric autoregulation.- The arep effect

Factors affecting afterload:

1. **Vessel diameter:**
   - Vasoconstriction
     - Increases peripheral resistance
     - Decreases stroke volume.
2. **Viscosity:**

Polycythemia

-Increased viscosity

-Decreases cardiac output

Anemia

-CO Increases.

FACTORS AFFECTING HEART RATE:

- **Vagal stimulation:** Decreases heart rate

- **Sympathetic stimulation:** Increases heart rate.

Tachycardia

⇩

Diastole duration shortens more than systolic

⇩

EDV Decreases

⇩

CO does not increase proportionately to that of increase in heart rate.

(Because unless associated with Increased venous return, increased heart rate does not increase proportionately.)

REGULATION OF CARDIAC OUTPUT:

1.Intrinsic or auto regulation

a)Frank starling mechanism

b)Rate induced regulation:Increased frequency force of contraction increases.

2.extrinsic regulation:

a)Afterload

b)Neural influences

c)Hormonal influences.

METHODS FOR MEASURING CARDIAC OUTPUT

1.FICK PRINCIPLE

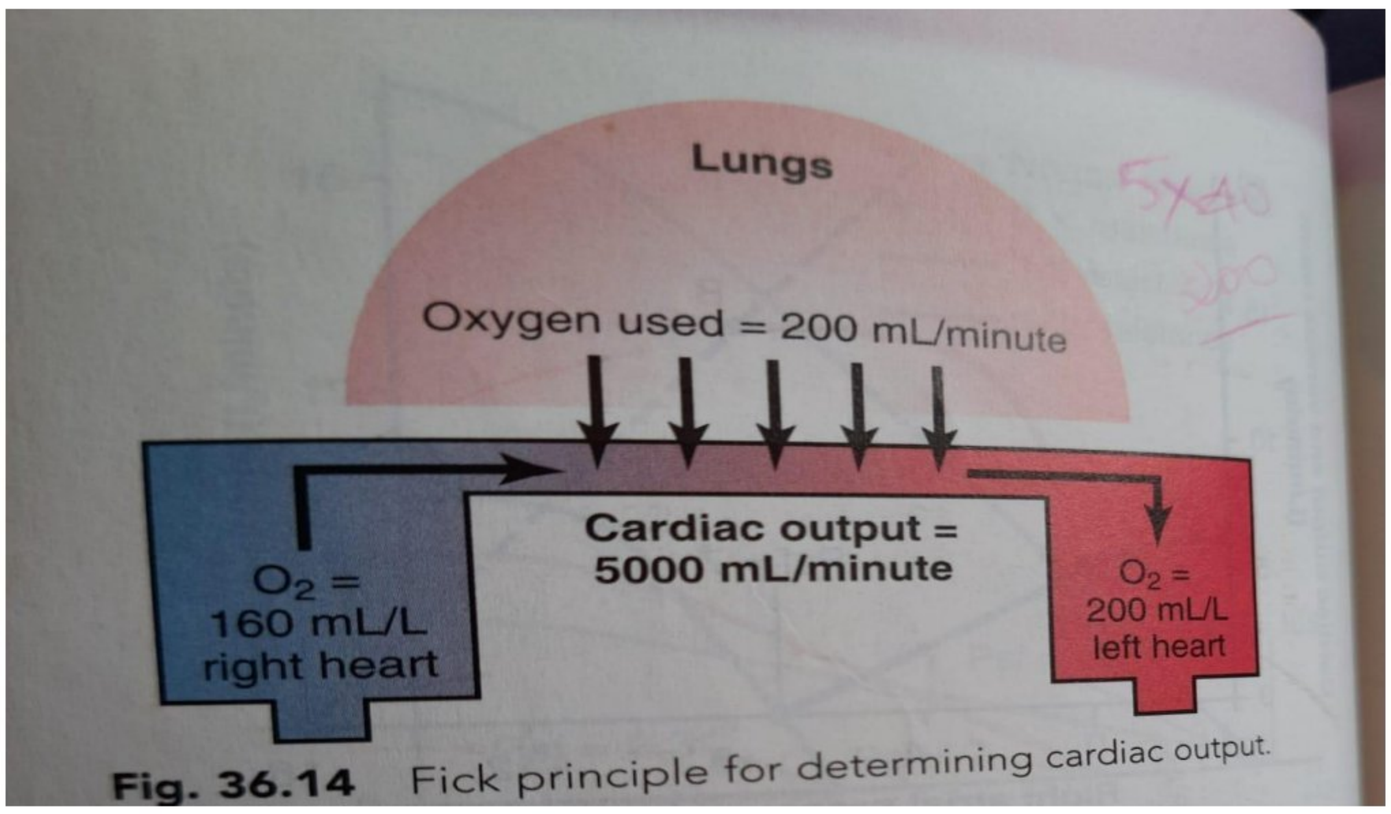


Fig. 36.14 Fick principle for determining cardiac output.

- 200/200-160=5 L
- Hence 5 lit of blood must flow through lungs each min-it is the measure of cardiac output.

