CARDIOVASCULAR SYSTEM

DR SARITHA

CVS

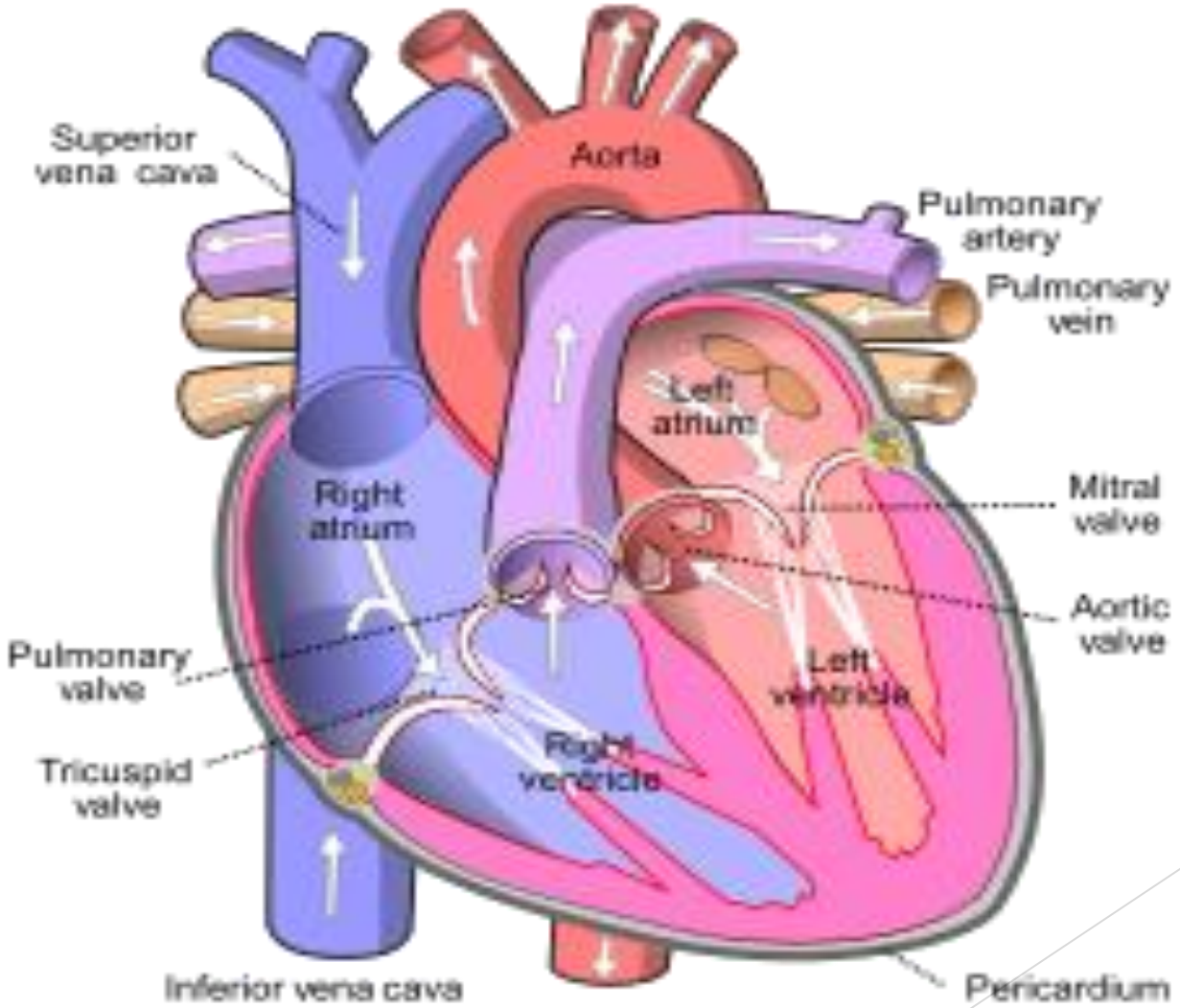
Deals with study of heart, blood vessels & blood

A)Heart-acts as the pump

B)Blood vessels-acts as the channels

C)Blood- circular fluid

- **WILLIAM HARVEY**-pioneer of CVS ,first to find out that blood is a constant circulatory fluid


Superior vena cava
Aorta
Pulmonary artery
Pulmonary vein
Left atrium
Mitral valve
Right atrium
Aortic valve
Pulmonary valve
Left ventricle
Tricuspid valve
Right ventricle
Inferior vena cava
Pericardium

HEART

- consists of 2 pumps.
- 1.Rt pump-pulmonary circulation-lesser circulation
- **2.Lt pump** –systemic circulation-greater circulation
- 2 pumps act simultaneously in series

- PULMONARY CIRCULATION-right ventricle---pulmonary artery----lungs----pulmonary veins---left atria
- SYSTEMIC CIRCULATION: Left ventricle---aorta---tissues----SVC &IVC---right Atria

- Heart is a conical structure with apex ,base & 3 surfaces –anterior, posterior &inferior
- Located in middle mediastinum between the lungs
- Heart is enclosed by fibro-serous covering called pericardium

Pericardium

2 layers. parietal & visceral layer

- Parietal continuous with coverings of larger vessels

  visceral layer reflected on to the surface of the heart to form the epicardium
- PERICARDIAL CAVITY:in between the parietal and visceral layers –filled with pericardial fluid
- Pericardial effusion

Layers of heart

- Heart made up of 3 layers

Epicardium-formed by visceral layer of pericardium

Myocardium-muscles of contractile unit,pacemaker& conducting system

Endocardium-has a single layer of endothelial layer, continues as endothelium of great vessels

- Myocardium of left ventricle is thick because

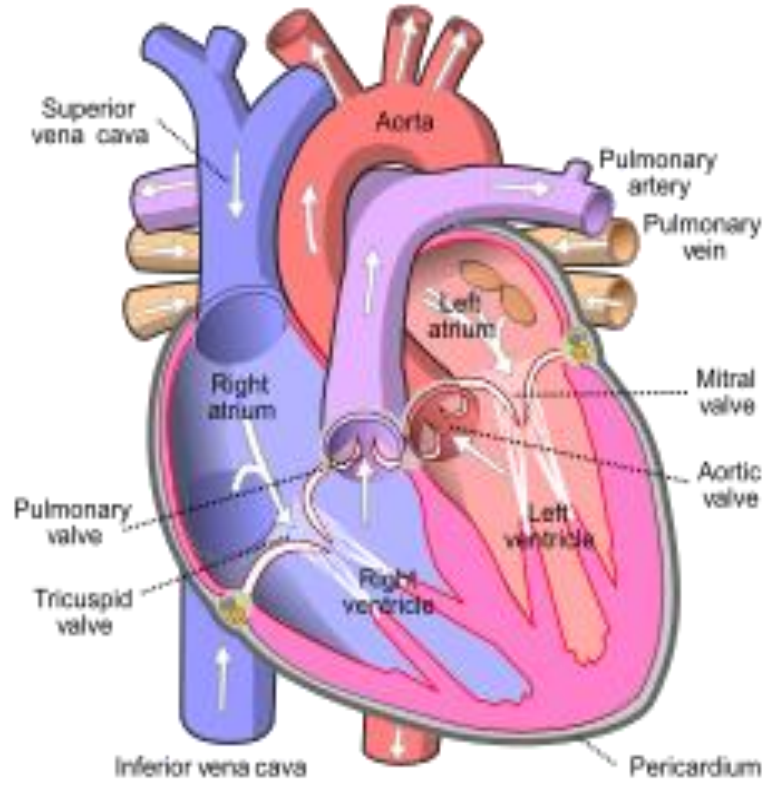
  - LV has to pump blood into systemic circulation- high pressure ,high resistance system

  - LV has to pump blood to a greater distance to all parts of body

- Apex of heart is @ left 5th intercostal space ½ inch medial to midclavicular line
- HEART : 4 chambers
- 2 ATRIA (right& left)
- 2.VENTRICLES (right&left)
- Atria & Ventricles are separated by a fibromuscular ring
- No direct connection between atria & ventricles.only connection through conducting /junctional tissues

ATRIA

- Right atria receives blood from SVC&IVC
- Left atria receives blood from pulmonary veins
- Interatrial septum separates right & left atria



VENTRICLES

- Right ventricle receives blood from rt atria & pumps blood into pulmonary arteries
- Left ventricle receives blood from left atria &pumps blood to aorta

VALVES

- **Atrioventricular valves:**
- Mitral valve: connects LA to LV
- Tricuspid valve:connect RA to RV
- Base of the valve connected to fibromuscular ring b/n atria & ventricle, apex connected to **chordae tendinae**, in turn connected to **papillary muscles**
- **Valves allow unidirectional flow**,they close when ventricles contract by the contraction of papillary muscles pulling cusps down

VALVES

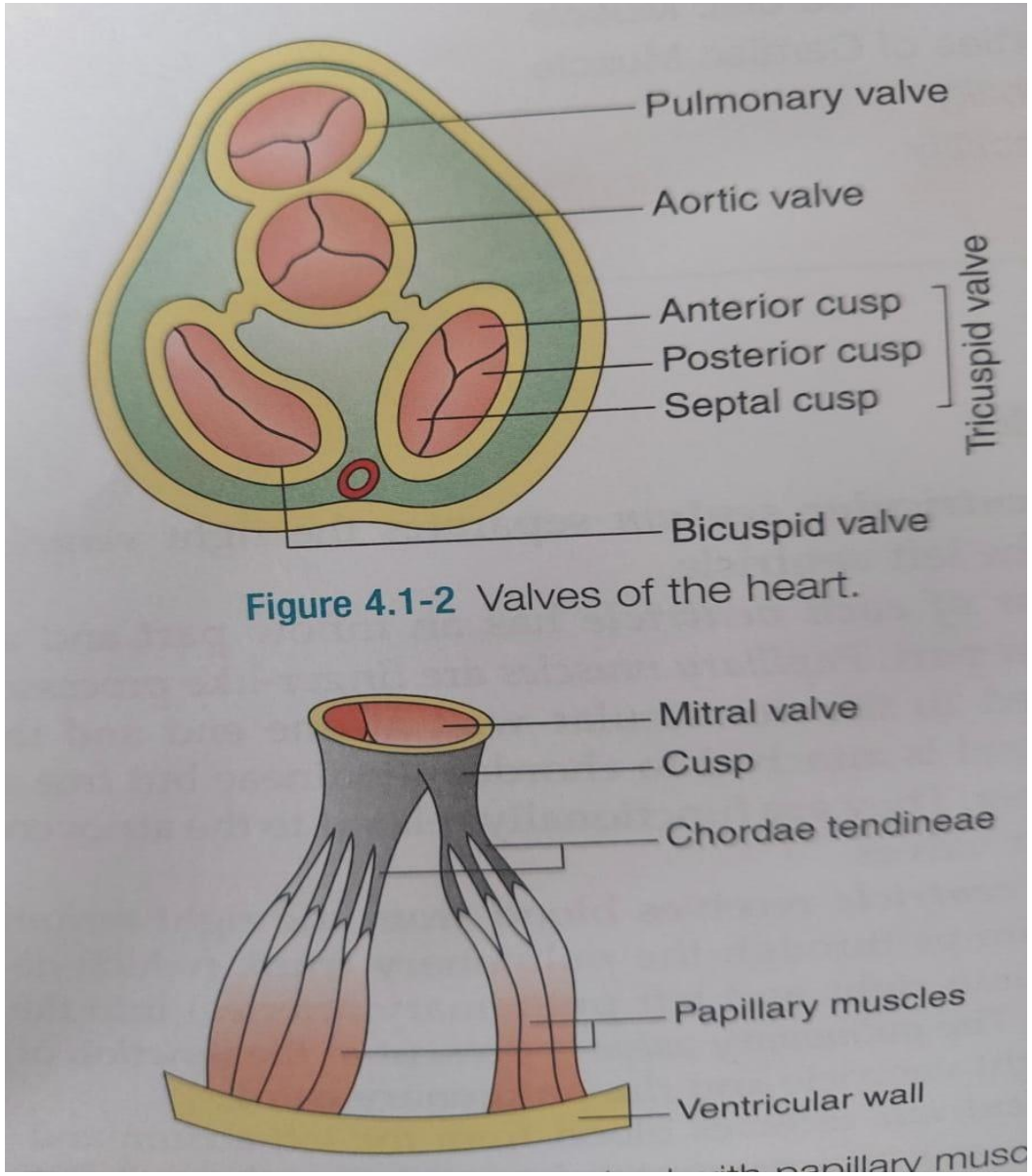


Figure 4.1-2 Valves of the heart.

- **SEMILUNAR VALVES**:
- Aortic valve &pulmonary valve
- Semilunar in shape
- Valves open when ventricles contract, all other times they remain closed preventing backflow of blood into the ventricles

JUNCTIONAL TISSUES

- Modified myocardium,loses its capacity to contract
- Acts as pathways of conduction
- SA node,AV node, Bundle of His, Purkinje fibres, internodal pathways

CARDIAC MUSCLE

Cardiac muscle fibres of 3 types

1.contractile or working muscle fibres-atria ,ventricle

2.nodal tissues or pacemaker tissues-SA node,AV node

3.conducting tissues-SA node,AV node,Bundle of His,Purkinje fibres

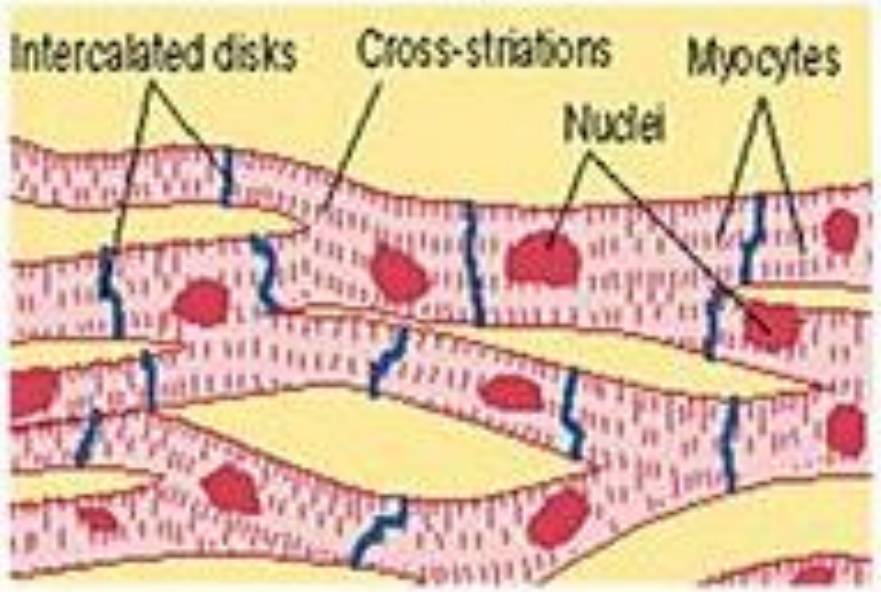
2&3 are junctional tissues

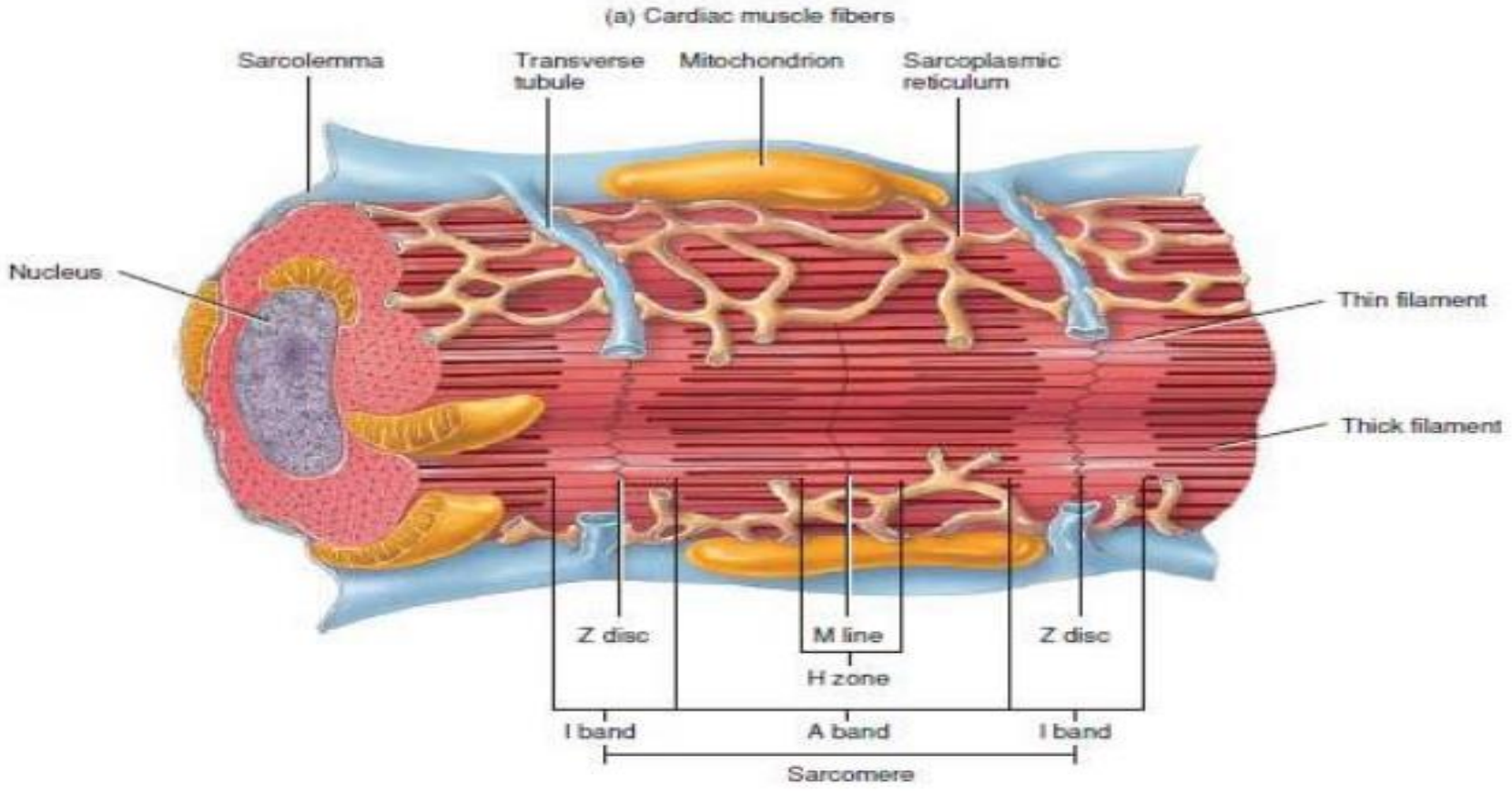
STRUCTURE OF CARDIAC MUSCLE

- Striated
- Rectangular,ribbon like with centrally placed nucleus
- Muscle fibres are connected to each other & branched
- At point of contact ,muscle fibres are fused together & form extensive infolding called INTERCALATED DISC-provide strong union between fibres that increase force of contraction

- Outer border of intercalated disc,two adjacent muscle fibres connected to each other by GAP JUNCTION-not an anatomical connection
- Gap junctions provide low resistance bridges for the spread of excitation or AP.thus cardiac muscle act as FUNCTIONAL SYNCYTIUM
- Two separate syncytium:atrial &ventricular
- Impulse conducted from atria to ventricle by conducting fibres

- SARCOMERE is the structural unit
- Sarcotubular system at Z line
- One triad or sometimes Diad per sarcomere
- T tubule & terminal cisterns


Intercalated disks
Cross-striations
Myocytes
Nuclei


(a) Cardiac muscle fibers
Sarcolemma
Transverse tubule
Mitochondrion
Sarcoplasmic reticulum
Nucleus
Thin filament
Thick filament
Z disc
M line
Z disc
H zone
I band
A band
I band
Sarcomere

PROPERTIES OF CARDIAC MUSCLE

- 1.AUTORHYTHMICITY/chronotropism
- 2.CONDUCTIVITY/Dromotropism
- 3.EXCITABILITY /Bathmotropism
- 4.CONTRACTILITY/ionotropism

1,2,3-electrical properties

4-mechanical

AUTORHYTHMICITY

- P cells or pacemaker cells have the property of self excitation & generate impulses to spread throughout heart
- P cells produces rhythmic impulses for the production of heart beat
- SA node is the PACEMAKER of the heart

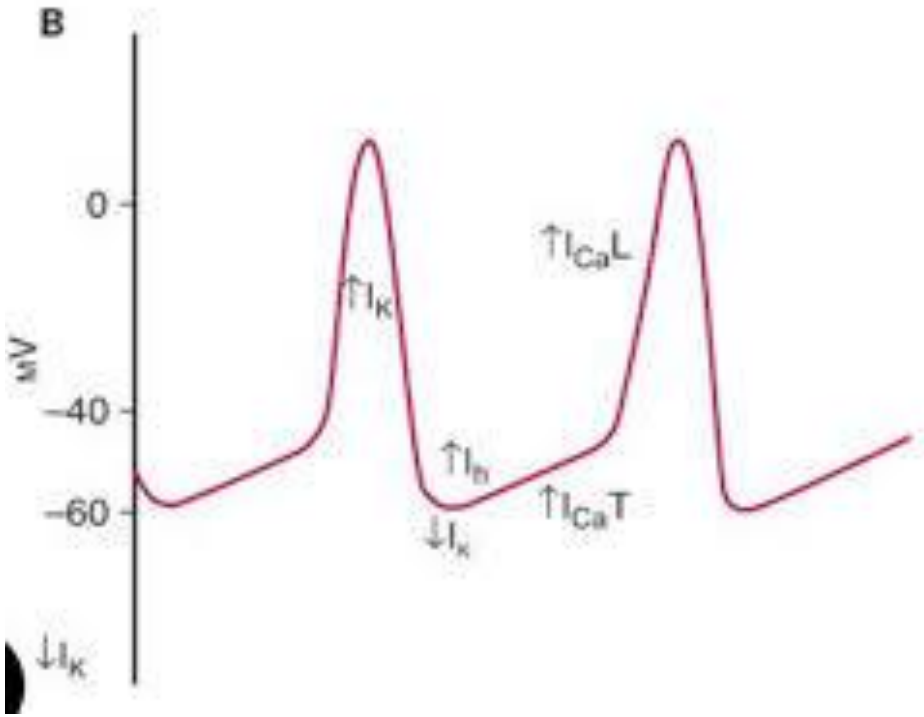
PACEMAKER

- SA NODE –why
- All parts of conducting system produces the impulse,but rate of impulse production is more in SA node or SA node produces impulses more rapidly with depolarisation spreads to other regions before they discharge spontaneously

ECTOPIC PACEMAKER

- A pacemaker other than SA node is called ectopic pacemaker
- In abnormal conditions AV Node,purkinje fibres ,atrial & ventricular fibres may discharge spontaneously

POTENTIAL of PACEMAKER CELLS



PACEMAKER POTENTIAL

- Electrical potential present in pacemaker cells only
- Change of potential from resting level to firing level in the pacemaker cell is called pacemaker potential or PREPOTENTIAL
- Slow spontaneous depolarisation to firing level
- Also called as diastolic depolarisation
- Prepotential triggers AP in pacemaker cells

- Prepotential prominent in SA node & AV NODE only
- Atrial & ventricular muscle fibres donot have prepotentials

IONIC BASIS

- REPOLARISATION-due to opening of K+channels(Ik).begins at the peak of depolarisation
- K + channels closes towards end of repolarisation,K+current decreases(I k decay)

- DEPOLARISATION -60 to -40 mv

closure of voltage gated K+ channels cause membrane potential to go up

b)channels open when membrane potential reaches -60mv—they open in the hyperpolarisation phase, mediated by cyclic Nucleotide gated channels-h channels (HCN channels)

called as f channels –funny channels becoz unusual opening

- They allow Na+(major)& K+ ions to enter—this forms first part of prepotential
- C)Transiently acting Ca2+ channels open to produce I ca2+ T completes prepotential & MP reaches the firing level

- DEPOLARISATION -40mv to peak

-long acting Ca2+ channels to produce I Ca2+ L & produce AP & impulse

- AP in a pacemaker tissue is a slow rising process
- AP in the SA & AV node largely depends on Ca2+ ,not Na+
- So there is no sharp, rapid depolarising spike before the plateau like

FACTORS affecting MP in pacemaker

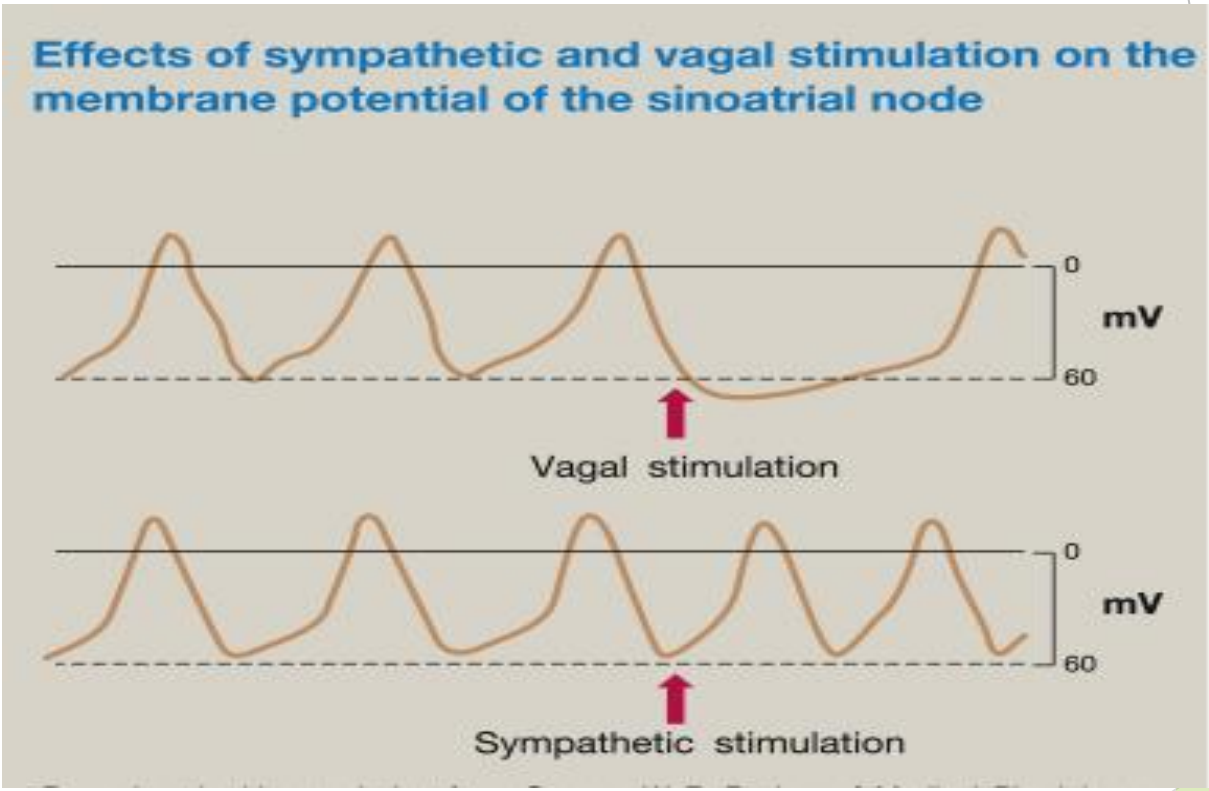
- 1.VAGAL STIMULATION (endocardial)

vagus N –parasympathetic supply of heart & releases Ach at nerve endings.

Ach on M2 receptors

1.Decreases cAMP—slows opening of Ca2+ channels---decreases Ca2+ influx---decreases HR

2.Open Ach activated K+ channels ---hyperpolarisation---prevents(Ik decay)---decreases HR


Effects of sympathetic and vagal stimulation on the membrane potential of the sinoatrial node
0
mV
60
Vagal stimulation
0
mV
60
Sympathetic stimulation

- 2.SYMPATHETIC stimulation(epicardial)

  Norepinephrine released acts on β 1 receptors—cAMP activation ---Ca2+ influx (through L type Ca2+ channels)---early opening of ca2+ channels ---- prepotential rises to firing level very fast---increase in HR

- 3.TEMPERATURE---increase in temperature---increase membrane permeability ---increase in Ca 2+ influx---increase in HR
- IONS:

K+ : increase in extracellular K+ --- K+ efflux will be less ------heart stops in diastole

occurs in renal failure

- Digitalis ---depresses nodal tissue & exerts an effect like vagal stimulation

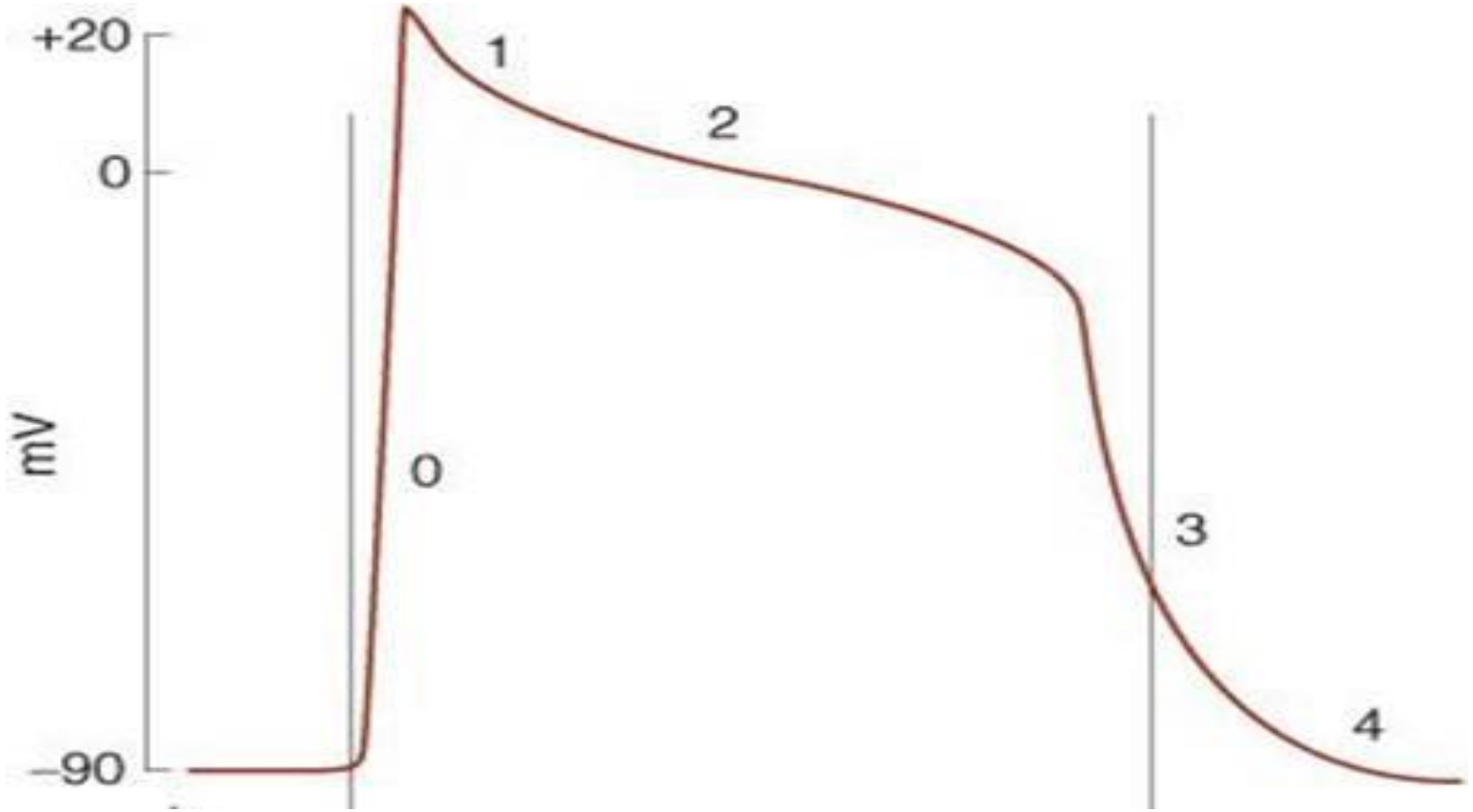
CONDUCTIVITY

- Impulse is conducted from SA node to different parts of the heart through conducting tissues

EXCITABILITY

- Property by which a tissue responds to a stimulus
- Contractile fibres exhibit excitability
- RMP of cardiac muscle is –90mv. k+ efflux through leaky channels responsible for RMP
- Stimulation produces a propagated AP ,responsible for initiating contraction

- A P of ventricle muscle –Plateau potential.
- prolonged about 250-300msec


+20
0
−90
mV
0
1
2
3
4

- PHASE 0-rapid depolarisation
- Phase 1-initial rapid repolarisation
- Phase 2-plateau phase
- Phase 3-final rapid repolarisation
- Phase 4.RMP

- Phase0

-stimulus applied to cardiac muscle ,rapid depolarisation occurs & reaches +20mv

-due to opening of voltage gated Na+ channels

-Inactivation gate closes at + 20mv

- Phase 1- initial rapid repolarisation

-30mv fall occurs very rapidly & MP falls upto -10mv

-due to closure of Na+ channels & also due to opening of transiently outwars acting K+ channel (I Kto) ---leads to K+ efflux

- PHASE 2 –plateau phase

-potential remains almost steady

-slow fall from -10 to -40mv

-due to opening of Long acting Ca2+ channels

-Inward flux of ca 2+ is balanced by K+ efflux ,but K+ efflux more

-K+ efflux through inward rectifying K+ channels

- PHASE 3-

-due to closure of Ca2+ channels

-K+ efflux occur through delayed rectifying & inwardly rectifying K+ channels

- Phase4 RMP

  Due to activity of Na+K+ ATPase pump .

DURATION of AP

Depolarisation: 1-2msec

Repolarisation:200-300 msec

REFRACTORY PERIOD

- Period following AP during which cardiac muscle doesnot respond to a stimulus
- 2, types :ABSOLUTE RP

  RELATIVE RP

-cardiac muscle has a long refractory period

- ABSOLUTE RP:cardiac muscle doesnot show any response to second stimulus

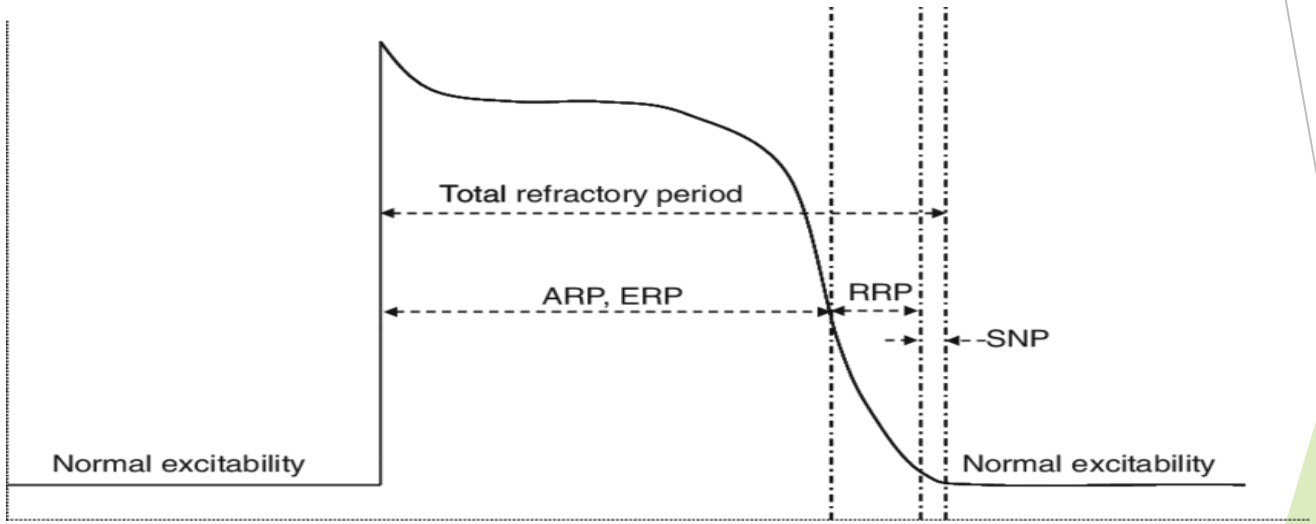
-it extends from phase 0 to half of phase 3 of AP

- RELATIVE REFRACTORY PERIOD:

- cardiac muscle respond if stimulus strength increase
- Extends from second half of phase 3 & phase4

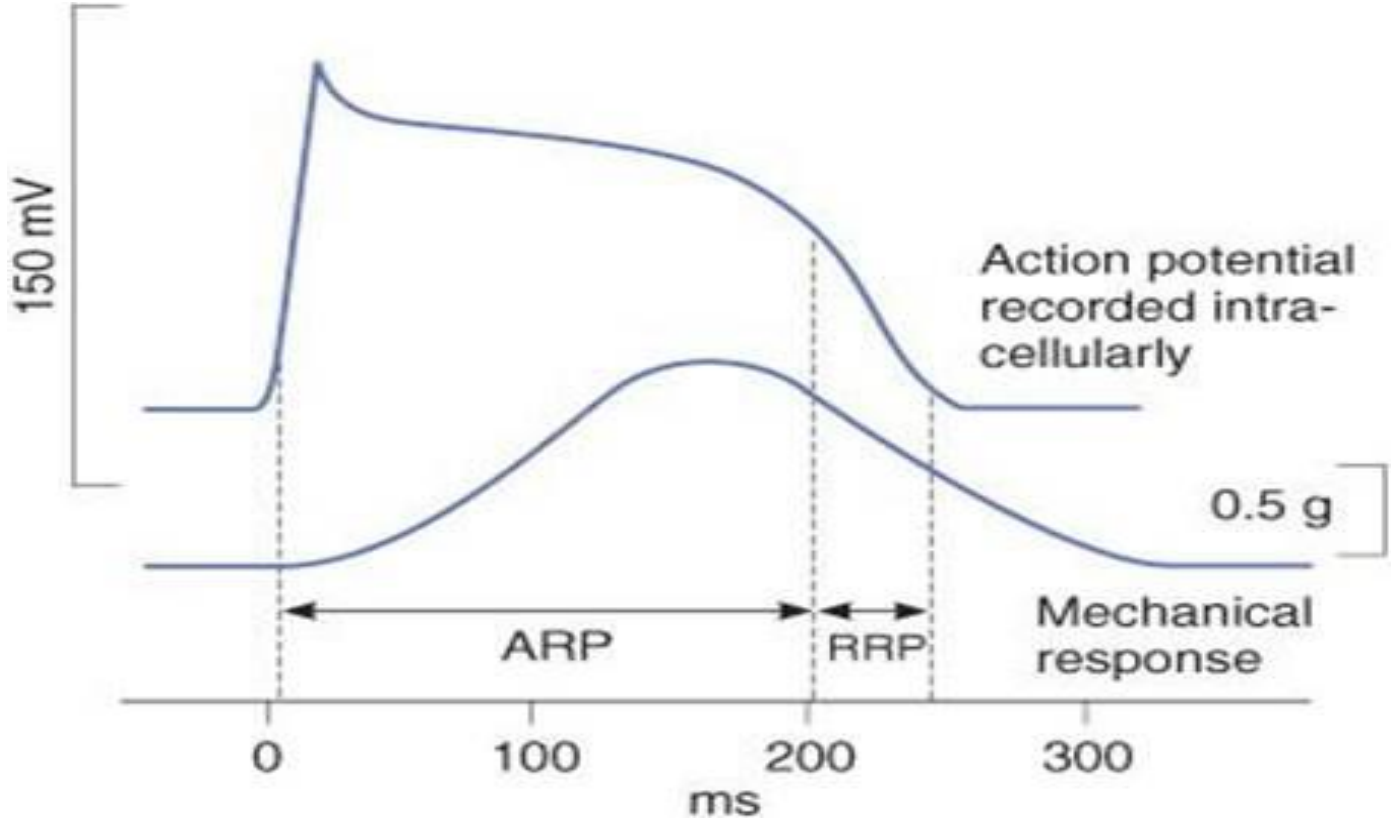
-

- Significance:
- Cardiac muscle cannot be tetanised due to long refractory period


Total refractory period
ARP, ERP
RRP
SNP
Normal excitability
Normal excitability

CONTRACTION

- Mechanical response
- Ability of cardiac muscle to actively generate force to shorten and to do work when sufficient stimulus is applied
- Mechanical response begins just after the start of depolarisation & last about 1.5 times as long as AP


150 mV
Action potential recorded intra-cellularly
0.5 g
ARP
RRP
Mechanical response
0
100
200
300
ms

EXCITATION- CONTRACTION

- Refers to the sequence of events by which an excited muscle leads to cross bridge activity by increasing sarcoplasmic calcium concentration
- As cardiac muscle act as a functional synctium AP spreads very rapidly

- Sequence of events during excitation –contraction coupling in the cardiac muscle similar to skeletal muscle
- When AP reaches sarcotubular system (T tubule),activation of DHPR channels----influx of calcium from extracellular fluid into sarcoplasm--- it triggers the release of Ca2+ from sarcoplasmic reticulum through RYR (calcium induced calcium release)

- Calcium release is a slow process & responsible for plateau phase—200 msec
- Strength of cardiac muscle contraction depends on great extent on ECF Ca2+
- Process of muscle contraction coupling –crossbridge cycling, sliding of filaments same as skeletal muscle

RELAXATION

- Relaxation occurs in diastole when levels of Ca2+ ions fall
- During diastole Ca2+ will move out the sarcoplasm by Na+ Ca2+ antiport in scarcolemma-calcium will move out Na+ will enter
- Gradient for entry of Na+ is created by Na+K+ATPase

Applied

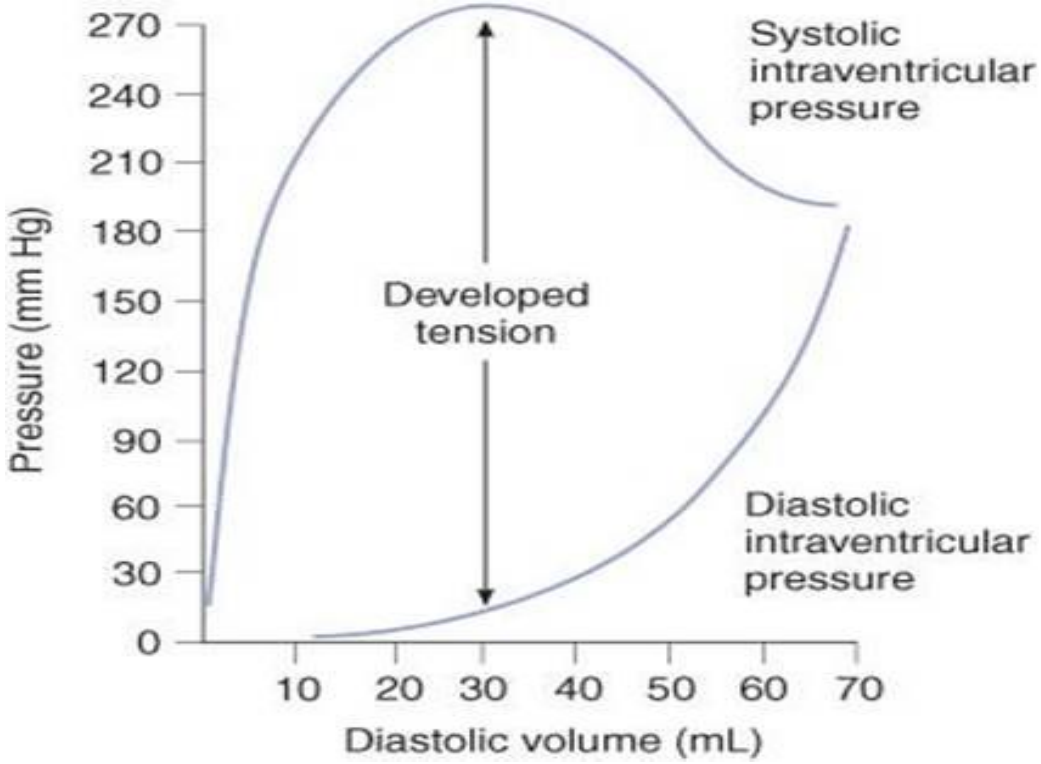
- Digitalis inhibit Na+K+ATPase –gradient not developed for the Na+ entry ---- so Ca2+ not transported outside----Ca level increases----force of contraction & myocardial contractility
- Digitalis used in CCF

LENGTH- TENSION

- Relation between initial length & total tension is CM similar to skeletal M
- Total tension developed is proportional to initial fibre length upto a physiological limits & then decreases
- Upper curve-total tension- systolic interventricular pressure
- Resting length at which tension developed on stimulation is maximal

- Initial length determined by degree of diastolic filling(end diastolic volume)
- Pressure developed during systolic depends on the volume of ventricle at the end of filling phase
- Developed tension increase when volume increases ,until it reaches a maximum then decreases

- Decrease is due to disruption of myocardial fibres
- Lower curve-passive tension –diastolic intraventricular pressure—increase with end diastolic volume
- Filling pressure increase only upto alimit


Pressure (mm Hg)
270
240
210
180
150
120
90
60
30
0
10
20
30
40
50
60
70
Diastolic volume (mL)
Systolic intraventricular pressure
Developed tension
Diastolic intraventricular pressure

FRANK STARLINGs LAW of HEART

- States that the greater the end diastolic volume greater the force of contraction within physiological limits
- Force of contraction increases as end diastolic volume increases upto certain limit then decreases

- MECHANISM
- Increase in end diastolic volume---increase in stretch of muscle fibres -----opens stretch sensitive calcium channels in muscle cell membrane---increase calcium influx----force of contraction increases
- Increase calcium also increase calcium release SR

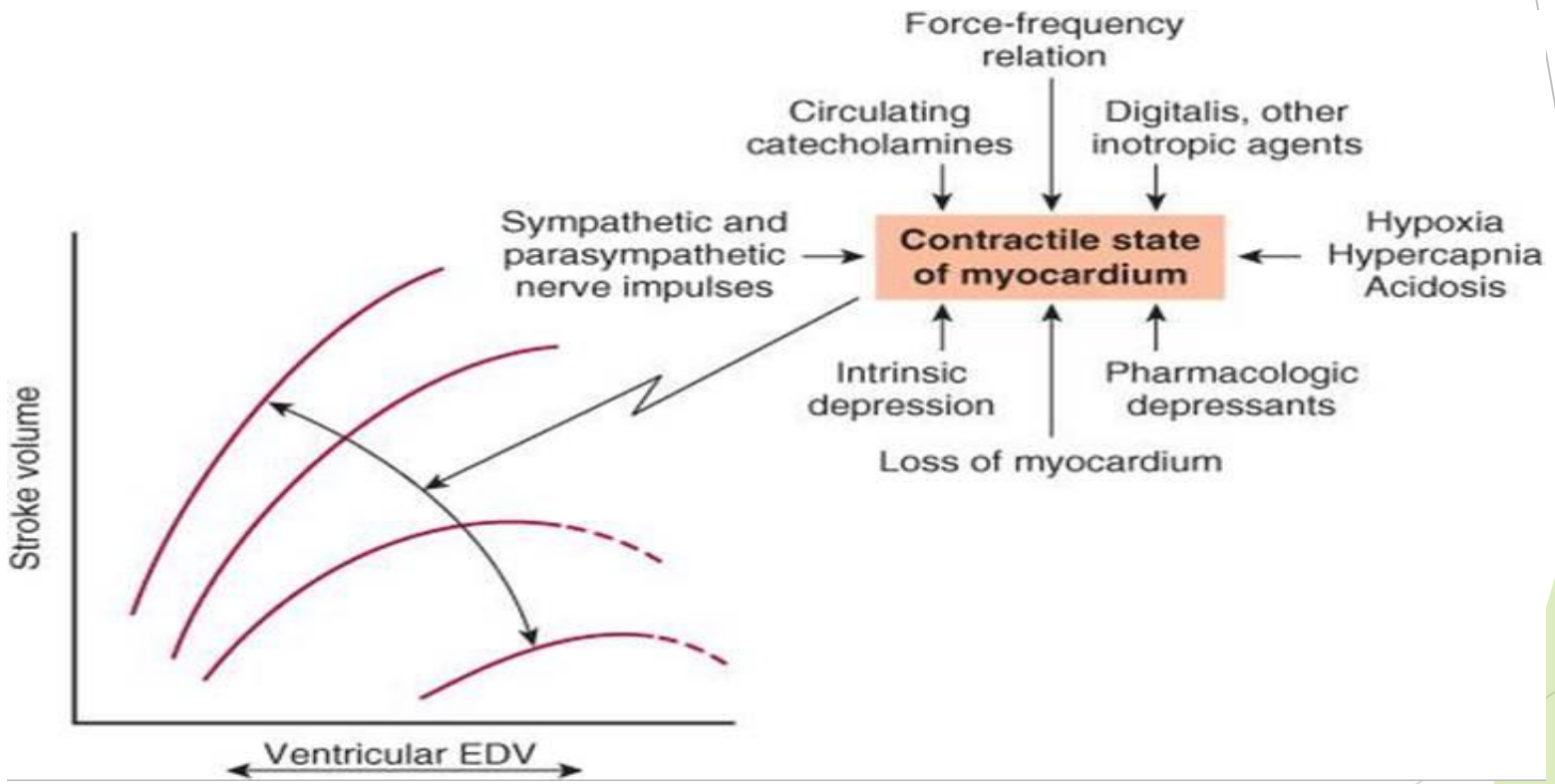
- Stretch of myocardium enhances affinity of troponin C for calcium---increase force of contraction

Factors increasing force of contraction

- Increase in EDV---increase in1) blood volume 2) venous return3) intrathoracic P
- Increase ECF ca2+ levels
- Catecholamines like adrenaline
- Stimulation of sympathetic system
- digitalis

Application

- Heterometric regulation of cardiac output


Force-frequency relation
Circulating catecholamines
Digitalis, other inotropic agents
Sympathetic and parasympathetic nerve impulses
Contractile state of myocardium
Hypoxia Hypercapnia Acidosis
Intrinsic depression
Pharmacologic depressants
Loss of myocardium
Stroke volume
Ventricular EDV

Differences B/n skeletal & cardiac muscle

	SKELETAL Muscle	Cardiac muscle
Branching of fibres	Absent	present
Connection b/n fibres	Absent	Functional syncytium
Sarcotubular system	2 triads per sarcomere at AI junction	1 triad per sarcomere at Z line
Control of action	voluntary	Involuntary

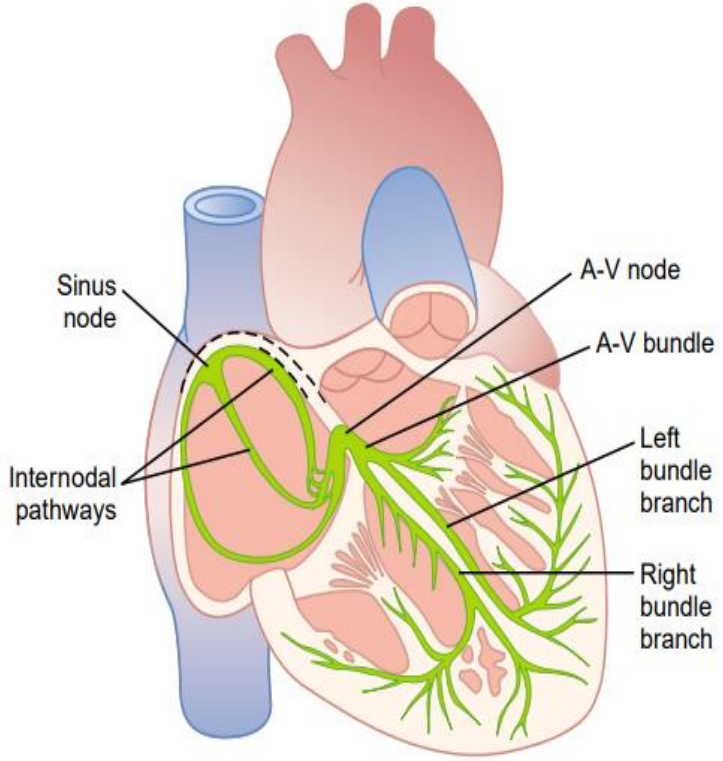
	SKELETAL MUSCLE	CARDIAC MUSCLE
AP shape & duration	Spike potential,5msec	Plateau potential (100-300)msec
ARP	1 to 3 msec	200msec
Autorhythmicity	Absent	present
Mechanical response & AP	Mechanical response at the end of repolarisation	Mechanical response start after depolarisation & overlaps electrical response & lasts 1.5 times

	Skeletal muscle	Cardiac muscle
contraction	AP reaches,T tubule depolarises,ca released from SR	AP reaches ,T tubule depolarises,ca enter from ECF & also released from SR (calcium induced Ca release)
Relaxation	Occurs ca returns to SR	Occurs Ca goes to ECF via Na+Ca2+ antiport
Dependance of ECF ca+ for contraction	No	Mainly dependant on ECF Ca+
length tension relationship	Fall in graph due to detachment of crossbridges	Due to destruction of myocardial fibres

	SKELETAL MUSCLE	CARDIAC MUSCLE
Duration of muscle twitch	.1 sec	1.5 times AP
All or none law	Obeyed by single muscle fibre	Obeyed by whole muscle
tetanus	possible	Not possible
fatigue	possible	Not possible

Conducting system of heart

- The heart beat originates in a specialised cardiac conducting system and spreads via this system to all parts of the myocardium
- Include SA node
- AV node
- Bundle of His
- Purkinje fibres



PECULARITIES

- Modified myocardium
- Lost the contractile property,have less striations,indistinct cell boundary connected by gap junctions
- It generate electrical impulses
- It conduct impulses to all parts of heart to produce rhythmical contraction of myocardium

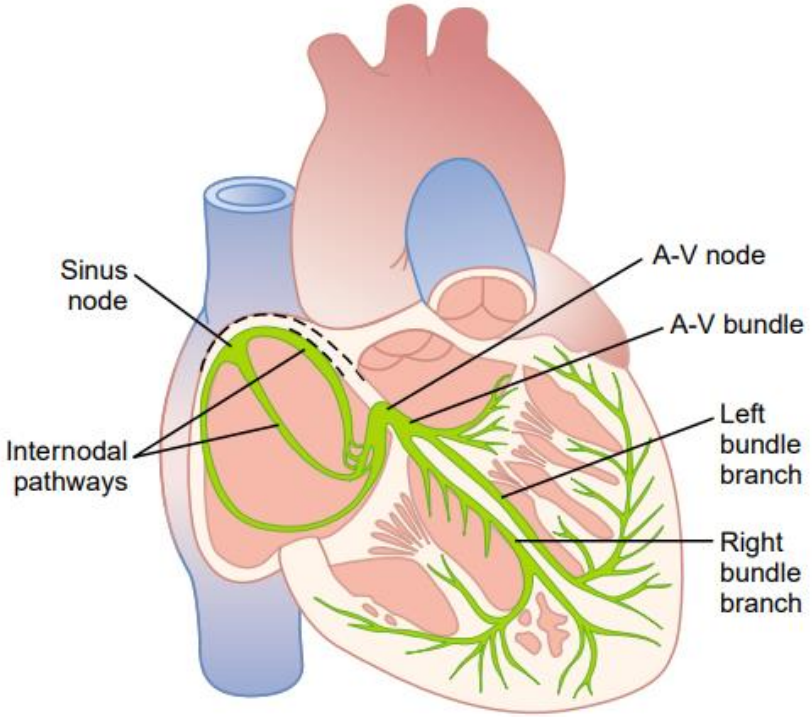
SA node

- Located at the junction of SVC with right atrium innervated by rt vagus
- The various parts of the conduction system , parts of the myocardium, are capable of spontaneous discharge.
- the SA node normally discharges most rapidly, with depolarization spreading from it to the other regions before they discharge spontaneously.
- The SA node is therefore the normal **cardiac pacemaker**, with its rate of discharge determining the rate at which the heart beats.

- The conduction system is composed for the most part of modified cardiac muscle with **fewer striations and indistinct boundaries.**
- The SA node contain small round cells with few organelles, which are connected by gap junctions. --- actual pacemaker cells (**P cells.**)
- **SA node fibres connect directly with atrial muscle,as impulse spread rapidly to atrium**

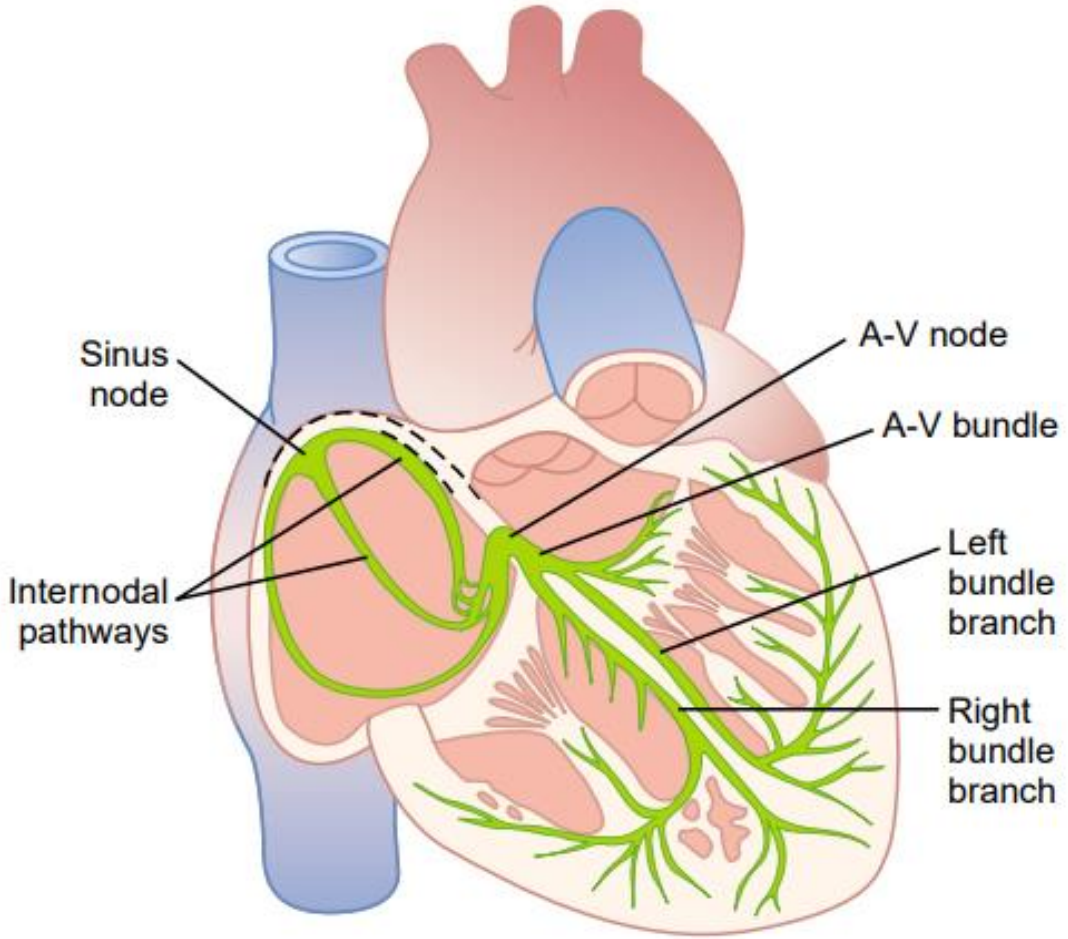
TNTERNODAL TRACTS

- 3 bundles of atrial fibers that contain Purkinje type fibers and connect SA node to AV node:
- 1)anterior internodal tract of Bachman,
- 2) middle internodal tract of Wenckebach,
- 3) posterior internodal tract of Thorel.
- Conduction more rapid in these bundles than atrial muscles



AV NODE

- AV node is located posteriorly on the right side of inter atrial septum near the opening of coronary sinus.
- Supplied by left vagus(PS) & left stellate ganglion (sympathetic)
- Contain P cells, less in number---rate of impulse production less
- Impulse reaching AV node not immediately transmitted --- AV nodal delay


Sinus node
A-V node
A-V bundle
Internodal pathways
Left bundle branch
Right bundle branch

BUNDLE OF HIS

- The atrial muscle fibers are separated from those of the ventricles by a **fibrous tissue ring (no conductivity)**
- normally the only conducting tissue between the atria and ventricles is the bundle of His.
- gives off a left bundle branch & right bundle branch at the top of the interventricular septum

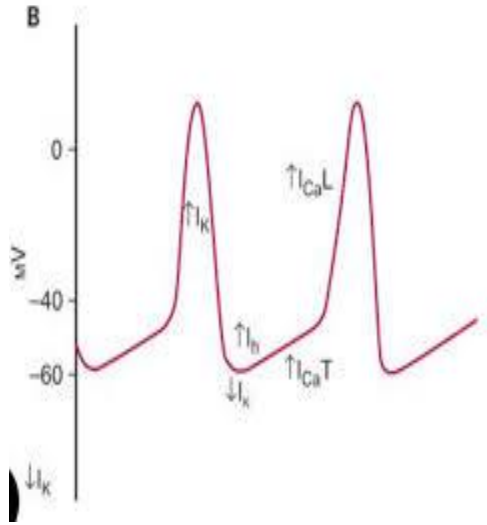
- The left bundle branch pierces septum & divides into an anterior fascicle and a posterior fascicle.
- The branches and fascicles run subendocardially down either side of the septum and divides into purkinje fibres
- PURKINJE fibers spread to all parts of the ventricular myocardium.

PURKINJE FIBRES

- Terminal branches of bundle fibres & spread to all parts of ventricles subendocardially
- Fast conducting fibres (C .V 4m/s)with large diameter
- Large amts of glycogen & more gap junctions
- Purkinje fibres is piercing from endocardium to myocardium---direction of impulse conduction from endocardium to myocardium

ORIGIN OF CARDIAC IMPULSE

- Cardiac impulse originated by the spontaneous depolarisation of pacemaker tissue
- Rate of rise of prepotential is more in SA node---SA node overrides other regions----SO PACEMAKER



- SA Node suppresses other regions of the conducting systems ---OVERDRIVE SUPPRESSION
- Rate of impulse production SA node—70-80/min

  AV node-40-60/min

  Bundle of His-15-40/min

Spread of cardiac impulse

- Depolarization initiated in the SA node spreads radially through the entire atria ,simultaneous depolarisation LA due to interatrial tract
- Depolarisation reaches the AV node through internodal tracts by 0.03 sec.
- Atrial depolarization is complete in about 0.1sec

Spread of cardiac impulse ctd

- Because of slow conduction in AV node, a delay of about 0.1 s **(AV nodal delay)** occurs before excitation spreads to the ventricles.

  This delay is shortened by stimulation of the sympathetic nerves to the heart and lengthened by stimulation of the vagi.

- Small size of fibres and decrease in the no: of gap jn

AV NODAL DELAY

- Causes: 1.AV node small diameter fibres,low conduction velocity

  2.number of gap junctions is less

  Presence of multiple branching systems in AV node

- ADVANTAGE:1.atrium gets enough time to empty blood to ventricle –so enough ventricular filling during relaxation of ventricle before V contraction

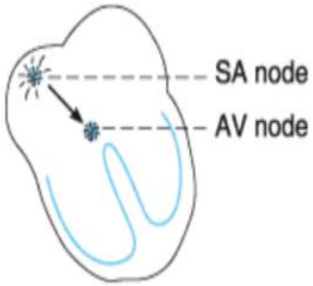
  2.When atrial rate is high in ATRIAL fibrillation---AV node cannot conduct above 230/min---ventricular rate can be maintained

- DISADVANTAGE:
- AV node most common site of heart disease--- abnormal delay in heart BLOCK

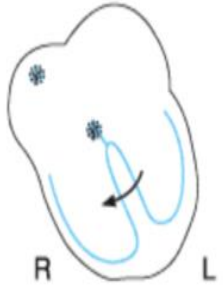
SPREAD OF CARDIAC IMPULSE

- Slow conduction of impulse in penetrating portion of bundle of his due to fibromuscular ring --- impulse passes through Bundle of his ,then impulse passes to bundle branches
- cardiac impulse reach ventricle by 0.16 sec

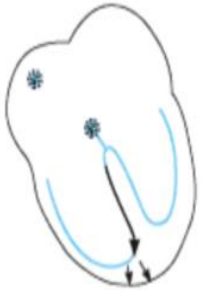
- The depolarization of the ventricular muscle spreads rapidly, starts at the left side of the interventricular septum and moves first to the right across the midportion of the septum.
- The wave of depolarization then spreads down the septum to the apex of the heart.
- It returns along the ventricular walls to the AV groove, proceeding from the endocardial to the epicardial surface .



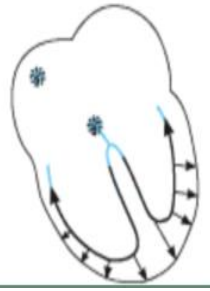
Atrial activation



Septal activation from left to right



Activation of anteroseptal region of the ventricular myocardium



Activation of major portion of ventricular myocardium from endocardial surfaces

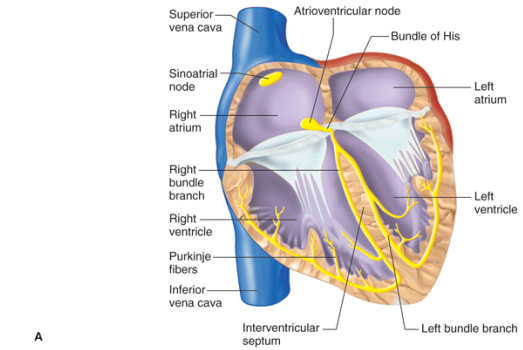
Late activation of posterobasal portion of the left ventricle and the pulmonary conus

- . The last parts of the heart to be depolarized are the **posterobasal portion of the left ventricle, the pulmonary conus, and the uppermost portion of the septum.**
- **RAPID & SIMULTANEOUS EXCITATION OF VENTRICULAR MUSCLE CAUSES SIMULATEOUS CONTRACTION OF VENTRICLES**
- **Whole heart depolarised by 0.22 sec to 0.26sec**

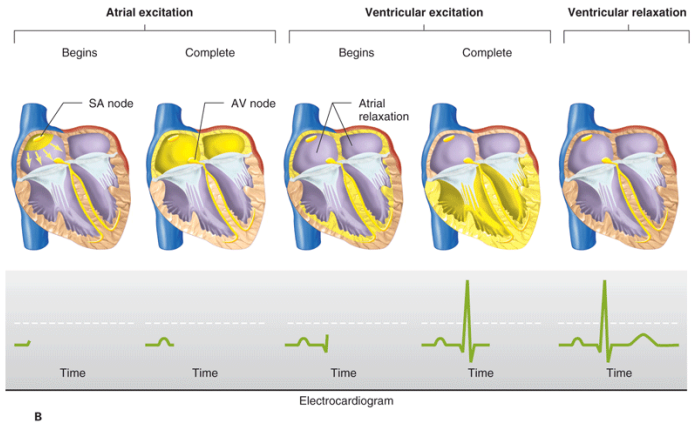
Table 30–1 Conduction Speeds in Cardiac Tissue.	
Tissue	**Conduction Rate (m/s)**
SA node	0.05
Atrial pathways	1
AV node	0.05
Bundle of His	1
Purkinje system	4
Ventricular muscle	1

REPOLARISATION

- In opposite direction (from epicardium to endocardium) & also apex to base


Superior vena cava
Atrioventricular node
Bundle of His
Sinoatrial node
Left atrium
Right atrium
Right bundle branch
Left ventricle
Right ventricle
Purkinje fibers
Inferior vena cava
Interventricular septum
Left bundle branch

A


Atrial excitation
Ventricular excitation
Ventricular relaxation
Begins
Complete
Begins
Complete
SA node
AV node
Atrial relaxation
Time
Time
Time
Time
Time
Electrocardiogram

B

APPLIED

- Sick sinus syndrome
- Ectopic beats (extra systole)
- Aberrant conducting pathway – Wolff Parkinson white syndrome
- Heart block

- ECTOPIC PACEMAKER:pacemaker other than SA node—AV node,Purkinje fibres,Ventricle
- Ectopic foci discharges produce premature beat—extrasystole
- SICK SINUS SYNDROME
- SA node incapable of impulse production—AV node produces—HR low

- WOLF PARKINSON WHITE SYNDROME: aberrant connection (pathway)between SA node & ventricle
- HR high—chest pain,dyspnoea

CONDUCTION DEFECTS (HEART BLOCKS)

- Due to defect in transmission of impulses across AV node
- FIRST DEGREE HEART BLOCK-conduction from SA node to AV node is slowed (more time needed).all imulses reach ventricles
- SECOND DEGREE HEART BLOCK-prolongation of transmission & certain impulses are prevented from reaching ventricles (mobitz type 1&type 2)

- THIRD DEGREE HEART BLOCK:no transmission of impulses across AV node
- Atria beats according to SA node
- Initially ventricles donot beat,but they beat after sometime on its own rhythm—ie ATRIA & VENTRICLE BEAT separately

CARDIAC CYCLE

- Defined as the events that occur in the heart from the beginning of one beat to the beginning of next beat
- Each cardiac cycle consists of simultaneously occuring atrial & ventricular cycles
- Duration of cardiac cycle when heart rate 75/' is 0.8sec

PHASES

- 2 phases

SYSTOLE-period of contraction of heart

DIASTOLE-relaxation of heart

ATRIAL CYCLE-

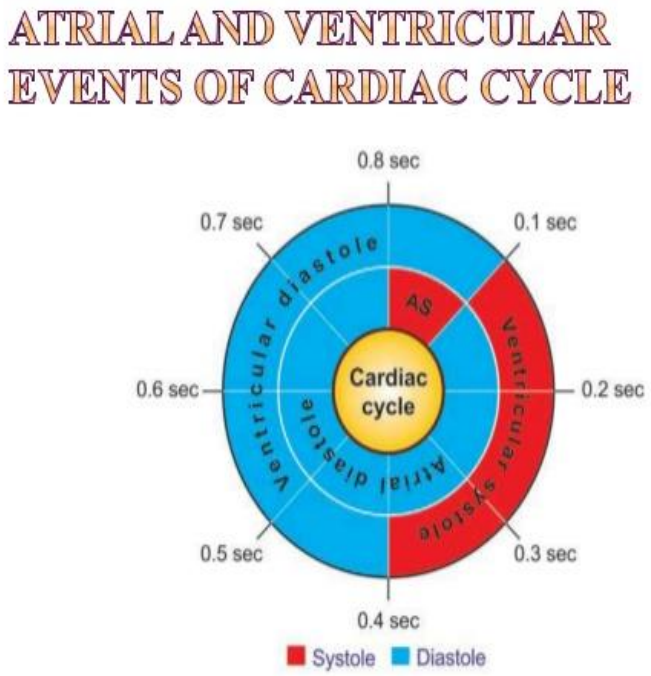
Atrial systole-0.1 sec

diastole-0.7sec

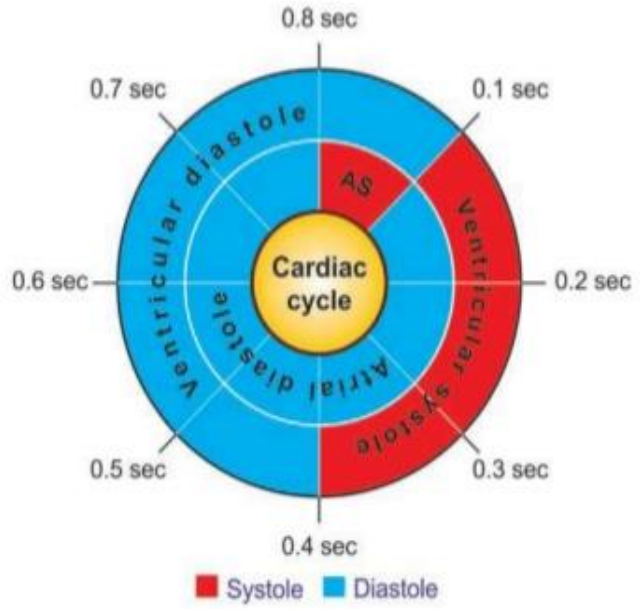
Ventricular cycle-

systole-0.3sec

diastole-0.5sec



ATRIAL AND VENTRICULAR EVENTS OF CARDIAC CYCLE



Phases of V systole

- ISOVOLUMETRIC CONTRACTION -0.05sec
- RAPID EJECTION PHASE-0.1sec
- REDUCED EJECTION PHASE-0.15sec

Phases of V diastole

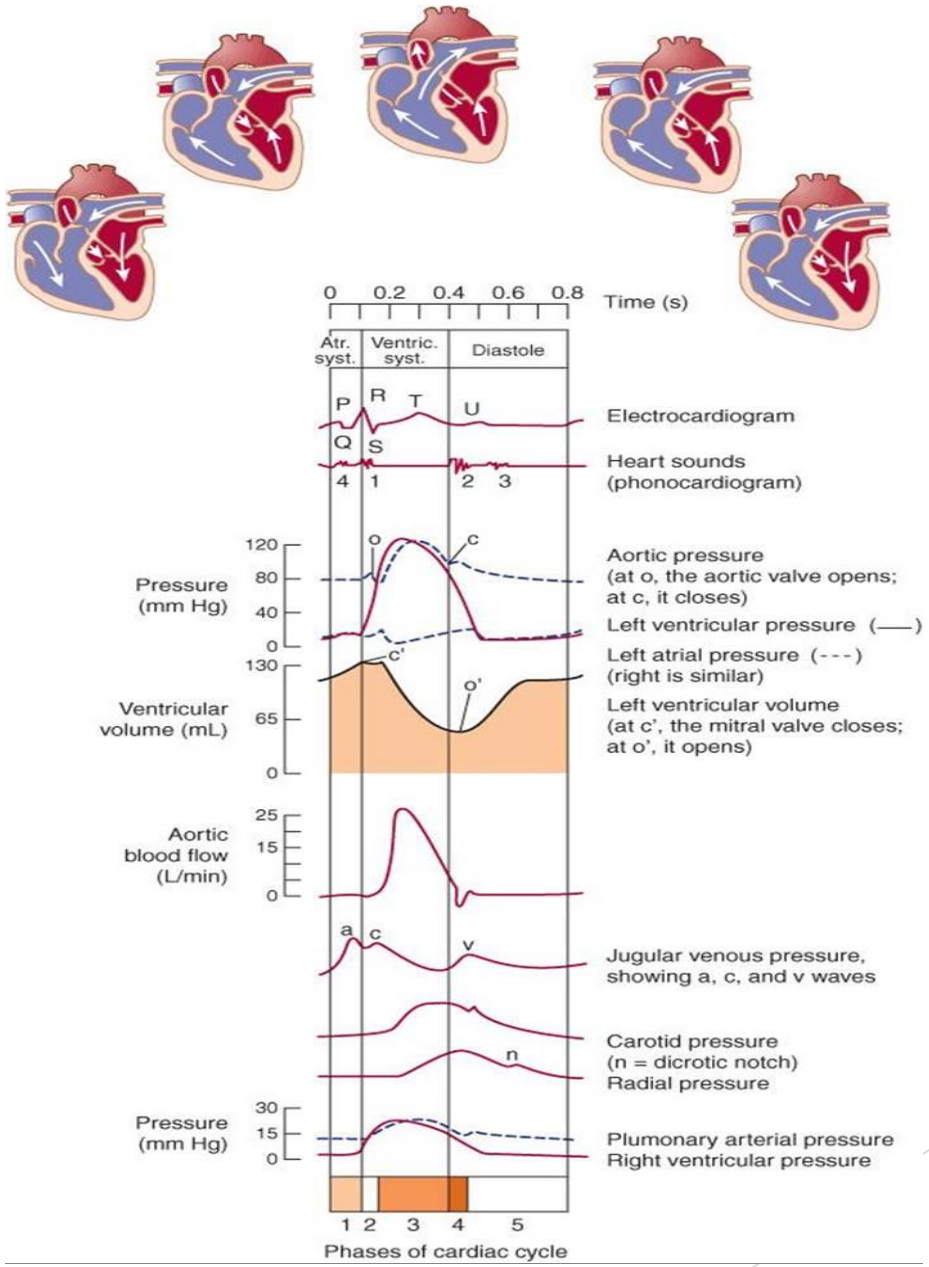
- 1.PROTODIASTOLE-.04sec
- 2.ISOVOLUMETRIC RELAXATION PHASE-.06sec
- 3.INITIAL RAPID FILLING PHASE-0.11 sec
- 4.REDUCED FILLING PHASE-0.19 sec
- 5.LAST RAPID FILLING PHASE-0.1sec

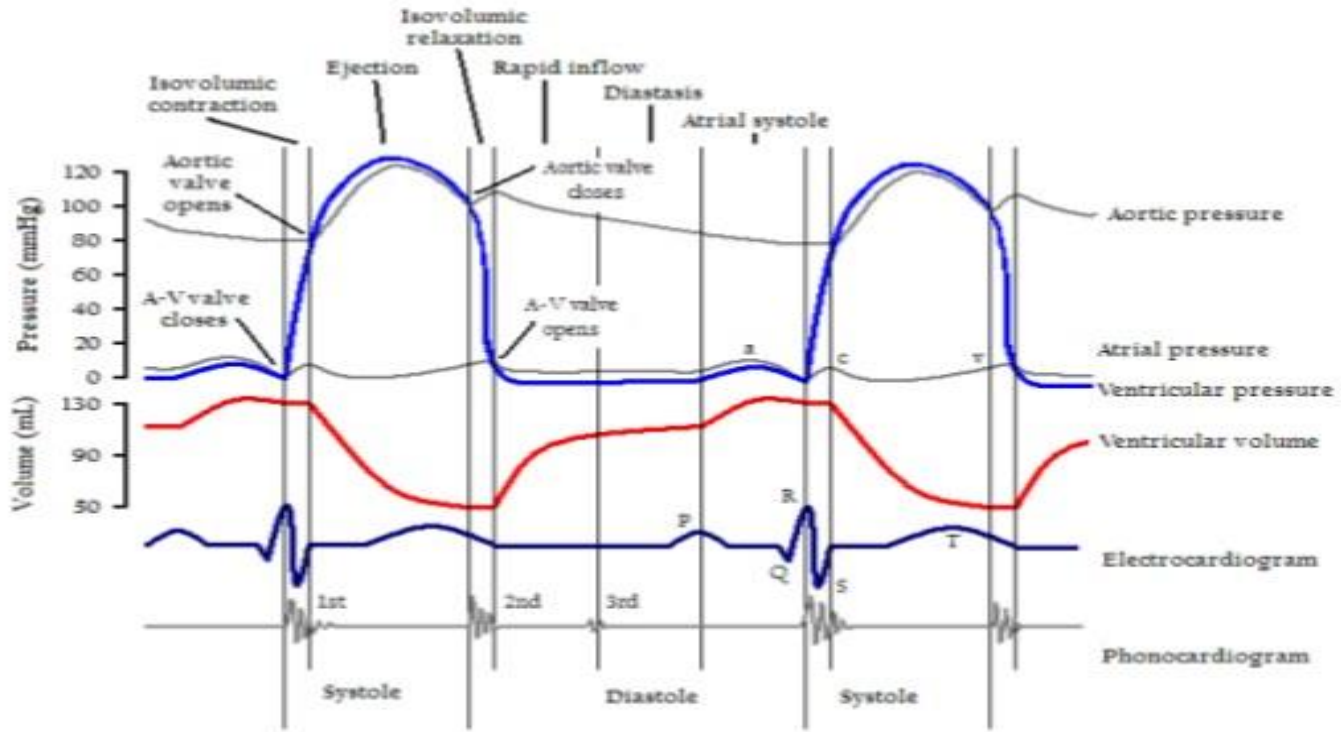
EVENTS OCCURING

- Pressure changes
- Volume changes
- Electrical changes
- Closure & Movements of Valves-heart sounds
- Pulse waves in the B V

WIGGERS DIAGRAM

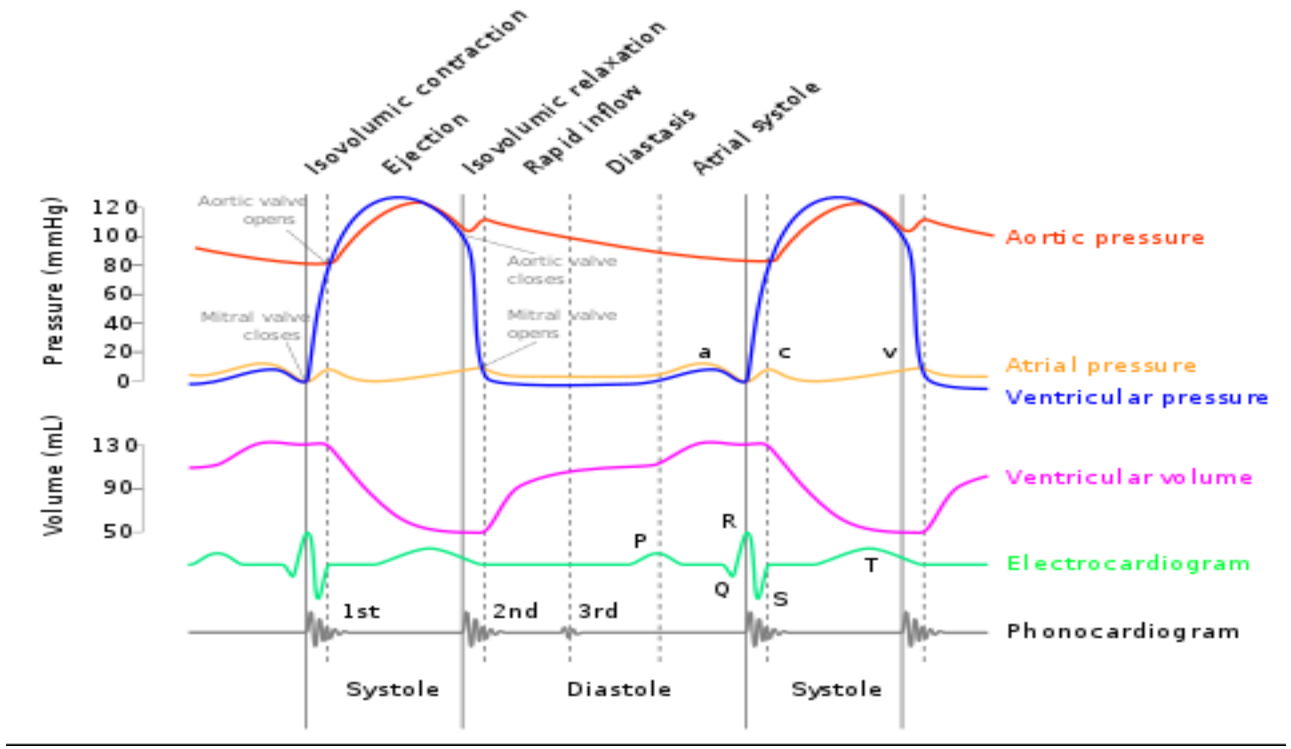
- Events of cardiac cycle expressed in diagram
- Includes ECG,pressure & volume changes in the heart & heart sounds
- Vertical lines separate the successive phases


0 0.2 0.4 0.6 0.8 Time (s)
Atr. syst.
Ventric. syst.
Diastole
P
R
T
U
Q
S
Electrocardiogram
4 1 2 3
Heart sounds (phonocardiogram)
120
80
40
0
Pressure (mm Hg)
o
c
Aortic pressure (at o, the aortic valve opens; at c, it closes)
Left ventricular pressure (——)
Left atrial pressure (- - -) (right is similar)
130
65
0
Ventricular volume (mL)
c'
o'
Left ventricular volume (at c', the mitral valve closes; at o', it opens)
25
15
0
Aortic blood flow (L/min)
a
c
v
Jugular venous pressure, showing a, c, and v waves
n
Carotid pressure (n = dicrotic notch)
Radial pressure
30
15
0
Pressure (mm Hg)
Plumonary arterial pressure
Right ventricular pressure
1 2 3 4 5
Phases of cardiac cycle


Isovolumic relaxation
Ejection
Rapid inflow
Isovolumic contraction
Diastasis
Atrial systole
Aortic valve opens
Aortic valve closes
A-V valve closes
A-V valve opens
Pressure (mmHg)
120
100
80
60
40
20
0
Volume (mL)
130
90
50
a
c
v
P
R
Q
S
T
1st
2nd
3rd
Aortic pressure
Atrial pressure
Ventricular pressure
Ventricular volume
Electrocardiogram
Phonocardiogram
Systole
Diastole
Systole

ATRIAL DIASTOLE

- Coincides with ventricular systole & most of ventricular diastole
- Atrial muscles relax & gradual filling of atria due to continous venous return from veins (SVC & INC)—causes increase pressure in atria---causes AV valve open---blood flow to ventricle
- 70% of ventricular filling occurs in A diastole


Isovolumic contraction
Ejection
Isovolumic relaxation
Rapid inflow
Diastasis
Atrial systole
Pressure (mmHg)
120
100
80
60
40
20
0
Aortic valve opens
Aortic valve closes
Mitral valve closes
Mitral valve opens
a
c
v
Aortic pressure
Atrial pressure
Ventricular pressure
Volume (mL)
130
90
50
Ventricular volume
P
R
Q
S
T
Electrocardiogram
1st
2nd
3rd
Phonocardiogram
Systole
Diastole
Systole

ATRIAL SYSTOLE

- AS occurs following the impulse generation in the SA node
- P wave occurs after impulse generation---at the peak of P wave, atria contracts & atrial systole occurs-----atrial pressure increases ---blood pushed into ventricles
- 30% ventricular filling occurs in atrial systole—venricular pressure increases
- Coincides with last rapid filling phase

- Large veins opening into atria doesnot have valves---atrial contraction causes some blood regurgitated into large veins----produces pressure changes in large veins---’a’ wave in JVP
- Atrial Contraction causes

1.increase in atrial P

2.increase in Ventricular P

3.P wave in ECG

4.’a’ wave in JVP

ATRIAL DIASTOLE

- Coincides with ventricular systole & most of ventricular diastole
- Atrial muscles relax & gradual filling of atria due to continous venous return from veins (SVC & INC)—causes increase pressure in atria---causes AV valve open---blood flow to ventricle
- 70% of ventricular filling occurs in A diastole

ISOVOLUMETRIC CONTRACTION

- Starts immediately after atrial systole
- IVC starts at the peak of QRS complex –in IVC ventricular pressure increases---AV valve close as ventricular pressure more than atrial P----(svc already closed)ventricle contracts as a closed chamber as both valve closed---no volume change
- 1st heart sound produced due to closure of AV valve

- Pressure in the ventricle increases—AV valve pushed up into atria –causes a slight pressure change in atria ‘c’ wave in JVP
- When Ventricular pressure above aortic & pulmonary pressure SLV open
- IVC ends when semilunar valve opens

Rapid Ejection phase

- SLV Valve opens at 80mm pressure,blood ejected to aorta & pulmonary A,
- But ventricular P increases bcoz ,ventricular contraction greater than the blood ejection .ventricular size decreases
- Ventricular P increases to 120mm Hg at left & 25mm in right
- At the end of rapid ejection maximum pressure attained
- Aortic P & pulmonary P also increases
- 2/3rd stroke volume ejected in this phase

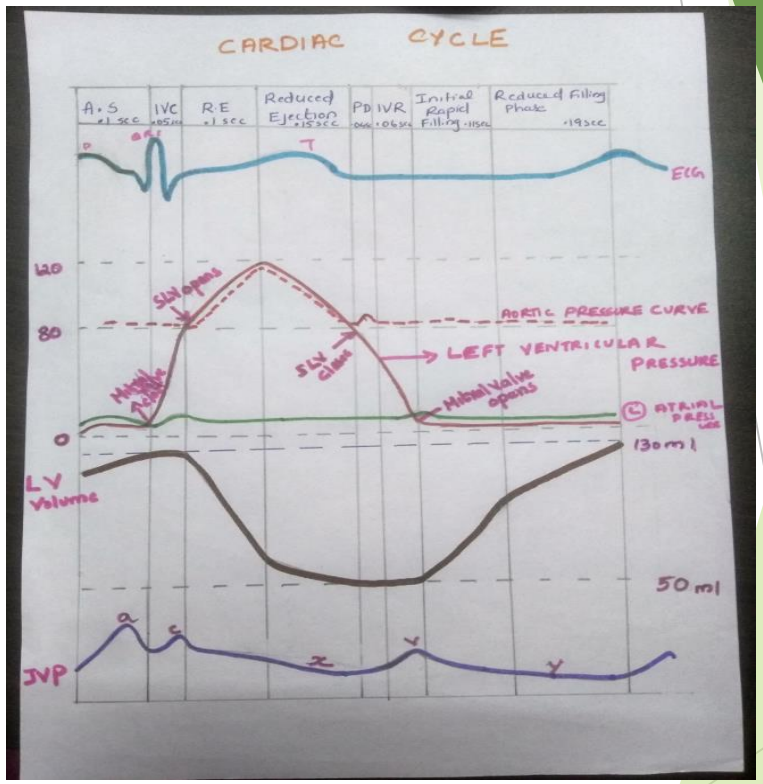
Reduced ejection

- Amount of blood ejected decreases-1/3rd
- Ventricle contracting but volume of blood in the ventricle is less, so pressure decreased
- Aortic pressure also falls bcoz ejection is less
- atria fills during this phase ,atrial P increases as AV closed
- At the end of reduced ejection phase,aortic pressure becomes more than V P ,tendency of backflow------ backflow prevented by SLV closure

- When aortic P equal to VP some filling occurs due to momentum of aorta
- AV valves are pulled down by the contraction of ventricular muscle & atrial P drops-x decent in JVP
- T wave of ECG is over by the time of reduced ejection
- About 50ml blood remains in each ventricle at the end of systole-Endsystolic volume

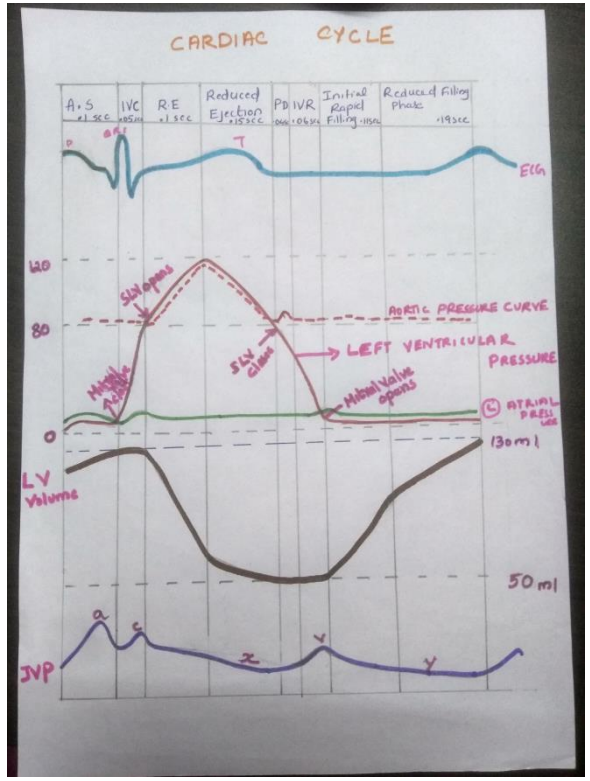
PROTODIASTOLE

- Ventricular relaxation starts
- During protodiastole SLV closes(VP just below aortic P)-2nd heart sound occurs
- Ventricular P decreases due to relaxation of ventricle
- At the end of protodiastole both AV valve & semilunar valve are closed ,ventricle begins to relax as a closed chamber



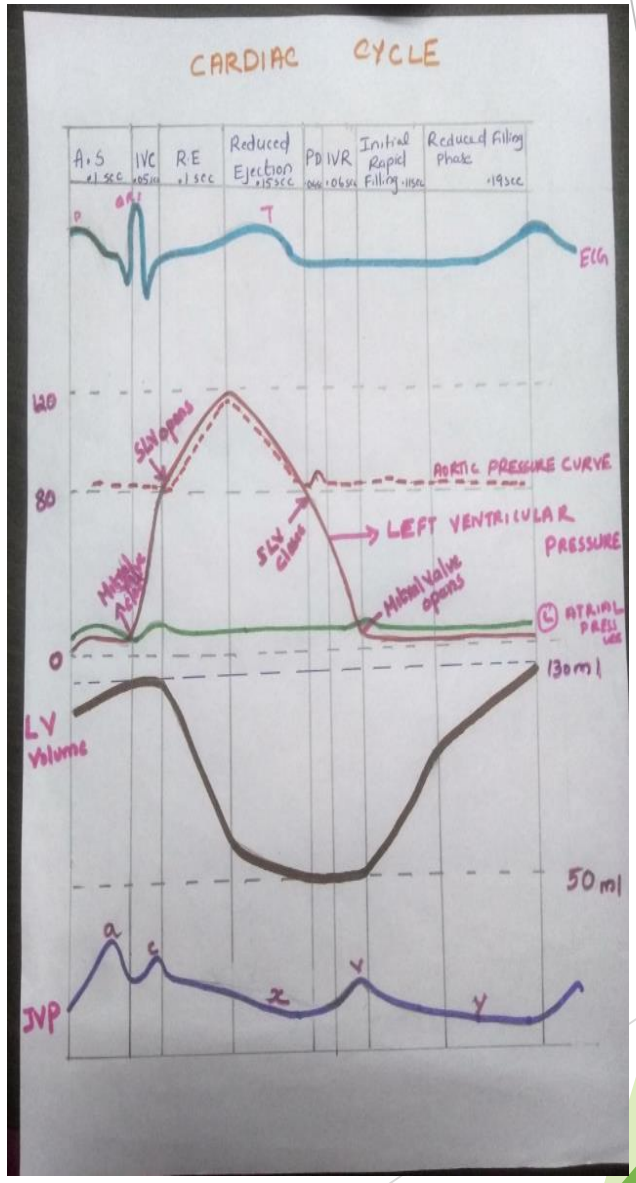
ISOVOLUMETRIC RELAXATION

- Ventricle relax as a closed chamber ,ventricular pressure falls to near 2-3mmHg
- Pressure in atria 4-5mm Hg at the end of IVR .this leads to opening of AV valve at the end of IVR(as VP below Atrial P)



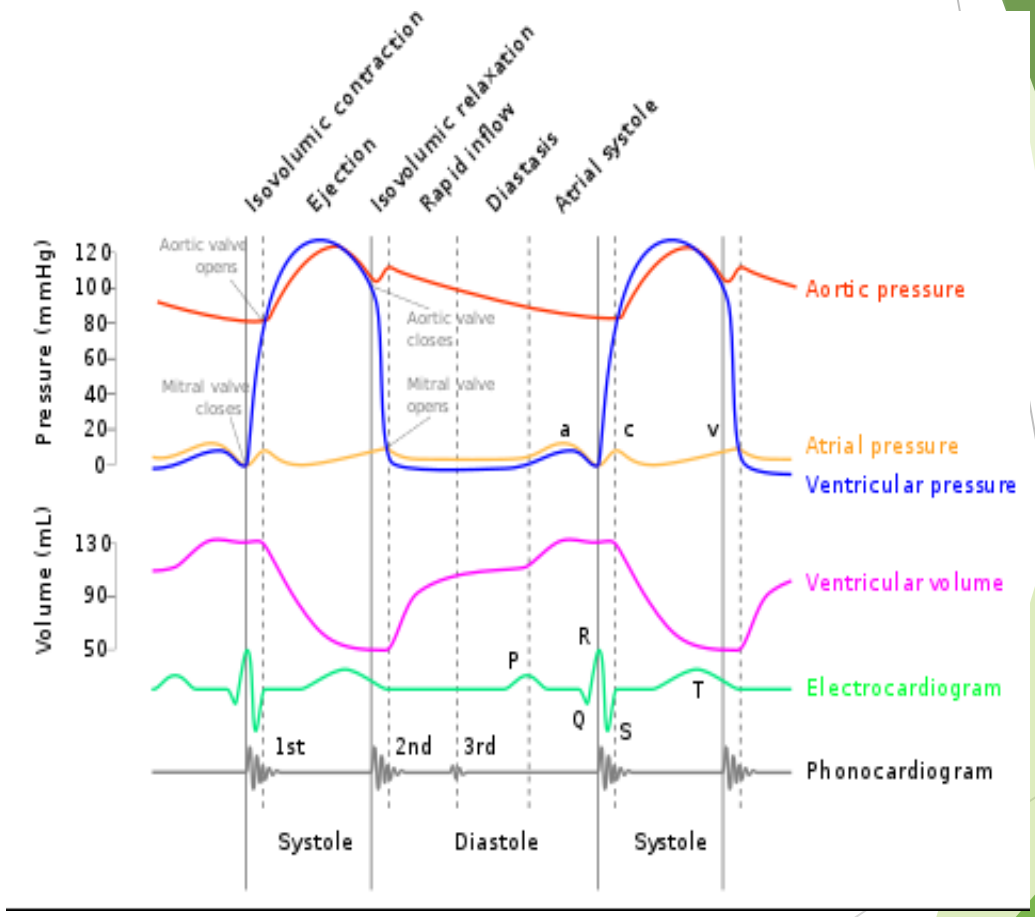
RAPID FILLING PHASE

- Due to increased pressure in atria blood flow to relaxing ventricle bcoz AV valve opened
- Atrial P decreases when AV opens(y decent),& then slowly increases
- Left Ventricular P decreases to near zero



REDUCED FILLING

- No turbulence & blood flow very slowly & smoothly
- Slow smooth ventricular filling is ‘diastasis’
- Ventricular pressure remain a little above zero



LAST RAPID FILLING

- Coincides with atrial systole
- End diastolic volume-volume of blood in the ventricle at the end ventricular diastole is 130ml

QUISCENT PERIOD

- Atrial systole occurs in last part of diastole only & all chambers of heart are in the relaxed state for a period of 0.4sec of cardiac cycle

VARIATION in duration of cardiac cycle

- Heart rate increases,duration of cardiac cycle decreases
- Slight increase in HR only diastasis affected
- If HR more than 180, diastole is shortened to a much greater degree
- Physiological & clinical significance

diastole heart rest& coronary blood flow to subendocardial portion of left ventricle

Ventricular filling occurs in diastole ,filling compromised—Cardiac output falls-cardiac failure

- HEART rate decreases--- diastolic period increases

- END DIASTOLIC VOLUME:amount of blood present in the ventricle at the end of diastole-130ml
- ENDSYSTOLIC VOLUME: after complete ejection ,some amount of blood remains in vrentricle - 50ml
- STROKE VOLUME:amount of blood ejected by each ventricle per beat 80ml

- EJECTION FRACTION:percentage of EDV ejected per heart beat-65%
- Measure of ventricular contractility

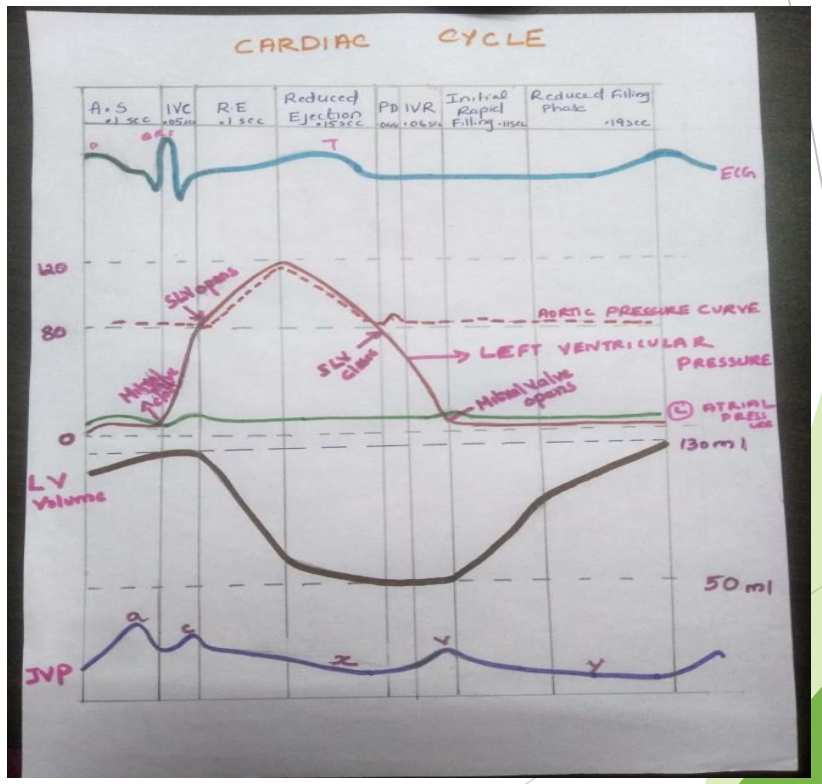
LEFT VENTRICULAR PRESSURE CHANGES

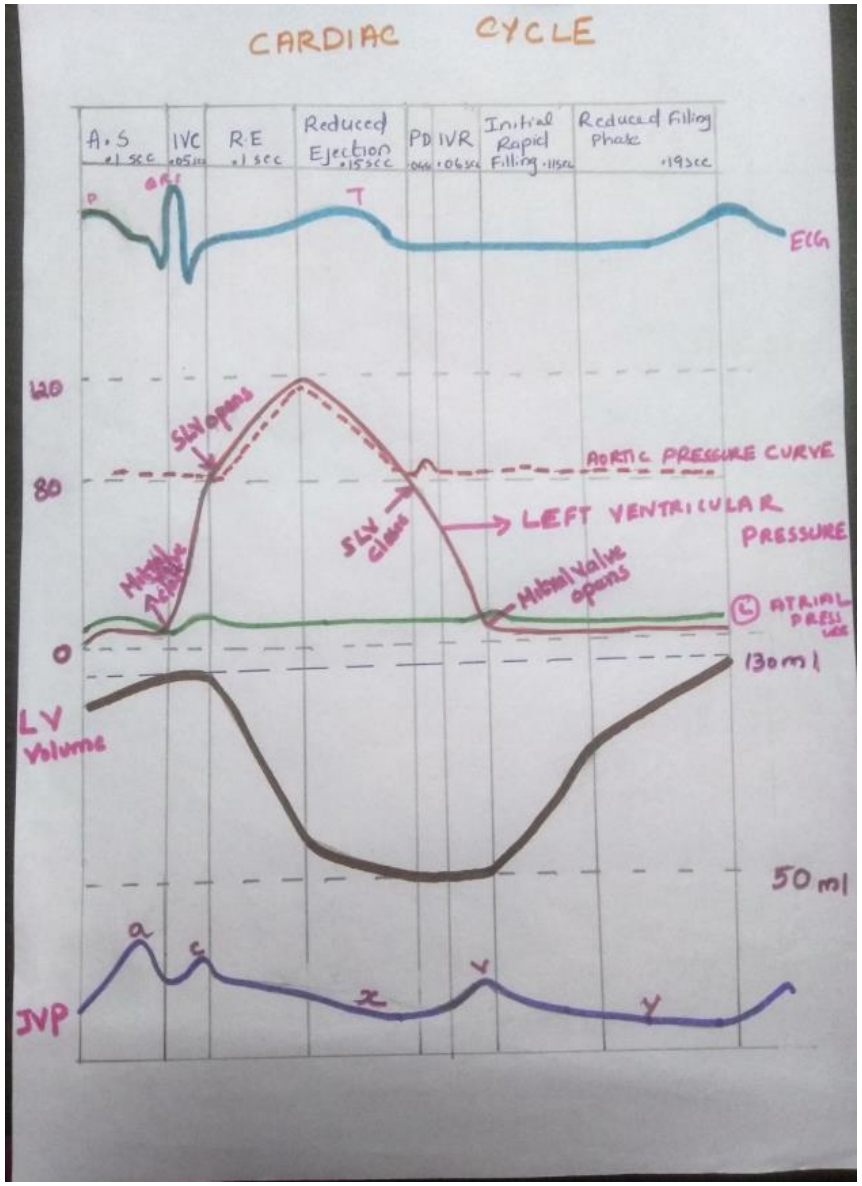
Intraventricular pressure can be measured by cardiac catheterization

ATRIAL SYSTOLE:

before AS (diastasis)pressure little above zero

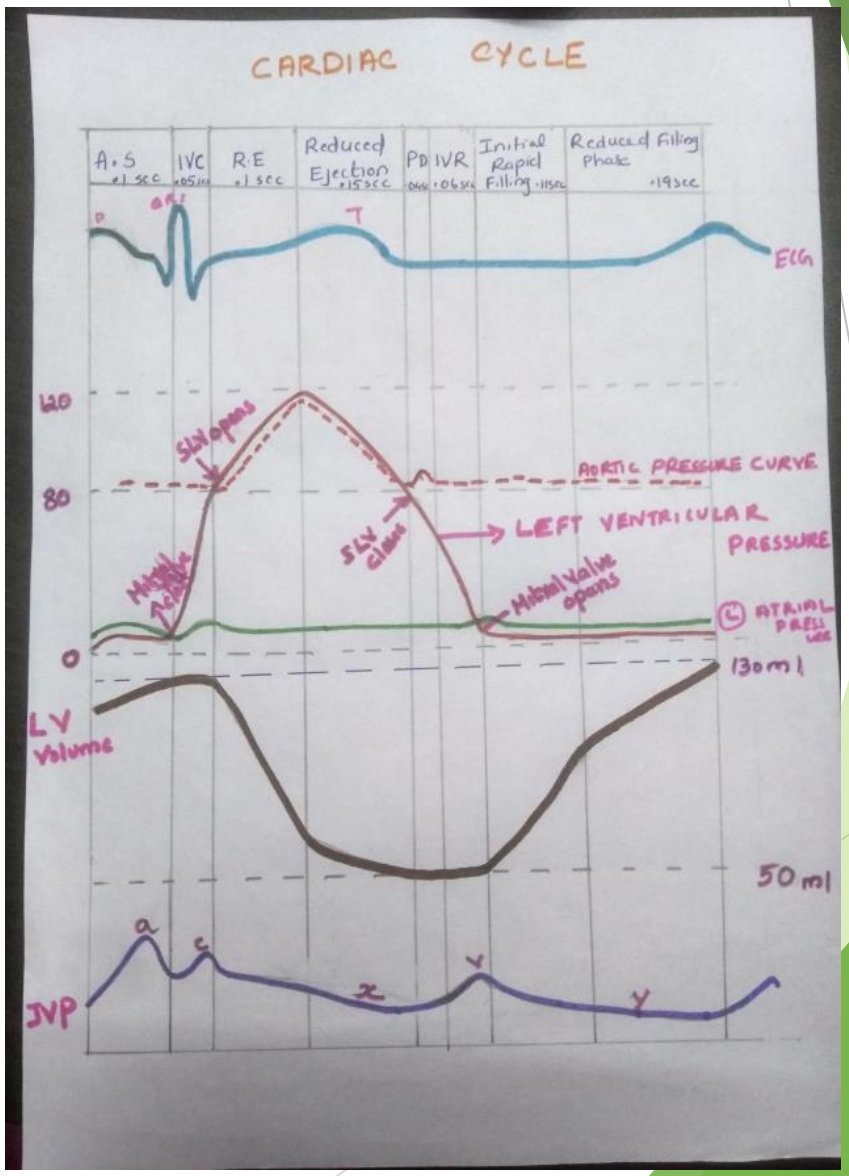
during AS ---increase in intraventricular pressure due to pumping of blood to the ventricles ---IVP becomes 7-8mm