CO=O2 absorbed per min(ml/min)÷arteriovenous O2 difference(ml/L).

In humans,

Mixed venous blood obtained through catheter inserted up the brachial vein

Subclavian vein

Pulmonary artery

- systemic arterial blood
- rate of oxygen absorbed by lungs using an oxygen meter.

2. INDICATOR DILUTION METHOD:

Indicator-injected into large systemic vein preferably into right atrium
(5 mg of cardiogreen dye at 0 time)

⇩

Right side of heart

⇩

Blood vessels of lungs

⇩

Left side of heart

⇩

Systemic arterial system.

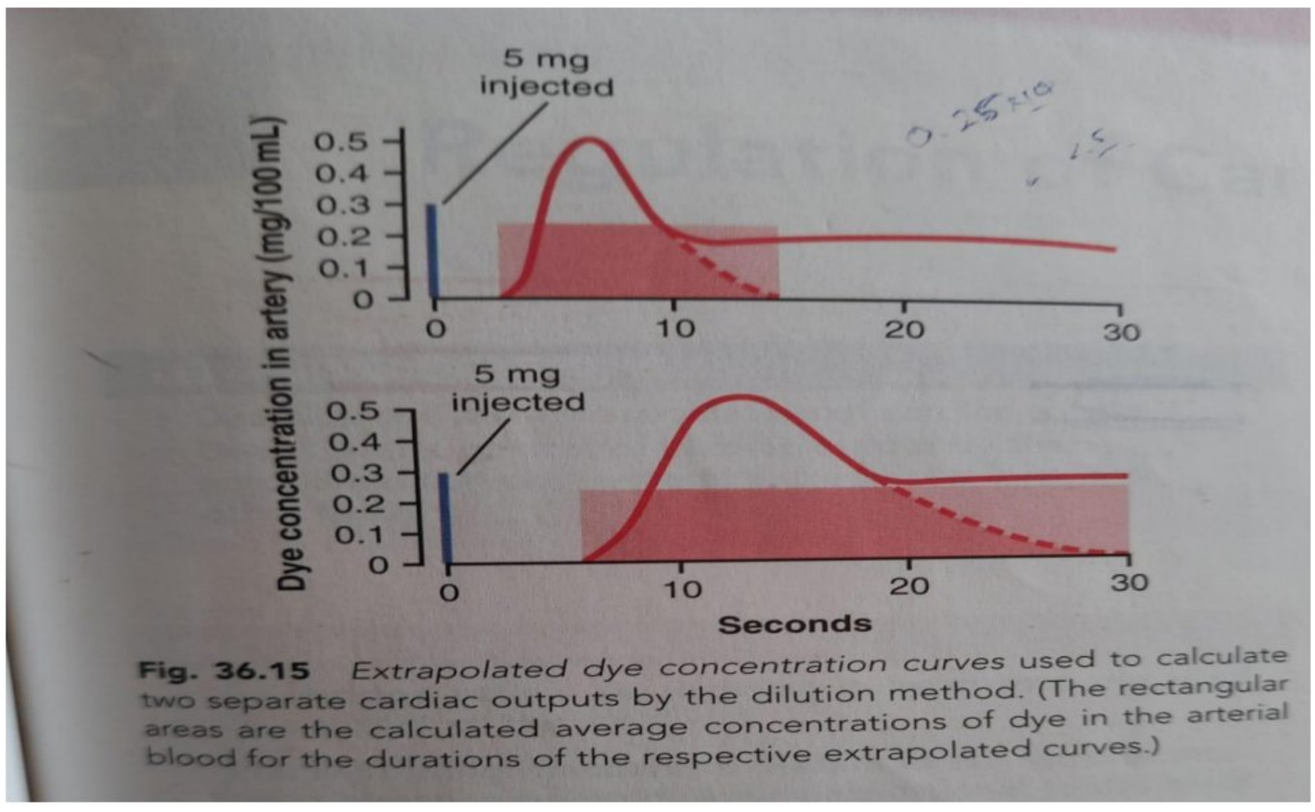


Fig. 36.15 *Extrapolated dye concentration curves* used to calculate two separate cardiac outputs by the dilution method. (The rectangular areas are the calculated average concentrations of dye in the arterial blood for the durations of the respective extrapolated curves.)

-Serial arterial samples were taken.

-None of the dye passed into the atrial tree for about 3 sec.

-In 6-7 sec, the concentration of dye rose rapidly.

-Concentration fell rapidly, but before concentration reached zero, some of the dye already circulated and returned through the heart for a second time.

-To measure CO, the early downslope of the curve is extrapolated to join the x-axis, given the time taken for a single circulation.

-Calculation of the mean concentration of dye in arterial blood for the duration of the curve was done by measuring the area under the initial and extrapolated curve and then averaging the concentration of dye for the duration of the curve.

CO=mg of dye injected ×60÷average concentration of dye in each ml of blood for the duration of curve × duration of curve in sec

3. OTHER METHODS:

- ECHOCARDIOGRAPHY
- THORACIC ELECTRICAL BIOIMPEDANCE METHOD.