CARDIAC CYCLE
A.S
IVC
R.E
Reduced Ejection
PD
IVR
Initial Rapid Filling
Reduced Filling Phase
ECG
120
80
0
SLV opens
AORTIC PRESSURE CURVE
SLV closes
LEFT VENTRICULAR PRESSURE
Mitral Valve opens
ATRIAL
130 ml
LV Volume
50 ml
JVP
a
c
x
v
y

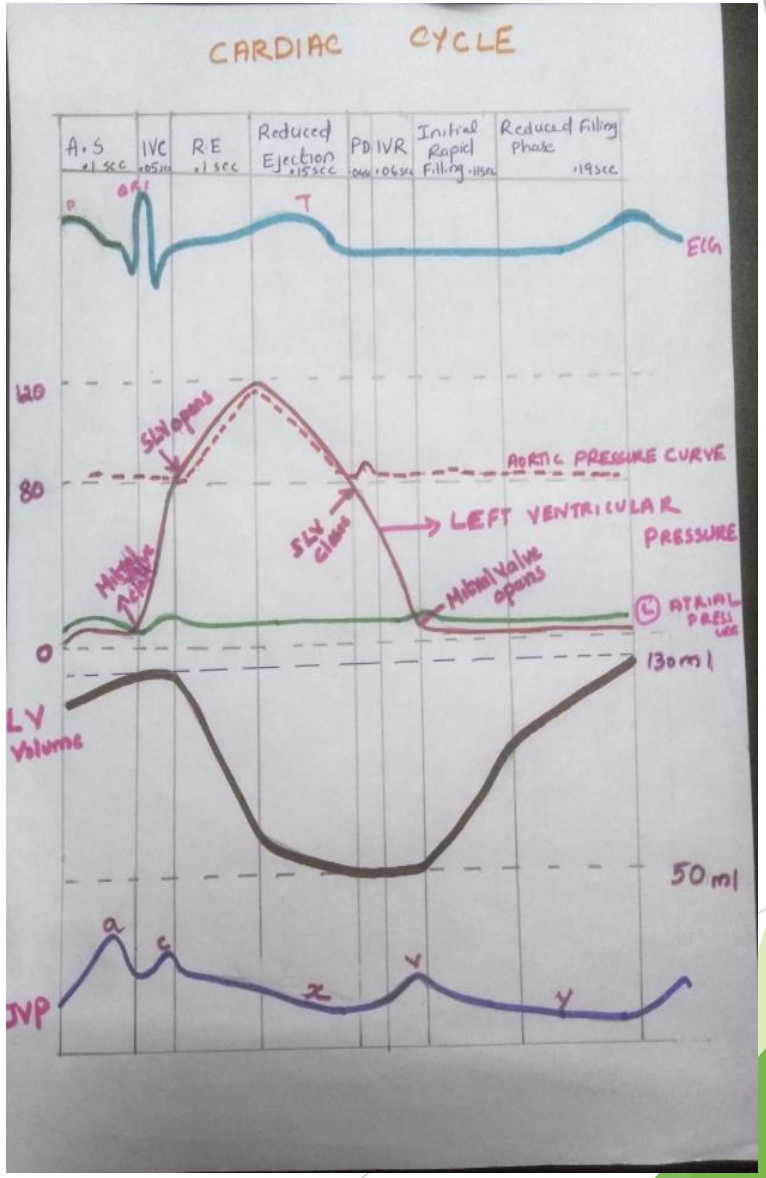
- **ISOVOLUMETRIC CONTRACTION PHASE**

ventricular pressure increase & becomes little above atrial pressure -AV closed----further increase in pressure due to contraction of ventricle & becomes little above aortic pressure (above 80mm)& semilunar valve open



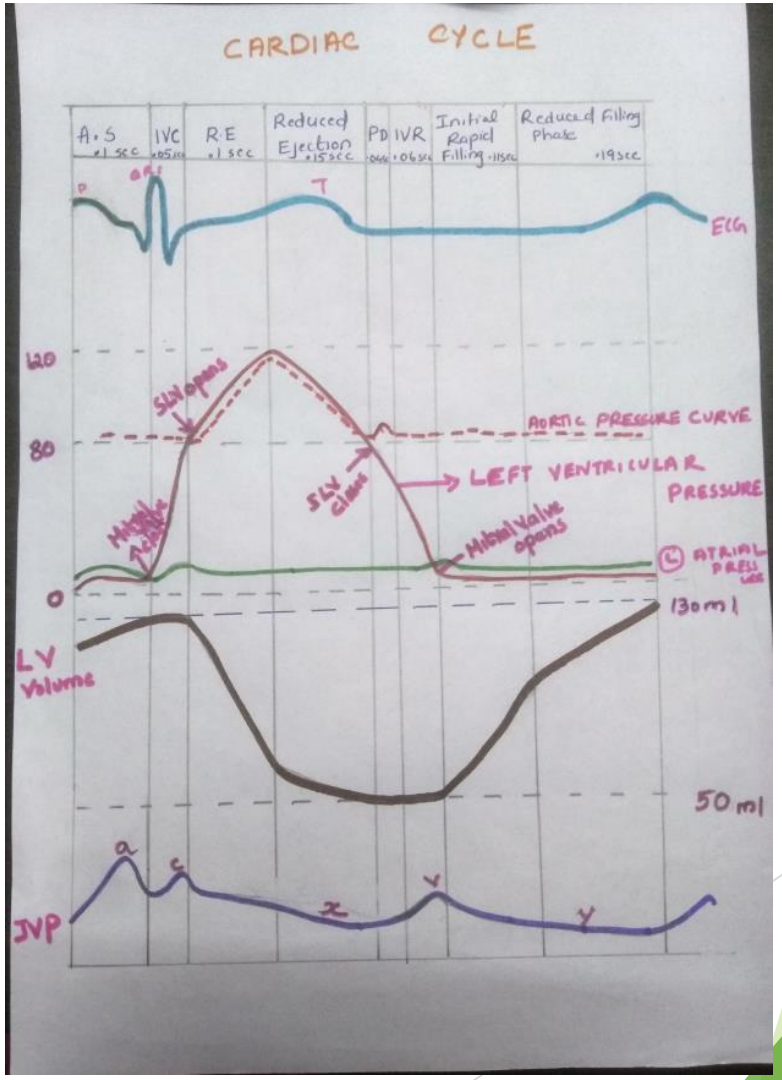
- **RAPID EJECTION PHASE**

  bcoz ventricular pressure is greater blood ejected into aorta & ventricle contract greater that the blood ejected,IVP rises to 120-130mmHg



- **SLOW EJECTION PHASE**

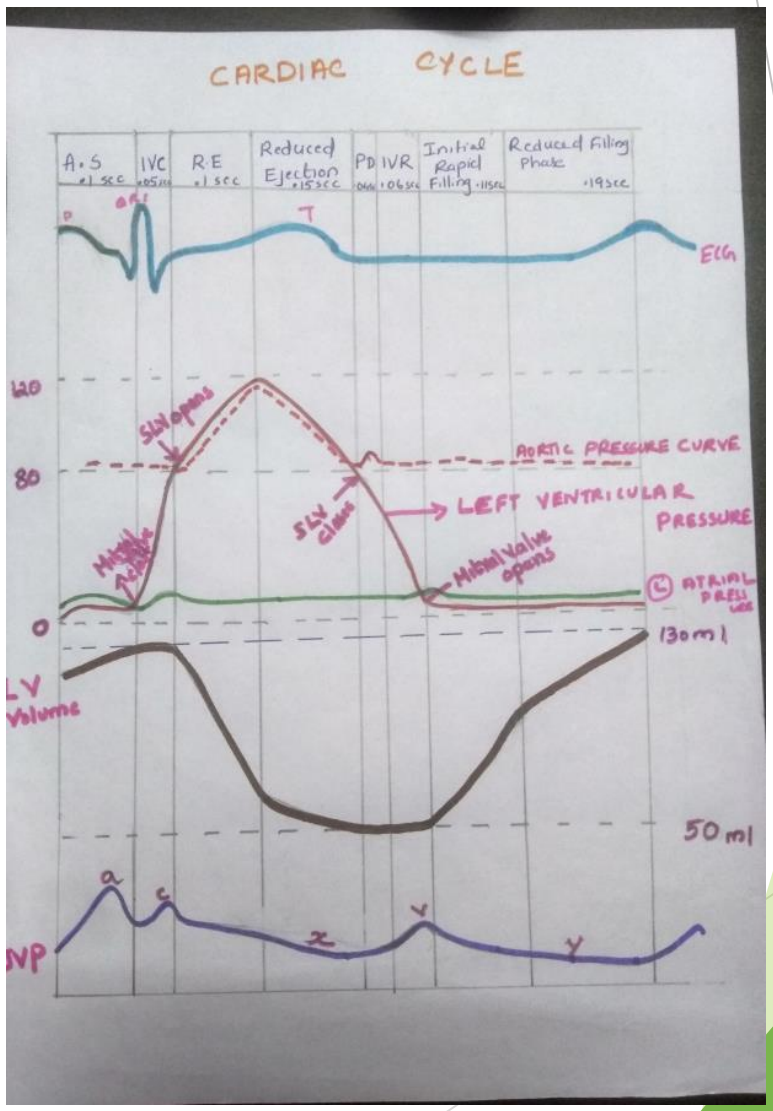
  due to less volume of blood,eventhough contracting VP decreases



VENTRICULAR DIASTOLE

- **PROTODIASTOLE**

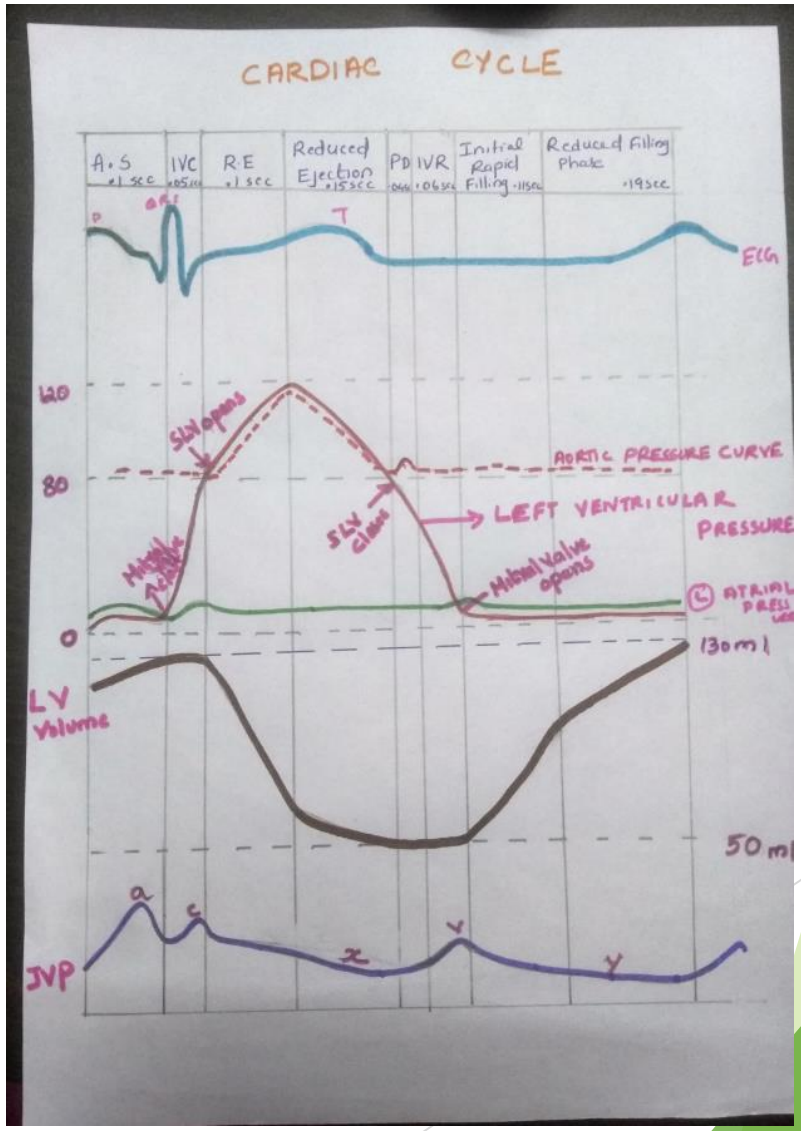
pressure falls below aortic pressure --- becomes less than 80mmHG) -semilunar valve closed



- ISOVOLUMETRIC RELAXATION PHASE

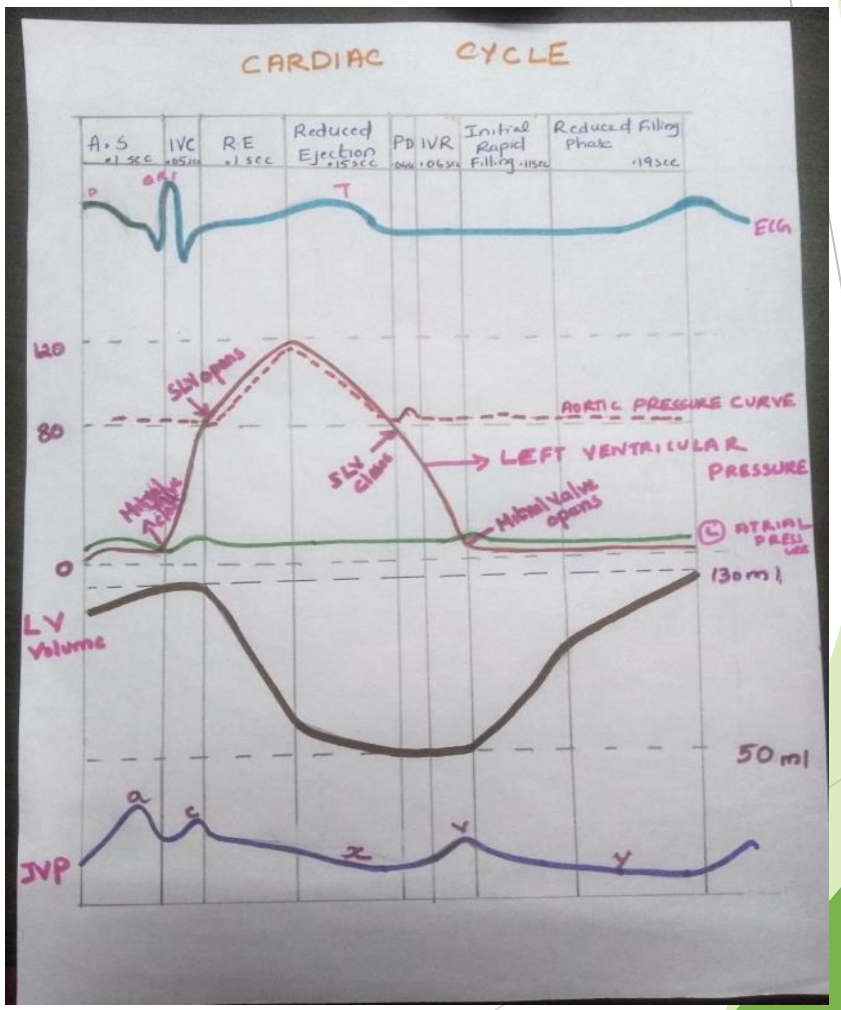
ventricle relaxing ---VP decreases from 80mmHg to 2-3mmHg.

When VP below atrial P ---AV valve open & rapid filling starts



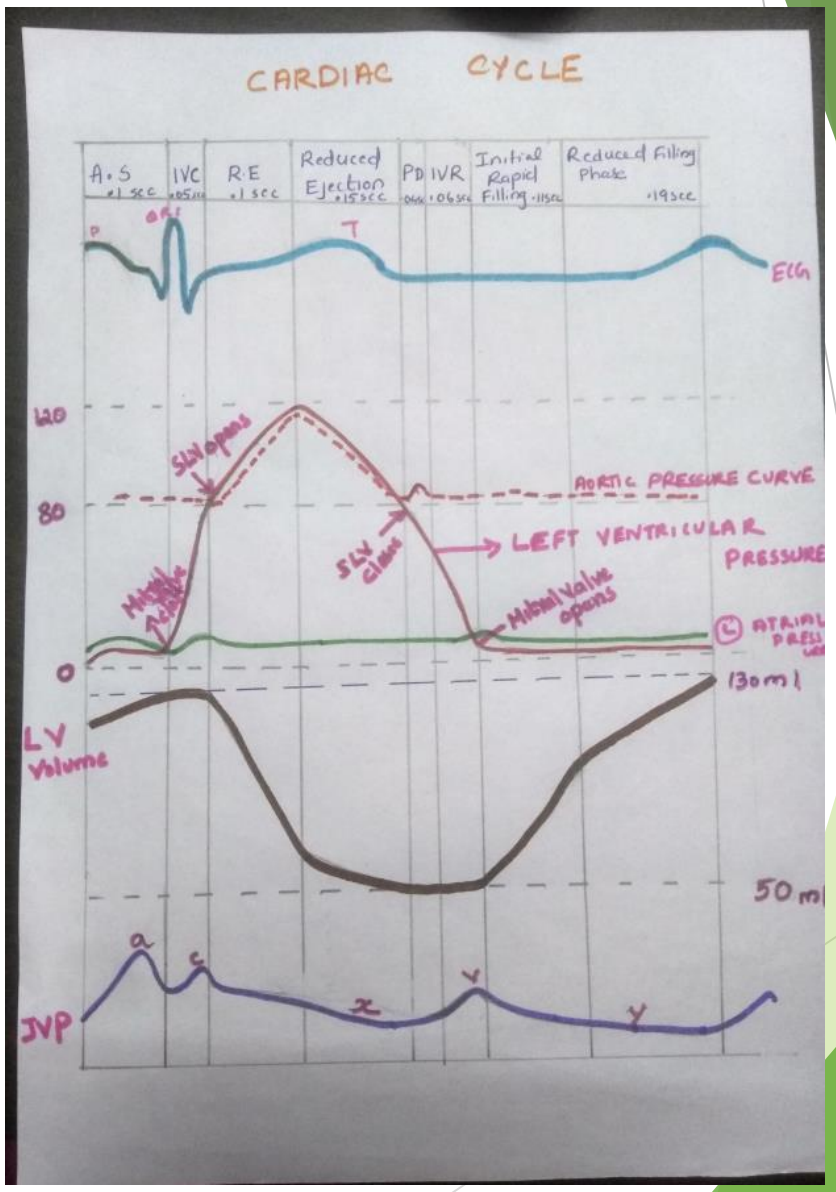
- **RAPID FILLING PHASE**

  ventricle is relaxing - so pressure decreases even though blood is filling



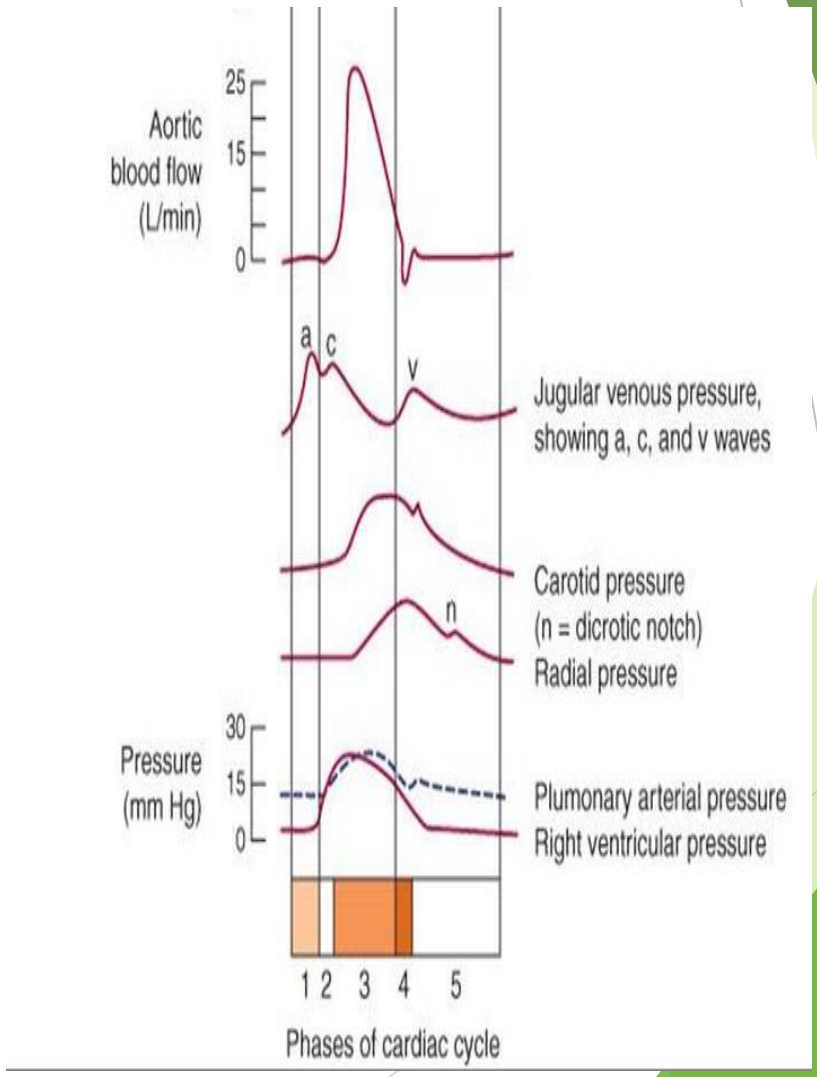
- **REDUCED FILLING or DIASTASIS**

Ventricular little above zero



RIGHT VENTRICULAR PRESSURE CHANGES

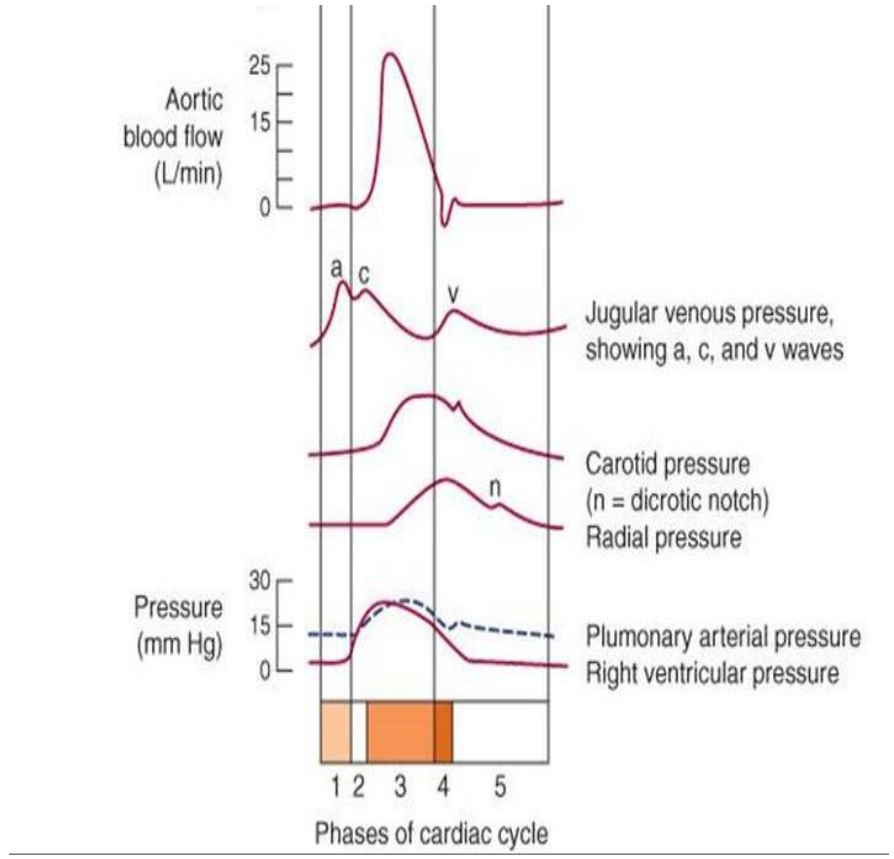
- Pressure changes similar to LV but less pressure bcoz LV wall thickness is more.
- Peak pressure is 25mmHG
- ATRIAL SYSTOLE: 6-7mmHg
- IVC -15mmHG (SLV opens when pressure exceeds pulmonary P(10-12mmHG)



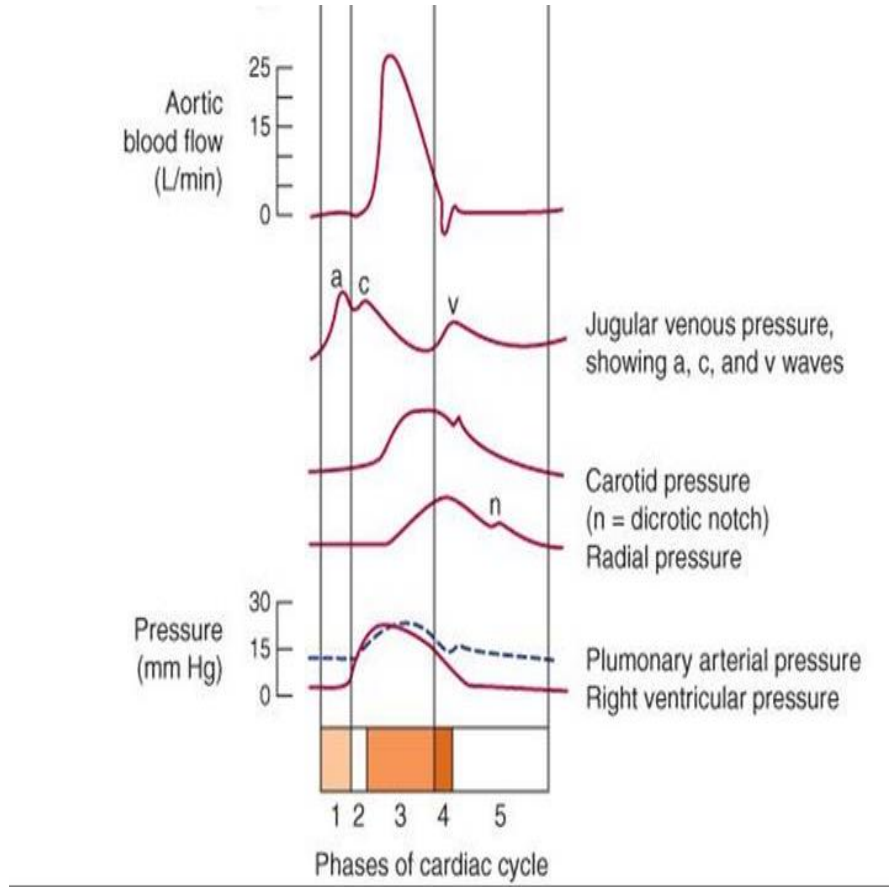
- **RAPID EJECTION**

Pressure increases & becomes 25mmHg

-PROTODIASTOLE:reduces becomes less than10mmHg less than pulmonary A --- SLV closes

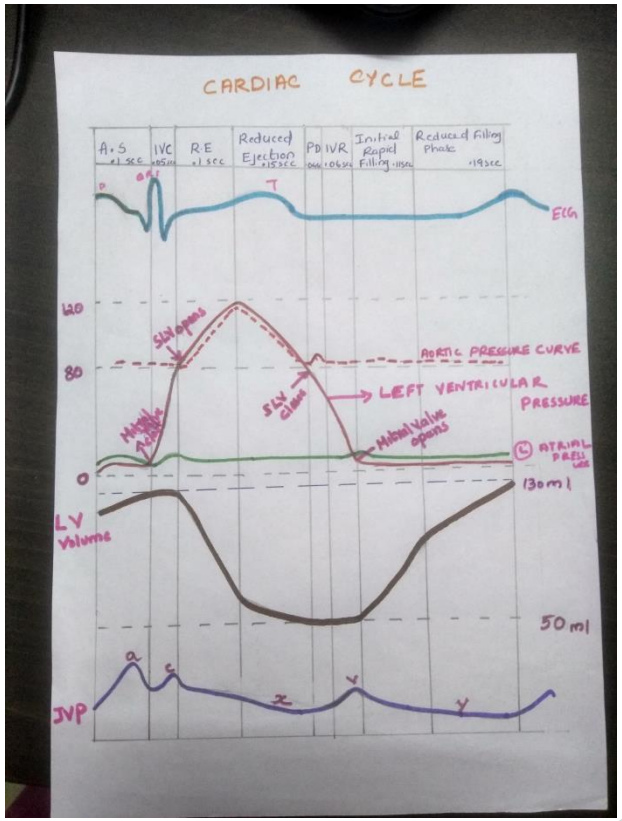


- IVR :pressure becomes 2mmHG
- Rest of the cycle:little above 0

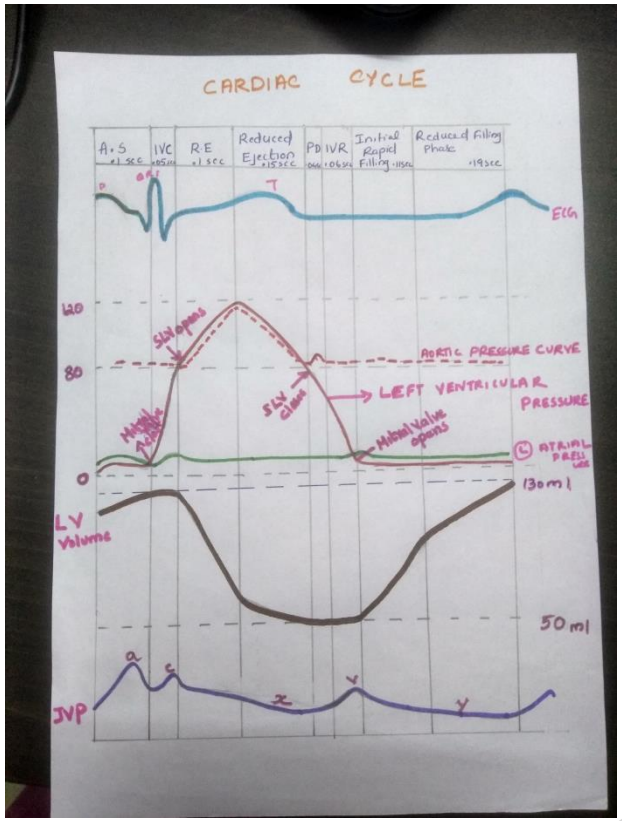


PRESSURE CHANGES IN AORTA

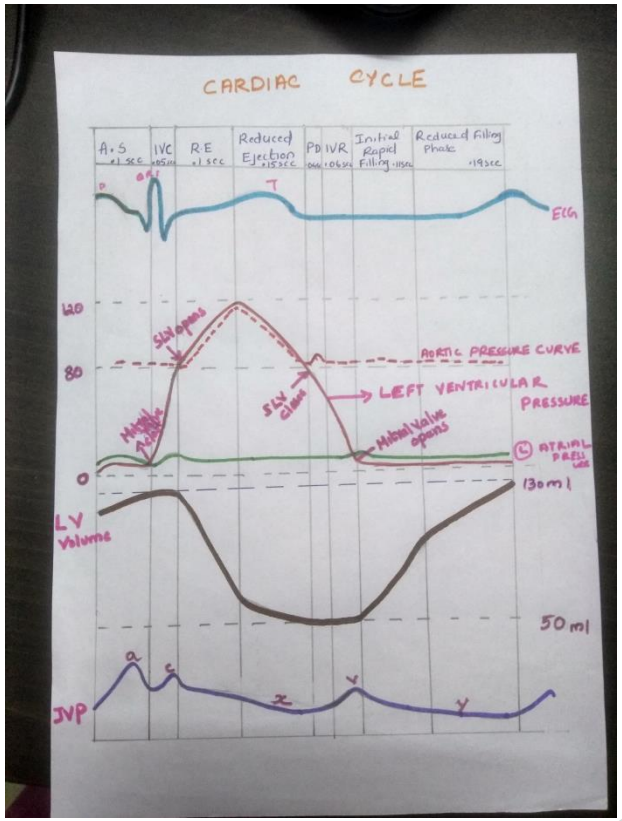
- Pressure changes in aorta varies between 80 to 120mmHg
- ATRIAL SYSTOLE:aortic pressure 80mmHg
- VENTRICULAR SYSTOLE:at first 80mmHg---ventricular pressure above 80mmHG -semilunar valve opens



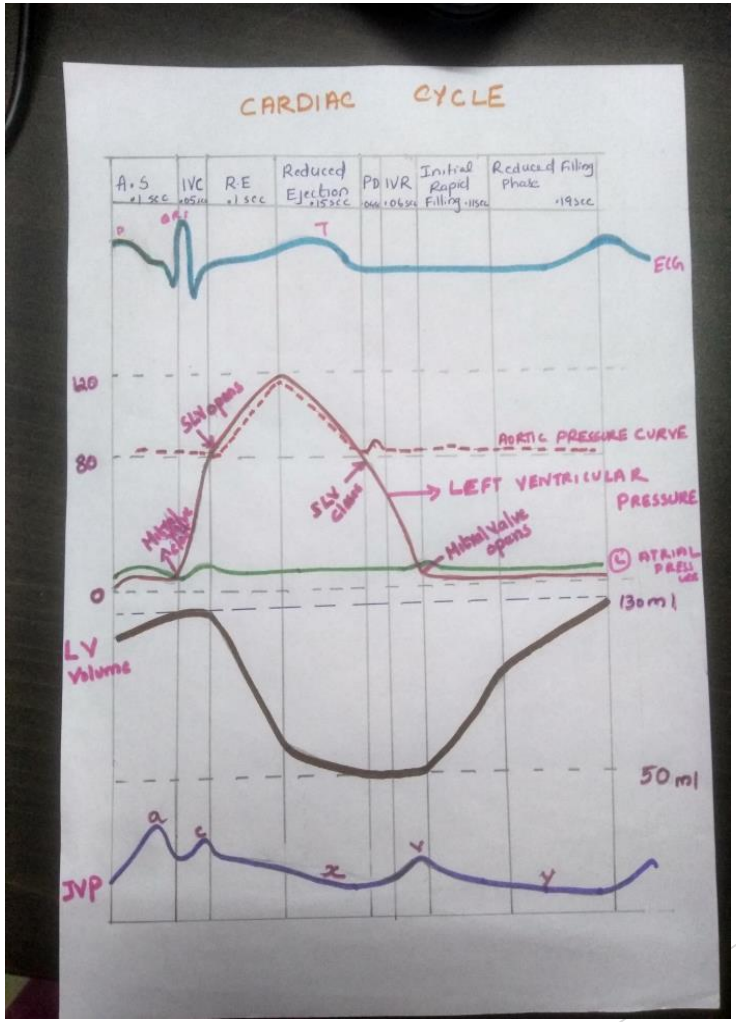
- RAPID EJECTION:blood flows to aorta----aortic pressure starts rising along with intraventricular pressure,but slightly less than IVP & becomes 120mmHg at the end of rapid ejection



- **REDUCED EJECTION:aortic pressure starts to decrease along with ventricular pressure**



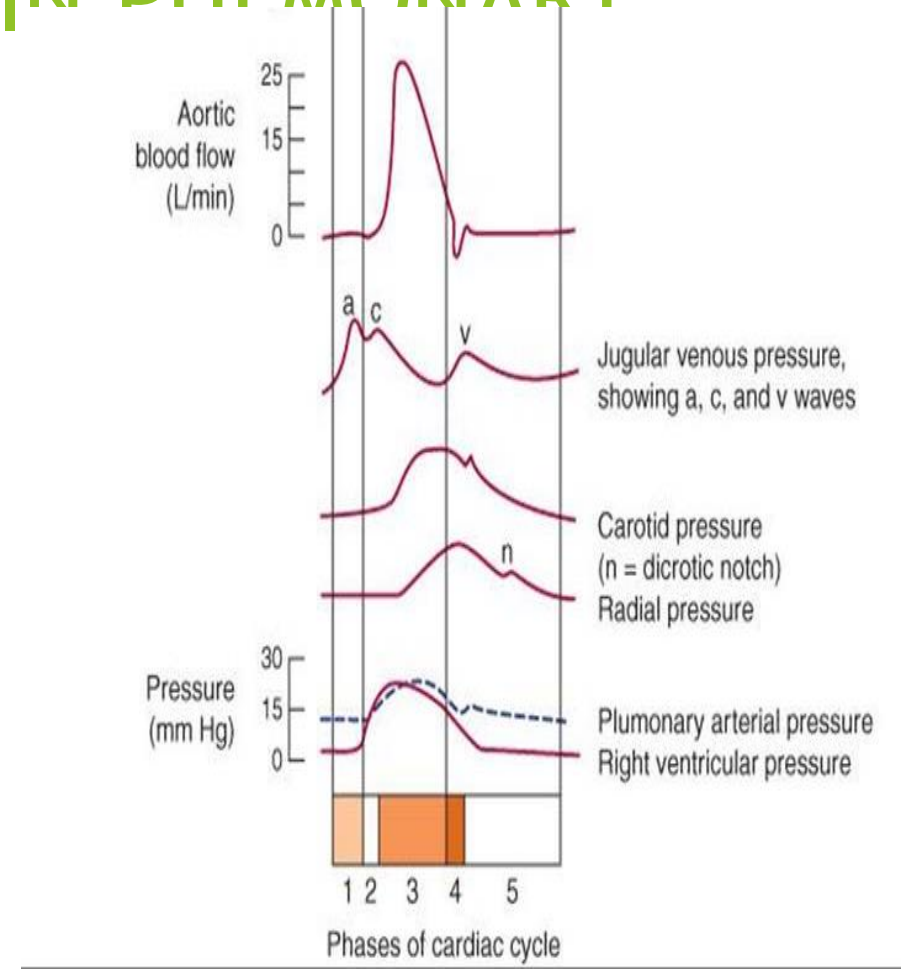
- PROTODIASTOLE:aortic pressure becomes 80mmHg ,slightly higher than LVP---causes backflow & closure of aortic semilunar valve---backflowing of blood collides against closed aortic valve----rise in aortic pressure---produces a notch INCISURA
- This sharp rise recordable even from peripheral arteries -DICROTIC NOTCH



- REST OF V
  DIASTOLE:aortic pressure declines to 80mmHg

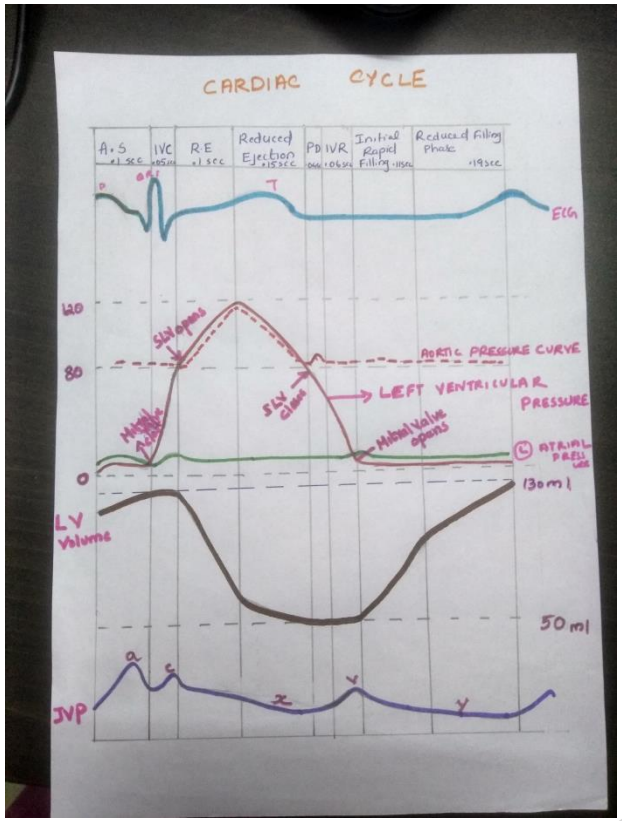
PRESSURE CHANGES IN PULMONARY ARTERY

- Pressure curve in pulmonary artery similar to aorta,but pressure is low
- Systolic 15-18mmHG
- Diastolic-8-10mmHG



VOLUME CHANGES IN THE VENTRICLE

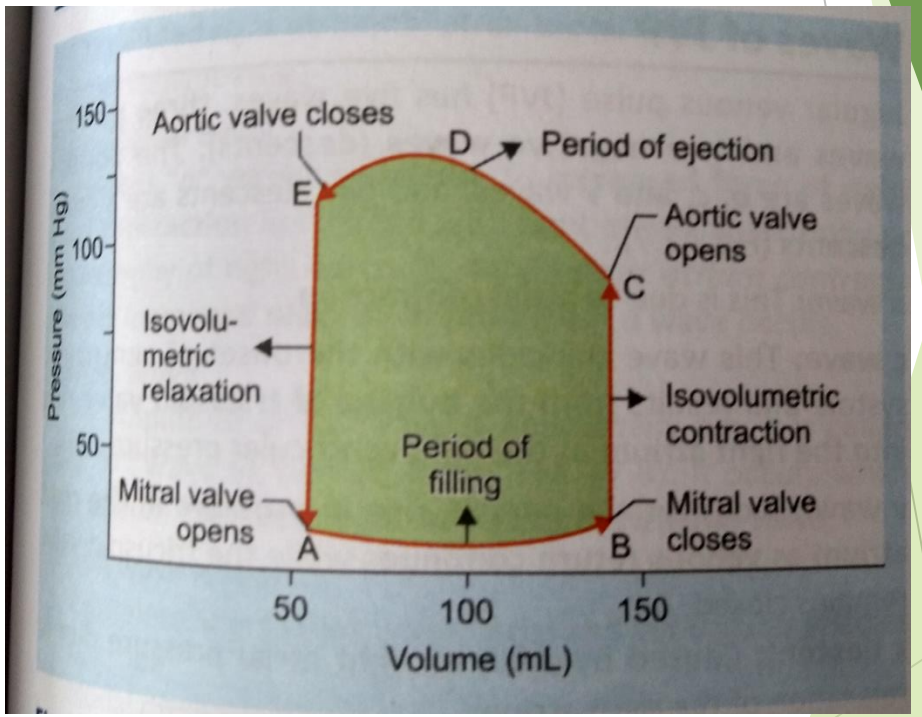
- ATRIAL SYSTOLE:30% of blood will enter the ventricle ,at the end of AS,ie EDV-blood is 130ml
- IVC-no change
- RAPID EJECTION-80ml ejected into aorta ----at the end of VS ,50ml left --ESV



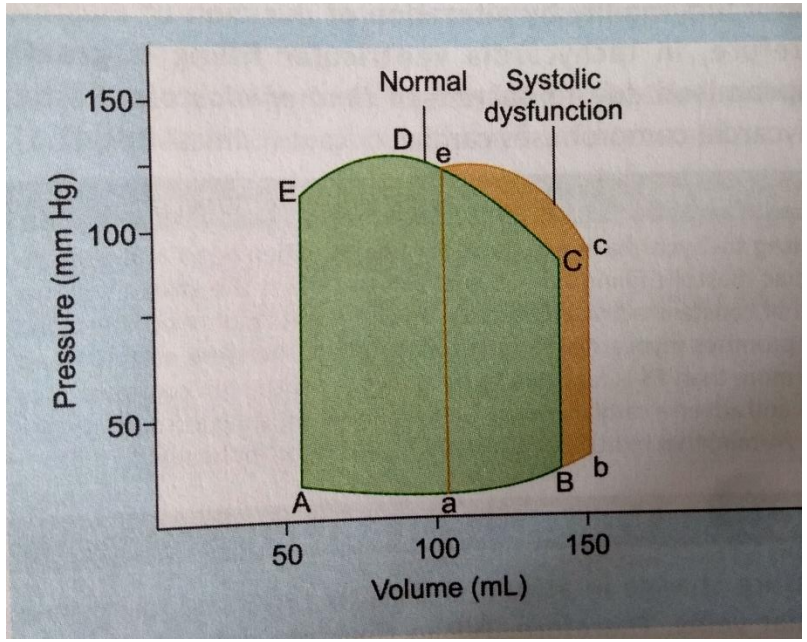
- IVR-no change in volume
- INITIAL RAPID FILLING & DIASTASIS:70% blood fill the ventricle

PRESSURE –VOLUME LOOP

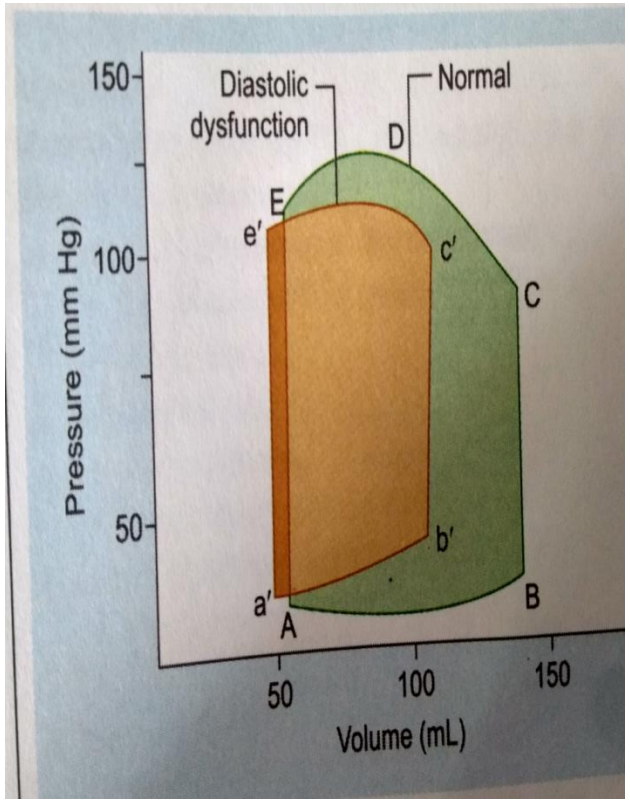
- Refers to graph obtained in the form of loop by plotting pressure in Y axis & volume in X axis



SYSTOLIC DYSFUNCTION



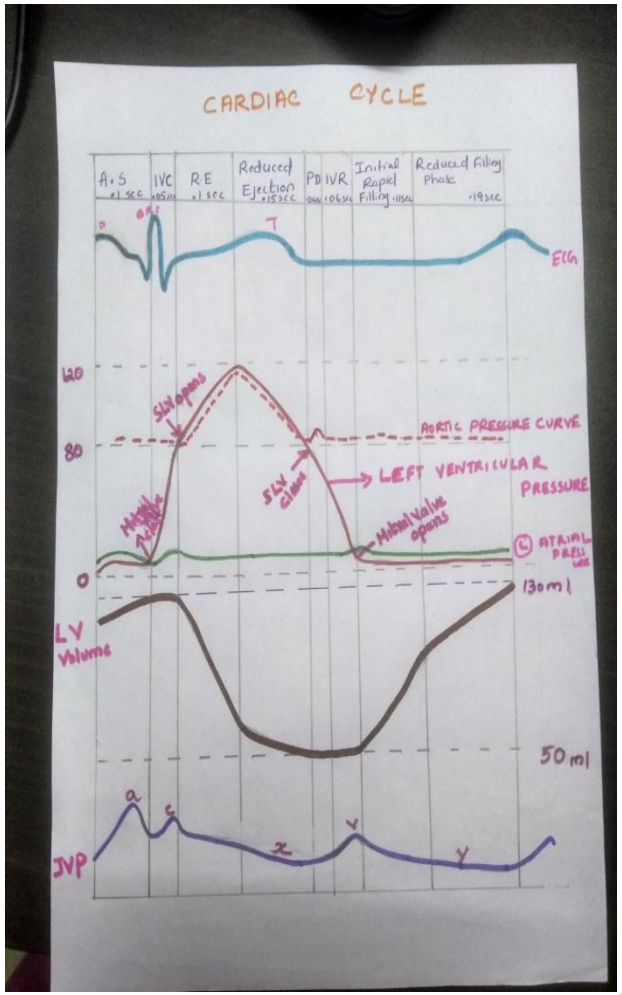
DIASTOLIC DYSFUNCTION



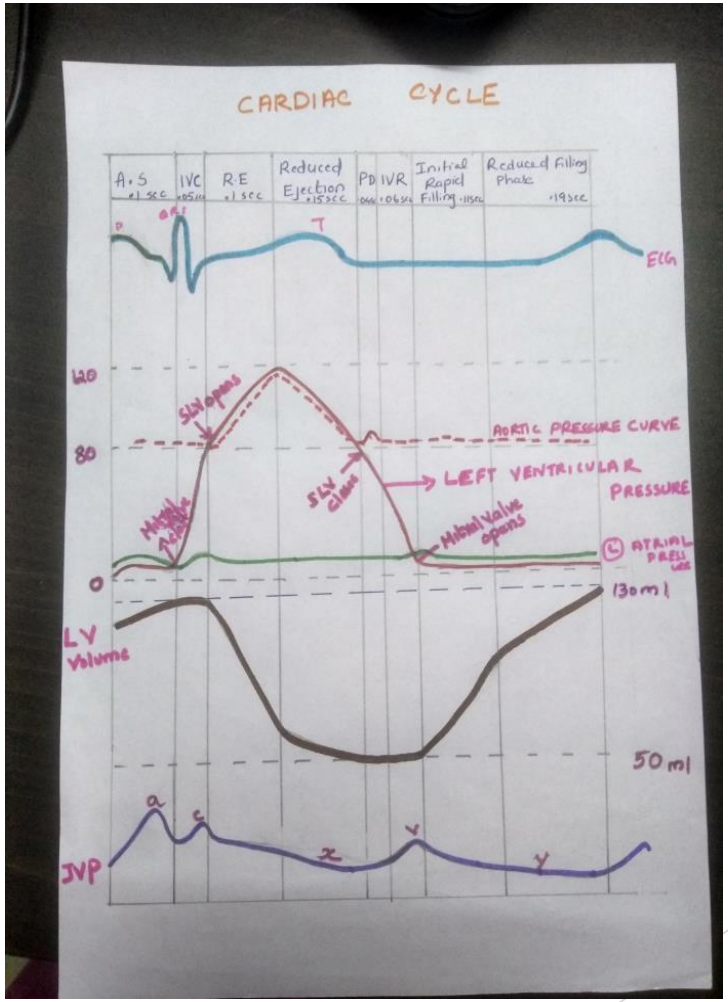
- **Diastolic dysfunction causes decrease in EDV**

PRESSURE CHANGES IN ATRIA

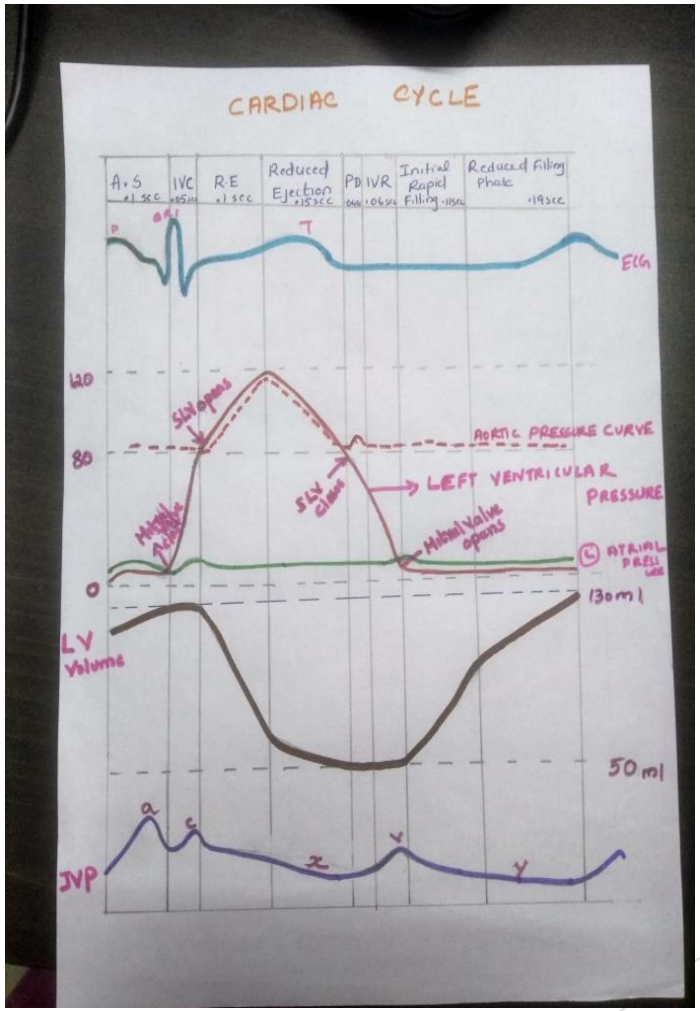
- Intraatrial pressure can be studied by intracardiac catheterization
- Left atrial pressure determined indirectly by measuring pulmonary capilliary wedge pressure
- Reflection of Right Atrial pressure is seen in jugular veins-JUGULAR VENOUS PRESSURE



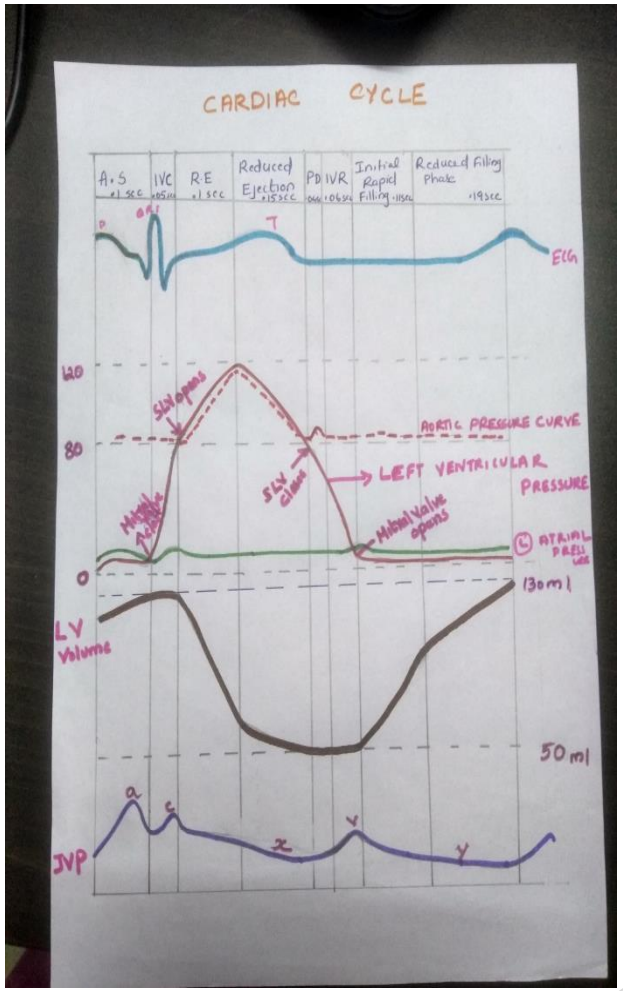
- ATRIAL SYSTOLE:before onset of AS ,intra atrial pressure slightly above zero & slightly greater than ventricular pressure
- During AS,atrial pressure rises 4-6mmHg (Rt)causes’ a wave.7-8mmHg(L)
- Immediately after atrial systole atrial pressure falls,bcoz atria starts relaxing



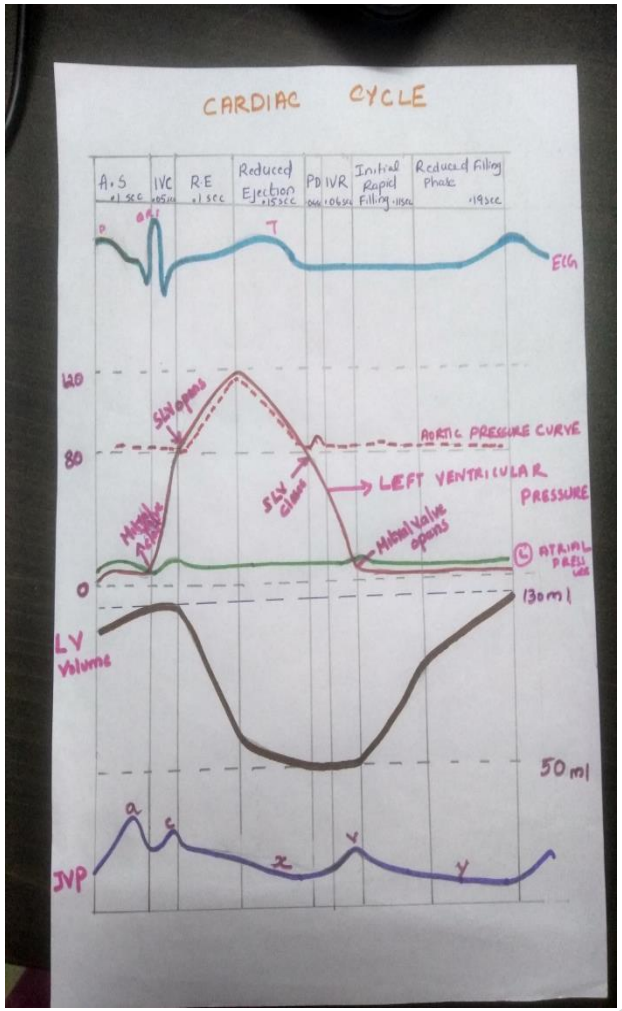
- **ISOVOLUMETRIC CONTRACTION:rise in intraventricular pressure causes AV valve to bulge into atria produces a small sharp rise in atrial pressure (C wave)**



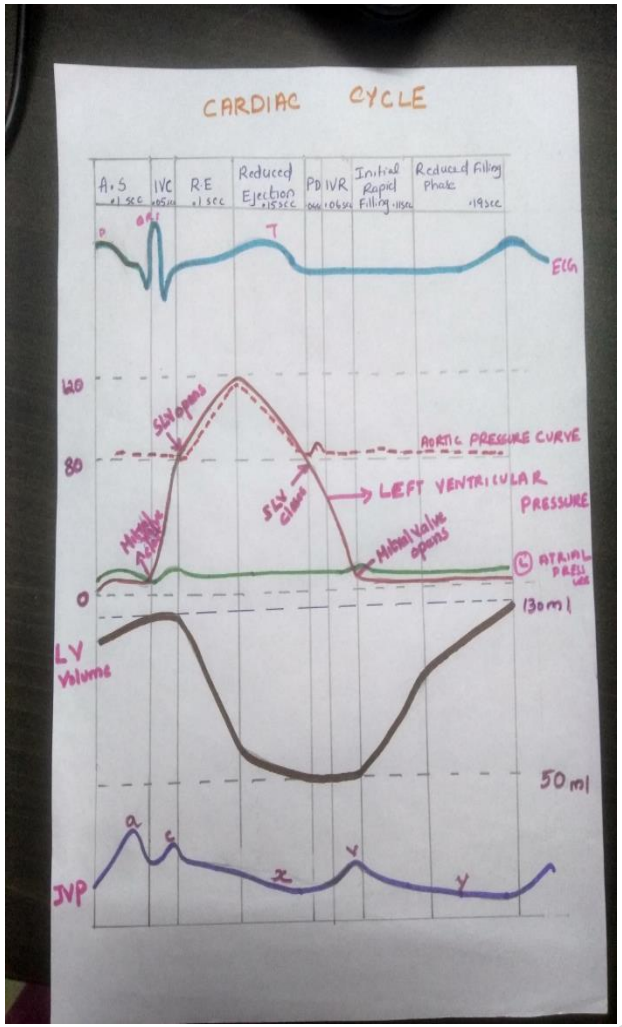
- EJECTION (RAPID):intra atrial pressure drops sharply in rapid ejection phase ,bcoz papillary muscles contract when the ventricular walls contract ---pull down the fibrous AV ring--- enlargement of atrial lumen---decreasing intra atrial pressure---X decent



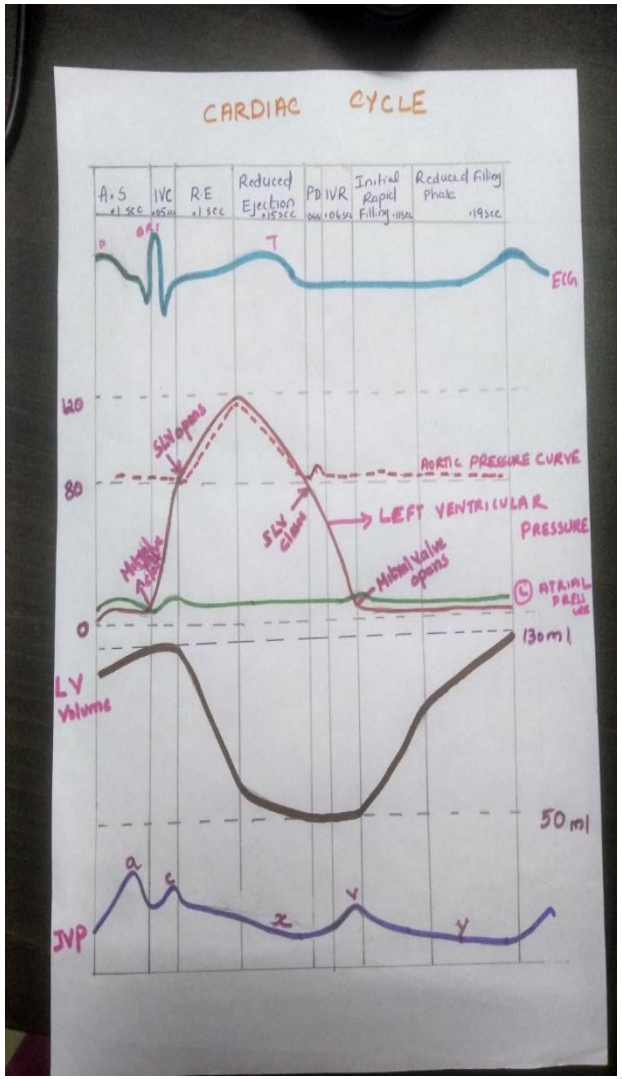
- **REDUCED EJECTION:as atria relaxing during this time,with blood flowing in from the great veins--- atrial pressure starts rising**



- ISOVOLUMETRIC RELAXATION:1.atrial pressure rise due to filling of blood in the atria when the AV valve remain closed ----v wave

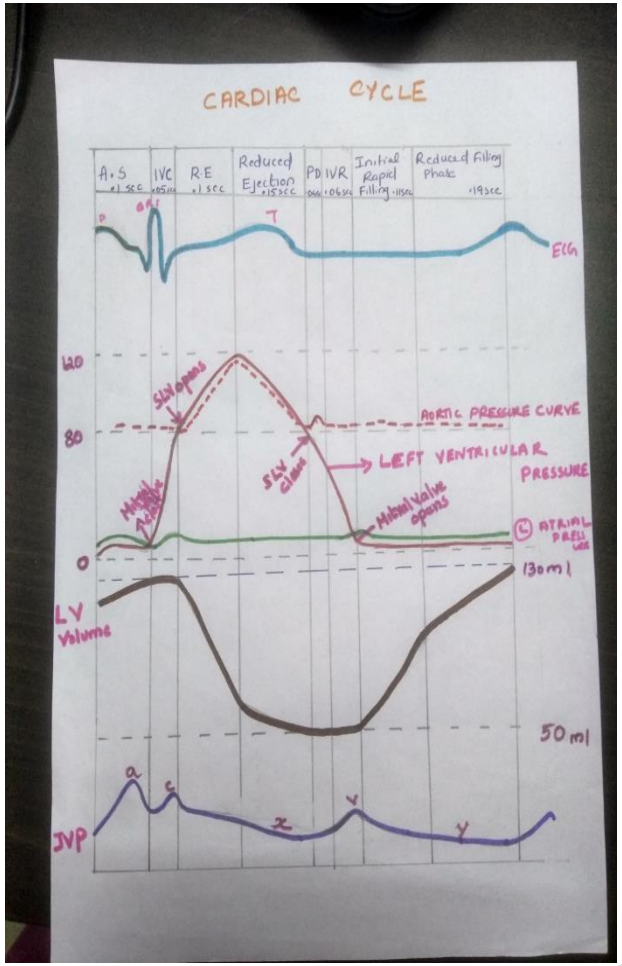


- RAPID FILLING PHASE:AV valve open & blood flow from atria to ventricle cause decrease in atrial pressure -atrialpressure drop sharply little above 0—Y decent



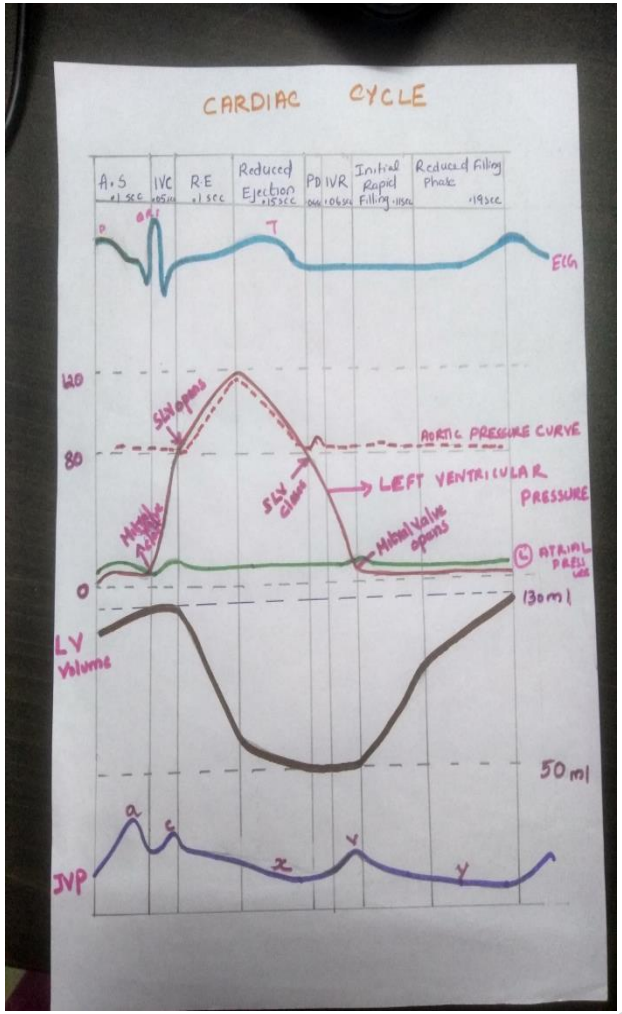
JUGULAR VENOUS PULSE

- Reflection of right atrial pressure seen in jugular vein
- Usually seen as pulse - jugular pulse
- Usually not seen above clavicle



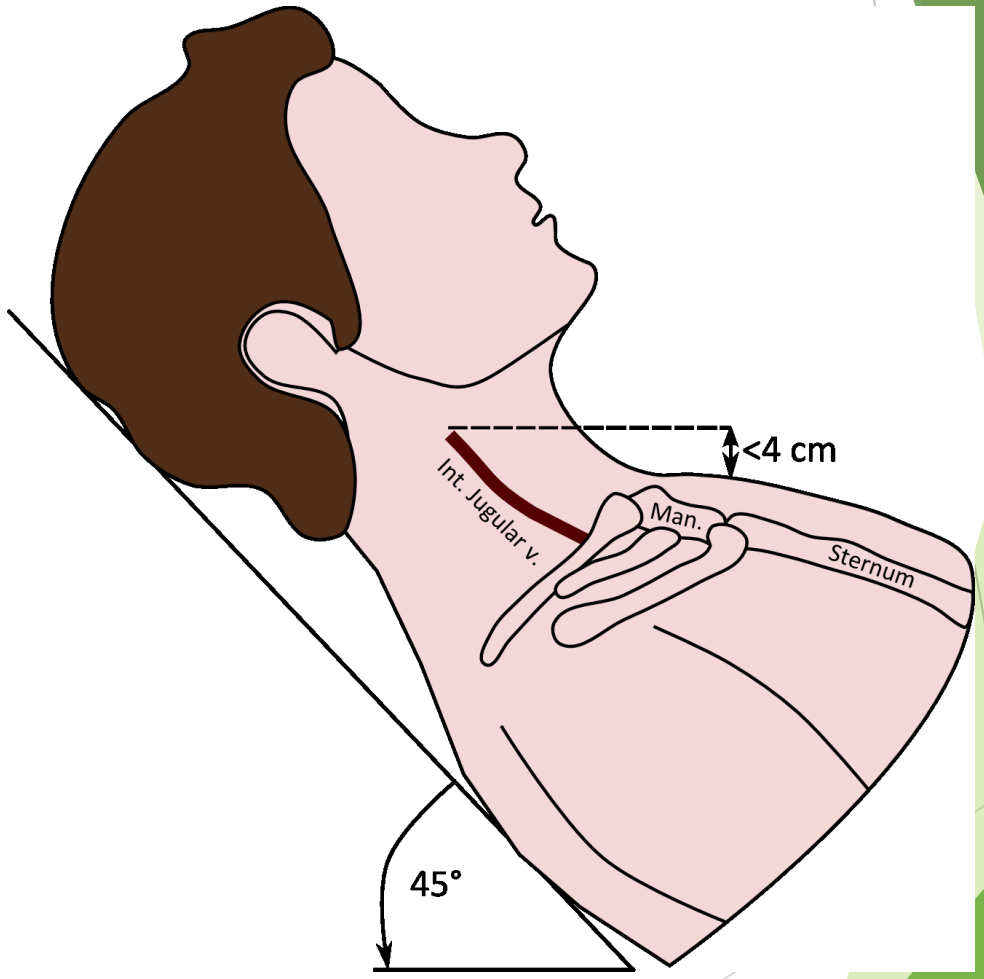
- Right atrial P are transmitted to great veins----right internal jugular vein reflects right atrial pressure
- Bcoz:it is in direct line with right atrium

  it has no valves

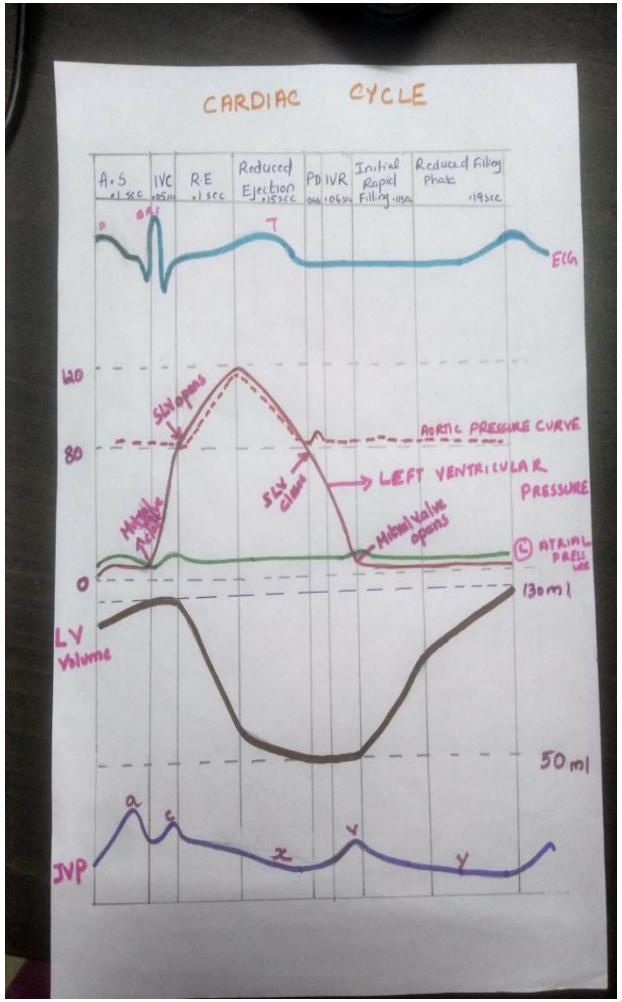


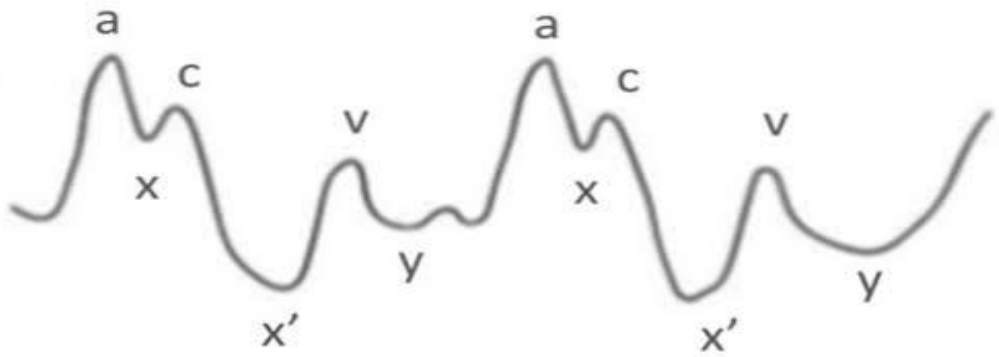
- Usually not seen above clavicle
- Visible in abnormal
- DEMONSTRATION:propped up position at 45degree inclination

vertical height from angleof louis to upper level of pulse—JVP



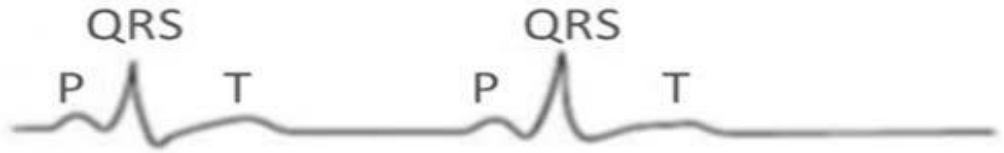
- 3 positive waves:a wave
  c wave
  v wave
- 2 negative waves:
  x decent
  y decent





Jugular venous pulse

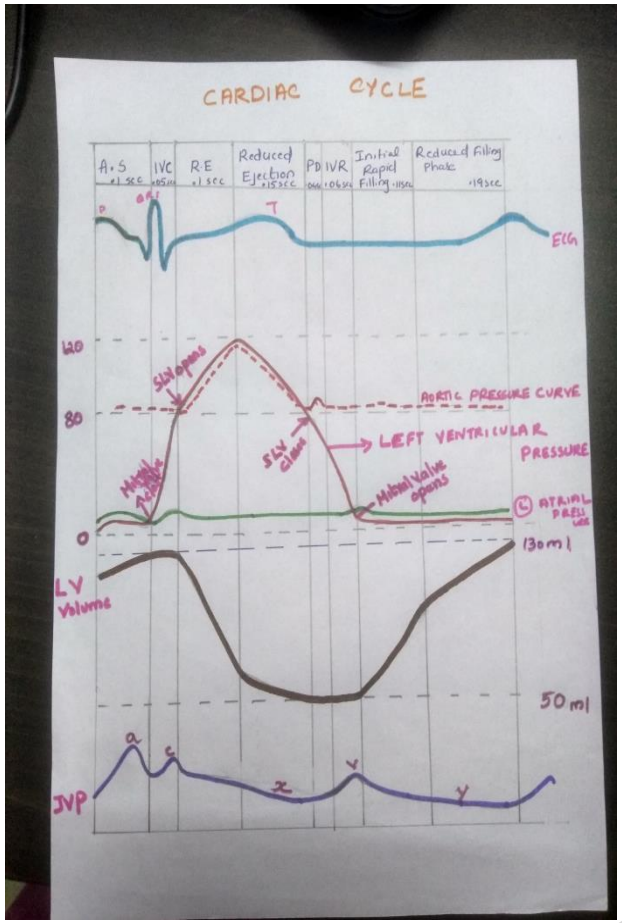
Phonocardiogram



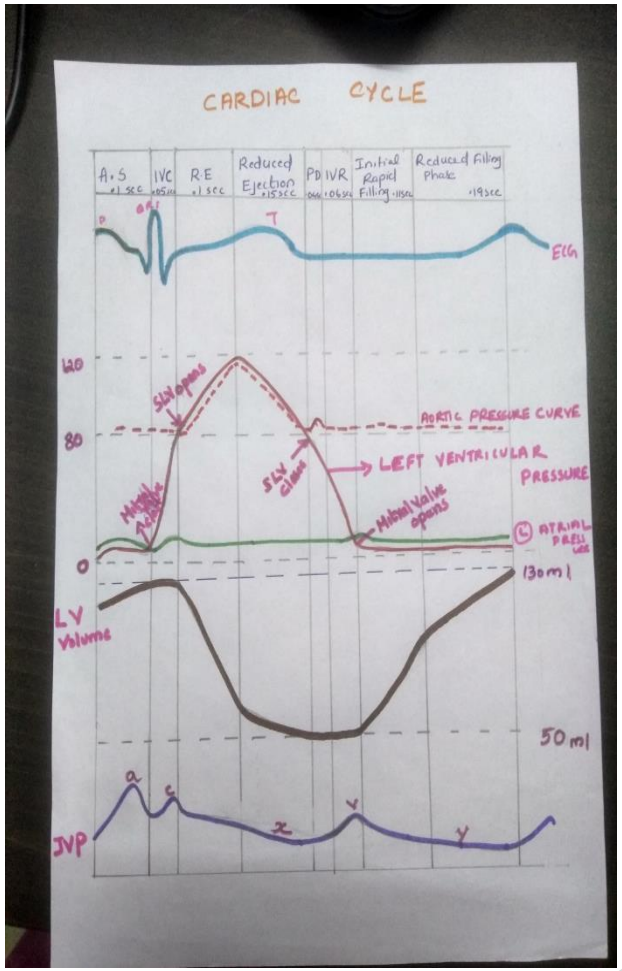
Electrocardiogram

- a wave: due to

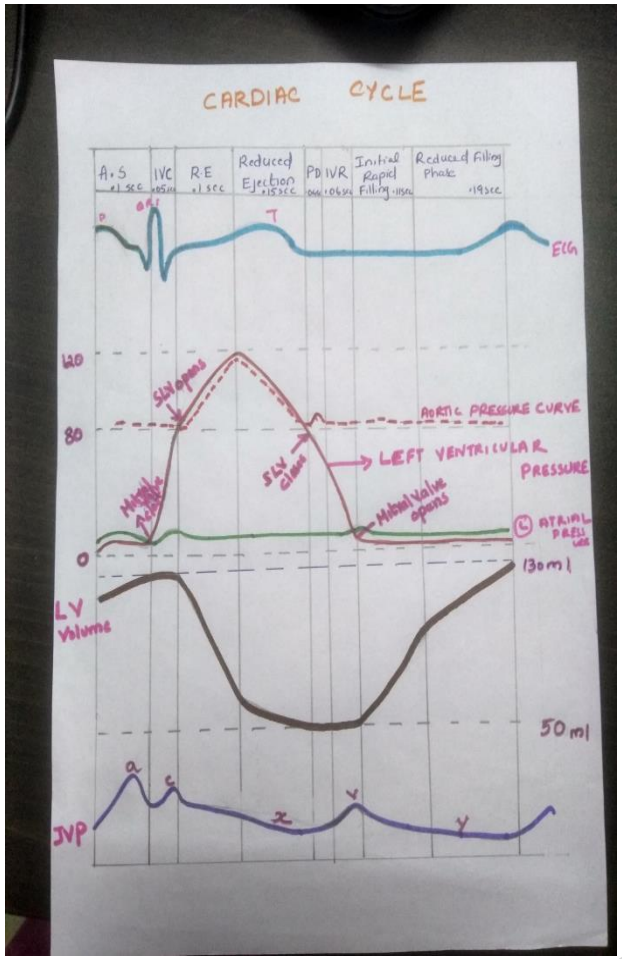
1. atrial systole & rise in P
2. regurgitation of blood into large veins due to atrial systole as no valves
3. Stagnation of blood in veins as blood cannot be emptied into atria as it is contracting



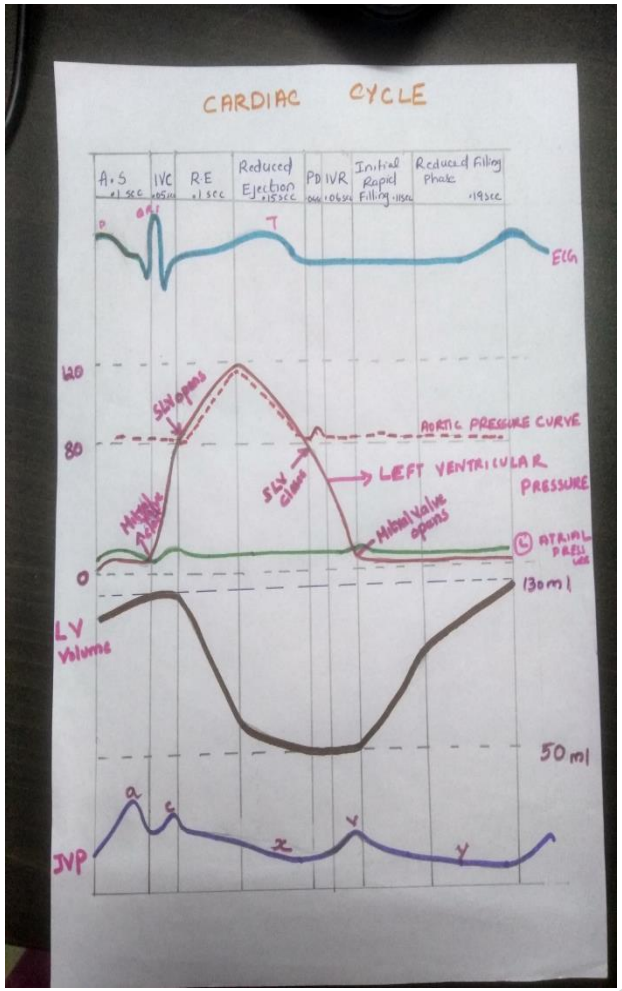
- c wave: due to pushing up of AV valve into atria during IVC
- x decent: due to pulling down of AV valves into ventricle ----enlargment of atrial lumen--- decrease in atrial pressure in rapid ejection phase



- V wave:due to filling of atria when the AV valve remain closed & rise in pressure in IVR
- Y decent:due to rapid flow of blood from atria to ventricle due to opening of AV valves



- Y decent:due to rapid flow of blood from atria to ventricle due to opening of AV valves



APPLIED

- **JVP elevated**:when right atrial P increased

  congestive heart failure

  tricuspid stenosis

a wave prominent

Tricuspid stenosis

Distended right atria

Complete heart block---Cannon wave

- **Absent a wave:** Atrial fibrillation---atria is not contracting as single unit

- Prominent v wave: Tricuspid regurgitation---rt atrial p increased due to regurgitation of blood from right ventricle

HEART SOUNDS

- are low frequency sounds that are heard during auscultation over the precordial region of the chest wall
- Produced during cardiac cycle
- Also heard by placing the ear directly over the chest wall
- Graphically recorded with phonocardiograph

- PRODUCTION:produced when valves are closed

  valvular, vascular & muscular vibrations are involved in the production of heart sounds
- FOUR HEART SOUNDS are produced during each cardiac cycle

  S1,S2 heard regularly

  S3 not easily audible

  S4 recorded by phonocardiograph only

- **AREAS OF AUSCULTATION:**

  Mitral area

  Tricuspid area

  Aortic area

  Pulmonary area

Heart sounds are best heard in these areas

S1-FIRST HEART SOUND

- **Soft low pitched** sound of relatively long duration .**15sec**, heard in **isovolumetric contraction** phase,frequency **25Hz**
- CAUSE:1)**valvular**-sudden closure of AV valves & vibrations of cusps & chordae tendinae

2) **vascular**: vibrations set up turbulence of the blood due to accelerations & decelerations caused by ventricular contractions

3)**Muscular:** vibrations set up in the ventricular muscle as it begins contracting from a relaxed state

- CHARACTERISTICS: low pitched ,sounds like LUBB,long duration
- INTENSITY:depends upon the force of contraction &tension developed in the ventricular muscle during isovolumetric contraction phase
- SITE OF AUSCULTATION: best heard in MITRAL & TRICUSPID AREA

- CORELATION WITH ECG & JVP: it coincides with spike of QRS complex of the ECG& just precedes c wave of the atrial pressure tracing & JVP
- COMPONENTS:S1 comprises two components ,the mitral (M1) which precedes the tricuspid (T1) by a few msec due to earlier closing of the mitral valve.splitting of S1 not normally heard

APPLIED

- Splitting of S1 widened & more apparent in complete right bundle branch block-when contraction of right ventricle is delayed
- REVERSE SPLITTING: in left bundle branch block (T1 precedes M1) due to delayed contraction of left ventricle

- Increased intensity of S1-exercise,anemia,ventricular hypertrophy
- Decreased –shock,pericrdial effusion,MI

SECOND HEART SOUND(S2)

- Shorter .12sec,sharper ,high pitched sound,sounds like DUBB,frequency 50Hz ,occurs during Protodiastole

- CAUSE:1.closure of semilunar valve &vibration of its cusps

2.vibration set up in the blood column of aorta & pulmonary artery

3 MUSCULAR -.Vibrations of in the walls of aorta & pulmonary artery

- **INTENSITY:depends upon blood pressure of aorta & pulmonary artery at the beginning of diastole**

- **CORELATION with ECG & JVP:coincides with protodiastole ,the upstroke V of jugular pulse tracing & end of T wave of ECG**

- COMPONENTS:Two components, AORTIC (A2) which preceds PULMONARY (P2) by a few secs-PHYSIOGICAL SPLITTING----P2 soft

- Aortic valve closes earlier than pulmonary valve due to pressure difference in them---pulmonary vascular resistance is 1/10 th systemic resistance, PV valve close later

-split is more apparent in inspiratory phase of respiration as more negative intrathoracic pressure venous return to R heart,R V takes more time to empty blood

APPLIED

- Splitting is widened in right bundle branch block
- Reverse splitting (P2 precedes A2) in left bundle branch block
- P2 louder than A2 in pulmonary hypertension

IMPORTANCE

- S1 marks beginning of ventricular systole
- S2 marks ending of ventricular systole
- Interval between S1 & S2 equals duration of ventricular systole
- Interval between S2 & S1 equals duration of ventricular diastole

THIRD HEART SOUND (S3)

- **CHARACTERISTICS: low pitched ,duration 0.1sec**

- **CAUSE: caused by vibrations set up in the ventricular walls & blood by either the rapid in rush of blood during the rapid filling phase or abrupt decline in the rate of blood flow immediately after the rapid filling phase.**

- CORRELATION with ECG: S3 appears between T & P waves of ECG
- SITE: can be heard at apex ,in children & probably in pregnancy ,not in adults normally

APPLIED

- **Heard in mitral regurgitation,LVF -increase in volume & rate of ventricular filling**

GALLOP RHYTHM

- When S3 is audible ,the rhythm of HS is like the gentle gallop of a horse called GALLOP or TRIPLE RHYTHM
- Eg in LVF,in pregnancy

FOURTH HEART SOUND(S4)

- **CHARACTERISTICS: short ,atrial sound heard before S1 duration-0.03 sec, normally not audiable**
- **CAUSE:by strong atrial contractions & is due to vibrations of atrial walls & blood rushing into ventricles in atrial systole**
- **CORRELATION with ECG:coincides with interval between the end of P wave & Q wave**

APPLIED

- when atrial pressure is high ,it is heard before S1
- It is heard in abnormal condition with hypertrophy & stiffness of atrium & ventricle----HYPERTENSION

CLINICAL SIGNIFICANCE of HS

- **Variation in HS,loudness, length,interval between sounds, splitting of sounds,additional sounds & murmurs gives valuable information in diagnosing heart sounds**

MURMERS

- Are adventitious abnormal sounds heard over precordium & blood vessels
- Murmers is heard over heart
- Bruit is heard over blood vessels

- CAUSE: blood flow is laminar ,non turbulent & silent upto a critical velocity ,above the velocity blood flow is turblent & creats sound

1. Blood flow speeds up when an artery or heart valve is narrowed-stenosis
2. Sound is heard when valve is not closed properly,incompetent & blood flows backward-regurgitation
3. Abnormal connection between Rt & Lt side eg:ASD,VSD,PDA
4. Acclerelated flows-hyperdynamic circulation-anemia

- BASED ON TIMING ,Murmurs are classified into

1.Systolic murmurs-PS,,AS,Anemia,MR TR (pansystolic)

2.Diastolic murmurs-TS,AR,MS,PR

3.Continous murmurs-PDA

- **BASED ON CAUSE,MURMERS are classified**

1.Valvular murmur

2.Hemic murmur

3.Anerysmic murmur

- Anemia –systolic murmur-due to reduced viscocity & increase in cardiac output producing vibrations of the blood due to turblence of flow

MITRAL stenosis

- Most common valvular lesion
- Caused by rheumatic heart disease due to streptococcal infection
- Antibodies are formed against valves & inflammatory lesion develop in valves----after healing scar tissue develop & willnot close properly----stenosis
- MS- late diastolic murmur

CARDIAC OUTPUT

- Defined as the volume of blood ejected by each ventricle in one minute.
- Also called minute volume
- Normal value ---5-6L/minute
- Right sided output &left side are equal ,ie CO equal to pulmonary outflow

- C.O = STROKE VOLUME X HEART RATE= 70X 72=5L/mt

- **STROKE VOLUME:** output per ventricle /beat=70ml

- **CARDIAC INDEX= C.O / surface area**

  C.I= 3.2L/m2/mt

  -Better index ---CO varies with the size of the individuals

DISTRIBUTION OF CARDIAC OUTPUT

- 75% distributed to the vital organs,25% skeletal muscle, skin & other organs

CARDIAC RESERVE

- maximum increase in the cardiac output above the normal value

- For healthy adults 300-400%
- Old age -200-250%
- Athletes---500-600%
- C R maximum during exercise & minimum during cardiac diseases

MEASUREMENT OF CARDIAC OUTPUT

- **By using FICK'S PRINCIPLE**
- **ECHOCARDIOGRAPHY**

FICK'S PRINCIPLE

- States that amount of substances taken up by an organ or whole body is equal to arteriovenous difference of that substance multiplied by blood flow through that organ
- Q= (Ax-Vx) F
- F=Q/Ax-Vx

- C O determined by pulmonary blood flow
- Pulmonary blood flow =rt ventricular output =Lt ventricular output

- Measurement of pulmonary blood flow by measuring the 1.amount of O2 taken by the blood from the lungs,

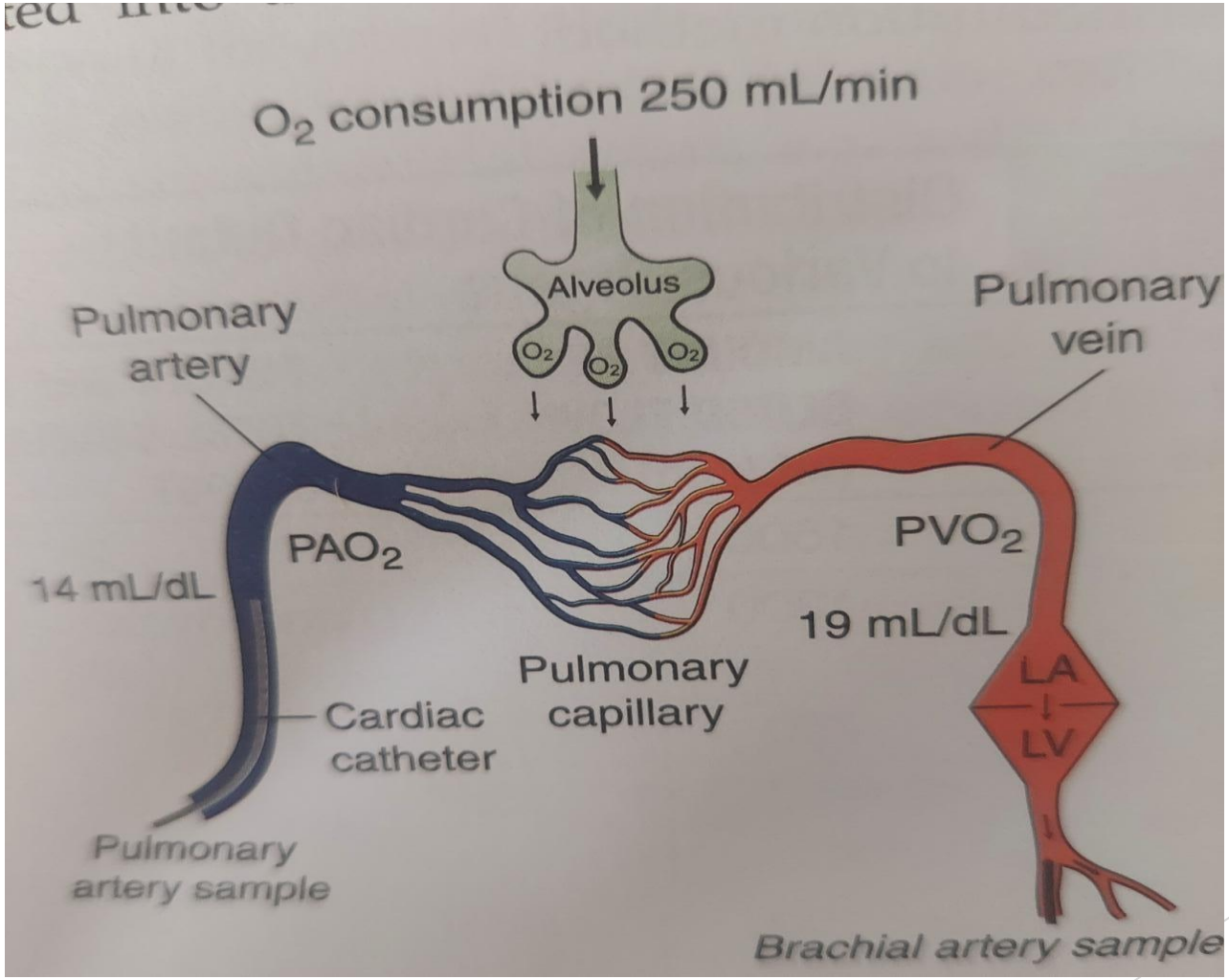
  2.O2 concentration in the venous blood (pulmonary artery)

  3.O2 concentration in arterial blood (pulmonary vein)

ARTERIAL BLOOD O2 : O2 taken by pulmonary vein--- reaches circulation ---used by body (MEASURING from pheripheral artery)

VENOUS BLOOD O2: reaches heart through veins----rt atria---- rt ventricle---pulmonary artery (by cardiac catherterisation)

Amount of O2 uptake /minute ---by spirometry =**250 ml/mt**


O_2 consumption 250 mL/min
Alveolus
O_2
O_2
O_2
Pulmonary artery
Pulmonary vein
PAO_2
14 mL/dL
PVO_2
19 mL/dL
Pulmonary capillary
Cardiac catheter
LA
LV
Pulmonary artery sample
Brachial artery sample

For example, if O_2 uptake is 250 mL/min, PVO_2 is 19 mL/100 mL and PAO_2 is 14 mL/100 mL, then

$$\text{Cardiac output} = \frac{250 \times 100}{19 - 14}$$

$$= \frac{25{,}000 \text{ mL/min}}{5}$$

$$= 5000 \text{ mL/min}$$

$$= 5 \text{ L/min}$$

DISADVANTAGES

- Invasive technique
- CO increased due to sympathetic stimulation
- Can cause fibrillation

ECHOCARDIOGRAPHY

- Ultrasonic evaluation of cardiac functions
- Non invasive ,using a transducer
- Useful in evaluating EDV, end systolic volume, cardiac output, valvular defects

VARIATION

- INCREASE
- Physiological
- 1.Excerise –CO increase by 700%----HR & SV increased
- 2.Anxiety & excitement—increase by 500% ---due to sympathetic stimulation
- 3.after eating:30% increase due to increase in metabolism
- 4.Pregnancy:CO increases by 50-60% as bld volume increases

- Pathological
- 1.Fever –increase in HR
- 2.Anemia –Hyperkinetic circulation
- 3.Thyrotoxicosis-due to increase in metabolism

- DECREASE:
- physiological
- Erect posture: bcoz of pooling of bld in the lower exterimities
- Pathological
- CCF,shock,Arrhythmias,Hemorrhage,Hypothyrodism

REGULATION OF CARDIAC OUTPUT

- CO= SV X HR
- So regulation of CO includes

  Regulation of Stroke volume & Regulation of HR

MECHANISMS

- **INTRINSIC /HETEROMETRIC**
- **EXTRINSIC/HOMOMETRIC**

INTRINSIC MECHANISM

- Cardiac output regulated by changes in cardiac fibre length is heterometric mechanism
- Force of contraction is proportional to the initial length of cardiac muscle fibre
- Force of contraction increases ----C O increases

FRANK STARLINGs LAW of HEART

- States that the greater the end diastolic volume greater the force of contraction within physiological limits
- Force of contraction increases as end diastolic volume increases upto certain limit then decreases

EXTRINSIC MECHANISM

- Regulation of cardiac output due to changes in contractility independent of length is called **extrinsic or homometric regulation**

REGULATION OF STROKE VOLUME

- END DIASTOLIC VOLUME:amount of blood present in the ventricle at the end of diastole-130ml
- STROKE VOLUME:amount of blood ejected by each ventricle per beat 80ml

- EJECTION FRACTION:percentage of EDV ejected per heart beat-65%

- SV/EDV X 100

REGULATION OF STROKE VOLUME

- Depends on 3 imp factors/determinants of SV
- 1.Preload-Heterometric regulation
- 2.afterload-Heterometric regulation
- 3,Myocardial contractility-Homometric

PRELOAD

- Freeload acting on the heart
- Load acting on the muscle just before contraction =the volume of blood present in the ventricle at the end of the diastole of the heart---**END DIASTOLIC VOLUME**

- **IF volume is more--stretching more---initial length more**
- **Increase in EDV(Preload)---Increase in force of contraction---increase in cardiac output—by Frank Starling s law**

- FACTORS AFFECTING EDV
- A)venous return
- B)Myocardial Compliance
- C) Atrial pump activity

VENOUS RETURN

- **AMOUNT OF Blood returning to the heart during 1 cardiac cycle**
- **VENOUS RETURN = CARDIAC OUTPUT**

- FACTORS CONTROLLING VENOUS RETURN
- A)Thoracic pump
- B) Cardiac Pump
- C)Muscle Pump
- D)Venous tone
- E)Posture
- F)Blood volume

THORACIC PUMP

- During inspiration intrapleural pressure more negative -6mmg ---increase in intrathoracic voume-----dilate the great veins----pressure in venacave decreases-----causes sucking force that sucks blood from below----increases VENOUS RETURN
- Descend of diaphragm suppresses the abdominal contents---increase in intra abdominal pressure ---- pushing blood from abdominal veins to rt atria--- increase in VR

CARDIAC PUMP

- FORCES THAT DEVELOPES IN THE HEART
- 1.‘VIS A TERGO’-forward push from behind---force acts from behind pushing the blood forwards

1. During systole ,ejection of blood from heart imparts a propelling force to the blood
2. Elastic recoil /windkessel effect of aorta also propel blood

So blood pushed into veins & to right artium

- **VIS A FRONTE**: suction force created by ventricle acting from the front pulls blood from great veins to Rt artium
- 1.Ventricular systolic suction - during ejection phase ,AV valve pulled down----increase in size of rt artium ----sucks blood from great veins to rt atrium
- 2.ventricular diastolic suction—opening of AV valve sudden fall in atrial pressure---sucks blood from veins

MUSCLE PUMP

- Important in exercise
- Activity of skeletal muscle act as pump pushing the blood forward
- Blood flows from superficial to deep veins only through communicating veins
- When skeletal Muscle contract ---deep veins compressed because of increased pressure,proximal valve open,distal valve closed ---blood propelled towards heart

- When skeletal muscle relaxes ----a negative pressure created in veins---due to back flow proximal valve closed----distal valve opened up---blood sucked up---blood filled up the veins again

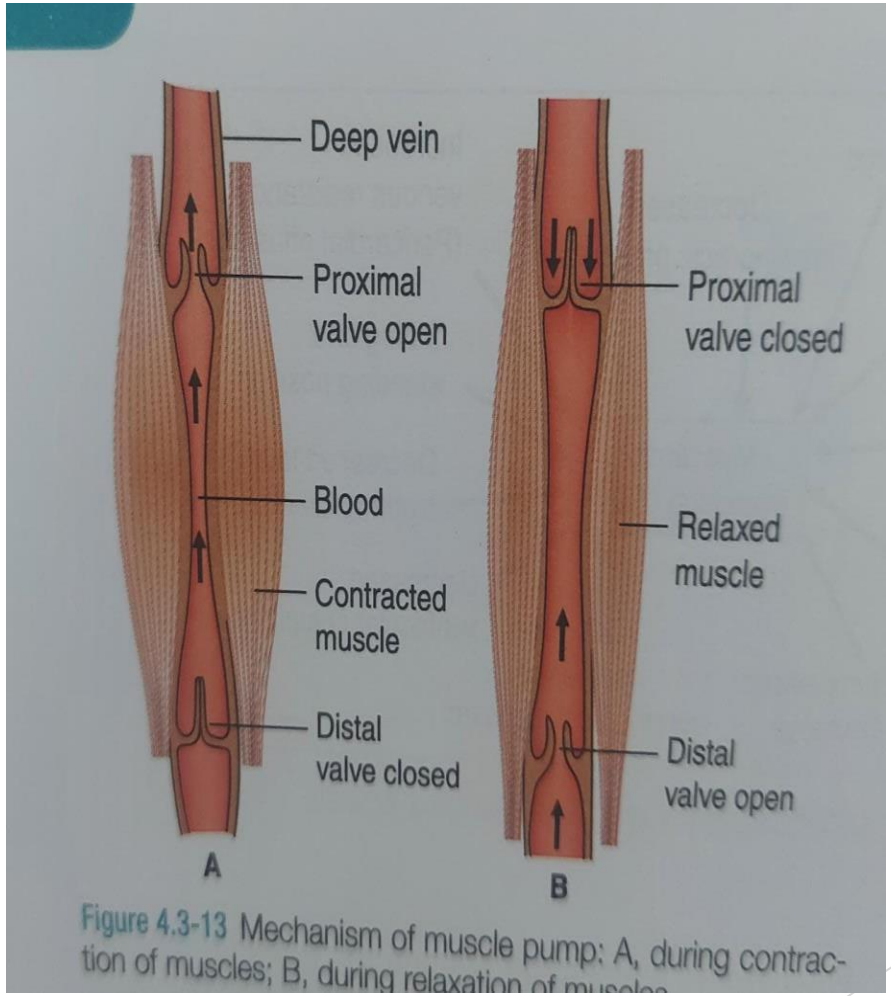


Figure 4.3-13 Mechanism of muscle pump: A, during contraction of muscles; B, during relaxation of muscles

- BLOOD VOLUME: increase in blood volume----- increase in mean circulating filling pressure----increase in venous return
- 5) VENOUS TONE:degree of contraction of veins of the body

Venous tone is increased-----increase in Venous return

Venus tone is less ----more blood pools in veins---- decrease in VR

- 6)POSTURE: influence in venous return due to effect of gravity
- Standing ----pooling of blood----decrease in venous return

MYOCARDIAL COMPLIANCE/VENTRICULAR COMPLIANCE

- Degree of stretchability of ventricle to accommodate blood
- **CARDIOMYOPATHIES & INFILTRATIVE DISEASE OF HEART**-- - restriction of filling due to increase in intrapericardial pressure ----heart cannot expand ----decrease in EDV

ATRIAL PUMP

- atria act as booster pump to fill 30 % of ventricular filling
- Significant when Increase in heart rate ---diastole is decreased

AFTERLOAD

- Resistance against heart pumping
- Determined by peripheral resistance
- TWO factors determining PR
- 1.CALIBER OF VESSEL—dilated vessel -less resistance-heart can pump more blood

  contricted vessel—more R

  Resistance vessel--- ARTERIOLES---caliber is small

MYOCARDIAL CONTRACTILITY

- Capacity of heart to contract without change in length
- When Myocardial contractility increases ---increase in force of contraction of myocardium without an increase in EDV,ie length of Cardiac muscle is remaining same----- HOMOMETRIC REGULATION of C O

- Positive ionotropic agents----increase force of contraction & myocardial contractility
- Negative ionotropic agents---decrease myocardial contractility

POSITIVE IONOTROPIC AGENTS

- 1.Ventricular Muscle Mass:increased can pump more blood. In athletes heart muscle is hypertrophied ---CO is more
- 2.Sympathetic Activity – sympathetic N secrete N E acts through B1 receptor ---increase Ca ----increase contractility
- 3.Catecholamines---increase Ca—increase contractility

- 4.HORMONES:

  Glucagon

  Insulin

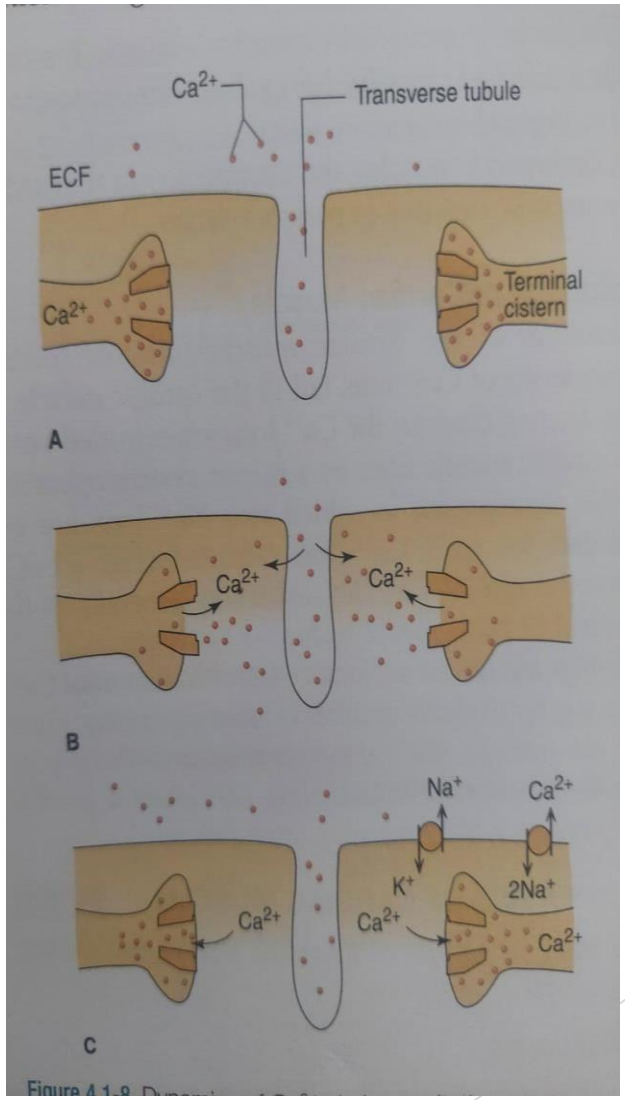
  Thyroxine

- 5.XANTHEINES

  Caffeines,Theophyllines

6. DIGITALIS & related drugs

-a Na+ K+ ATPase inhibitor---decrease Na + secretion ---increase Ca concentration ---increase myocardial contractility



NEGATIVE IONOTROPIC AGENTS

- HYPOXIA,HYPERCAPNEA,ACIDOSIS
- **BARBITURATES,QUINIDINE,PROCAINAMIDE**
- **MYOCARDIAL INFARACTION**
- **HEART FAILURE**
- **PARASYMPATHETIC STIMULATION: willnot affect ventricular contractility much--**

  atrial contractility decreased

- HOMOMETERIC REGULATION : EJECTION FRACTION is more

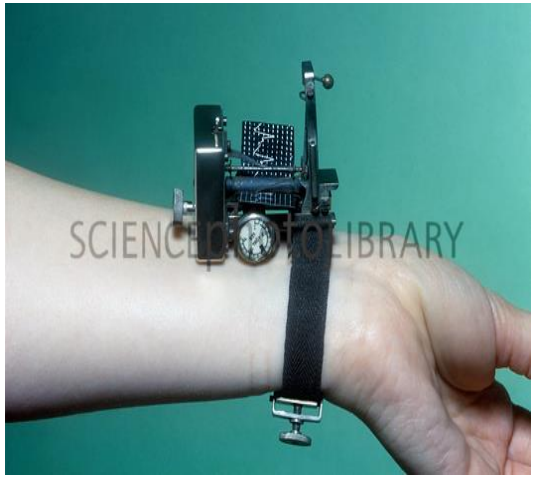
Arterial pulse

- Defined as expansile pressure wave transmitting along the systemic arteries when blood is ejected during systole into the already filled aorta .
- Blood (SV 70ml)ejected into aorta during systole set up a pressure wave which is transmitted along artery to periphery
- pressure wave expands arterial wall as it travels & expansion is palpated as pulse

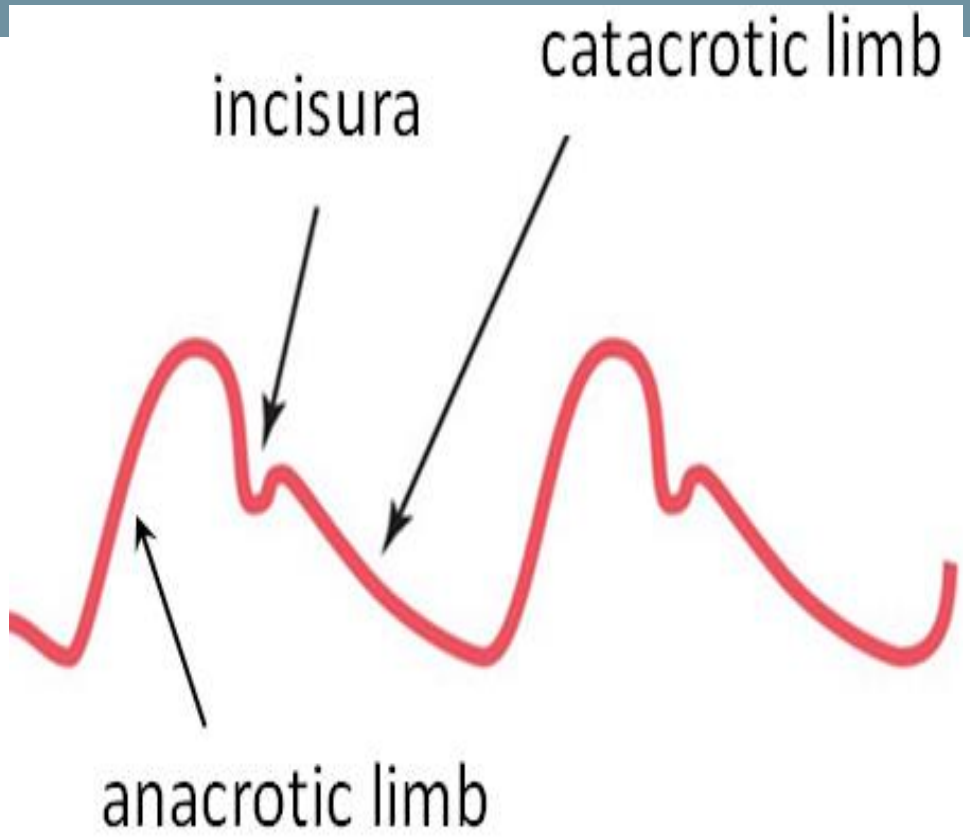
Arterial pulse

- Radial pulse : 0.1 sec after peak of ejection
- Velocity of pulse wave inversely related to elasticity of vessel wall
- aorta: 4 m/sec,
- large arteries: 8 m/sec,
- small arteries: 16 m/sec.
- Central pulse tracing, Peripheral pulse tracing

Sphygmograph



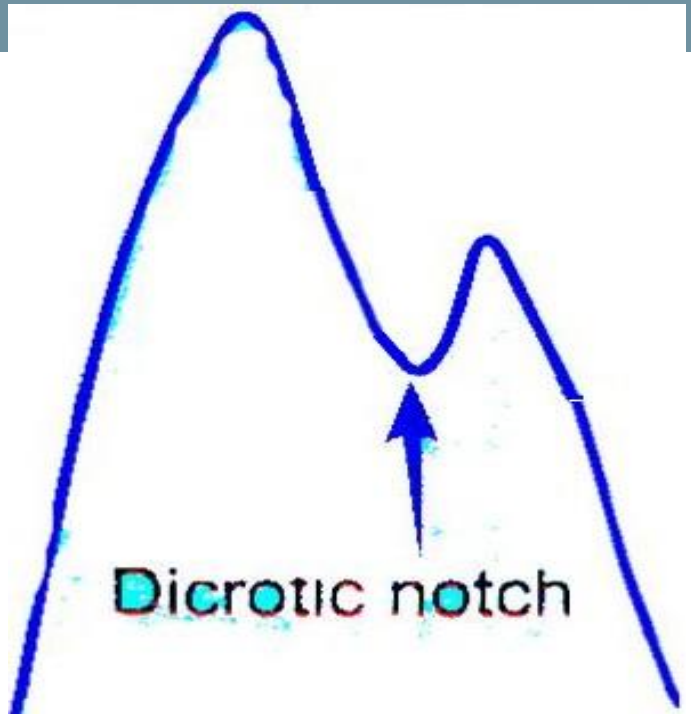
Pulse tracing



- **TWO LIMBS:**

  1. **ASCENDING LIMB –ANACROTIC LIMB- steep & smooth**
  2. **DESCENDING LIMB-CATACROTIC LIMB- gradual fall,not smooth,shows oscillations**

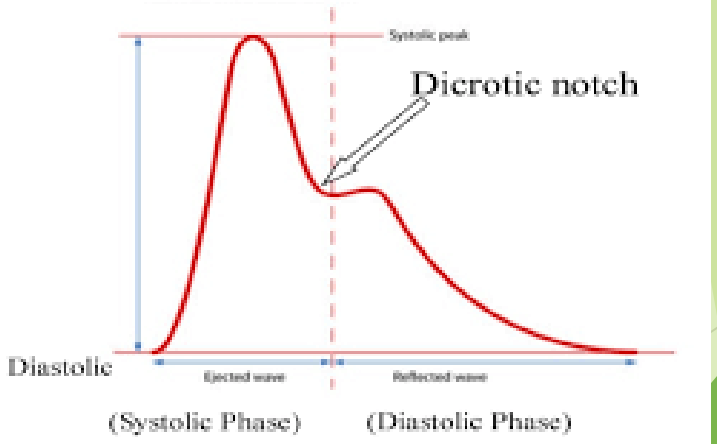
Pulse tracing



Systole

Primary wave or percussion wave – maximum ejection phase of ventricular systole

Later fall – reduced ejection p



Dicrotic notch

- present in descending limb of pulse tracing. produced due to vibrations set up when the aortic valve closes corresponds to insisura

Dicrotic wave

- Follows dicrotic notch.
- -- rebound of blood from closed aortic valve causes increase in pressure

Factors affecting pulse

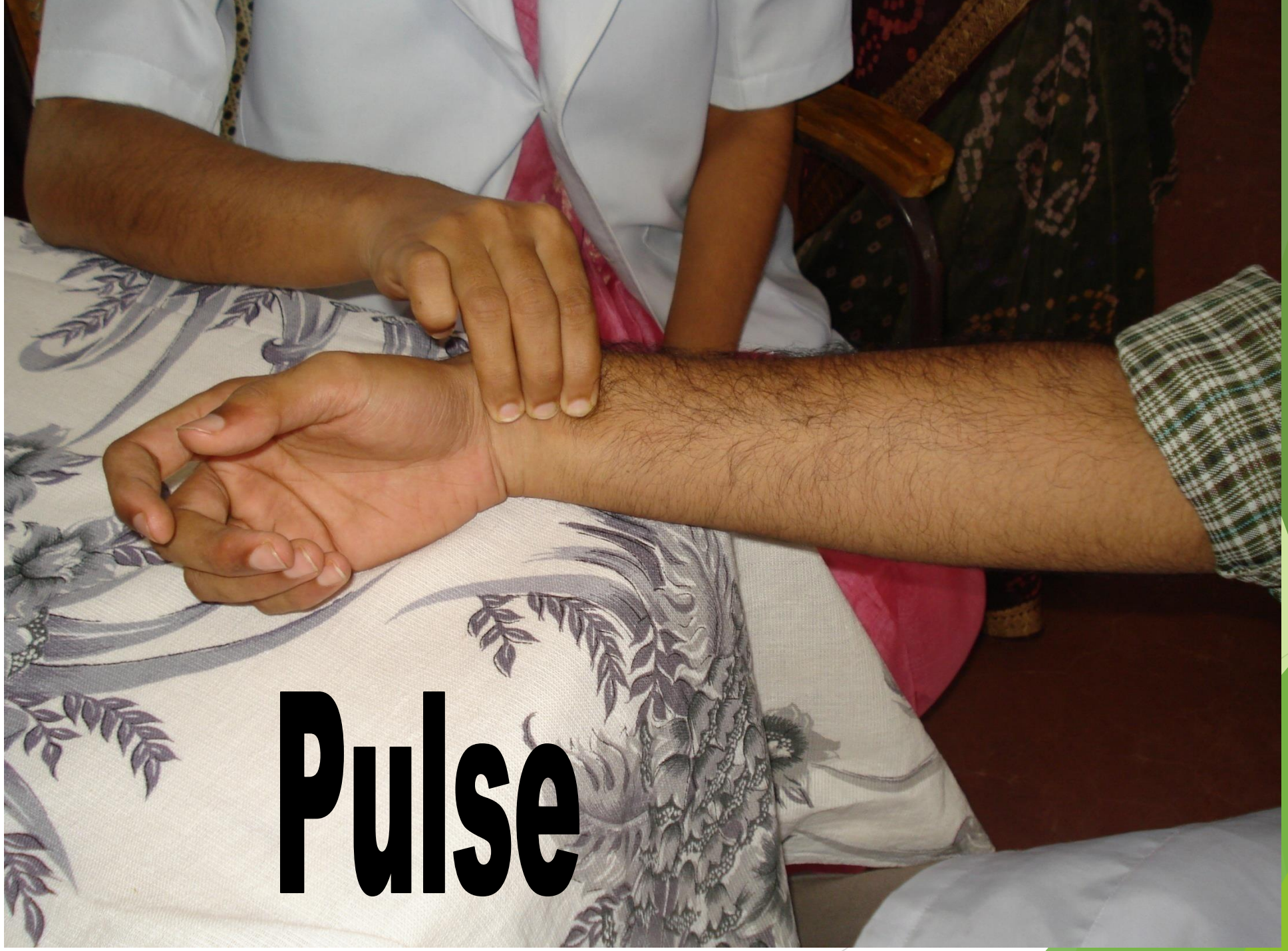
- Stroke volume
- Compliance of vessels

VARIATIONS

- A large primary wave when cardiac output is high
- Small primary wave -cardiac output is less
- In the arterioles ,the pulse waves reduced (damping effect) due to increased resistance & reduced elasticity

Arterial pulse

- Rate -- NL pulse rate =60-90/min
- Rhythm
- Volume-strength of pulse
- Character – tension & pulsewaves
- Condition of vessel wall
- Radiofemoral delay
- pulse deficit -pulse rate < heart rate


Pulse

- Rate -no of beats per min counted for 1 min.

  Increase in pulse rate -tachycardia -seen in exercise,fever

  Decrease in pulse rate - bradycardia-seen in sleep,heart block

- **Rhythm** -regularity of occurrence of pulse wave (regular/irregular
- Regularly irregular-heart block
- Irregularly irregular-Atrial fibrillation

Volume -extent of displacement of palpating finger-gives assesment of pulse pressure

(Normal/high/low volume)

- High volume pulse -fever, aortic regurgitation
- Low volume pulse - sleep,shock

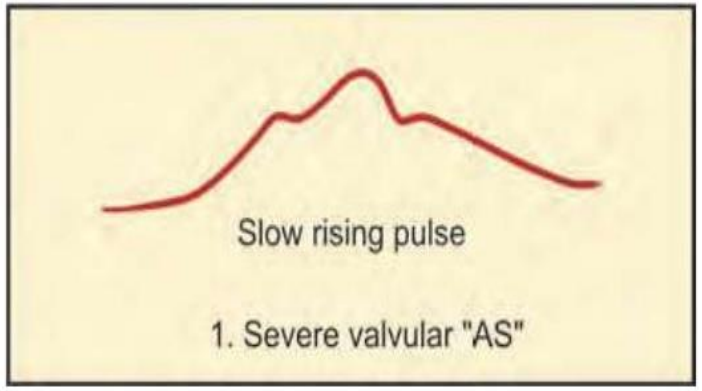
- **CHARACTER:** wave pattern of the pulse with respect to its volume &time

Abnormal character

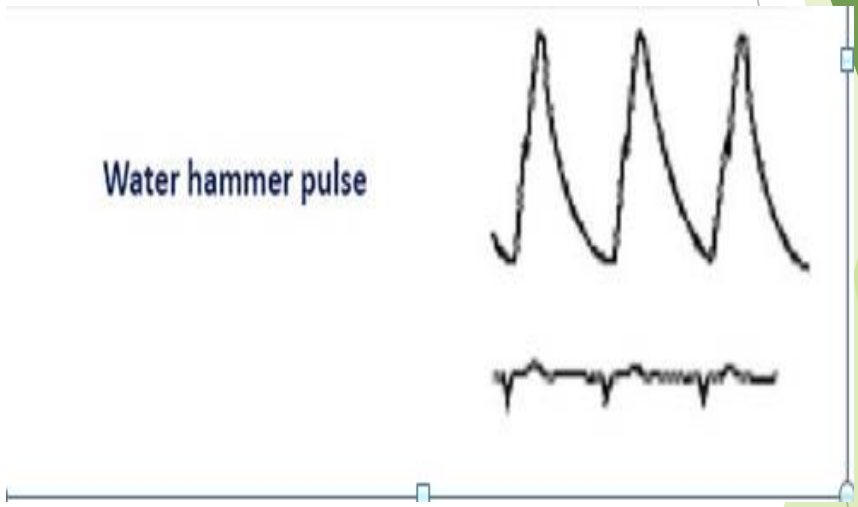
- Aortic stenosis – anacrotic pulse
- Aortic regurgitation –collapsing (water hammer) pulse
- Bisferiens pulse
- Pulses alternans -LVF
- Pulses paradoxus - pericardial effusion


Collapsing pulse
(Aortic regurgitation)

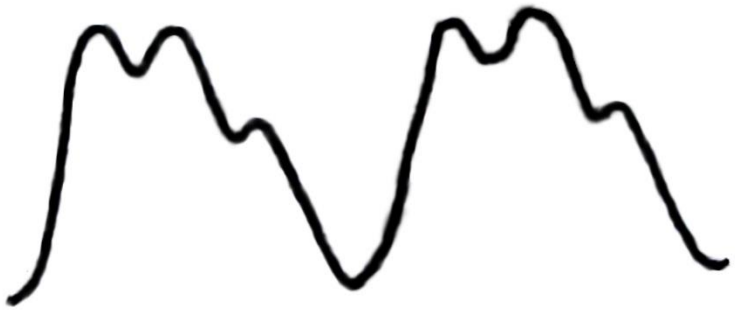
Anacrotic Pulse (Parvus et Tardus)



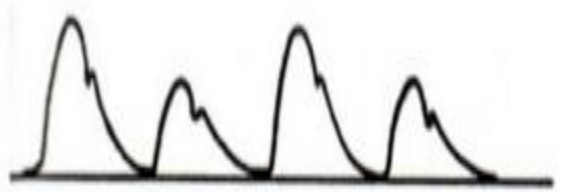
A low amplitude pulse (parvus) with a slow rising and late peak (tardus).



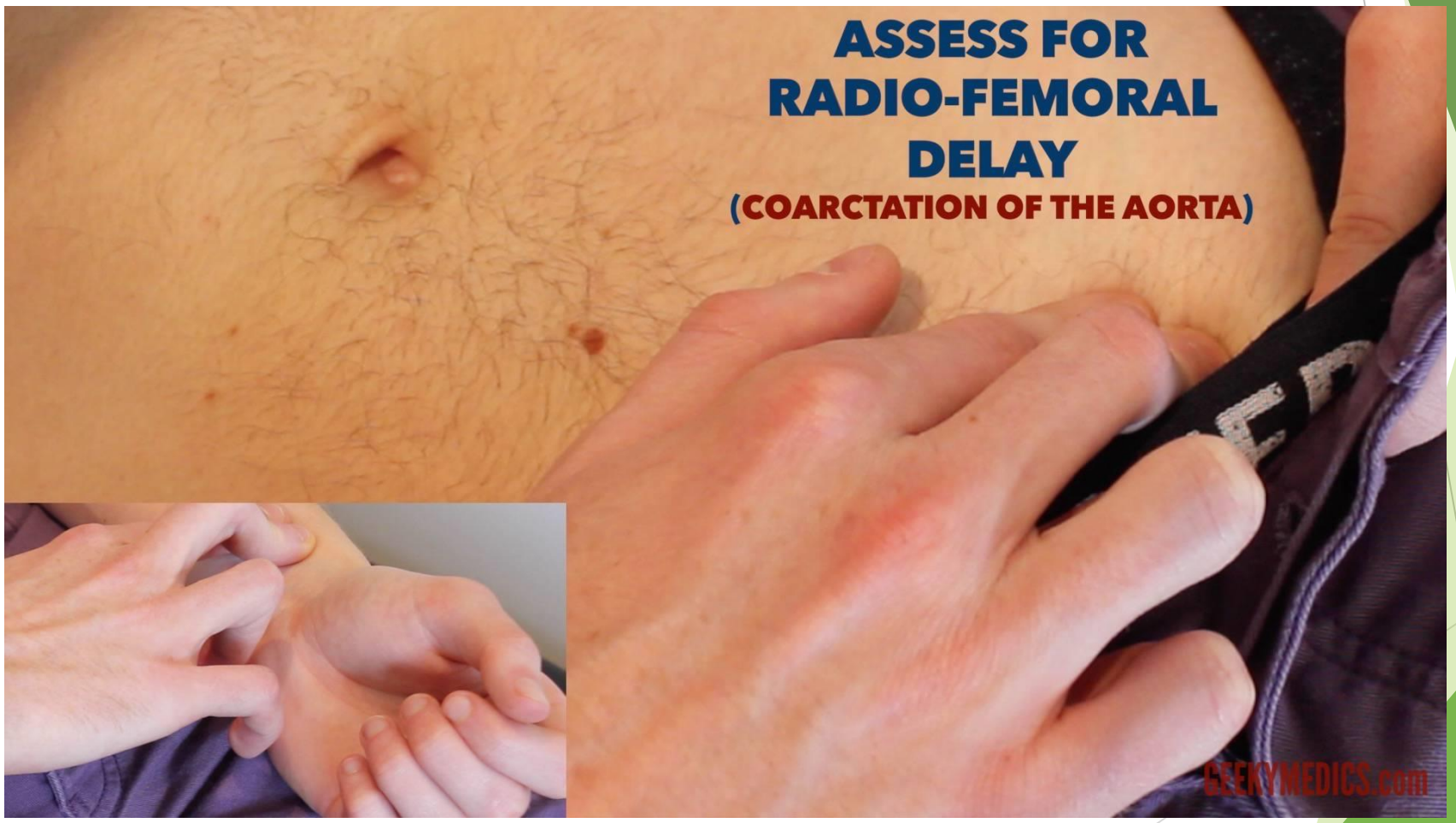
Pulsus bisferience



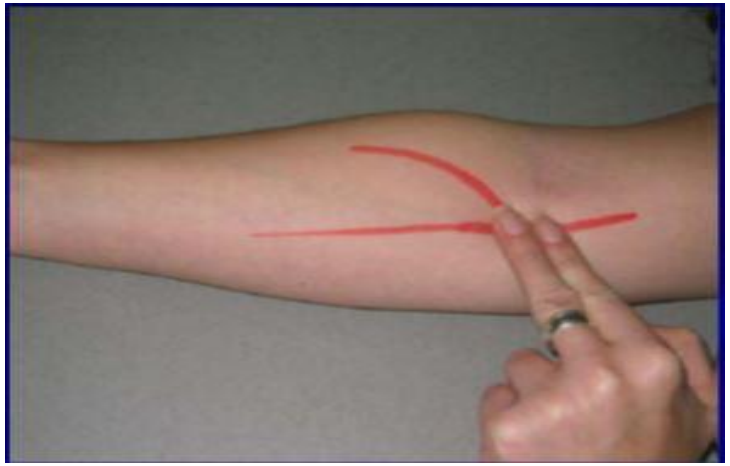
Pulsus Alternans

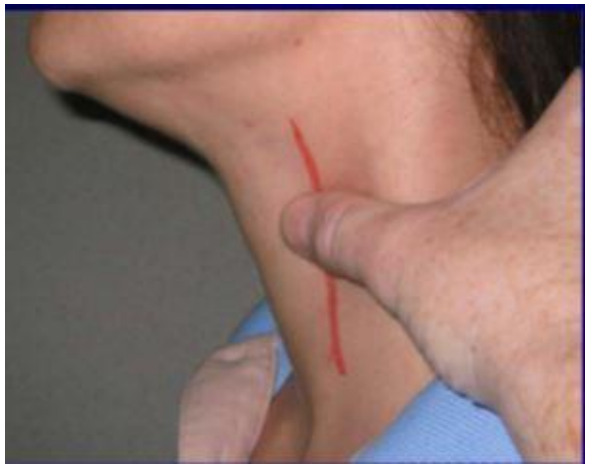


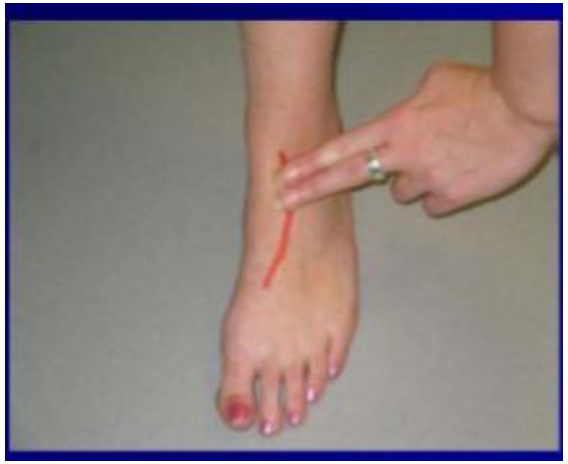
- Condition of vessel wall- thickened & palpable/soft & not palpable
- Pulse deficit-pulse rate < heart rate
- Radiofemoral delay-coarction of aorta

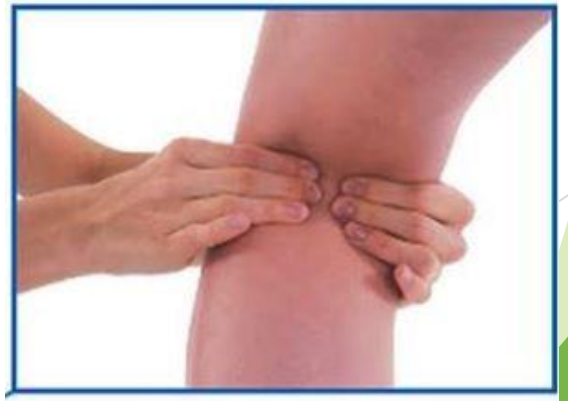

ASSESS FOR
RADIO-FEMORAL
DELAY
(COARCTATION OF THE AORTA)
GEEKYMEDICS.com

Peripheral pulses









Popliteal pulse

Arterial pulse

More medial

- better Felt than seen
- Independent of respiration
- Hard to obliterate by pressure

Venous pulse

- More lateral
- better seen than felt
- Definite upper level
- Falls during inspiration
- Obliterated by pressure

Heart rate

Heart rate

- rate of discharge of SA node
- HR is the number of heart beats/min.

- Normal heart rate in adults= 60-100 beats/min

VARIATIONS

- TACHYCARDIA----HR more than 100
- BARDYCARDIA---HR less than 60

Variations

- Age –increased HR in infants &old age (vagal tone)
- Respiration -↑ inspiration& ↓expiration(sinus arrhythmia)
- Body temperature –

  For each 1°F rise in temp HR ↑10beats/min
- Exercise- ↑ HR
- Food intake-↑ HR- due to↑ in metabolism

- Diurnal variation – more in day ,less at night
- Emotions – excitement,anxiety,anger

<u>↓HR</u>

- Sleep, athletes
- Myxoedema
- Heart block

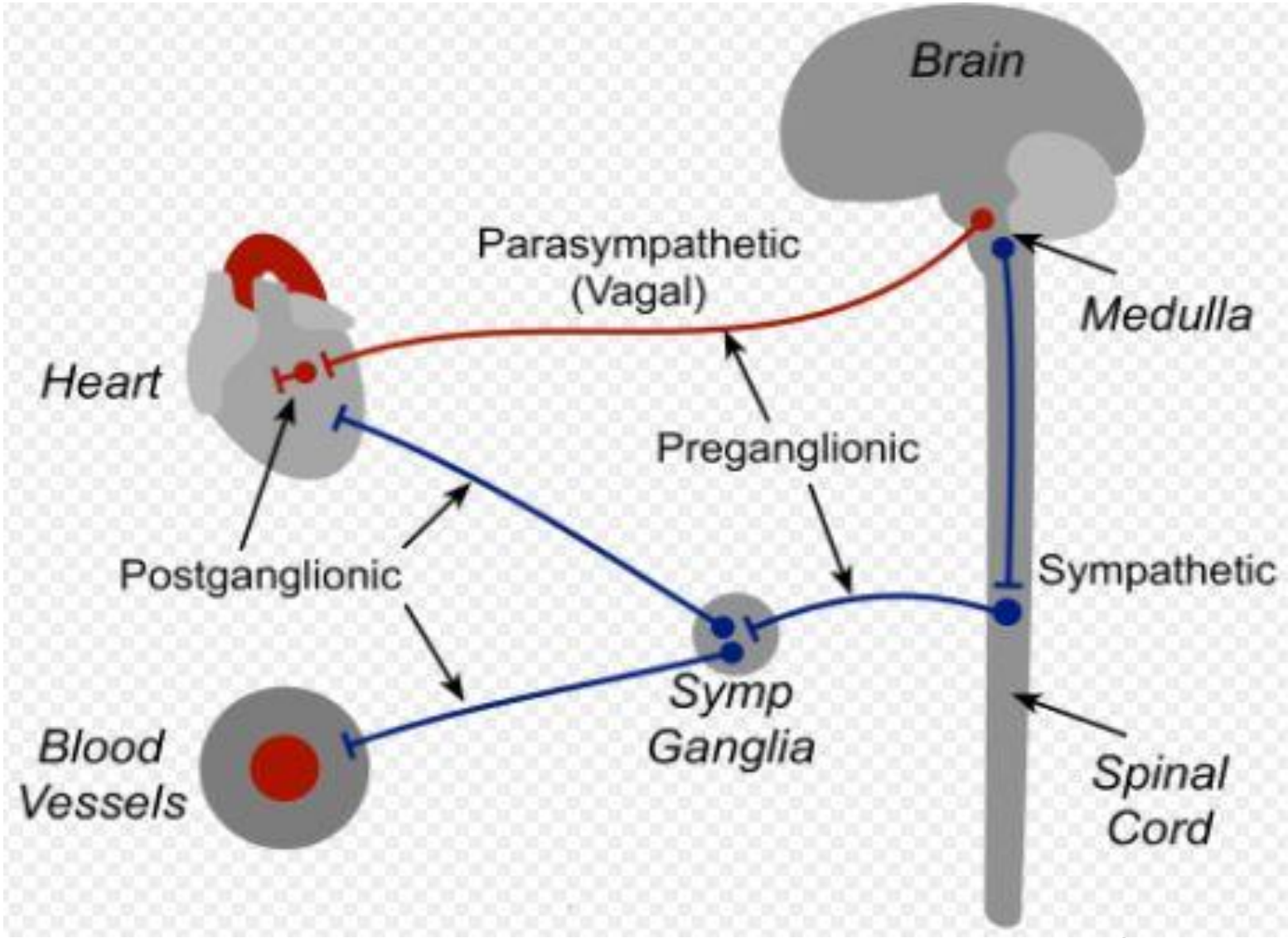
REGULATION OF HEART RATE

- NEURAL REGULATION
- HUMORAL REGULATION

Neural Regulation of heart rate

1. Autonomic Regulation
2. Medullary Regulation
3. Reflex regulation

AUTONOMIC INNERVATION OF HEART



SYMPATHETIC Regulation

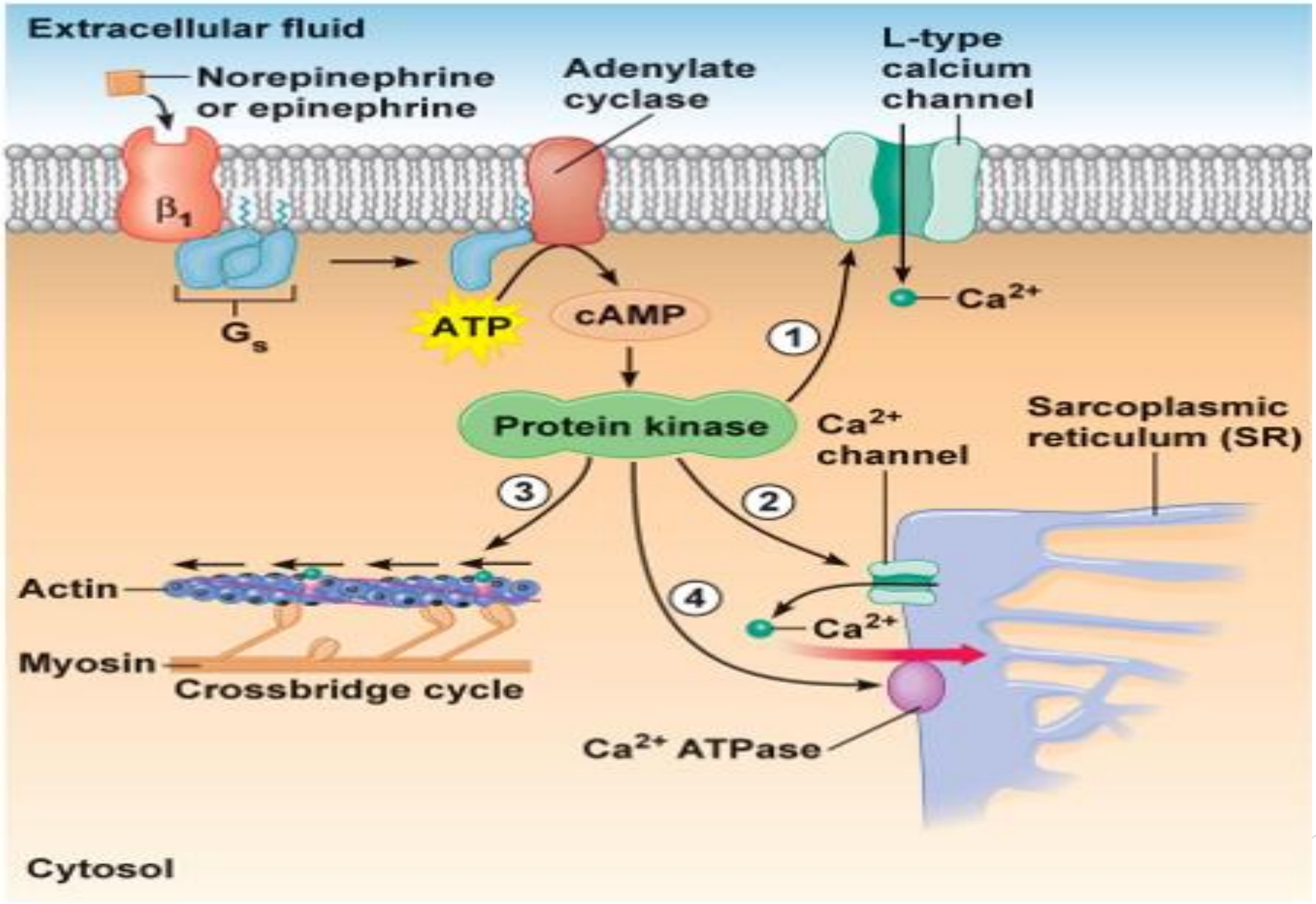
- Sympathetic supplying heart arise from IML grey column in T1 –T5 spinal segment
- Post ganglionic fibres supply nodal tissues, muscles of atria, ventricles
- Rt supply SA node, RA,RV
- Lt supply AV node & Lt side of heart

symp Regulation

- Sympathetic stimulation causes
- +ve chronotropic effect (increase HR)
- +ve inotropic effect (increase contractility
- +ve dromotropic effect (↑conductivity)
- +ve bathmotropic effect (↑ excitability)

Sympathetic -mechanism

- Sympathetic stimulation- NE - adrenergic β1 receptor
- –activation of adenyl cyclase & ↑cyclic AMP –
- calcium channel to open for a long time
- Pre potential rises to firing level very fast


Extracellular fluid
Norepinephrine or epinephrine
Adenylate cyclase
L-type calcium channel
β_1
G_s
ATP
cAMP
Ca^{2+}
1
Protein kinase
Ca^{2+} channel
Sarcoplasmic reticulum (SR)
3
2
Actin
4
Myosin
Crossbridge cycle
Ca^{2+}
Ca^{2+} ATPase
Cytosol

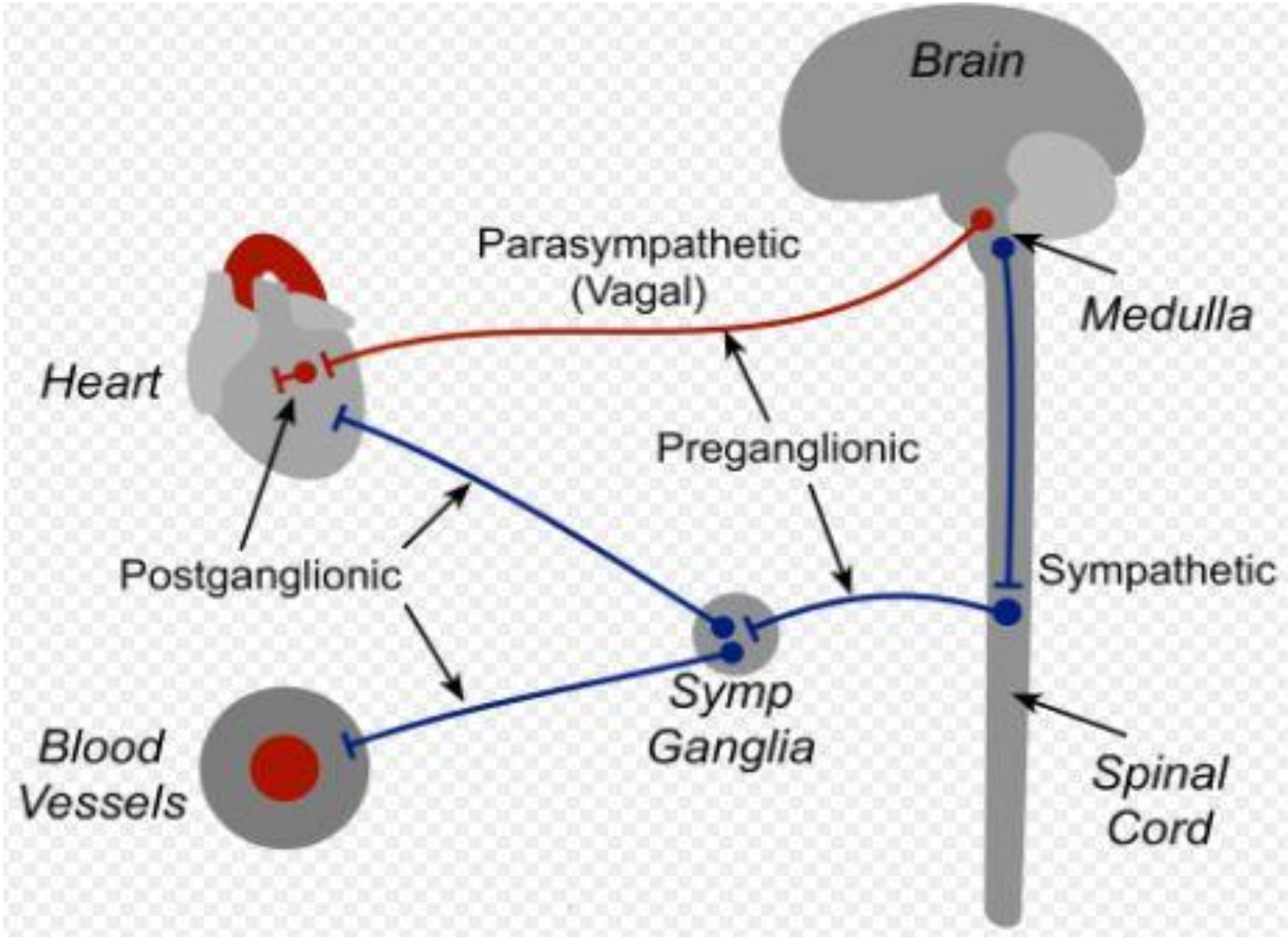
- Discharge of intermediolateral neurons are controlled by medullary cardiovascular centres

PARASYMPATHETIC REGULATION

- The cardiac parasympathetic fibers originate in the medulla oblongata, in cells that lie in the dorsal motor nucleus of the vagus

- Preganglionic fibers arise from medulla travel in vagi to synapse in ganglia with in heart. Postganglionic fibers supply mainly SA node & AV node mainly.

AUTONOMIC INNERVATION OF HEART



- Rt vagus –SA node
- Lt vagus-Av node
- No vagal fibers are distributed to ventricle

Parasympathethic effects

- PS fibres release ACh
- SA node, AV node, atrial muscles
- -ve chronotropism

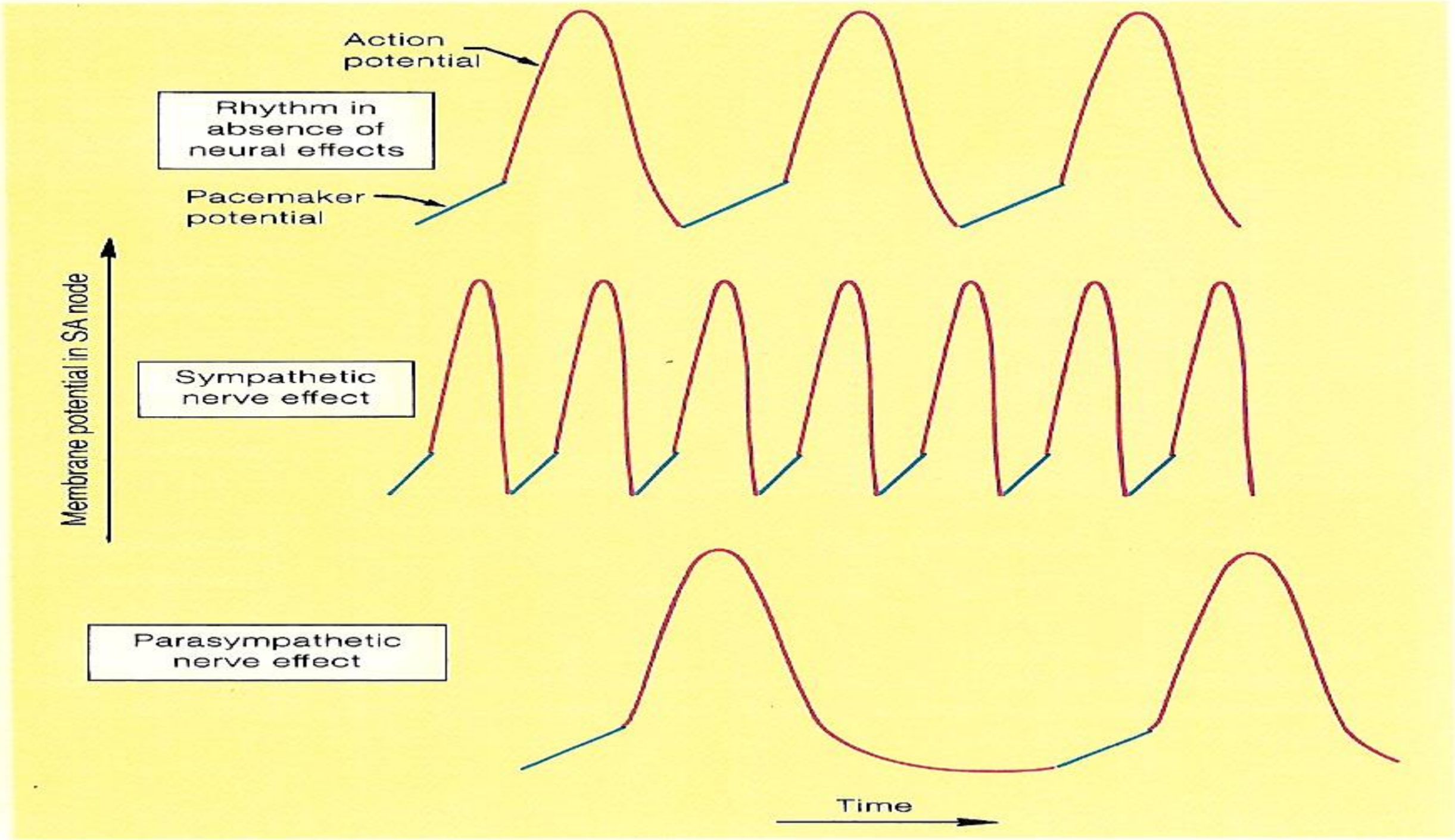
  -ve inotropism

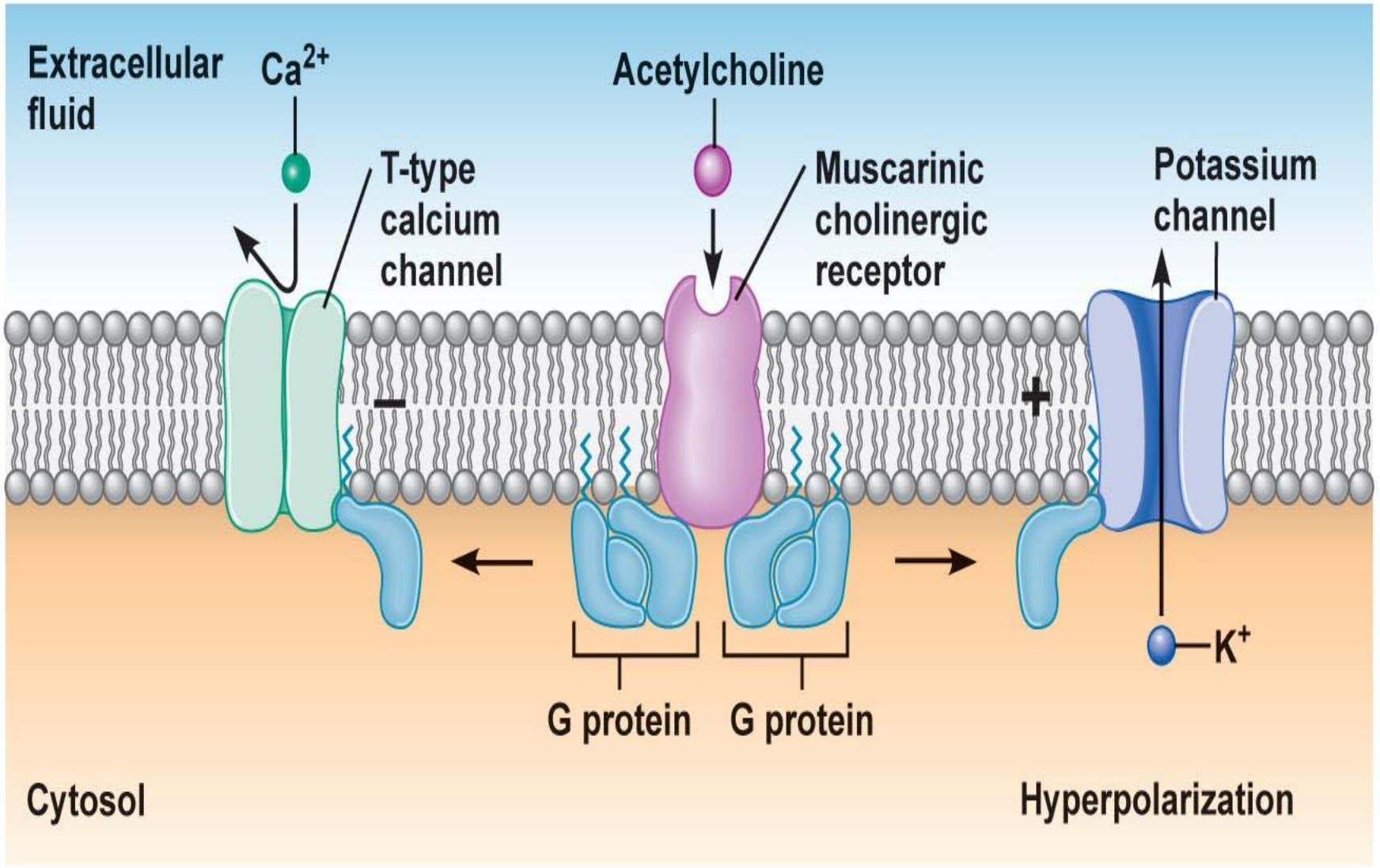
  -ve dromotropism

  ve bathmotropism

Mechanism of action

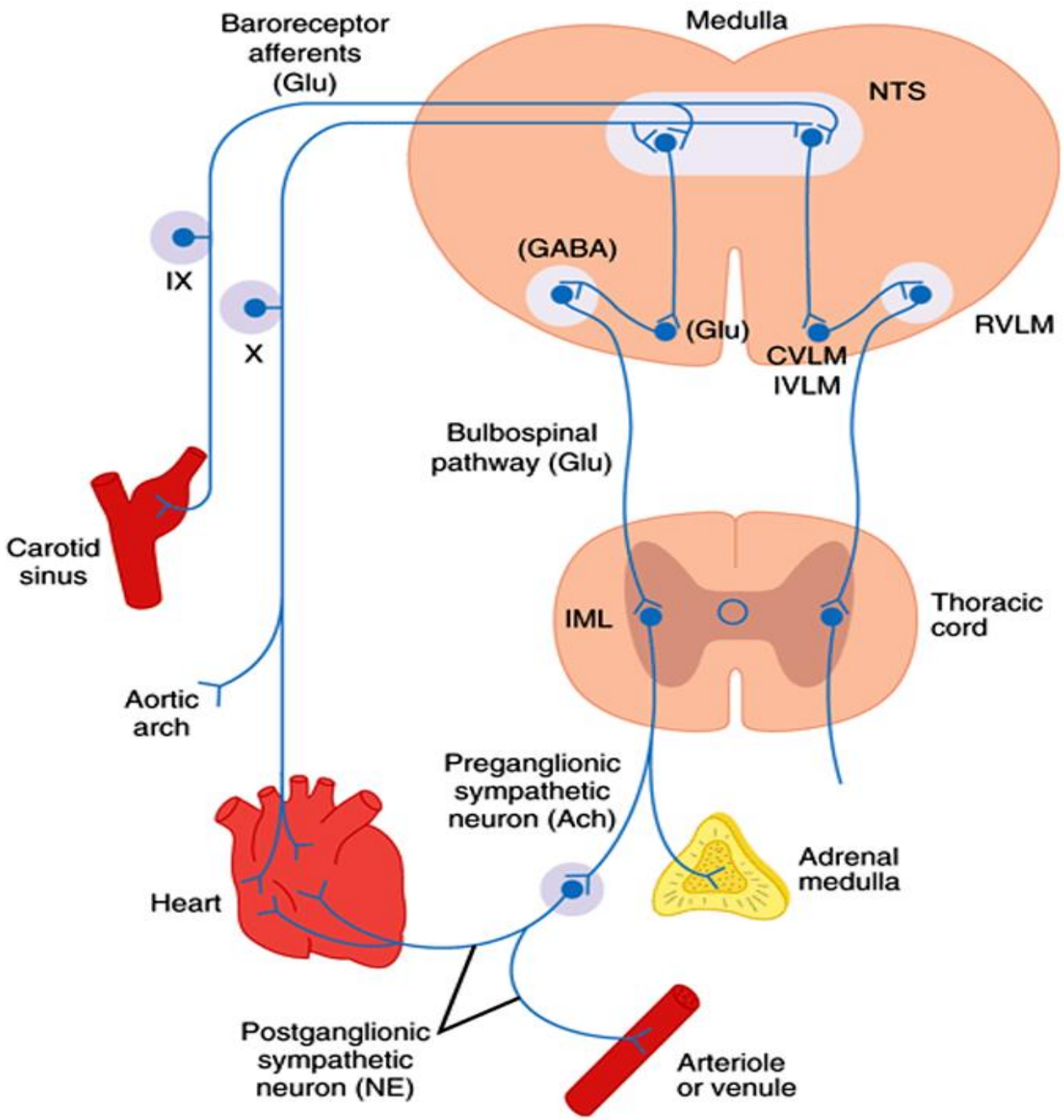
- Ach - **increase K^+ conductance** of nodal tissue via M_2 muscarinic receptor
- which via G protein open a special set of K^+ channel (Ach regulated K channel) --- hyperpolarization
- Activation of M_2 receptor decreases the cyclic AMP in the cell and **slows the opening of Ca^{2+}**


Action potential
Rhythm in absence of neural effects
Pacemaker potential
Membrane potential in SA node
Sympathetic nerve effect
Parasympathetic nerve effect
Time



(b) Parasympathetic

MEDULLARY CONTROL



Medullary centres

- VASOMOTOR area
- CARDIOINHIBITORY area
- SENSORY area -NTS

Vasomotor center

1) Vasoconstrictor/pressor area

Rostral ventrolateral medulla(RVLM)

- Stimulation of pressor area
- ↑ HR, force of contraction, Vasoconstriction –↑ BP, Venoconstriction

2) Vasodilator area/depressor area

Caudal ventrolateral medulla (CVLM)

intermediateventrolateral medulla(IVLM)

Sensory area

Nucleus tractus solitarius (NTS)-afferents from receptors via IX,X.

1) NTS → excitatory projections to CVLM or depressor area (excitatory NT glutamate)

2) CVLM → inhibitory projections to RVLM or pressor area (inhibitory transmitter GABA)

RVLM /Pressor area

RVLM---TONICALLY ACTIVE

continuous slow discharge of excitatory signals from RVLM through sympathetic nerves to heart& BVs

Cardioinhibitory area/cardiac vagal center

- Dorsal motor nucleus of vagus
- Afferents -NTS, Resp center
- Preganglionic fibers run in cardiac vagal branches & postganglionic fibers are distributed to SA node, AV node & atria.
- Responsible for vagal tone

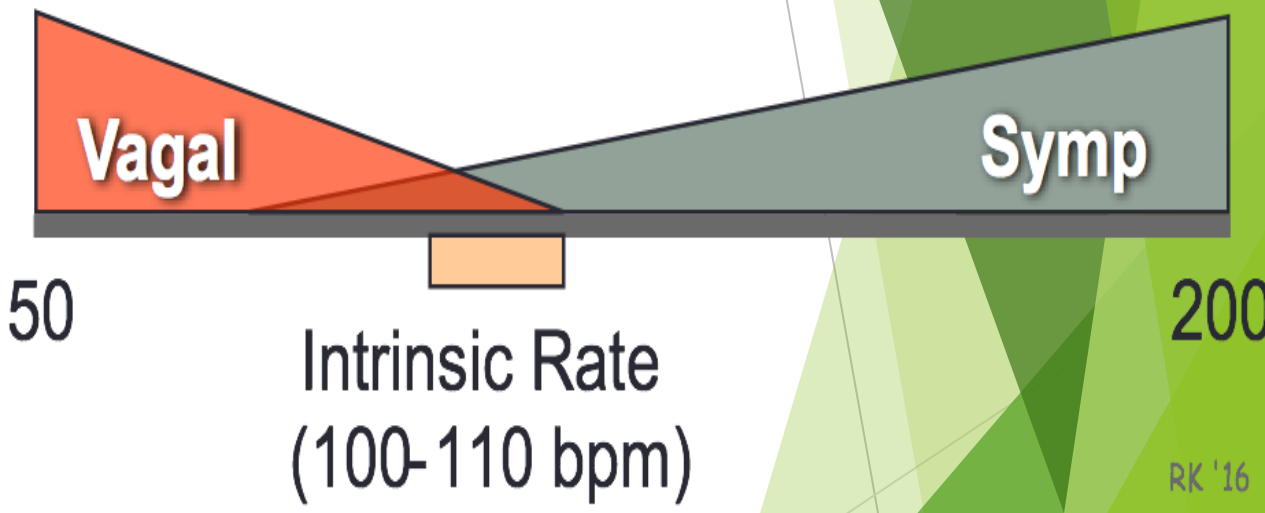
Vagal tone

- Continuous inhibitory effect of vagus on heart which keeps HR low. (predominant)
- Due to Impulses from cardioinhibitory region of medulla passing down vagus nerves to heart
- vagi is cut- HR ↑.

SYMPATHETIC TONE

- Seen in blood vessels that have little PS activity
- TONIC CONTRACTION OF BLOOD VESSELS by the stimulation of RVLM through sympathetic nerves
- (moderate amount)

symp & parasympathetic blocked -**intrinsic heart rate--**100 beats/min.



- Normally vagal tone > sympathetic tone

- Vagal tone ↓in new born ,inspiration – HR more
- Vagal tone ↑ in athletes- HR less

Table 33–2 Factors Affecting the Activity of the RVLM.
Direct stimulation
CO_2
Hypoxia
Excitatory inputs
Cortex via hypothalamus
Mesencephalic periaqueductal gray
Brain stem reticular formation
Pain pathways
Somatic afferents (somatosympathetic reflex)
Carotid and aortic chemoreceptors
Inhibitory inputs
Cortex via hypothalamus
Caudal ventrolateral medulla
Caudal medullary raphé nuclei
Lung inflation afferents
Carotid, aortic, and cardiopulmonary baroreceptors

3)Reflex Regulation

- Afferents from arteries – baroreceptor reflex, chemoreceptor reflex
- Afferents from heart & pulmonary circulation
- Other reflexes – Cushing's reflex, sinus arrythmia

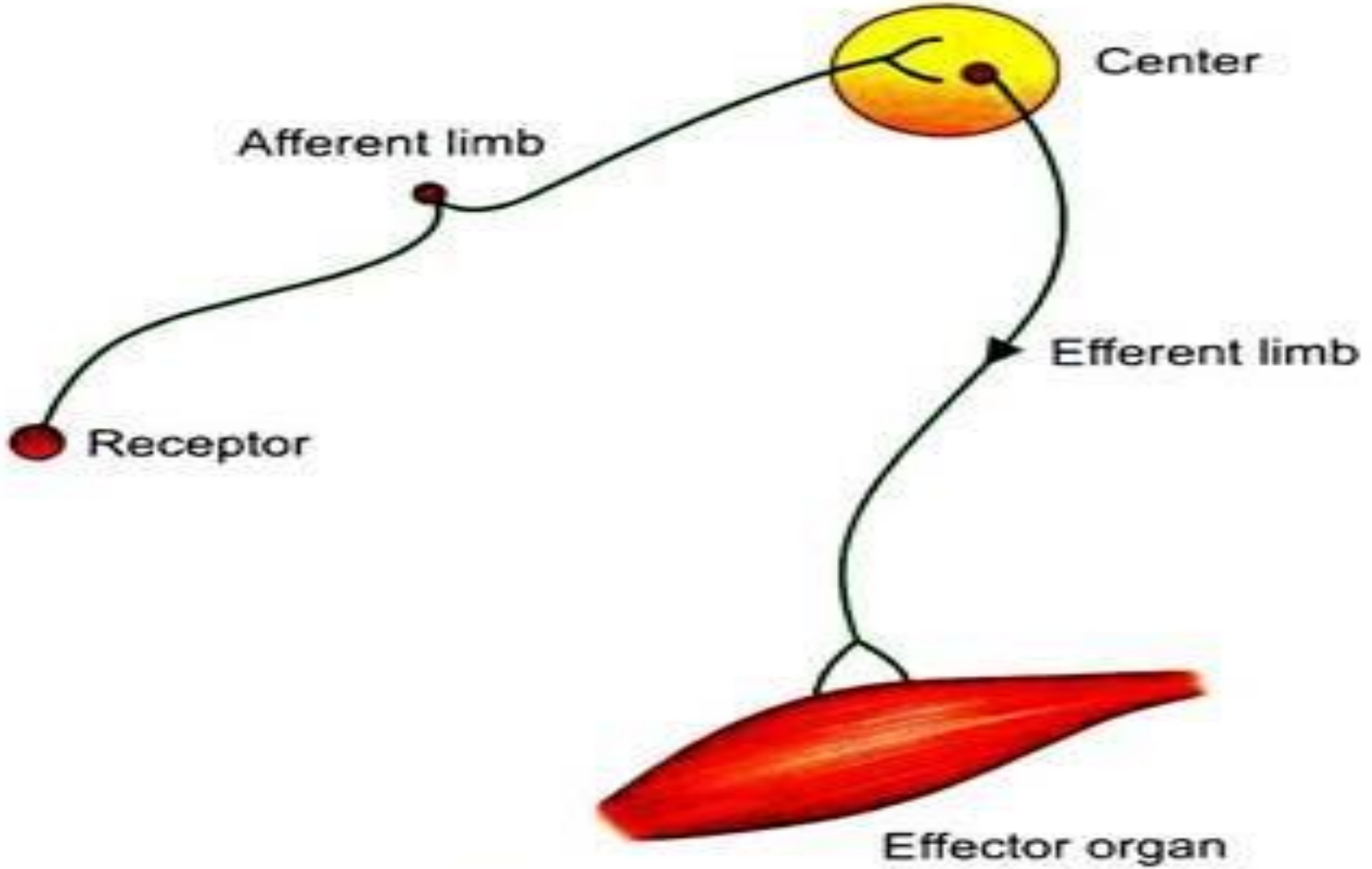


Fig. 9.11: Basic reflex arc

Reflex control

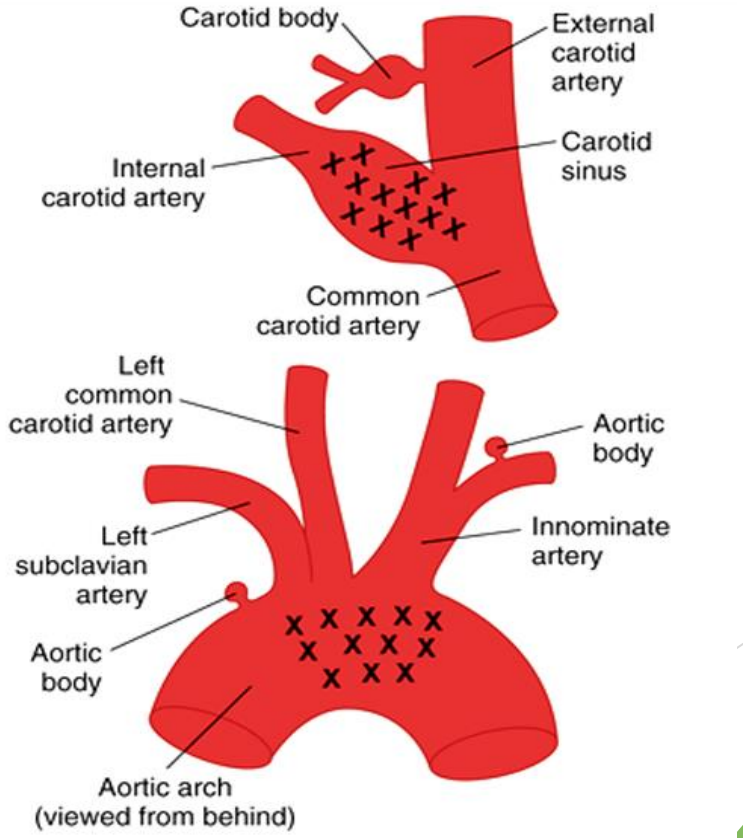
- 1.BARORECEPTOR REFLEX
- 2.CHEMORECEPTOR REFLEX
- 3.Bainbridge reflex
- 4.coronary chemoreflex
- 5.CNS ISCHEMIC RESPONSE (CUSHING S REFLEX)

Baroreceptor mechanism

- Stretch receptors in walls of heart & blood vessels
- carotid sinus
- aortic arch

Carotid sinus – 9th nerve

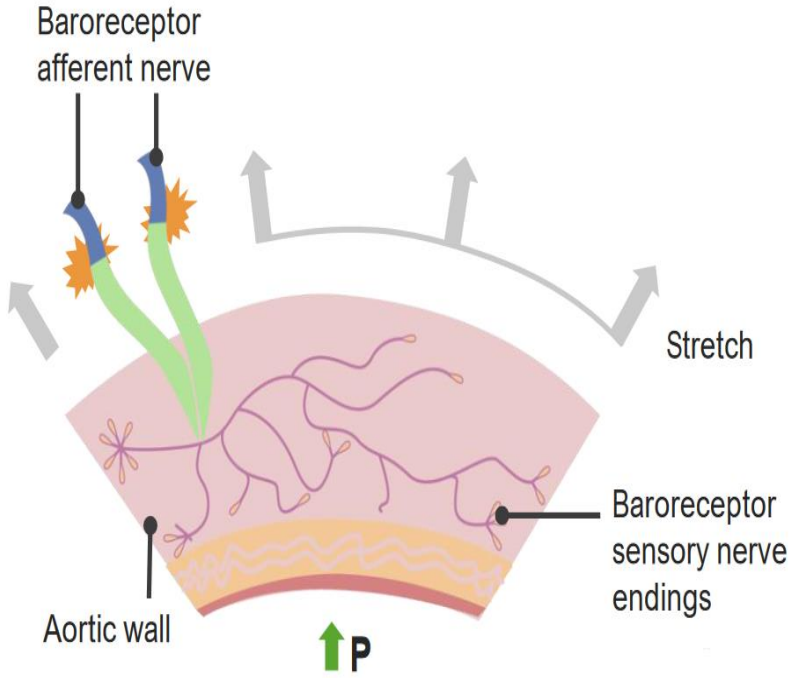
Aortic arch –10th

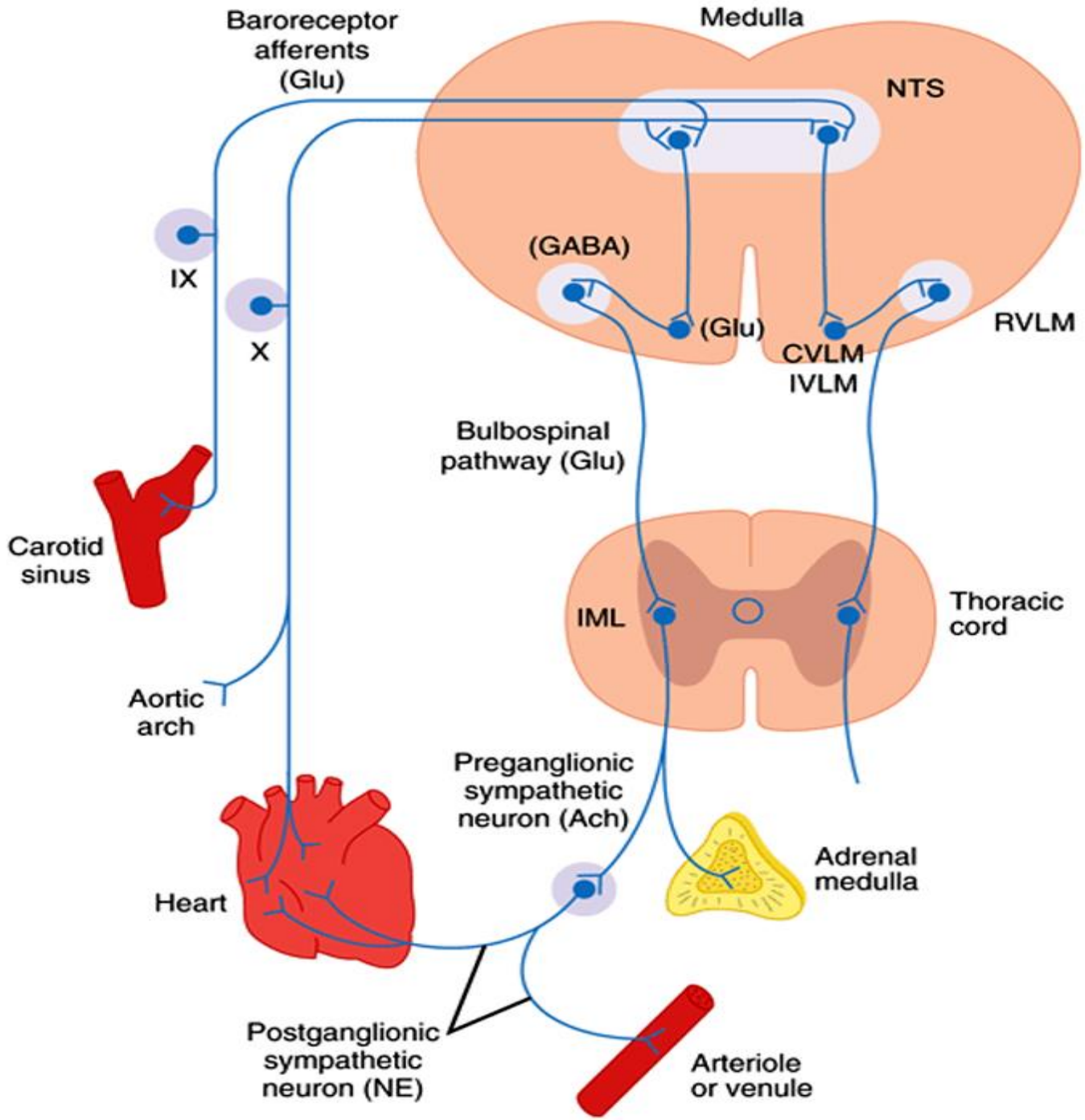


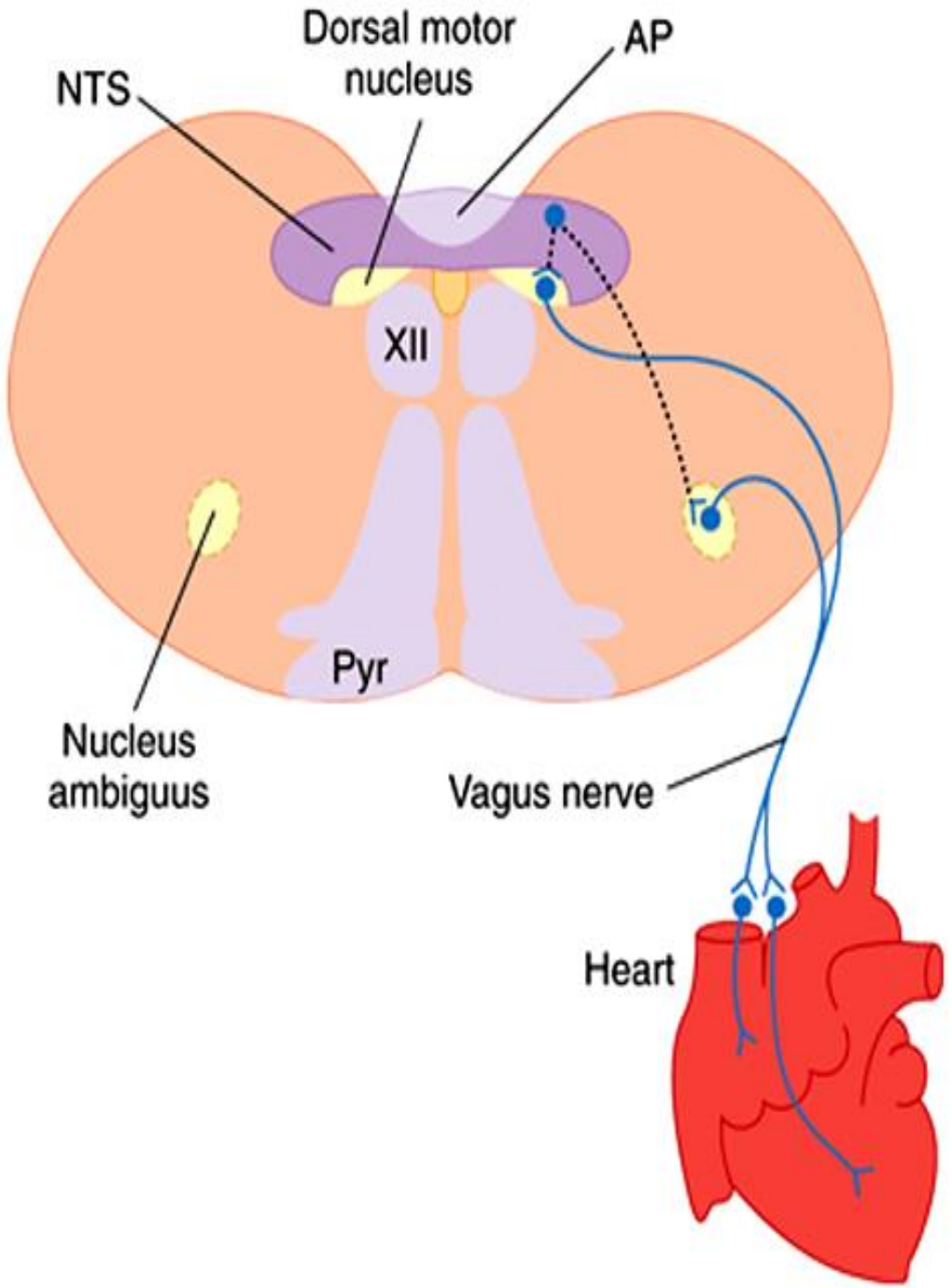
Baroreceptor mechanism/sinoaortic reflex

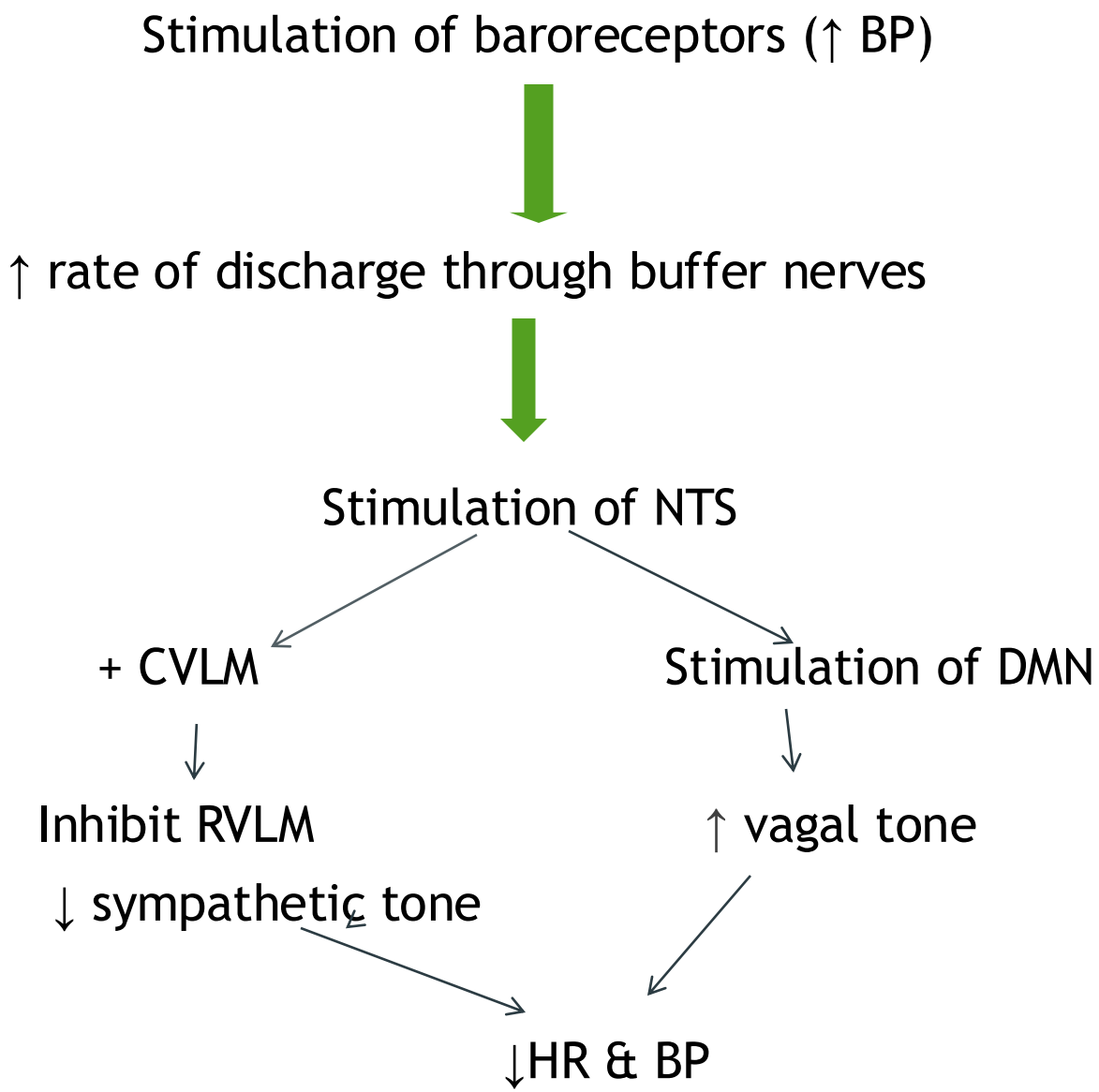
- **baroreceptors** -stretch receptors in the walls of the heart and blood vessels
- They are branched, coiled &intertwined ends of myelinated nerve fibers
- stimulated by distention of the structures in which they are located, & discharge at ↑ rate when the pressure in these structures rises.

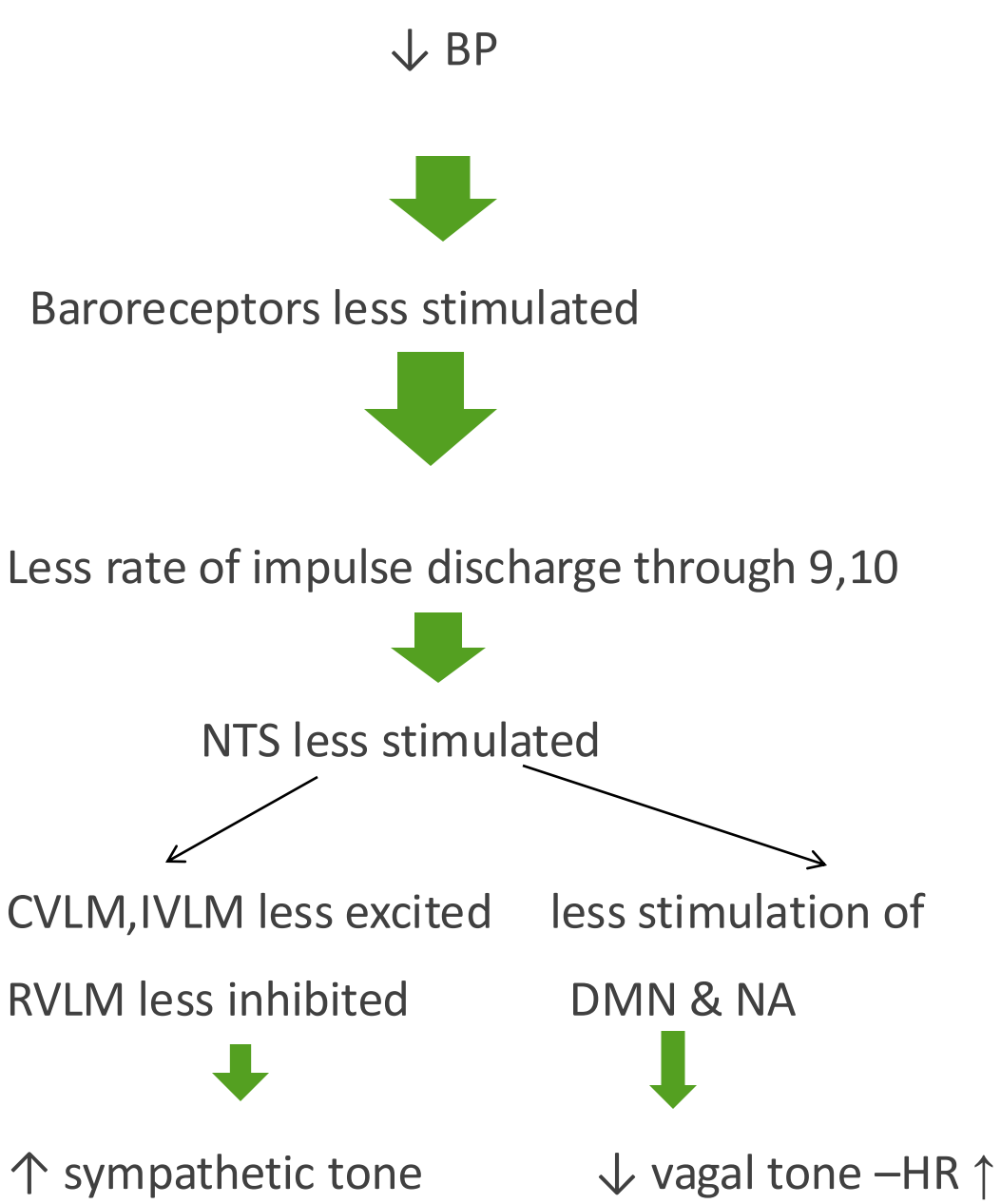
- Normal BP –impulse discharge through these nerves at low frequency – vagal tone
- ↑ BP – reflex↓ HR,BP fall
- ↓ BP – reflex ↑ HR




Baroreceptor afferents (Glu)
Medulla
NTS
IX
X
(GABA)
(Glu)
RVLM
CVLM
IVLM
Bulbospinal pathway (Glu)
Carotid sinus
IML
Thoracic cord
Aortic arch
Preganglionic sympathetic neuron (Ach)
Adrenal medulla
Heart
Postganglionic sympathetic neuron (NE)
Arteriole or venule


Dorsal motor
nucleus
AP
NTS
XII
Pyr
Nucleus
ambiguus
Vagus nerve
Heart


Stimulation of baroreceptors (↑ BP)
↑ rate of discharge through buffer nerves
Stimulation of NTS
+ CVLM
Stimulation of DMN
Inhibit RVLM
↓ sympathetic tone
↑ vagal tone
↓HR & BP


↓ BP
Baroreceptors less stimulated
Less rate of impulse discharge through 9,10
NTS less stimulated
CVLM,IVLM less excited
RVLM less inhibited
↑ sympathetic tone
less stimulation of
DMN & NA
↓ vagal tone –HR ↑

BARORECEPTOR REFLEX

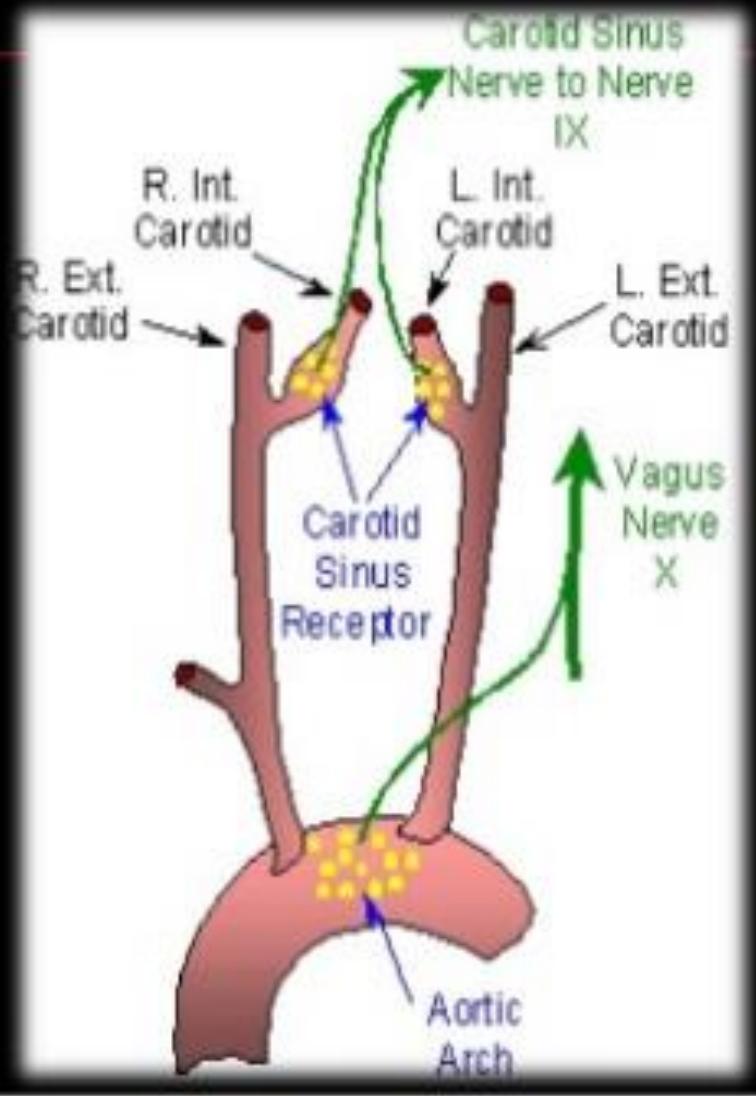
Net effect

↓ Peripheral resistance

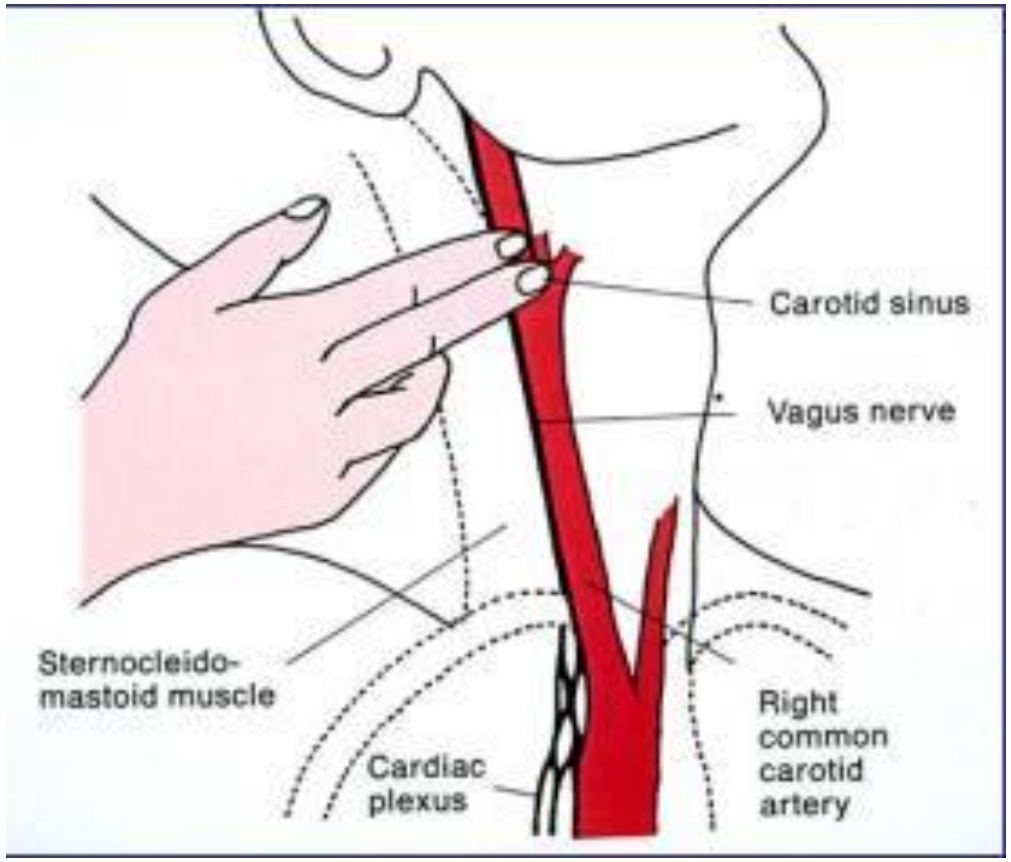
↓ Myocardial contractility

↓ Heart rate (Bradycardia)

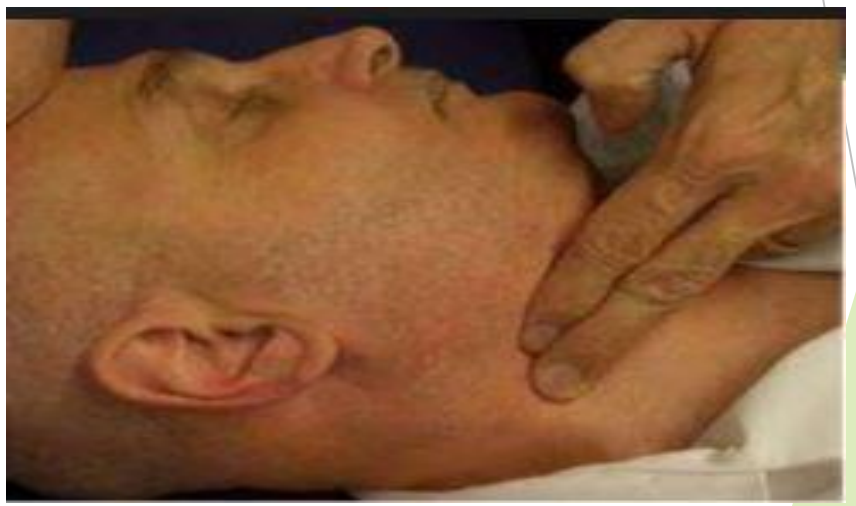
↓ Fall in BP



Carotid sinus massage



- Slows HR in paroxysmal tachycardia



MAREY'S LAW

- States that heart rate is inversely proportional to blood pressure.
- When BP increased –reflex bradycardia
- When BP decreased-reflex Tachycardia
- Reverse of this law is not applicable

Marey’s law

HR inversely Proportional to BP

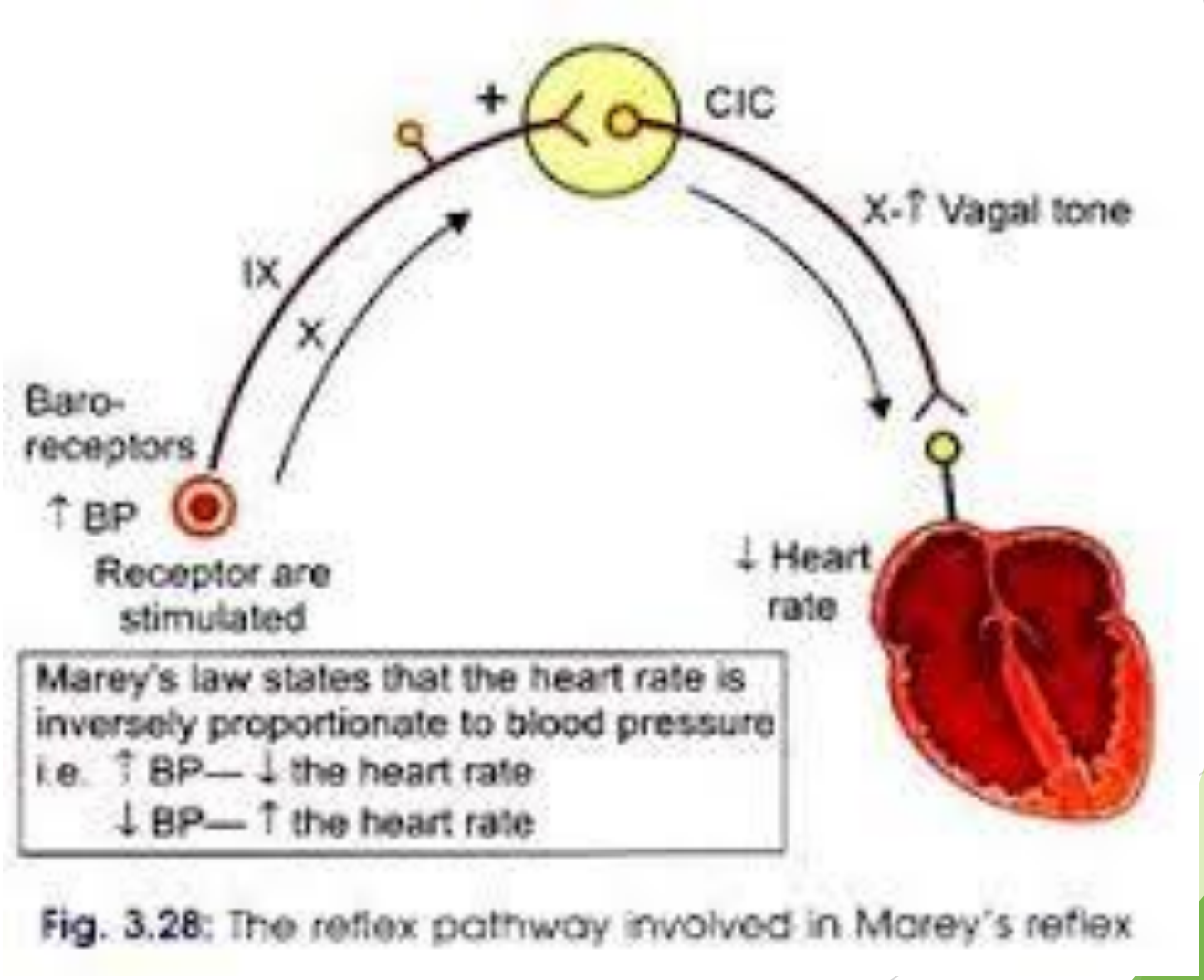


Fig. 3.28: The reflex pathway involved in Marey's reflex

Baroreceptors

- High pressure baroreceptors-

  carotid sinus, aortic arch

- Low pressure baroreceptors – cardiopulmonary receptors

  atrial stretch receptors, pulmonary receptors

Reflexes from Atrial Stretch Receptors

- Low pressure receptors
- Respond to changes in blood volume
- Location -myelinated nerve endings where veins enter atria
- type A- discharge during atrial systole
- type B – discharge during peak atrial filling ---stimulated by↑ Venous Return
- **Receptors –tachycardia producing atrial receptors**

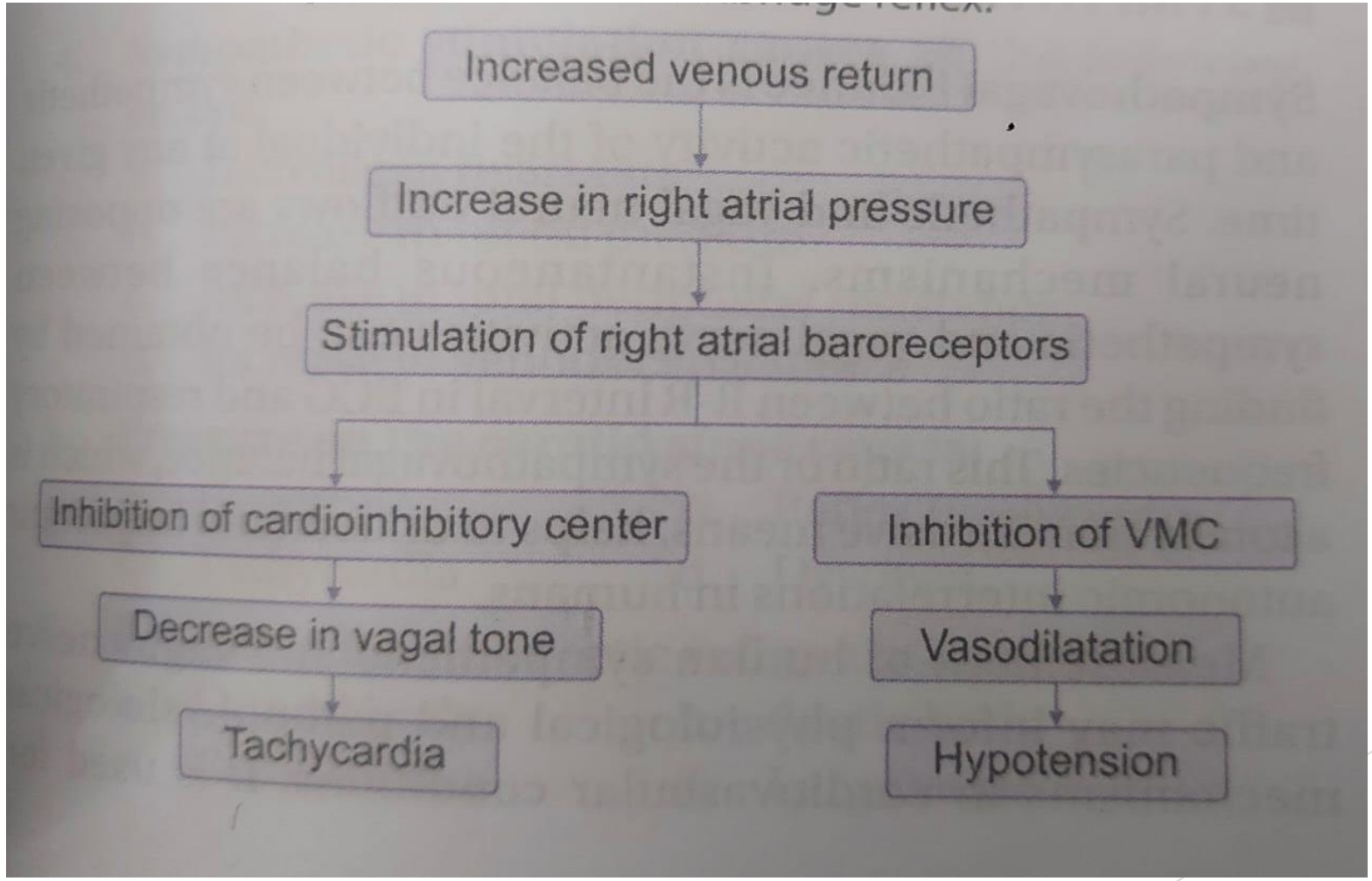
Atrial stretch reflex

↑ Venous return

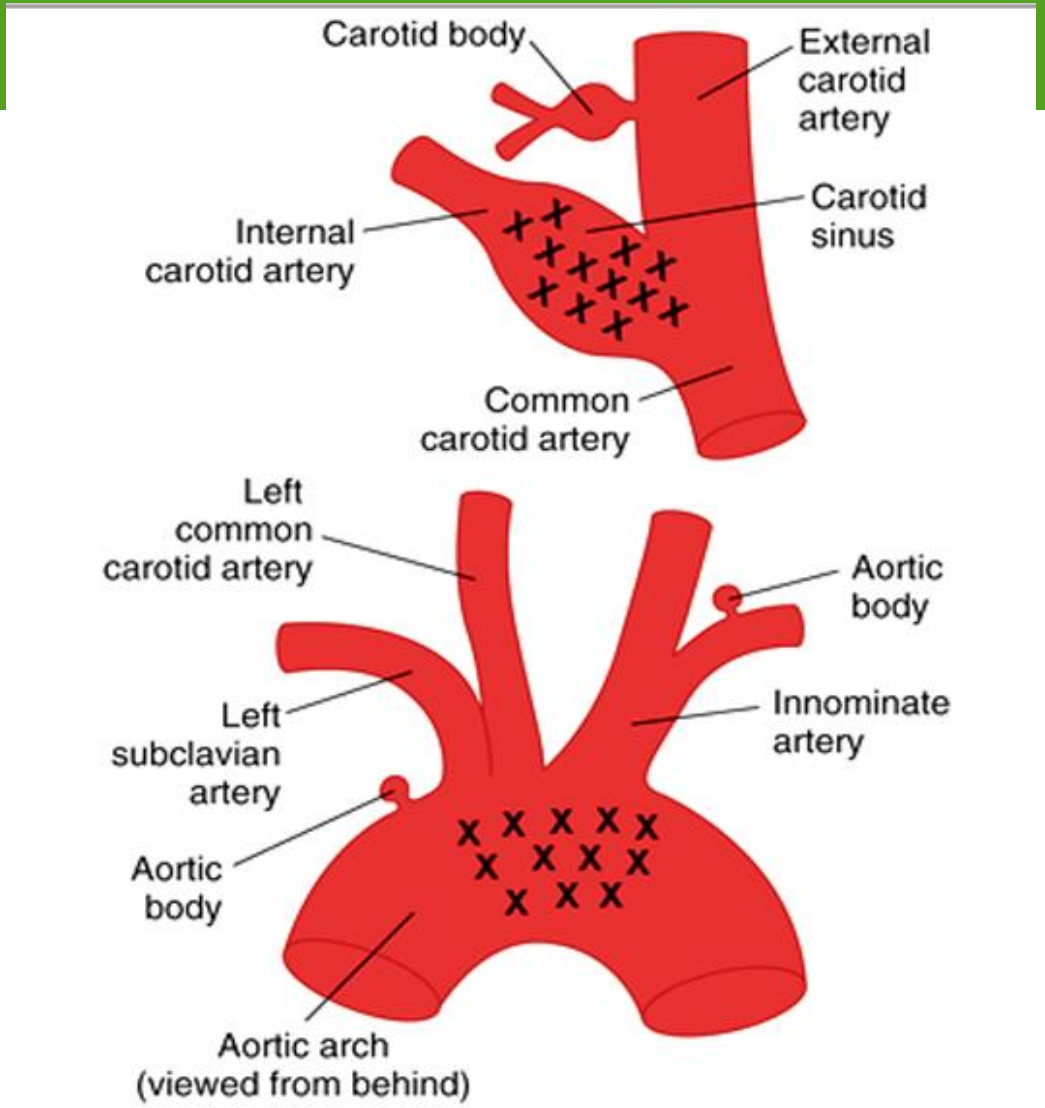
↑ filling of atria- atrial distension

Stimulation of type B receptor

Tachycardia


Increased venous return
Increase in right atrial pressure
Stimulation of right atrial baroreceptors
Inhibition of cardioinhibitory center
Decrease in vagal tone
Tachycardia
Inhibition of VMC
Vasodilatation
Hypotension

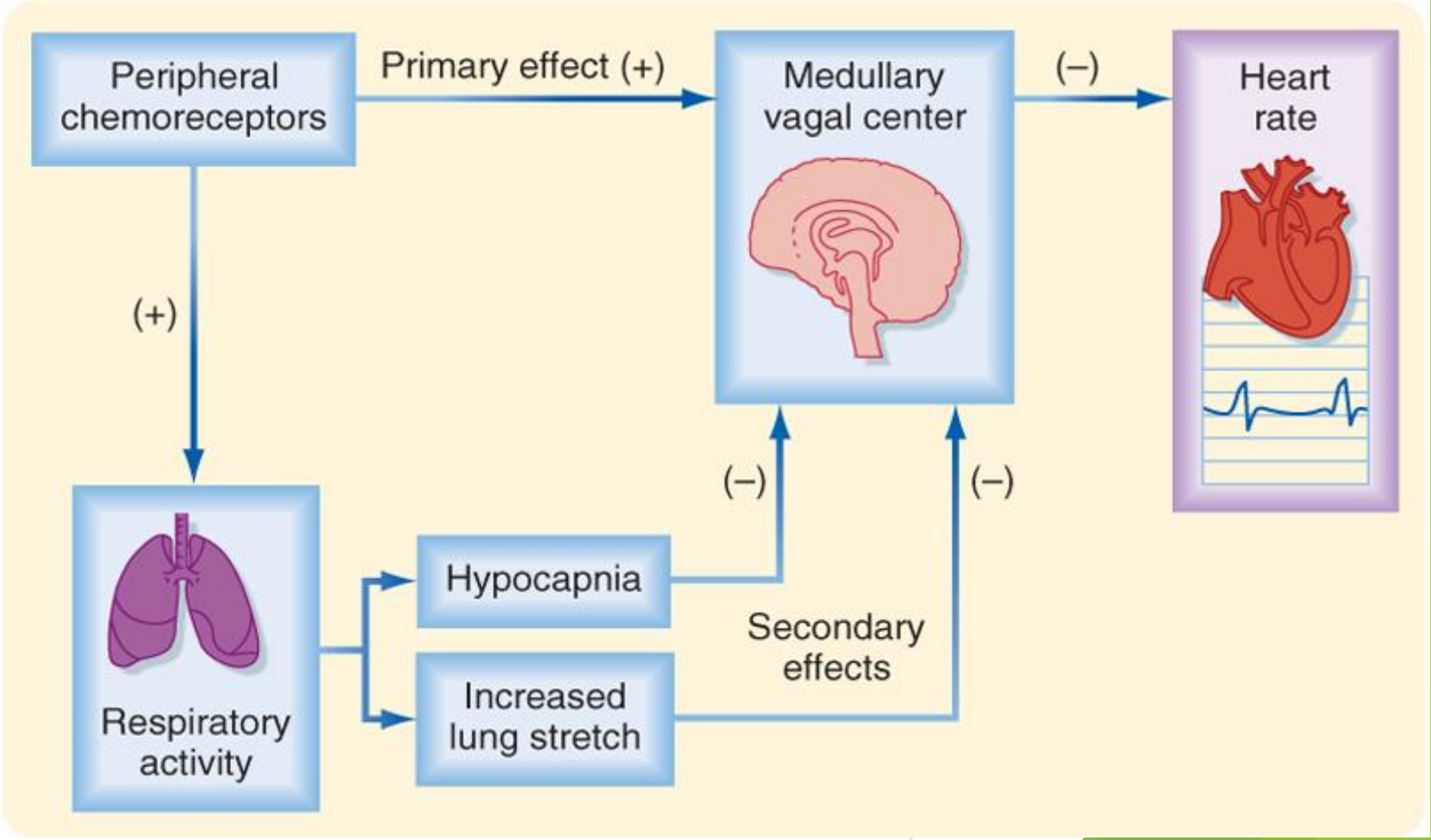
chemoreceptors



Chemoreceptor mechanism

- **Carotid body, aortic body**
- Respond to changes in blood chemistry

  ↓02,↑CO2, ↑H+ **(primary effect on resp centres)**
- stimulate VMC –vasoconstriction,
- **HR changes are variable**
- Direct effect ↑ vagal nerve activity –bradycardia
- But indirect effect of hypoxia –↑ breathing ,↑ catecholamine secretion from the adrenal medulla- both leading to tachycardia & ↑C.O


Peripheral chemoreceptors
Primary effect (+)
Medullary vagal center
(–)
Heart rate
(+)
(–)
(–)
Hypocapnia
Secondary effects
Increased lung stretch
Respiratory activity

Bainbridge Reflex

- Rapid infusion of blood or saline in anesthetized animals sometimes produces a rise in heart rate & hypotension if the initial heart rate is low.
- True reflex abolished by vagotomy
- Receptors maybe tachycardia producing atrial receptors
- Physiological significance - prevent accumulation of blood in great veins & atria

Bainbridge reflex

Infusion of saline/blood
(with initial slow HR)

Increased venous filling of atria

Stimulation of tachycardia producing type B receptor

↑Heart rate

Reflexes from LV receptors

- LV Distension stimulates stretch receptors- bradycardia ,hypotension
- Probable role in vagal tone

Coronary chemoreflex/Bezold-Jarisch reflex

- Introduction of chemicals like serotonin, veratridine, capsaicin into coronary arteries supplying LV –
- hypotension, bradycardia, apnea followed by rapid breathing
- receptors - unmyelinated C nerve endings
- Afferent – vagus
- May be Cause of hypotension following MI

Pulmonary chemoreflex

- Similar to coronary chemoreflex
- Pulmonary baroreceptors - walls of pulmonary trunk & its division-rt & lt pulmonary artery
  Receptor –juxta capillary J receptors

CNS Ischemic Response

- When blood flow to brain decreased (brain ischemia)----- hypoxia, hypercapnia & lactic acid secretion-----
- stimulates the RVLM & CIC ----increase in BP & bradycardia
- Operates only when ABP falls below 60mmHg

Cushing s reflex

- Increase in intra cranial tension leads to increased BP & bradycardia

↑ intracranial tension (↑ CSF pressure)

↓

compression of blood vessels supplying VMC

↓

Hypoxia & hypercapnia stimulate RVLM

↓

Vasoconstriction , ↑ BP

↓

↑ cerebral blood flow(relieves ischemia)

Activation of baroreceptor reflex due to increase in BP

↓

Reflex bradycardia

Sinus arrythmia

HR ↑ - inspiration & HR ↓ -expiration

- spillover" of signals from the medullary respiratory center into the adjacent VMC during inspiratory and expiratory cycles of respiration
- spillover signals cause alternate ↑ and ↓ in the number of impulses transmitted through the sympathetic & vagus nerves to the heart

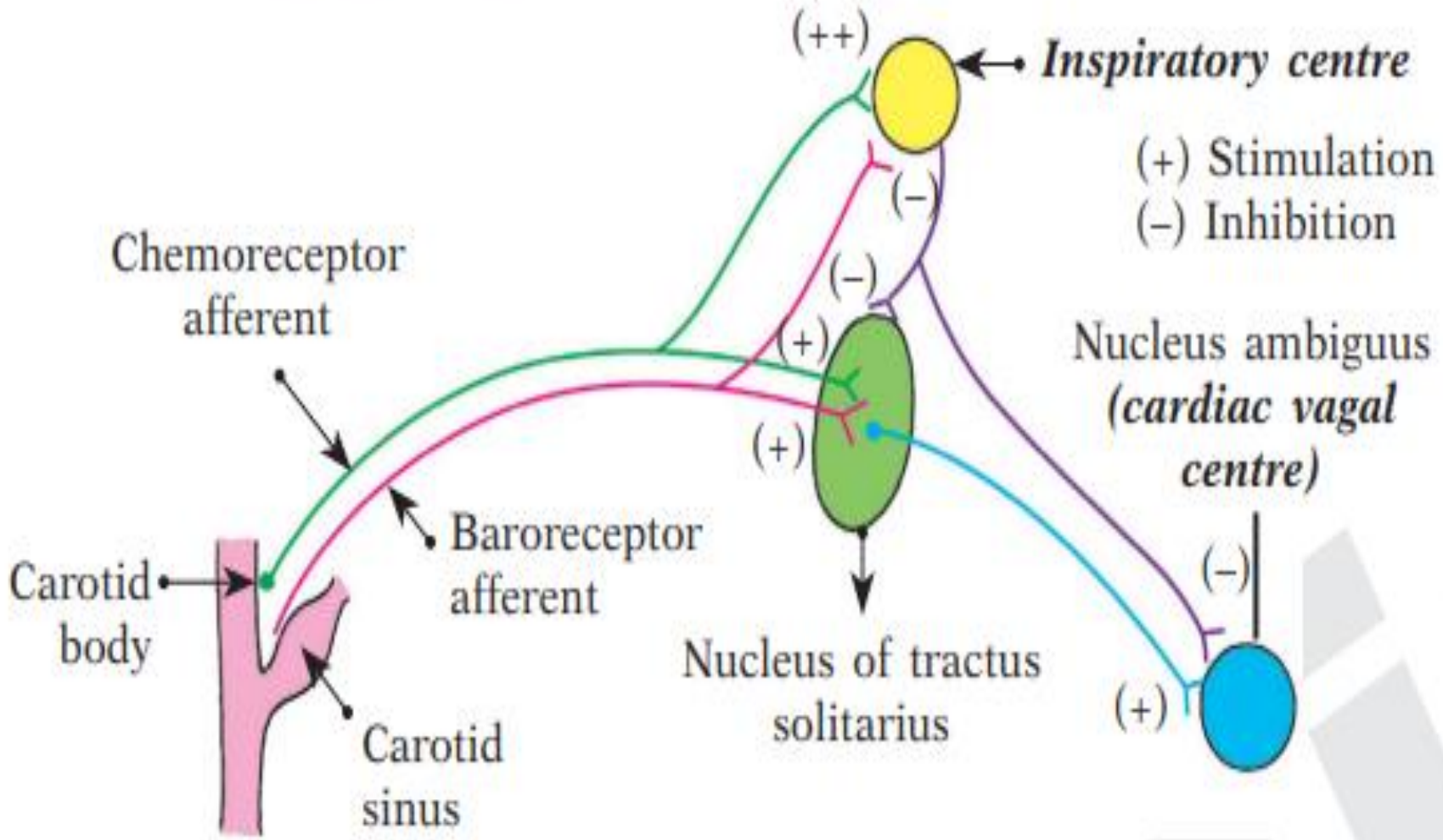


Fig. 5.42 Pathways relating interaction of cardiac and respiratory reflexes

Sinus arrythmia

inspiration

↓

stimulation of pulmonary stretch receptors

↓

Vagal afferents inhibit cardioinhibitory center in medulla

↓

↑ HR (vagal tone↓)

- Expiration –no inhibitory impulses to cardiac vagal center ----------increased vagal tone -------sinus bradycardia

Sinus arrythmia

inspiration

↓

↑ Venous return

↓

stimulation of atrial stretch receptors

↓

↑ HR

Oculocardiac reflex

- Pressure on eye balls causes reflex ↓HR

by ↑ vagal tone

Afferent – trigeminal

Efferent –vagus

Useful in slowing HR in paroxysmal tachycardia

Regulation by higher centre

Hypothalamus-stimulation of posterior group (sym centre)–increase in HR

anterior –decrease HR

cerebral cortex– limbic system-excitement – increase HR

fear –decrease HR

Humoral Regulation

1)Hormones- catecholamines,Thyroid hormones,insulin,glucagon,,GH

- Thyroid hormones-increase no β receptor,increase myosin ATPase activity,increase sensitivity of β receptor to catecholamines
- Epinephrine-through β1 receptor increase HR

2)CHEMICALS

- role of O_2,CO_2,H^+
- hypoxia ↑ HR
- Hypercapnea and acidosis decrease HR
- High body temp ↑ HR

Table 33–3 Factors Affecting Heart Rate.
Heart rate accelerated by:
Decreased activity of arterial baroreceptors
Increased activity of atrial stretch receptors
Inspiration
Excitement
Anger
Most painful stimuli
Hypoxia
Exercise
Thyroid hormones
Fever
Heart rate slowed by:
Increased activity of arterial baroreceptors
Expiration
Fear
Grief
Stimulation of pain fibers in trigeminal nerve
Increased intracranial pressure

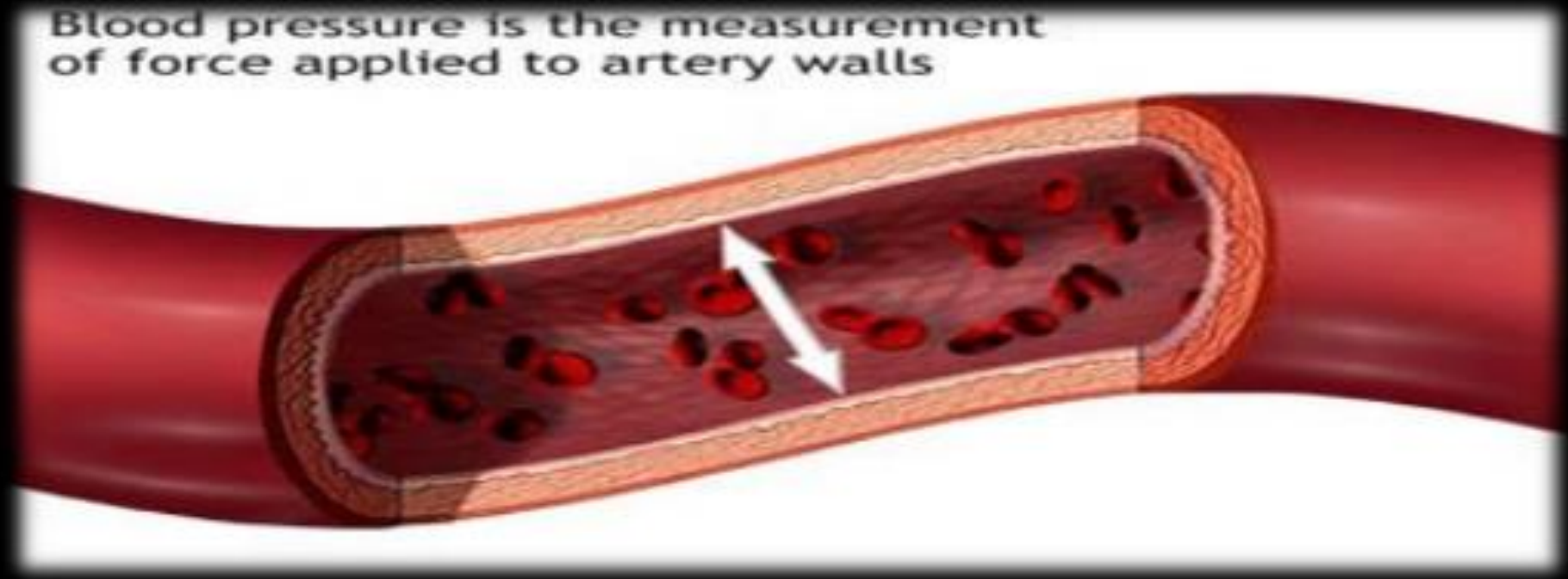
Tachycardia

- Within physiological limits an increase in heart rate increases cardiac output
- Beyond physiological limit, when heart beats rapidly diastolic phase is much reduced ---initial fibre length smaller ----↓stroke volume ----- ↓cardiac output

Blood pressure

ARTERIAL BLOOD PRESSURE

Definition : Arterial blood pressure can be defined as the lateral pressure exerted by the moving column of blood on the walls of the arteries.



Blood pressure

Systolic pressure

Maximum pressure produced during systolic phase of cardiac cycle.

100-140mmHg

Diastolic pressure

Minimum pressure produced during diastolic phase of cardiac cycle. NL value 60-90mmHg

PULSE PRESSURE

DIFFERENCE BETWEEN SYSTOLIC & DIASTOLIC BP = 40-50mm Hg

MEAN ARTERIAL PRESSURE

Average pressure produced during cardiac cycle

Diastolic BP +1/3 PULSE PRESSURE (93mmHg)

90 - 100 mm Hg

Variations in BP - physiological

- Age- increase in old age (loss of elasticity of blood vessels)
- Sex-before menopause, BP is low in females (progesterone cause vasodilation)
- After meals-BP increases (increase in metabolism & blood flow)
- Diurnal variation-less during morning
- Sleep-marked fall in BP by 20mm Hg (dippers)
- Environment temp- Hot -vasodilation-decrease BP
- Cold ❄ —vasoconstriction—increase BP

Variations in BP - physiological

- Exercise-SBP increase in all types of exercise due to increase in SV & HR

  DBP decrease in moderate & severe exercise due to vasodilation & reduction in PR .Mild exercise increase in sym activity-vasoconstriction –increase in DBP
- Emotions –BP increases due to symp discharge(white coat HTN)
- Posture- sudden standing from lying down--- venous pooling--- decrease in CO---decrease in SBP

  less baroreceptor reflex--- vasoconstriction---DBP increased

DETERMINANTS OF BP

- 1. cardiac output
- 2.Peripheral resistance
- 3.elasticity of blood vessel
- 4.blood volume
- 5.volume of vascular space

Factors affecting CO

- CO = SV X HR

Stroke volume

PRE LOAD
AFTER LOAD
MYOCARDIAL CONTACTILITY

- 1.CARDIAC OUTPUT : determines SBP.
- CO = SV X HR
- SV is determined by myocardial contractility
- Change in myocardial contractility or HR affects systolic BP

PERIPHERAL RESISTANCE

Factors determining PR –

- diameter of blood vessels
- viscosity &
- velocity of blood flow
- Normal condition –velocity & viscosity donot change, calibre of blood vessel is major determinant
- Peripheral resistance determines DBP

Determinants of BP

3)ELASITICITY of blood vessels: determine SBP

- As we age, atherosclerosis cause elasticity to decrease.

- SBP increases in elderly, DBP not affected –so wide pulse pressure upto 60-70mm Hg

- 4) Blood volume: Mean arterial pressure increases
- 5) Volume of vascular space: decrease in vascular space due to vasoconstriction ---increase in SBP

MEASUREMENT OF BP

- DIRECT-canula introduced into an artery & connected to manometer
- INDIRECT -Sphygmomanometer

SPHYGMOMANOMETER

- Mercury manometer, a cuff & hand pump
- Mercury manometer has reservoir & manometer
- BP cuff-rubber bag of 10X11 cm size ,wrapped in linen cloth,width 12.5 cm in adults & 8cm in children
- Hand pump –provided for inflating cuff,a valve for deflation


fidelis


AccuSure
ARTERY
AccuSure
RIGHT ARM
LEFT ARM


MEDTECH
10-08 00:07 PM
SYS
mmHg
120
DIA
mmHg
80
PUL
/min
72
SYS-
mmHg
139
DIA-
mmHg
85
WHO
15
PUL-
/MIN
NOVACHECK BP-09N
SET
START
STOP
MEM

STETHOSCOPE

- Chest piece & ear piece connected by rubber tube
- Chest piece has diaphragm & bell adjusted by lock
- Ear piece fit in external auditory canal


Search inside image

KOROTKOFF sounds

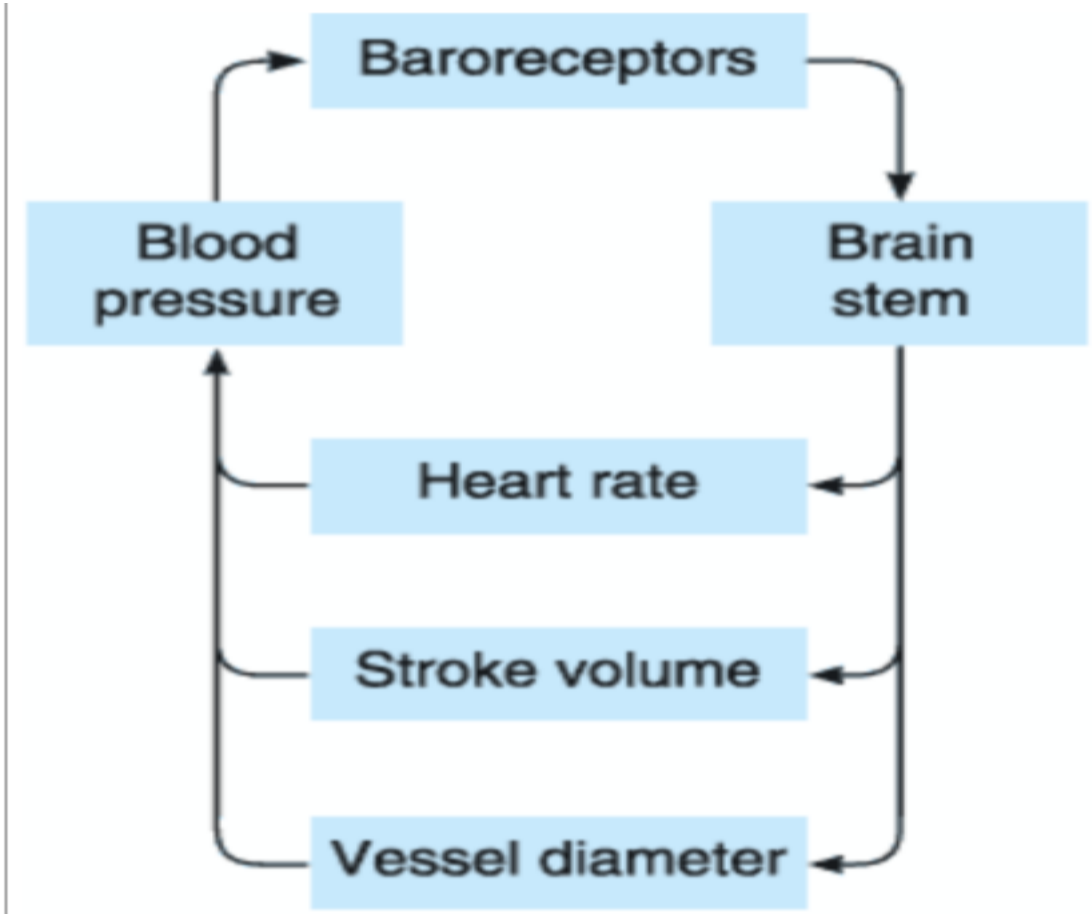
- Series of sounds heard during deflation
- 5 PHASES
- Phase 1:sudden appearance of clear tapping sound
- Phase 2.soft ,murmuring
- Phase 3:murmering,more loud & clear
- Phase 4:soft & mufled
- Phase5:disappears

Regulation of BP

- 1) short term regulation (rapidly acting)
- 2) intermediate regulation
- 3) long term regulation

Short term regulation

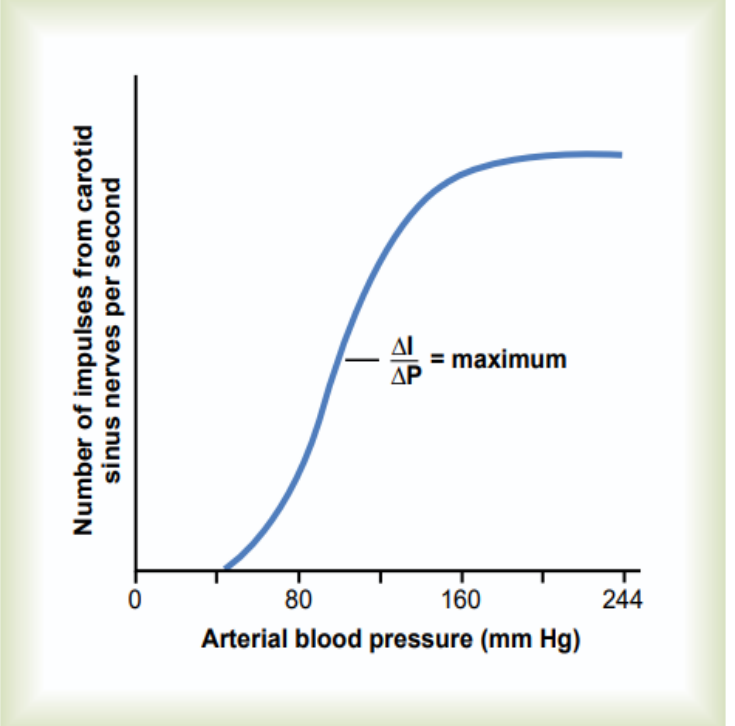
- Within seconds to minutes
- Short lasting response
- 1)Baroreceptor reflex (also include low pressure atrial stretch receptors & Bainbridge reflex)
- 2)chemoreceptor reflex
- 3) CNS ischemic response & Cushing s reflex



Feedback control of blood pressure.

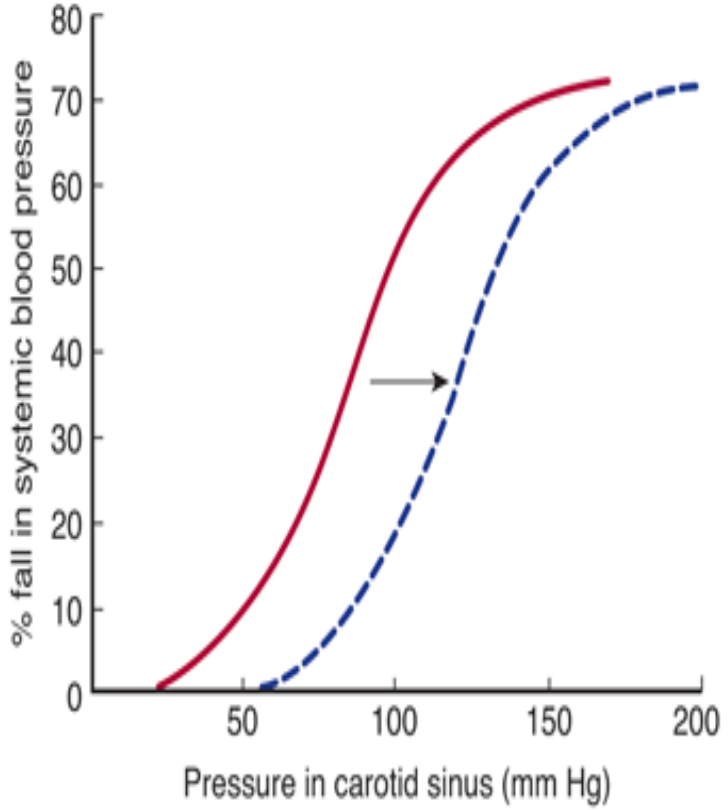
Baroreceptor reflex

- Works within BP range of 60 – 180 mm Hg
- Max BR stimulation at 180 mm Hg
- BR more sensitive to pulsatile pressure change than constant



Resetting of BR

- Baroreceptor exposed to high BP constantly for long period---BR discharge less (high BP considered nl by BR)----RESETTING OF BR



Applied aspect

- Neurogenic HTN-

  baroreceptor nerves denervated, lesion of NTS
- Carotid sinus massage

ATRIAL STRETCH REFLEX

a. As low pressure receptors:

Increased venous return

↓

Increased atrial filling

↓

Stimulation of type – B receptors

↓

Sympathetic inhibition

↓

Vasodilation, ↓ BP, but ↑ HR

- REFLEX endocrine changes occuring due to atrial stretch
- 1.when atria stretches---impulse to NTS-----inhibition of posterior pituitary----ADH secretion decreased----urine output increased—decrease in BP
- Atria stretches ---ANP produced---Na & water excretion---decrease in BLD volume---decrease in BP

CNS Ischemic Response

- When blood flow to brain decreased (brain ischemia)----- hypoxia, hypercapnia & lactic acid secretion----- stimulates the RVLM & CIC ----increase in BP & bradycardia
- Operates only when ABP falls below 60mmHg

↑ intracranial tension (↑ CSF pressure)

↓

compression of blood vessels supplying VMC

↓

Hypoxia & hypercapnia stimulate RVLM

↓

Vasoconstriction , ↑ BP

↓ ↑ cerebral blood flow(relieves ischemia)

Activation of baroreceptor reflex due to increase in BP

↓

Reflex bradycardia

Chemoreceptor mechanism

- Carotid body,aortic body
- Respond to changes in blood chemistry

  ↓02,↑CO2, ↑H+

  - stimulate VMC –vasoconstriction ---increase in BP
  - Direct effect ↑ vagal nerve activity –bradycardia
  - But hypoxia –↑ breathing ,↑ catecholamine secretion -tachycardia, ↑CO

Intermediate regulating mechanism

- Correct BP by altering blood volume. act in few minutes and remain functional for days to months.
- 1. capillary fluid shift mechanism
- 2. Stress relaxation reverse stress relaxation mech
- 3. Renin Angiotensin mechanism

Capillary fluid shift mechanism

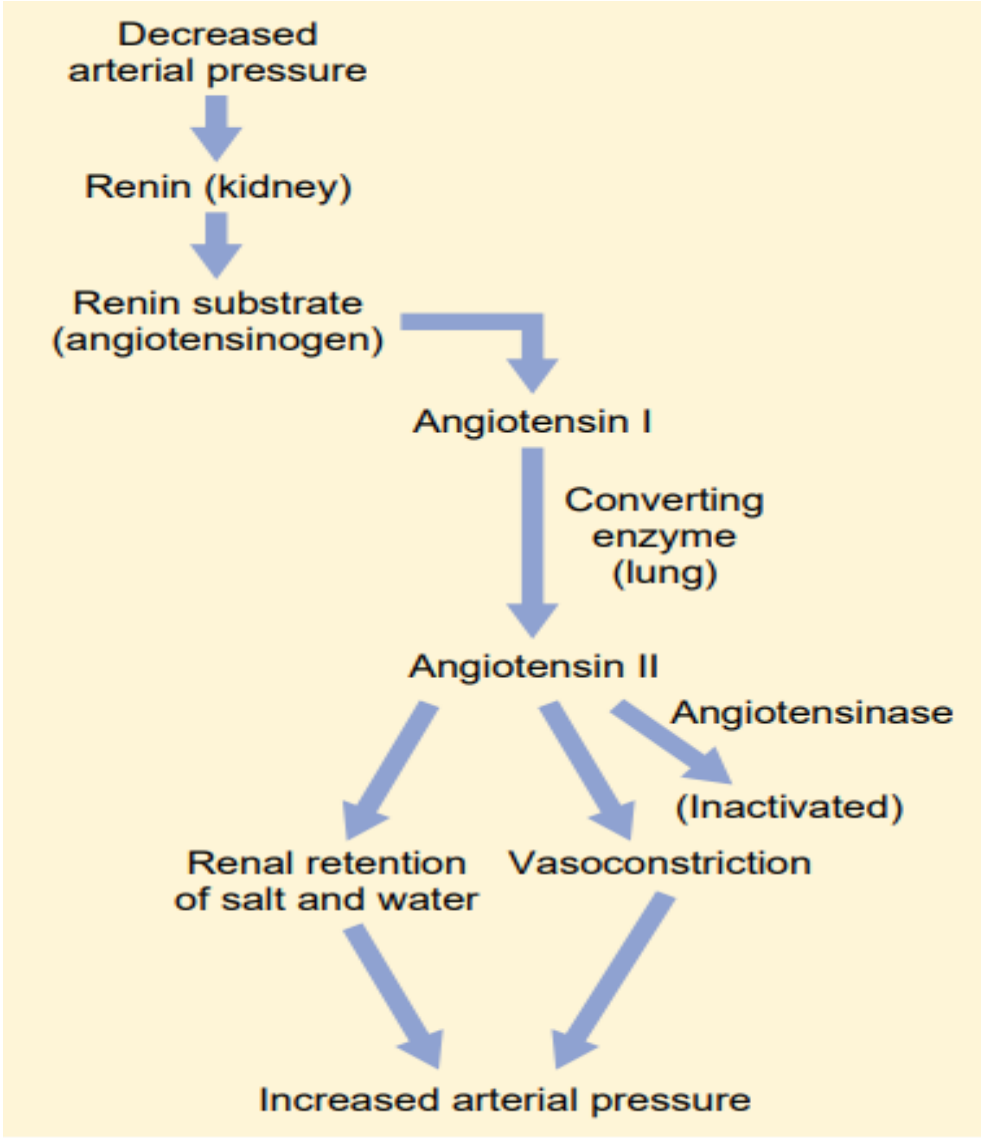
- Capillary hydrostatic pressure increased in arterial end
- More filtration
- Fluid shifts out of capillaries returning blood volume & thereby BP to normal
- 2 times more effective than BR mechanism

Stress relaxation mechanism

- Vascular tone readjustment
- Stretch continued for some time leads to relaxation of smooth muscles (vasodilation) --- Venous return normalize- CO normal-- BP to normal
- Reverse stress relaxation when BP is less

Renin angiotensin aldosterone mechanism

- Decrease in BP----renal perfusion pressure decreases----stimulation of JGA



Angiotensin II

- 1)Potent vasoconstrictor---increase BP
- 2)Stimulates aldosterone secretion from adrenal cortex---Na &water retention---increase blood volume---BP increase
- 3)Stimulates ADH secretion---H20 retention—BP increase
- 4)Stimulates catecholamine release from adrenal medulla—increase BP
- 5)Increase Na reabsorption from PCT (direct effect)—increase BP

Long term mechanisms

- Slow to start its action.last for days to years
- Basic mechanism by regulating blood volume
- Kidney plays important role
- Direct (renal body fluid mech) & indirect mechanism (through action of hormones)
- Directly act on glomerular capillaries
- (capillary fluid shift mech – renal body fluid feedback mechanism)

- Pressure natriuresis and diuresis

DIRECT- pressure diuresis & natriuresis

1) Directly act on glomerular capillaries - capillary fluid shift mech – renal body fluid feedback mechanism

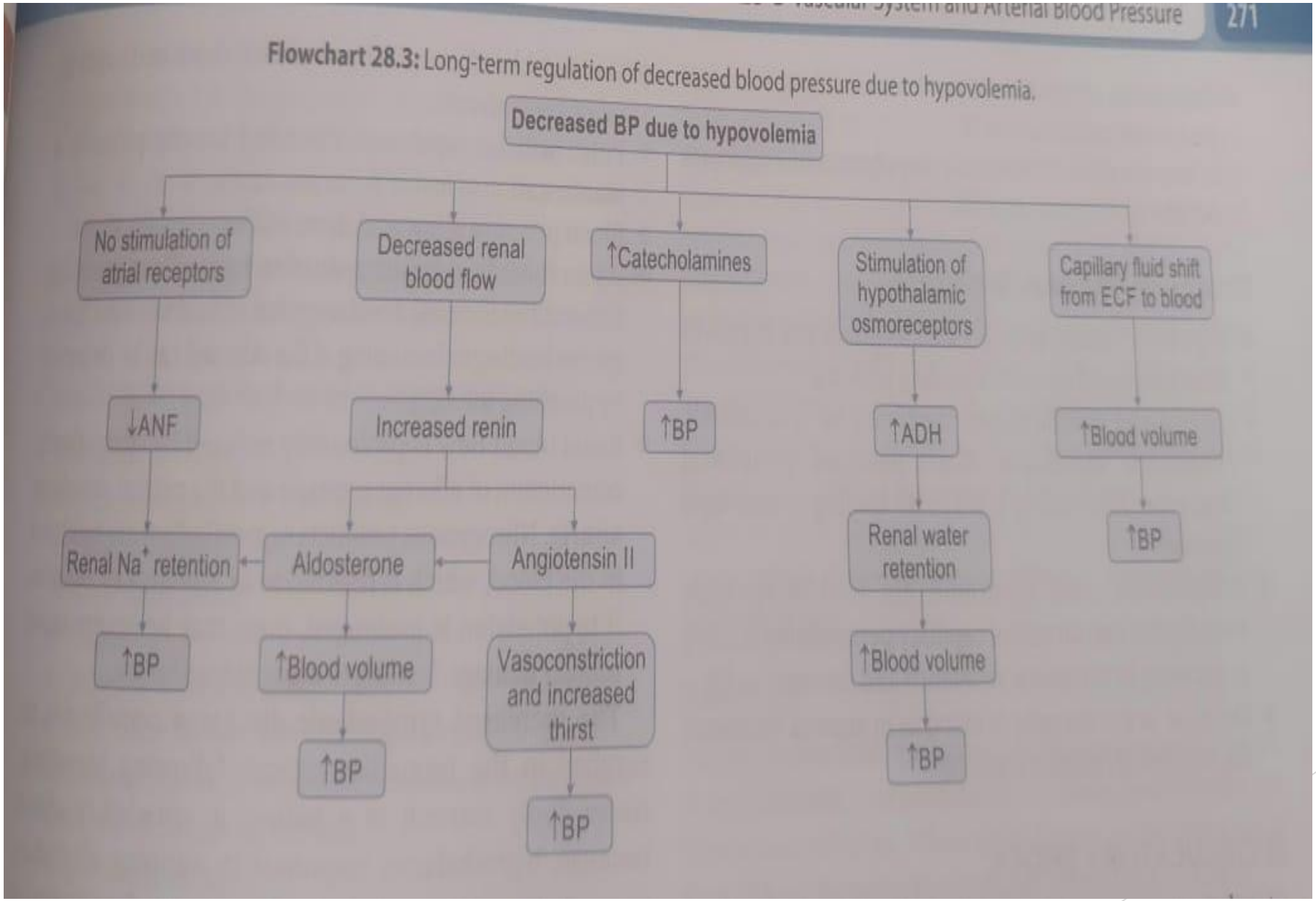
High ECF volume---increase in B P---- increase in GFR--- loss of Na+ & H20---decrease in ECF volume ----decrease in BP -Pressure natriuresis and diuresis

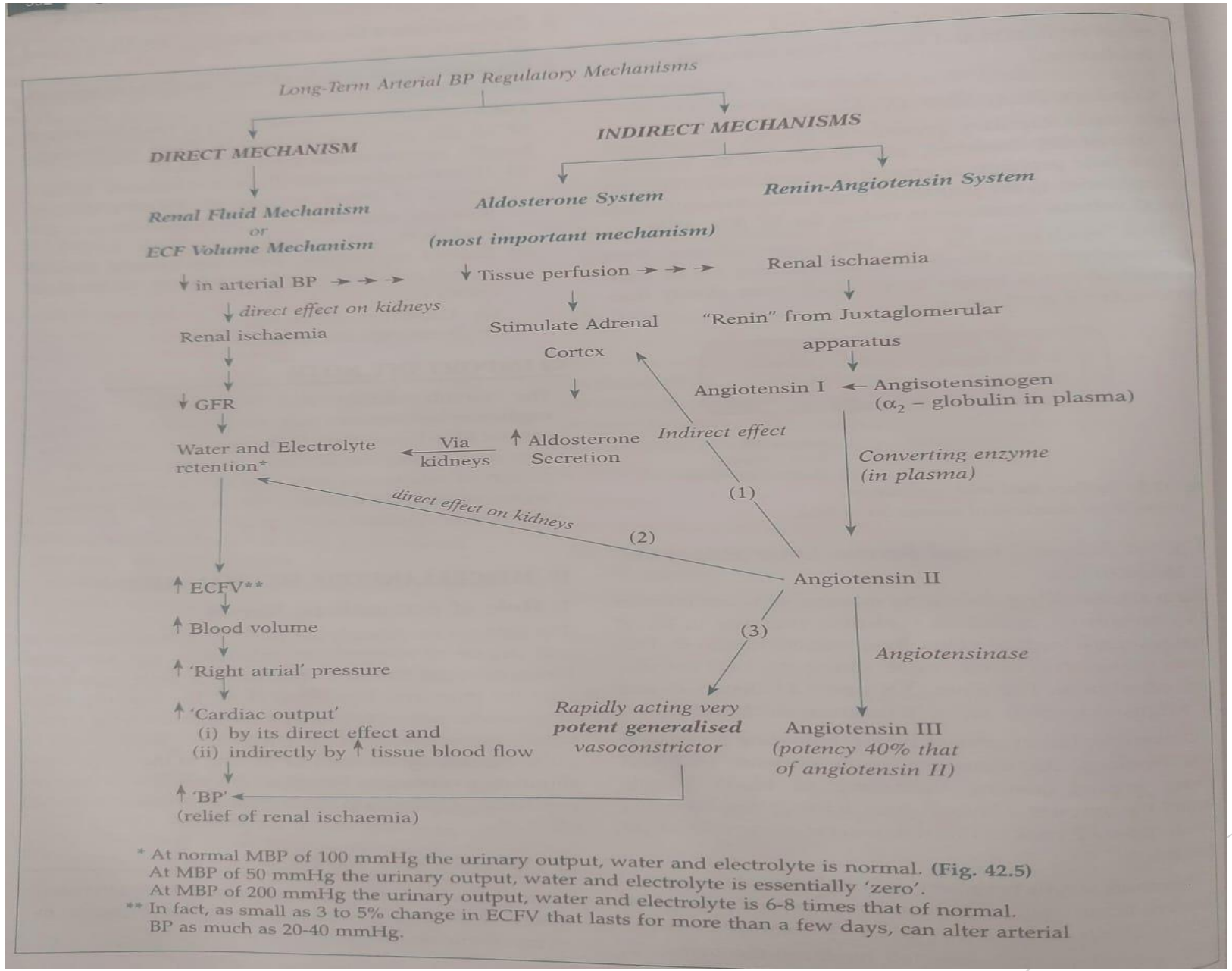
- Very strong mechanism

INDIRECT MECHANISM

- 1.RENIN –ANGIOTENSIN MECHANISM
- 2.ATRIAL NATRIURETIC PEPTIDE (ANP)-secreted by atrium - 1. Na+ & water excretion 2. vasodilation
- 3.CATECHOLAMINES- increases BP .Epinephrine –increases SBP. Norepinephrine –increases SBP & DBP
- 4. ADH- increases water reabsorption, vasoconstriction

Flowchart 28.3: Long-term regulation of decreased blood pressure due to hypovolemia.





* At normal MBP of 100 mmHg the urinary output, water and electrolyte is normal. **(Fig. 42.5)**
At MBP of 50 mmHg the urinary output, water and electrolyte is essentially 'zero'.
At MBP of 200 mmHg the urinary output, water and electrolyte is 6-8 times that of normal.
** In fact, as small as 3 to 5% change in ECFV that lasts for more than a few days, can alter arterial BP as much as 20-40 mmHg.

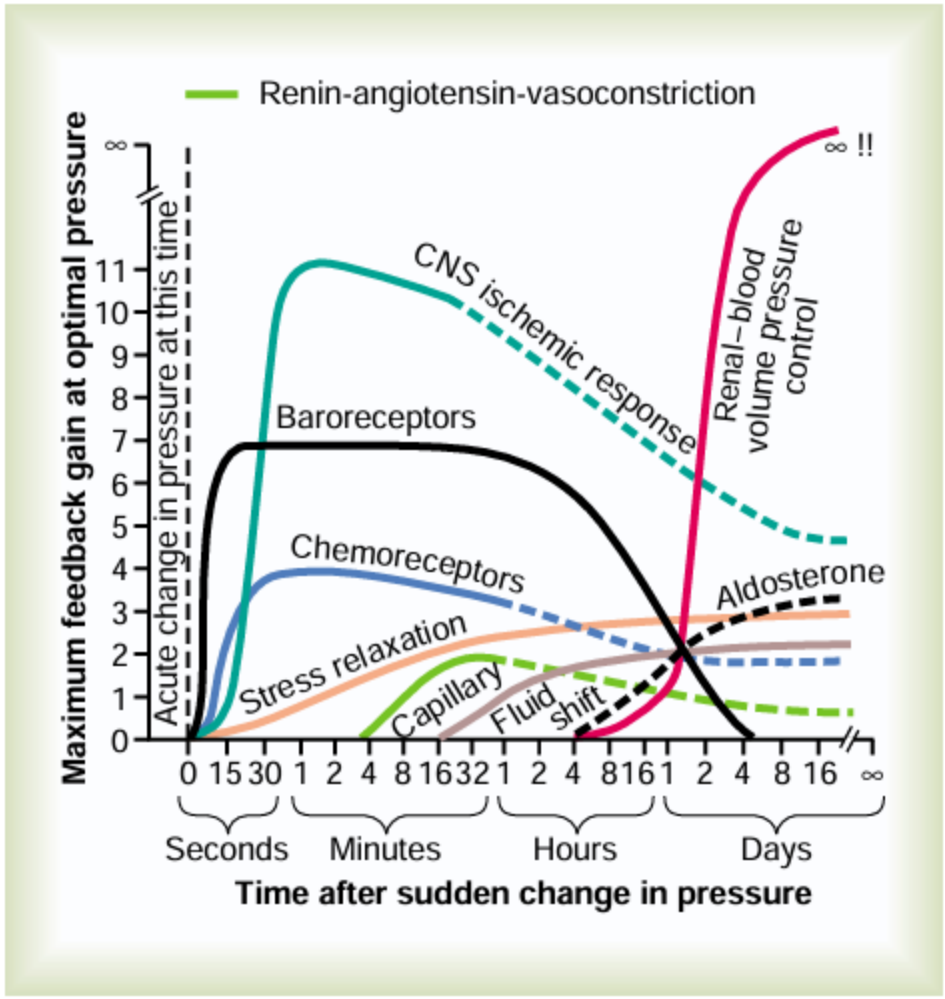


Figure 19–15

Approximate potency of various arterial pressure control mechanisms at different time intervals after onset of a disturbance to the arterial pressure. Note especially the infinite gain (∞) of the renal body fluid pressure control mechanism that occurs after a few weeks' time. (Redrawn from Guyton AC: Arterial Pressure and Hypertension. Philadelphia: WB Saunders Co, 1980.)

Respiratory waves in BP

- Traube Hering waves – fluctuations in BP synchronised with respiration

  With each cycle of respiration, arterial BP usually rise and

  fall 4 -6 mm Hg in a wave like manner
- MAYER waves – vasomotor waves – some larger waves 10-40 mm Hg at times are noted during hypotension.
- chemoreceptor discharge contribute to this
- Reflex oscillation of 1 or more nervous reflex mechanisms

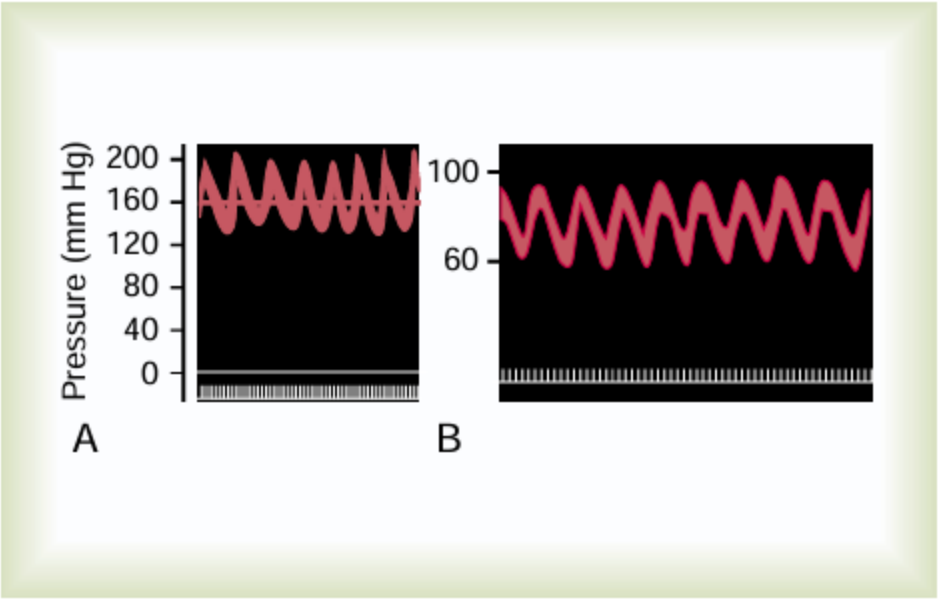


Figure 18–11

A, Vasomotor waves caused by oscillation of the CNS ischemic response. *B*, Vasomotor waves caused by baroreceptor reflex oscillation.

Local regulation of CVS

- Autoregulation
- Vasodilator metabolites
- Vasoconstrictor metabolites
- Products secreted by endothelium

Autoregulation

The capacity of tissues to regulate their own blood flow

- Organs like kidneys, brain, mesentry, skeletal muscle, myocardium
- Intrinsic capacity of an organ or tissues to regulate their own blood flow over a wide range of arterial pressure by adjusting vascular resistance
- Myogenic theory
- Metabolic theory

Myogenic theory

- When vascular smooth muscle is stretched, it responds by contraction.(intrinsic contractile response of smooth muscle to stretch)
- Vasoconstriction---decrease blood flow
- Can occur in the absence of neural or hormonal influence
- Law of Laplace

Metabolic theory

- Vasodilator metabolites – when blood flow decreases ,these metabolites accumulate and blood flow return to normal

1. Hypoxia, hypercapnia, acidosis,
2. increase in temperature,
3. K^+ ,
4. lactate,
5. histamine, adenosine
6. Prostacyclin ,NO produced by vascular endothelium

- When blood flow increase---metabolites washed off

Vasoconstrictor metabolites

- Serotonin from platelets
- Decrease in tissue temperature
- Thromboxane A 2
- Substances secreted by endothelium-
- endothelins
- Thromboxane A 2

Clinical application-HYPERTENSION

- Hypertension – sustained elevation of systemic arterial pressure above normal limits
- Systolic >140mm of Hg, Diastolic > 90 mmof Hg in young adult

- JNC 8 (joint National committee for HYPERTENSION)classification

JNC8 classification of hypertension

Category	Systolic		Diastolic
Normal	< 120	and	< 80
Prehypertension	120-139	or	80-89
High Blood Pressure/Hypertension			
Stage 1 Hypertension	140-159	or	90-99
Stage 2 Hypertension	≥ 160	or	≥ 100

Etiologic Classification

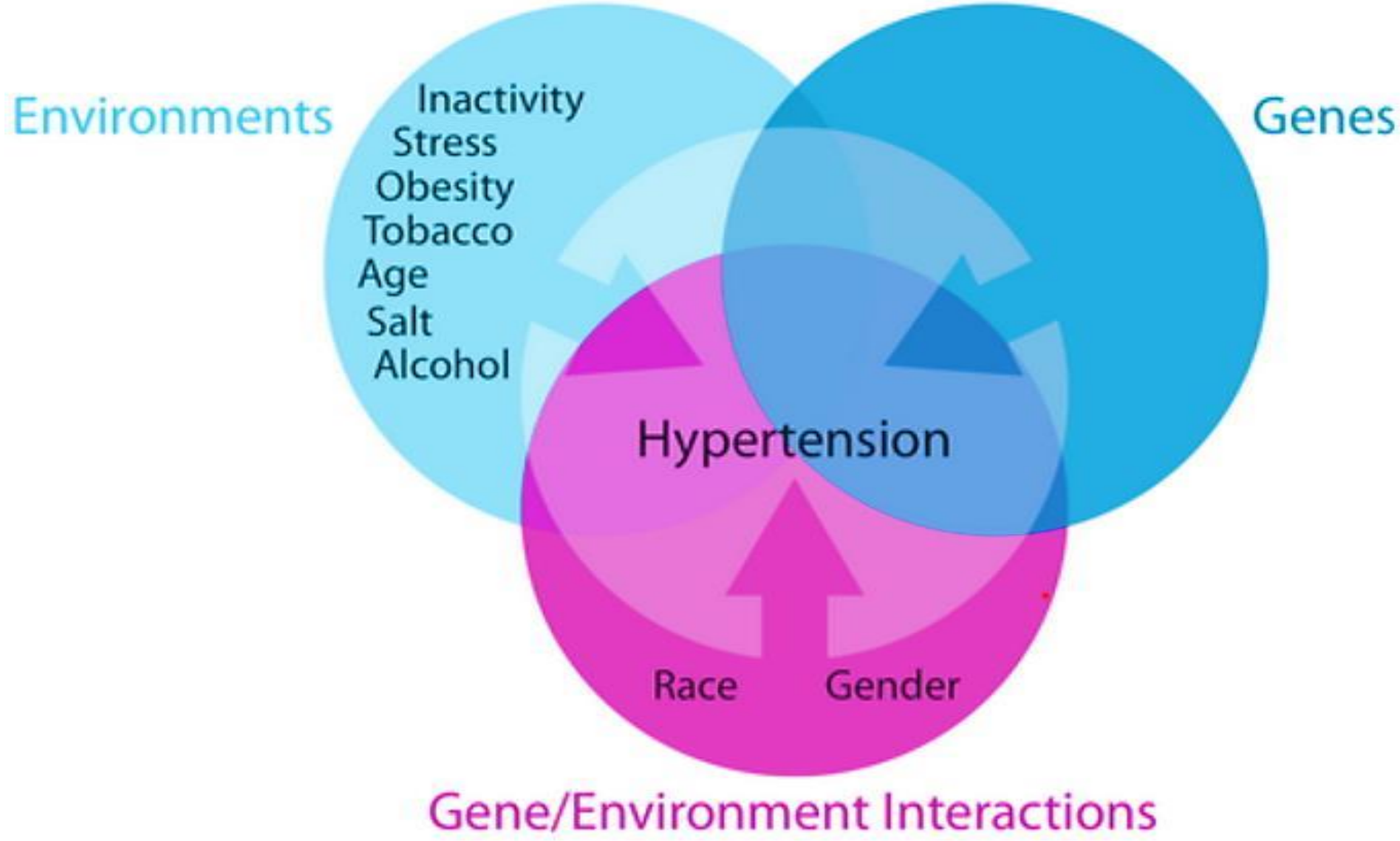
- **Primary/Essential Hypertension (88%)**
  unknown cause

- **Secondary Hypertension (12%)**
  underlying cause present

Primary/ Essential Hypertension

- Polygenic in origin
  - Environmental factors involved
  - Salt intake, obesity

- Usually symptomless, sometimes headache


Environments
Inactivity
Stress
Obesity
Tobacco
Age
Salt
Alcohol
Genes
Hypertension
Race
Gender
Gene/Environment Interactions

Risk factors

- Heredity
- Obesity
- Stress
- Smoking

Obesity

- Cardiac output is increased
- Sympathetic nerve activity is increased (especially in kidneys)
- Angiotensin II and aldosterone levels are increased 2-3 fold
- Renal-pressure natriuresis mechanism is impaired

Secondary hypertension

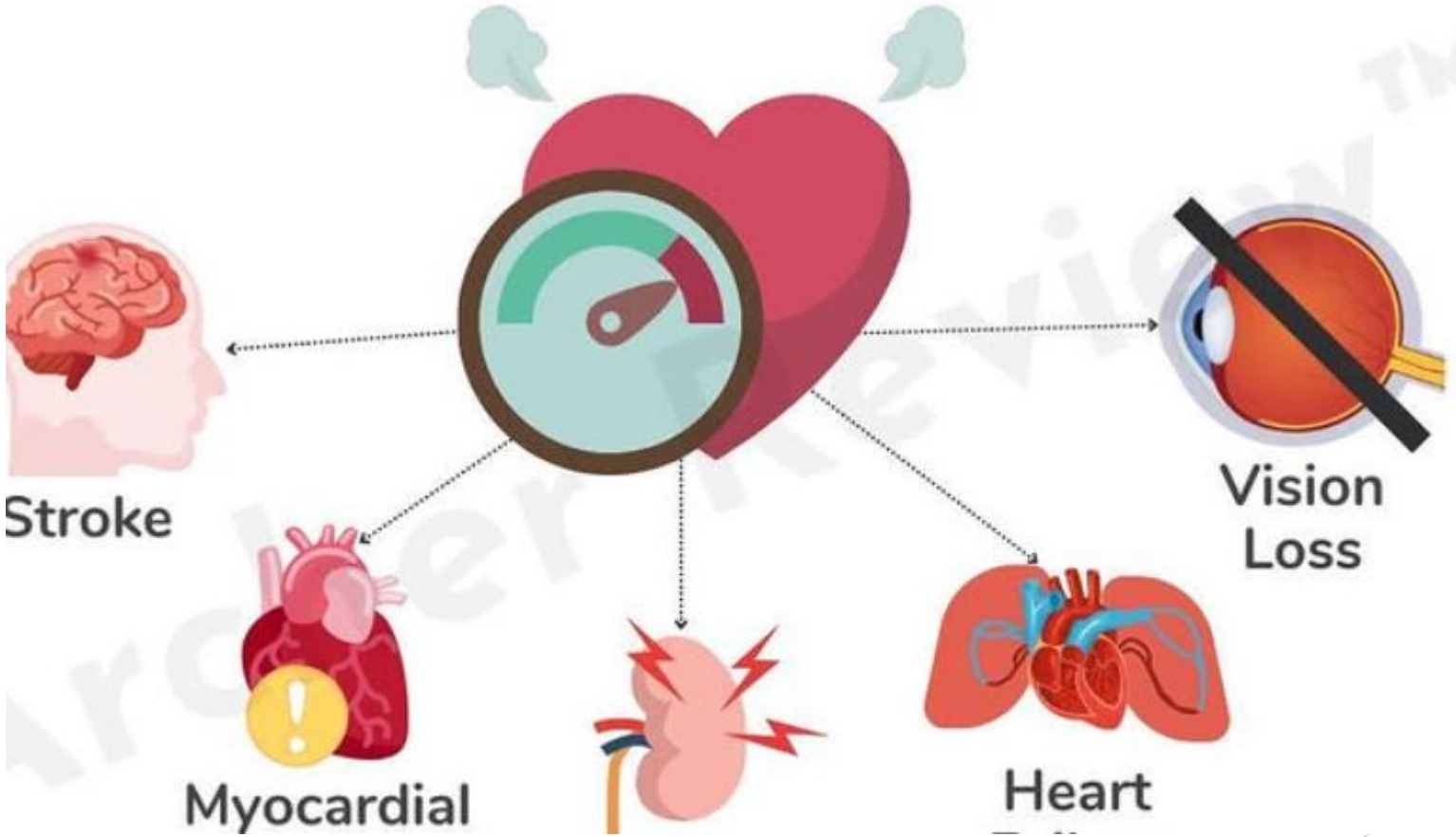
- 1.**Renal -**renal artery stenosis---constriction of renal artery ----increased renin secretion &decreased secretion of Na+
- 2. **endocrine** -pheochromocytoma-increase in NE,cushing syndrome,thyrotoxicosis-increase in CO
- 3.**neurogenic HTN-**lesion of NTS
- 4.severe polycythemia –increase in viscosity

Table 32–12 Estimated Frequency of Various Forms of Hypertension in the General Hypertensive Population.	
	Percentage of Population
Essential hypertension	88
Renal hypertension	
Renovascular	2
Parenchymal	3
Endocrine hypertension	
Primary aldosteronism	5
Cushing syndrome	0.1
Pheochromocytoma	0.1
Other adrenal forms	0.2
Estrogen treatment ("pill hypertension")	1
Miscellaneous (Liddle syndrome, coarctation of the aorta, etc)	0.6

complications

- High BP makes heart to do extra work ,causes strain to CVS leading cardiac failure
- Enlargement of heart (concentric hypertrophy)
- Atherosclerosis & MI
- Rupture of cerebral vessels leading to stroke
- Renal failure
- retinopathy

Complications

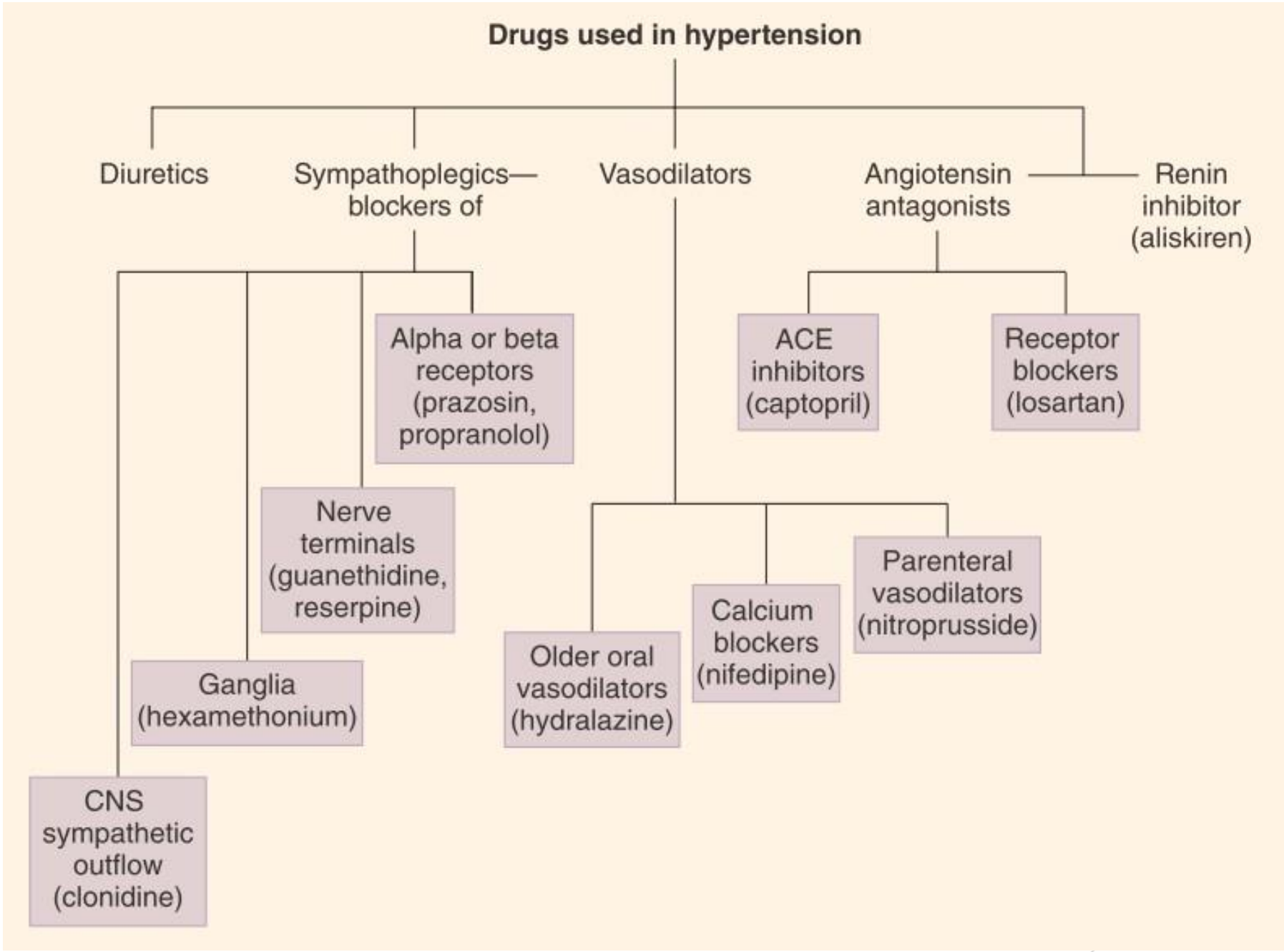


Non pharmacological treatment

- Life style modifications
  - Correct obesity
  - Restrict salt intake
  - Regular physical exercise
  - Quit smoking
  - Reduce alcohol intake
  - Diet low in saturated fat

Pharmacological

- Antihypertensive drugs
  - Diuretics-fruosemide (lasiz),thiazide
  - Angiotensin converting enzyme (ACE) inhibitors
  - Angiotensin receptor blockers (ARBs)
  - Calcium channel blockers
  - Beta-blockers


Drugs used in hypertension
Diuretics
Sympathoplegics—blockers of
Vasodilators
Angiotensin antagonists
Renin inhibitor (aliskiren)
Alpha or beta receptors (prazosin, propranolol)
Nerve terminals (guanethidine, reserpine)
Ganglia (hexamethonium)
CNS sympathetic outflow (clonidine)
Older oral vasodilators (hydralazine)
Calcium blockers (nifedipine)
Parenteral vasodilators (nitroprusside)
ACE inhibitors (captopril)
Receptor blockers (losartan)

- HYPOTENSION-persistant reduction in arterial BP below 100mmHg (systolic),60mmHg (diastolic)
- Sudden hypotension will cause loss of consciousness ---fainting /syncope

Circulatory shock

- A collection of different entities characterised by inadequate tissue perfusion with relative or absolute inadequate cardiac output

- Types of shock
  - hypovolemic
  - distributive /vasogenic /low resistance
  - cardiogenic
  - obstructive

Hypovolemic shock

- Hemorrhagic shock
- Dehydration – burns ,diarrhoea
- Traumatic shock- crush injury

- Cold shock - hypotension, rapid thread pulse, cold ,pale clammy skin, intense thirst, rapid respiration, restlessness

Distributive shock

- Blood volume normal ,capacity of vessel is increased by marked vasodilation (warm shock)

Causes –

- neurogenic shock – marked reduction in sympathetic tone as in deep anaesthesia , brain damage
- Pronounced increase in vagal tone (vasovagal syncope or emotional fainting)
- anaphylactic shock – release of histamine (vasodilator , increase in capillary permeability)

septic shock

- endotoxins released from Gram(-) organisms
- Release of inflammatory mediators – cytokines,leukotrienes etc
- Vasodilation and increase in cap permeability
- Platelet adhesion and coagulation factors activation leads to DIC
- Multi organ failure

CARDIOGENIC SHOCK

- Impaired pumping activity of left ventricle

Causes :

- MI (>40% of the myocardium if involved)
- cardiac failure
- cardiac arrhythmias
- severe valvular disorders

OBSTRUCTIVE SHOCK

- Impairment of ventricular filling during diastole
- due to extra cardiac obstruction like cardiac tamponade , constrictive pericarditis , pulmonary embolism

Clinical features

- Reduced blood volume – reduced C.O – increase in heart rate
- Hypotension if severe hemorrhage
- With moderate hemorrhage, (5 -15 ml/kg body weight),pulse pressure is reduced but MAP is normal
- Skin is cool and pale grayish tinge because of stasis in capillaries and some cyanosis
- Decreased stimulation of baroreceptors result in increased sympathetic output

- Respiration may be rapid
- Intense thirst if conscious
- Oliguria (renal artery constriction)

Stages of shock

- Stage 1 or non progressive shock/reversible shock
- Progressive shock
- Refractory shock

Compensatory mechanisms occur in reversible shock

- Hypovolemia - if blood loss is atleast 10-15 % of blood volume
- Rapid comp mech – to maintain blood flow to heart and brain.
- 1) baroreceptor mech
- 2) chemoreceptor mech
- 3) CNS ischemic response

- BARORECEPTOR -- stretched less , sympathetic output is increased – reflex tachycardia
- Vasoconstriction sparing heart and brain
- Vasoconstriction more in skin , kidneys and viscera

SKIN-cold ,clammy skin

Kidney

- renal ischemia-release of Angiotensin II---intense thirst
- constriction of aff arterioles--GFR decreased
- constriction of eff arteriole-- RPF decreased to a greater extent .
- Oliguria
- retention of Nitrogenous substance ---uremia

- Blood shunted to heart from splanchnic circulation
- Stimulation of sympathetic lead to restlessness , apprehension
- Vasodilatation in coronary circulation due to local metabolites
- Generalized cerebral ischemia result in apathy

Rapid compensatory mech

- CHEMORECEPTOR ACTIVATION:

  Hypoxia result in chemoreceptor stimulation (increase in rate and depth of respiration)

  Vasoconstriction &increase in HR

  CNS ISCHEMIC RESPONSE:when BP reduces less than 50mmHG---more powerful sympathetic stimulation

compensatory through kidney

- Renin Angiotensin Aldosterone mechanism

Other mechanisms

- 1.STRESS—increases ACTH------aldosterone increases
- 2.capillary fluid shift
- 3.decrease in blood volume stimulate ADH----water retention
- 4. Hemorrhage is a potent stimulus for adrenal medullary secretion---increase in catecholamines
- 5.stress relaxation,reverse stress relaxation

Long term comp

- Plasma volume returns to normal in 12 -72 hrs, after a moderate hemorrhage by mobilising tissue fluid & Na+ & water retention
- RESTORATION OF PLASMA PROTEINS
- Pre formed albumin from common pool enter intra vascular space
- rest of the protein loss is replaced by albumin production in 3-4 days
- Erythropoeisis, RBC s replaced in 4-8 wks

Progressive shock

- If 15 – 20% of circulating blood vol is lost ,compensatory mechanism is not sufficient

Various positive feedback mechanisms operate

- 1. cardiac depression – low BP causes decrease in coronary blood flow.
- 2. vasomotor failure - diminishes blood flow to brain causing depression of VMC & cardiac centres of medulla - vasodilation and bradycardia

- 3.peripheral circulatory failure- capillary blood flow is greatly reduced causing hypoxia of tissues.
- Vasodilator metabolites cause peripheral pooling.Blood stagnates and there is sludging of blood in the capillaries.
- 4. tissue hypoxia when continued increase cap permeability -release of toxins,tissue enzymes. Bacteria enter circulation thru damaged GI mucosa.

5.At cellular level

- Lack of nutrients especially to liver – decrease in Na K pump activity ,
- Mitochondrial activity decreased
- Lysosomal damage leading to release of hydrolases

Refractory shock

- Irreversible shock
- Generalised cellular deterioration
- Lack of high energy phosphate – ATP especially liver,heart
- Slow necrosis of tissues
- Patchy necrosis in liver, kidney tubules (ATN), lungs (ARDS), heart (cardiac failure),DIC in septic shock

Management

- Correct the cause
- Aid the physiological compensatory mechanisms to restore normal tissue perfusion

GENERAL MEASURES (if in early stage)

- Kept in cold room – to preserve cutaneous vasoconstriction
- Never wrap in blankets (aggravates shock)
- Foot end of the bed to be raised by 6 -12 inches (Trendenlenburg position) – helpful in hemorrhagic and neurogenic shock

Management

- Replacement therapy in hypovolemic shock – plasma, whole blood (not in cardiogenic or obstructive shock)
- Plasma substitutes or expanders like dextran helpful
- Electrolyte solution of appropriate concentration

- DRUGS – sympathomimetic drugs in anaphylactic and neurogenic shock -Dopamine,NE,E can be given
- Glucocorticoids, adrenaline in anaphylactic shock

- Treat the cause in obstructive shock.
- In shock due to burns, plasma is the treatment of choice to restore the fundamental defect

CARDIAC FAILURE/HEART FAILURE

- State of heart in which cardiac performance is too low to maintain the cardiac output to meet the demands of tissues

Types

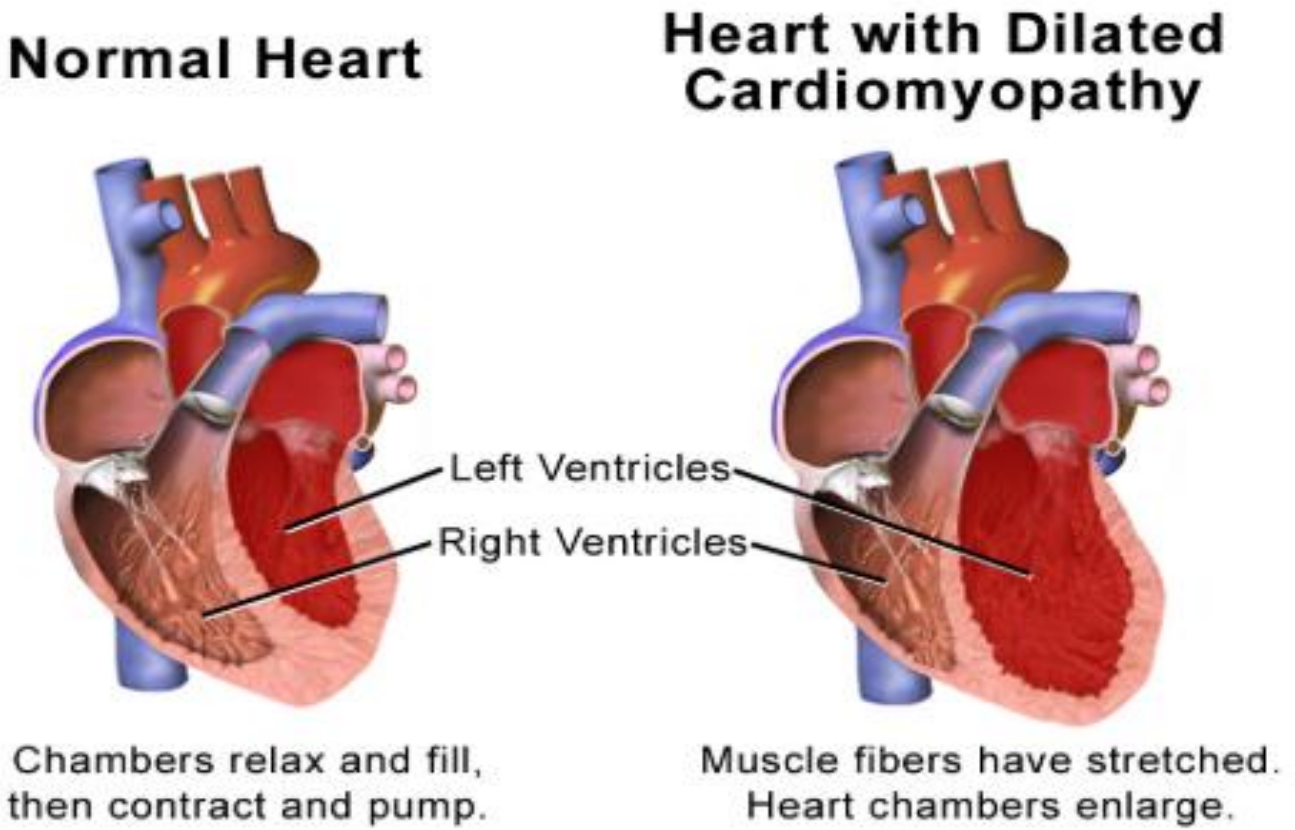
- Systolic failure
- Diastolic failure

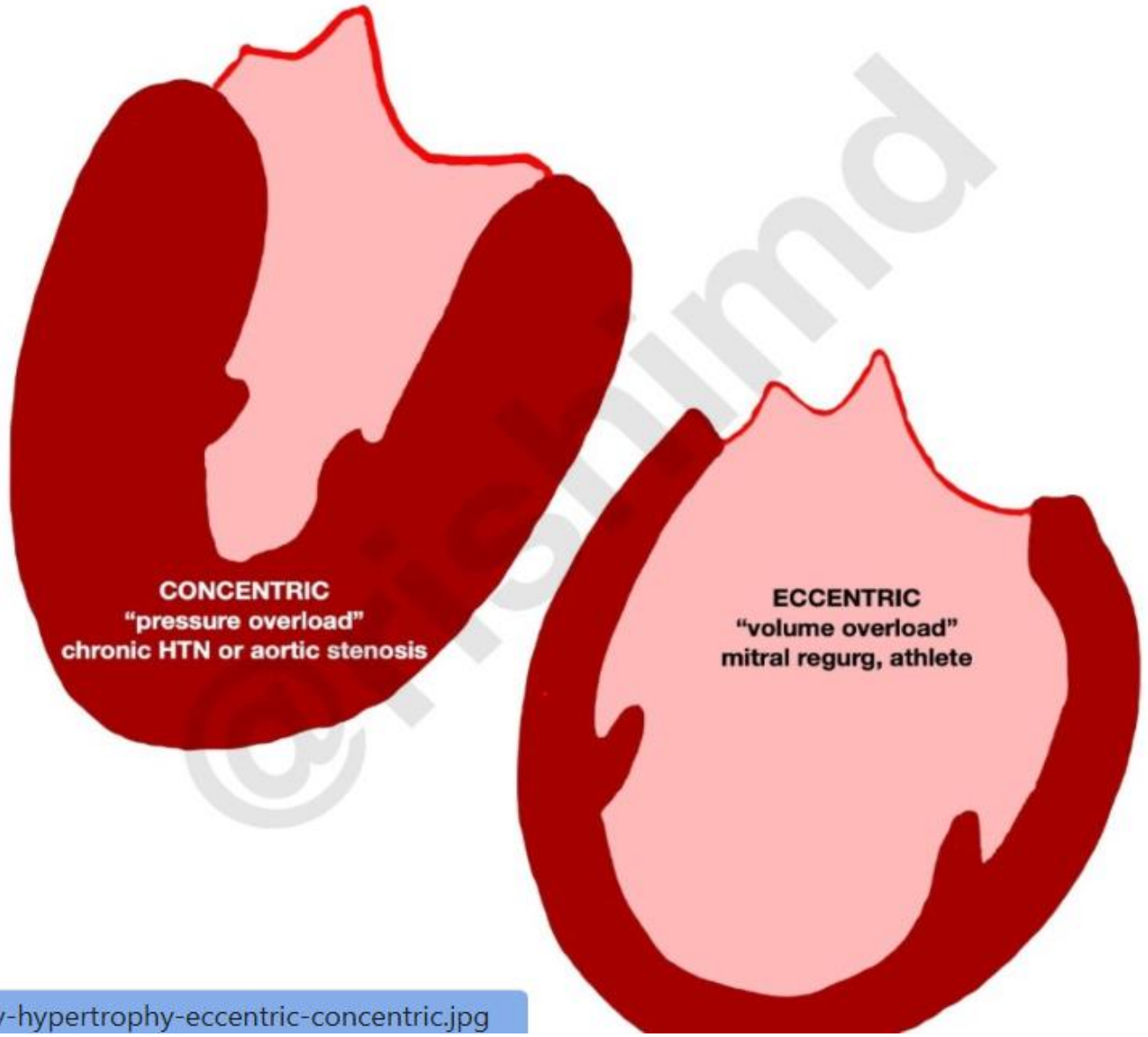
CAUSES(systolic) –

- reduced muscle mass- MI
- Dilated cardiomyopathy

CAUSES (diastolic)-

concentric hypertrophy as in Hypertension,AS


Normal Heart
Heart with Dilated Cardiomyopathy
Left Ventricles
Right Ventricles
Chambers relax and fill, then contract and pump.
Muscle fibers have stretched. Heart chambers enlarge.



2/05/lv-hypertrophy-eccentric-concentric.jpg

Clinical features -LHF

Forward failure

- Decreased CO, SBP ----tachycardia,cool peripheries (BR reflex)
- Exertional Dyspnea
- Renal ischemia,liver,cerebral also

Backward failure

- Left atrial diln
- Pulm edema ---hypoxia
- Orthopnea,PND
- Peripheral edema

LHF - signs

- Crepitations on auscultation
- S3,S4

RHF-

forward failure

Same as in left

Less flow to pulmonary circulation

Backward failure

- Increased JVP
- Tender hepatomegaly,splenomegaly
- Peripheral edema

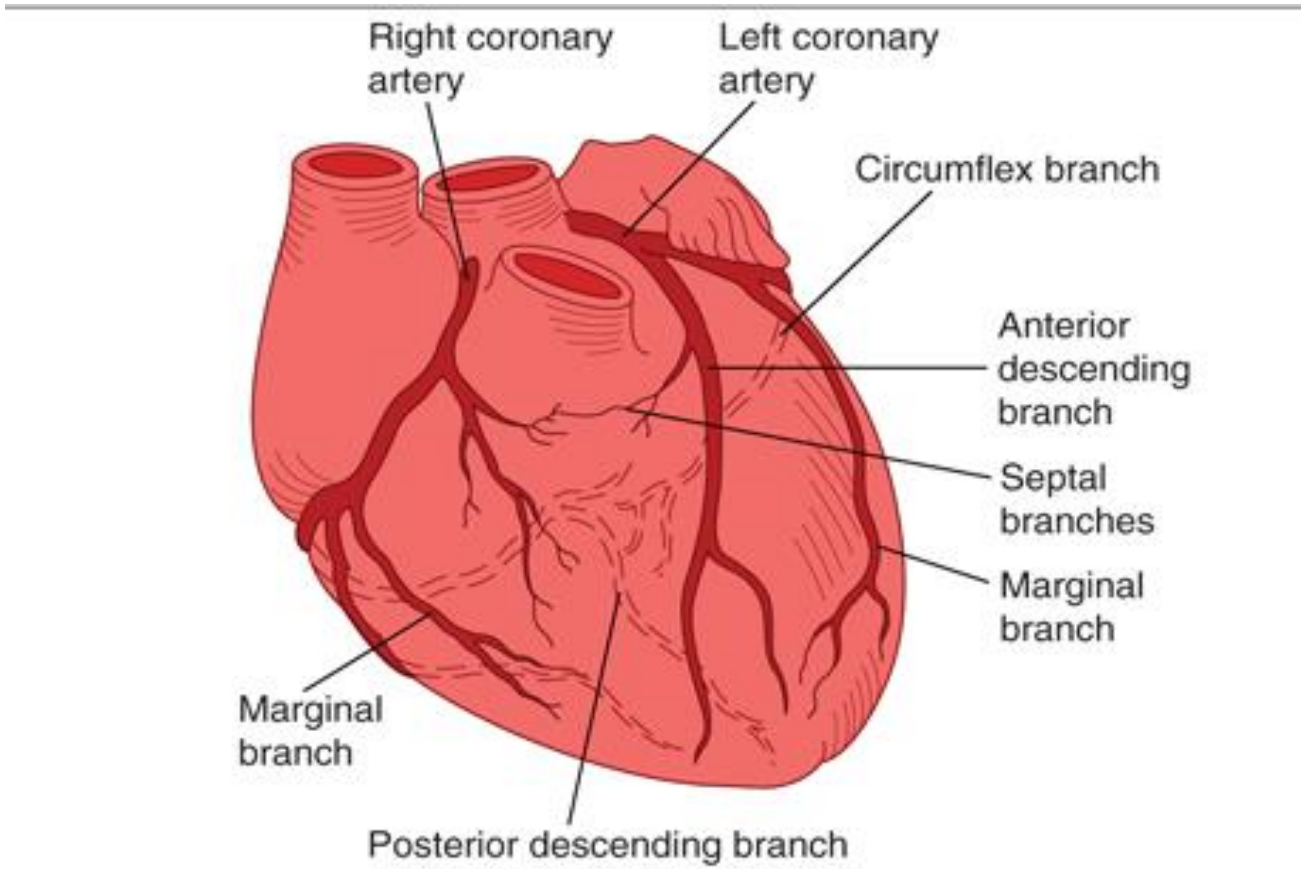
CLINICAL FEATURES

- Due to low cardiac output:
- Cardiomegaly
- Dyspnea-due to tissue hypoxia & increased pulmonary hydrostatic pressure
- Orthopnea-increased pulmonary P
- Oedema,raised JVP,hepatomegaly-Rt heart failure

TREATMENT

- REST
- To improve myocardial contractility- drugs like digitalis
- To control fluid retention-salt restriction
- To reduce after load-vasodilator drugs-ACE inhibitors
- Beta blockers,alpha blockers

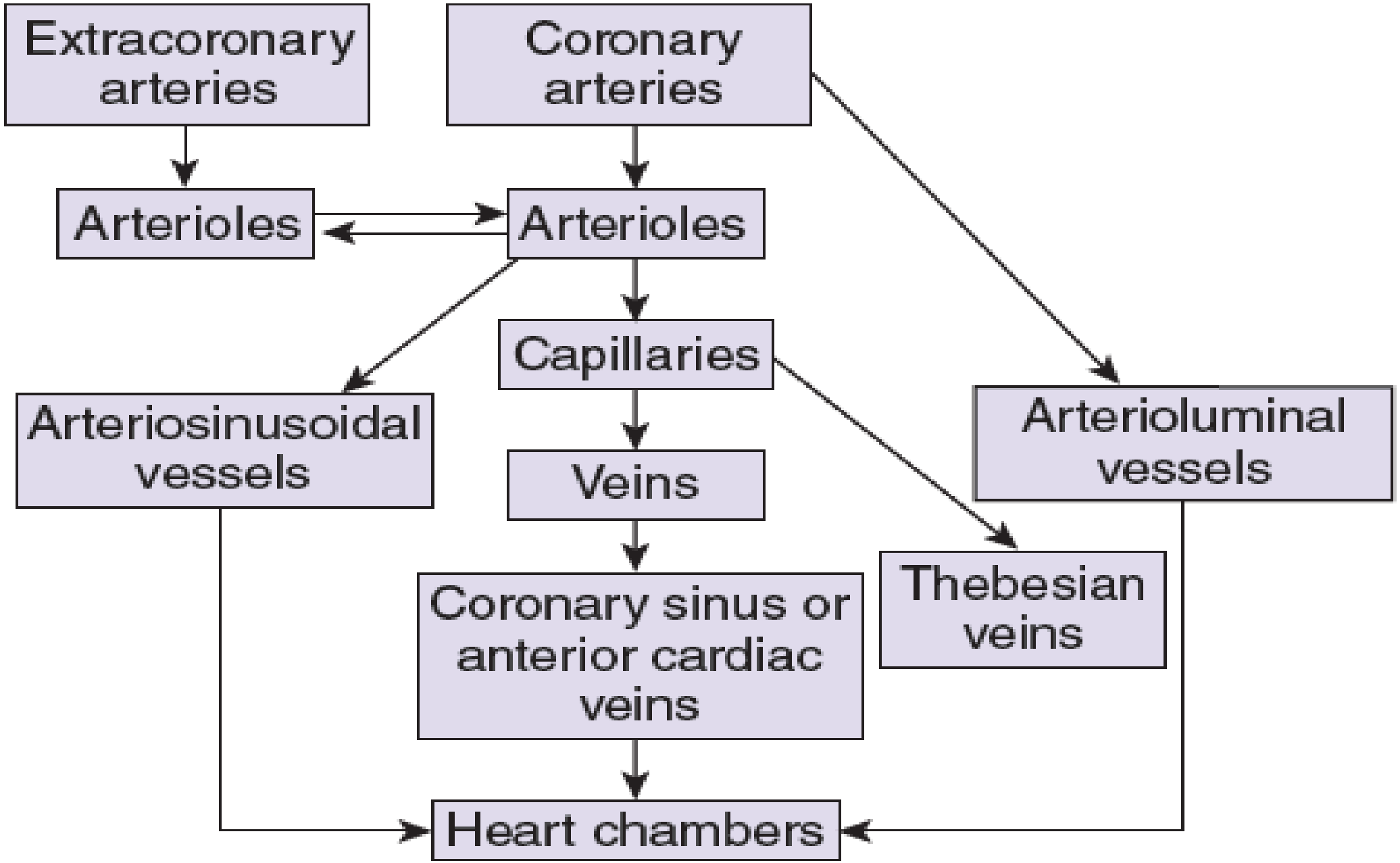
Coronary circulation


Right coronary artery
Left coronary artery
Circumflex branch
Anterior descending branch
Septal branches
Marginal branch
Marginal branch
Posterior descending branch

- Coronary arteries arise from aortic sinuses
- RCA – supply RA ,greater part of RV, post part of LV, posterior iv septum , conducting system of heart including SA node.
- LCA – greater part of LV, LA ,small part of RV, anterior part of iv septum

VENOUS DRAINAGE

- **FROM LEFT VENTRICLE--- CARDIAC SINUS----- RA**
- **FROM RIGHT VENTRICLE----ANTERIOR CARDIAC VEINS-----RA**


Extracoronary arteries
Coronary arteries
Arterioles
Arterioles
Capillaries
Arteriosinusoidal vessels
Arterioluminal vessels
Veins
Thebesian veins
Coronary sinus or anterior cardiac veins
Heart chambers

Dominance

- 50% -RCA
- 20% -LCA
- 30% - dual supply

Coronary blood flow – Characteristic features

1. Normal coronary blood flow - **250ml/min** (5% of C.O)- 70ml/100g/mt

↑3-4 fold in exercise

2. Oxygen utilization of myocardium very high- 8ml/min/100g.

- Even at rest 70-80% of O2 is extracted from each unit of coronary blood
- So when heart requires additional oxygen as in exercise - ↑ coronary blood flow
- Resting A-V 02 difference is high

  (19-6=13ml%)

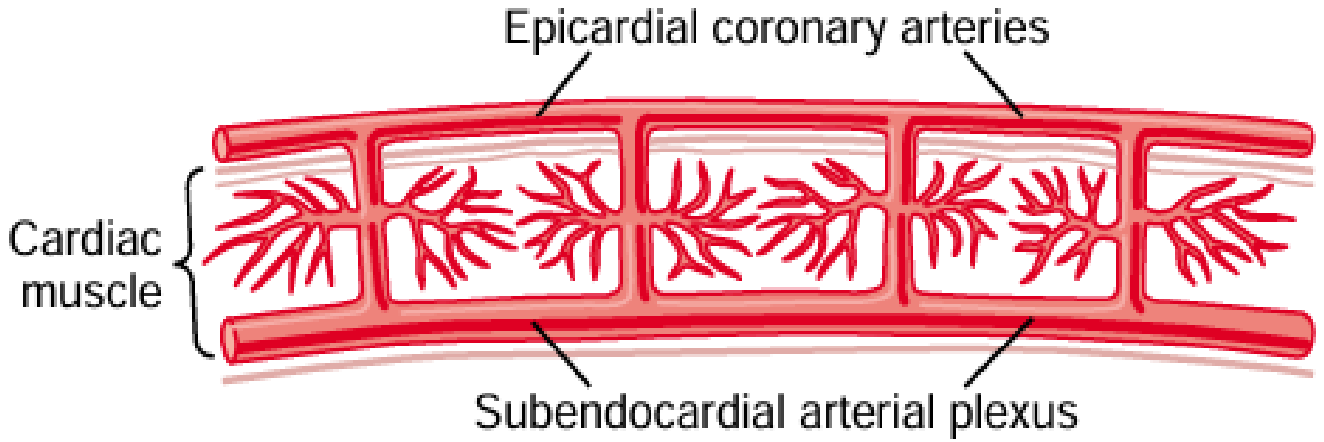
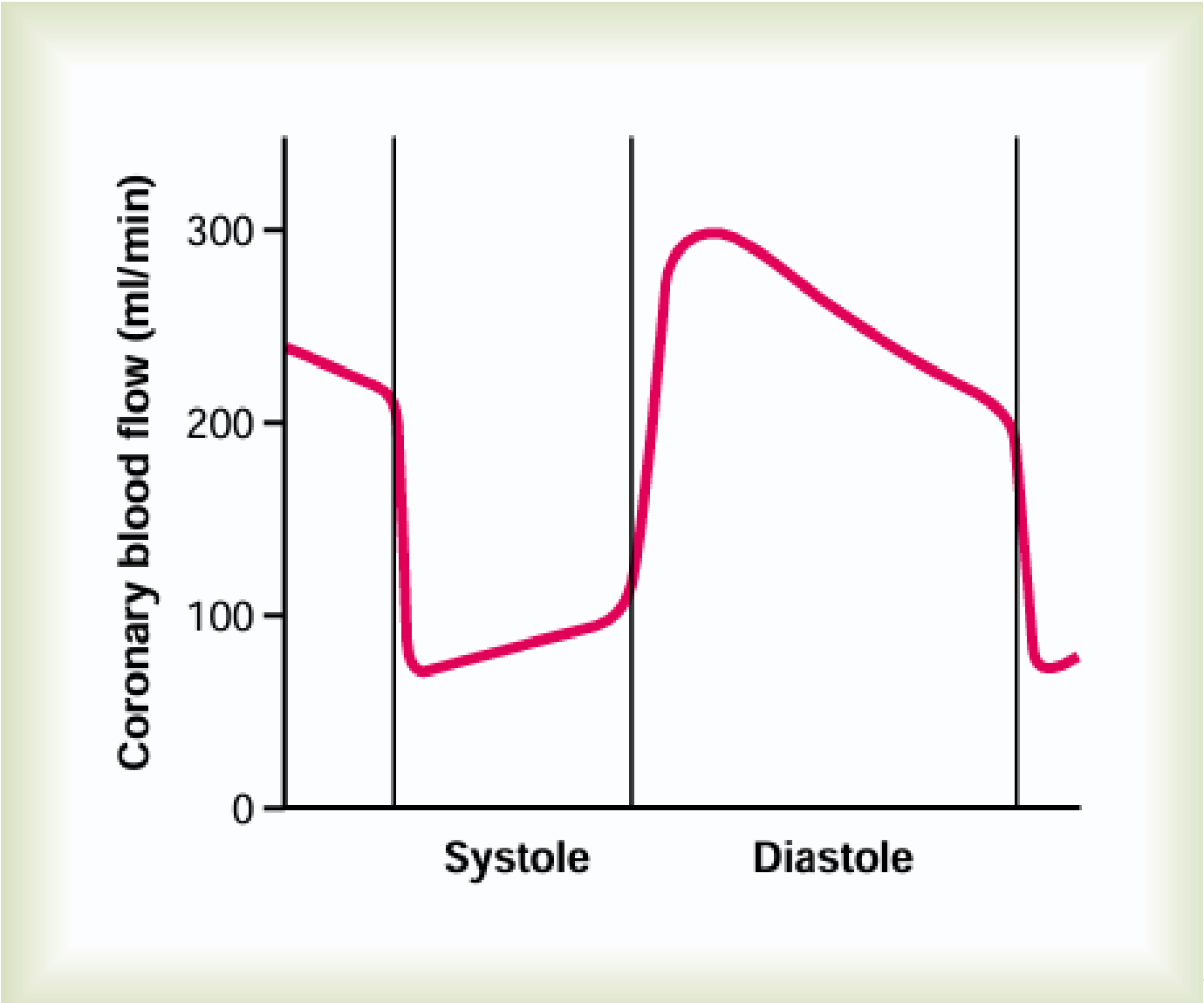


Figure 21–5

Diagram of the epicardial, intramuscular, and subendocardial coronary vasculature.

3.Phasic coronary flow

- blood flow↑ during diastole & ↓ during systole (80% blood flow during diastole)
- Blood flow through coronary vessels determined by pressure head b/w aorta & ventricles, resistance due to compression of intramuscular coronary vessels by myocardium


Coronary blood flow (ml/min)
300
200
100
0
Systole
Diastole

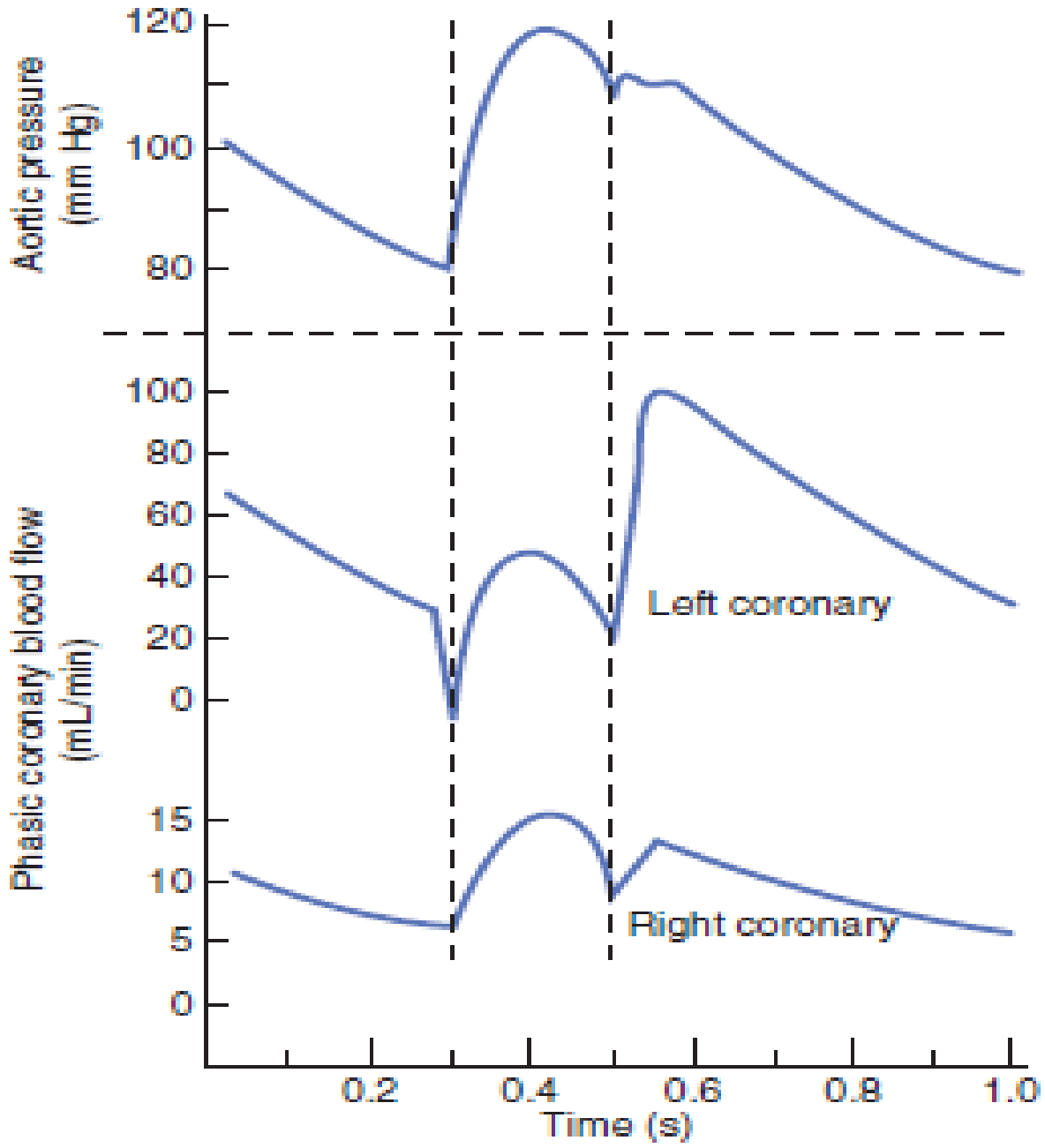
Pressure gradient b/w ventricle & aorta

Phases of cardiac cycle	Pressure (mmHg)			Pressure gradient b/w aorta & Ventricle	
	Aorta	RV	LV	Aorta & RV	Aorta & LV
Systole	120	25	121	95	-1
Diastole	80	0	0	80	80

Phasic flow in LV

- During systole –cor vessels compressed & pressure of LV slightly higher than aortic P –pressure head during systole is zero or-1----- no/low blood flow through the coronaries in the subendocardial region during systole

- During DIASTOLE, the pressure in LV is almost zero, pressure in aorta is around 80mmHg .so pressure head is high & no obstruction of blood flow----sub endocardial region of LV recieves blood during diastole


Aortic pressure (mm Hg)
120
100
80
Phasic coronary blood flow (mL/min)
100
80
60
40
20
0
Left coronary
15
10
5
0
Right coronary
0.2
0.4
0.6
0.8
1.0
Time (s)

Phasic flow in LV- explanation of diagram

- During systole – IVC - intraventricular pressure in LV is high which compresses the coronary artery branches passing through them& also aortic pressure is low---flow through vessels esp cap ↓(0)
- Rapid ejection phase –aortic pressure ↑-flow↑
- Reduced ejection phase – aortic pressure ↓- flow ↓

- Diastole – pressure gradient is high ,no obstruction to flow -CBF↑

Phasic flow in rt ventricle

Aortic P > RV pressure throughout

Systolic RV pressure is lower than LV – so less compression of coronary vessels

- Blood flow occur during both systole, diastole

Clinical importance of phasic blood flow

- 1.Heart rate & Coronary blood flow

  Tachycardia→ shortened diastole → ↓Coronary flow

- 2.Subendocardial region of LV receives no blood supply during systole so it is prone to ischemia & common site of MI

- 3.Aortic stenosis – LV Pressure> aortic P, Severe Compression of coronary vessels during systole – chance for MI

Compensatory mechanisms/protective mechanisms

a.High capillary density than epicardium

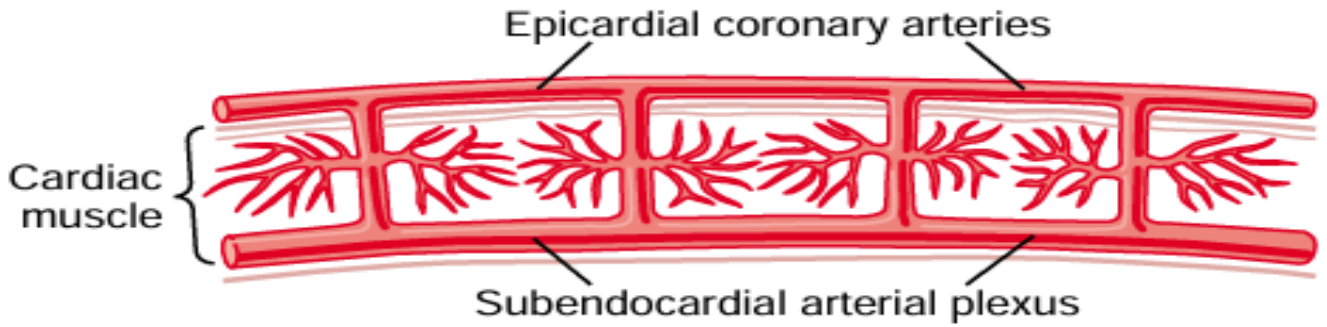
b. Minimum diffusion distance b/w cap &myocardial cell

4.Coronary arteries are end arteries –only minimal anastomosis present.if it gets blocked,area supplied by them suffer ischemia

5. Myocardium has high capillary density 6-7 times more than in skeletal muscle

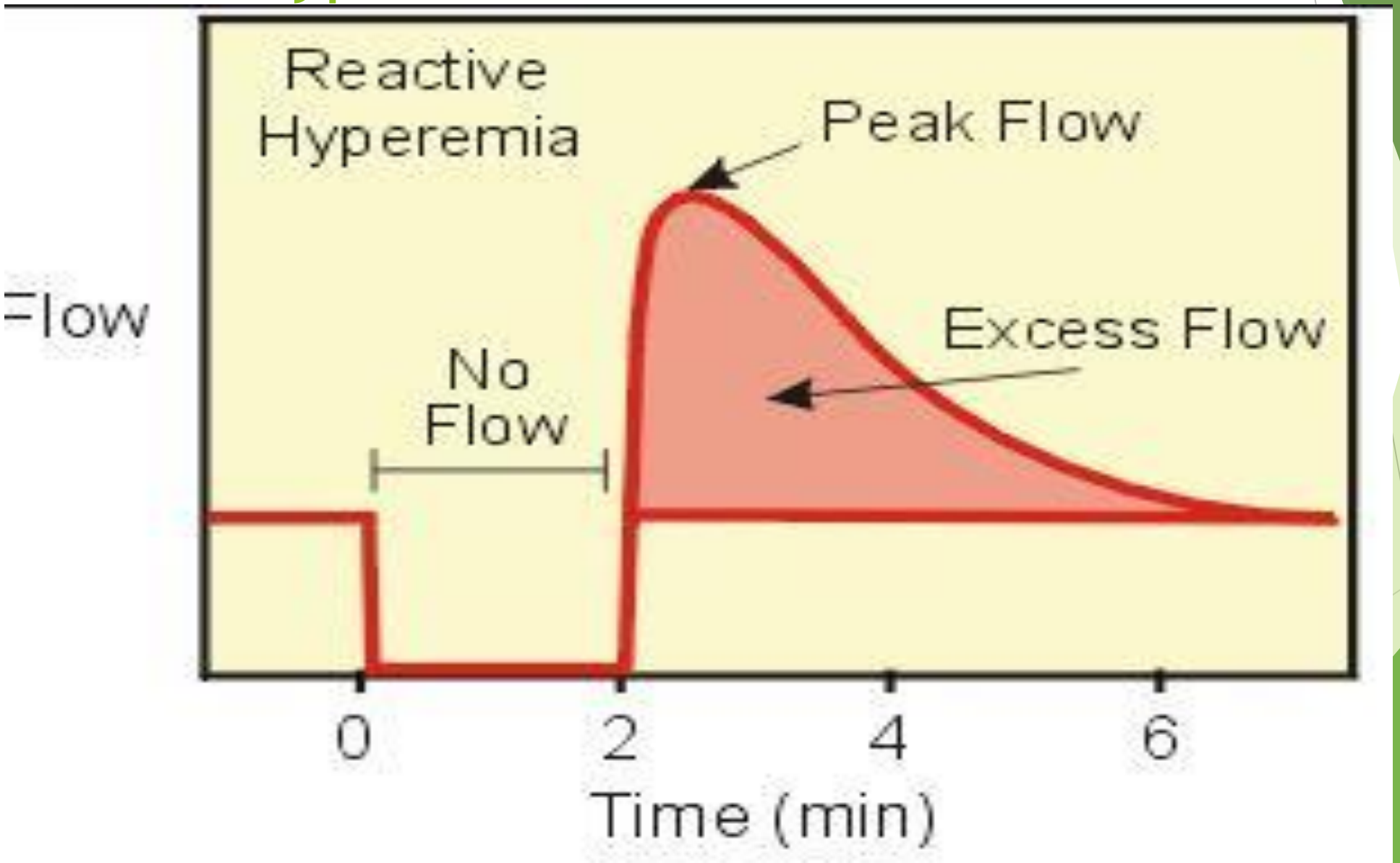
6. Regional variation of supply

- subepicardial portions of myocardium are better perfused than subendocardium because of greater compression of intramural vessels during systole



7. Autoregulation – Coronary blood flow remains constant between 60-150mmHg mean arterial pressure by adjusting resistance
8. Reactive hyperemia – if coronary atery is briefly occluded→release of obstruction→↑blood flow (due to adenosine release by hypoxia)

reactive hyperemia



9)Resting heart obtain 2/3rd of energy requirements from **fatty acids**

Coronary blood flow – Characteristic features

1. Normal coronary blood flow - 250ml/min (5% of Cardiac output).
2. Oxygen utilization of myocardium very high
3. Phasic coronary flow
4. Coronary arteries – end arteries.
5. Myocardium has high capillary density

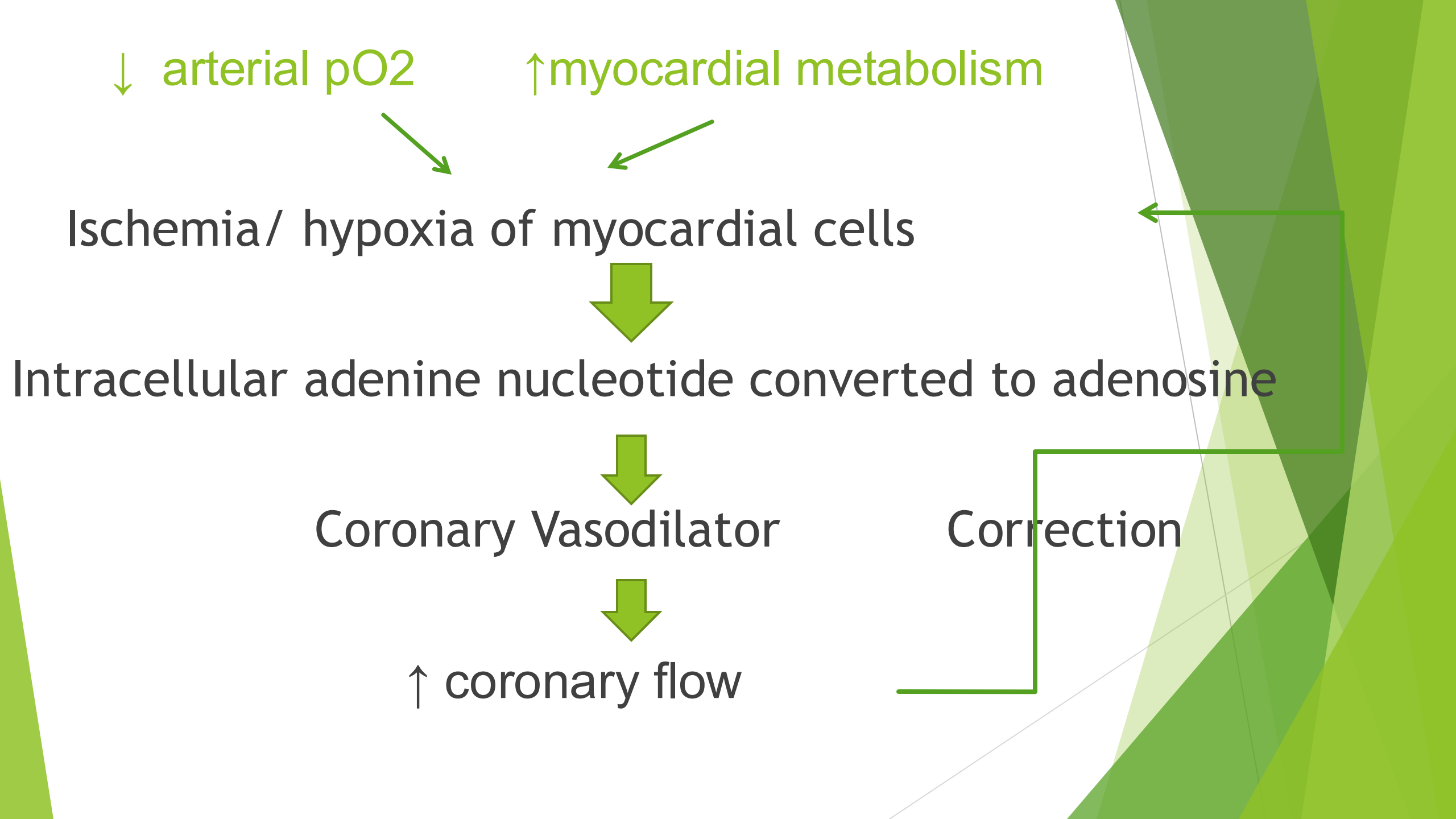
6. Regional variation of supply
7. Autoregulation – Coronary blood flow remains constant between 60-150mmHg mean arterial pressure by adjusting vascular resistance
8. Reactive hyperemia
9.N supply

Regulation of coronary blood flow

2. Chemical factors/Metabolic factors

O_2 lack , ↑ CO_2, ↑ H^+, ↑ K^+, lactate, adenosine - coronary vasodilation -↑ flow

Hypoxia directly cause vasodilation &also indirectly through adenosine


↓ arterial pO2
↑myocardial metabolism
Ischemia/ hypoxia of myocardial cells
Intracellular adenine nucleotide converted to adenosine
Coronary Vasodilator
↑ coronary flow
Correction

3.ROLE OF ENDOTHELIAL CELLS

- Endothelial cells release vasodilator substances like EDRF,Prostacyclin(PGI2),EDHF ---contribute the regulation of coronary blood flow

Neural control

Sympathetic stimulation –release NE

- ά receptors – Vasoconstriction
- β receptors - vasodilation

NET Direct effect –vasoconstriction

Indirect effect –Vasodilation

NET OVERALL EFFECT(DIRECT&INDIRECT)-VASODILATION

Sympathetic stimulation –indirect effect

Stimulation of sympathetic nerves

↓

Release of noraderenaline

↓

↑HR & force of contraction

↓

Release of vasodilator metabolites

↓

Vasodilation

Parasympathetic stimulation

- Sparse innervation
- Vagal stimulation release Acetylcholine – coronary Vasodilation

Factors increasing coronary flow

- Mean aortic pressure – force for driving blood into coronary artery

↑mean aortic pressure ↑ blood flow

- Exercise-increased sympathetic activity –increases blood flow 4 times
- Emotion-Excitement—increased CBF due to symp activity
- Hypotension-reflex sym stimulation (vasodilation) –CBF increase

Factors affecting coronary blood flow

- Metabolic factors - ↑heart metabolism ↑O2 consumption---hypoxia

Vasodilation adenosine (VD)

- Temperature-Hyperthermia –increased metabolism—increases CBF

- Heart rate—
- Hormones –

1. Thyroid—increase metabolism increase CBF
2. NE,E ---Increase CBF
3. Acetylcholine—vasodilation-increase CBF

Measurement of coronary blood flow

- 1)Nitrous oxide method (Kety method);

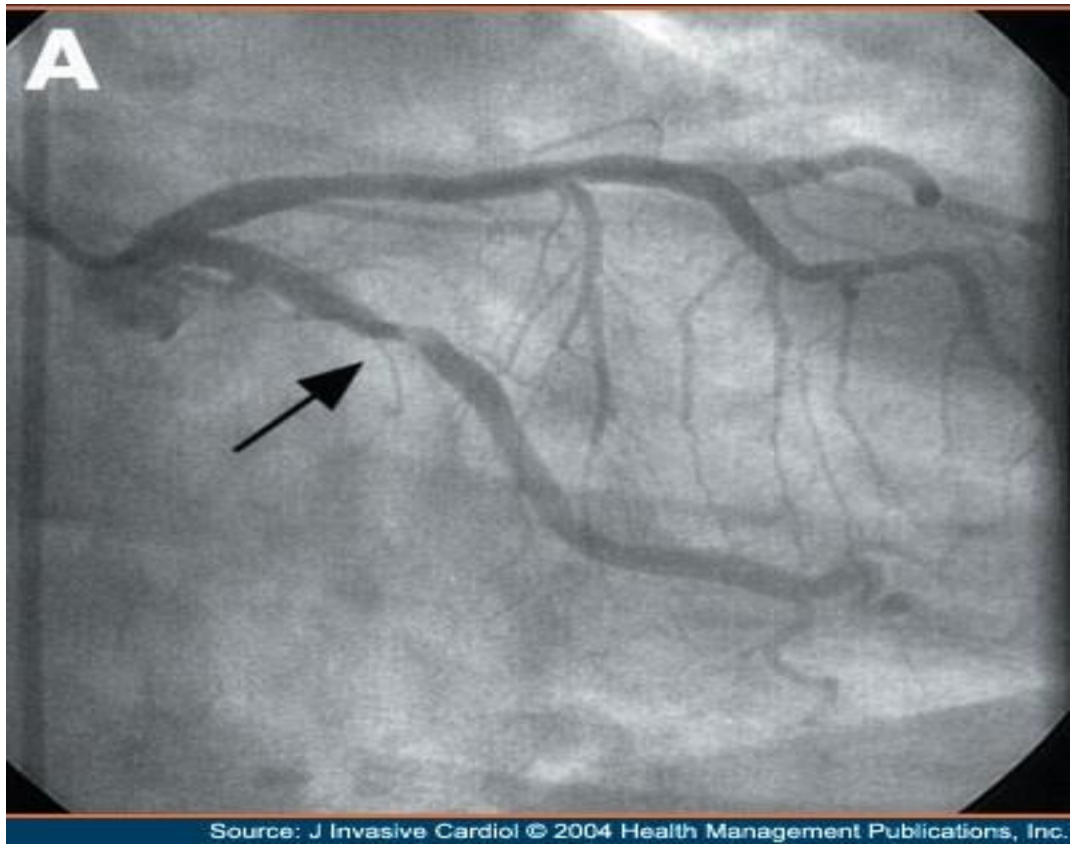
  based on Fick's principle

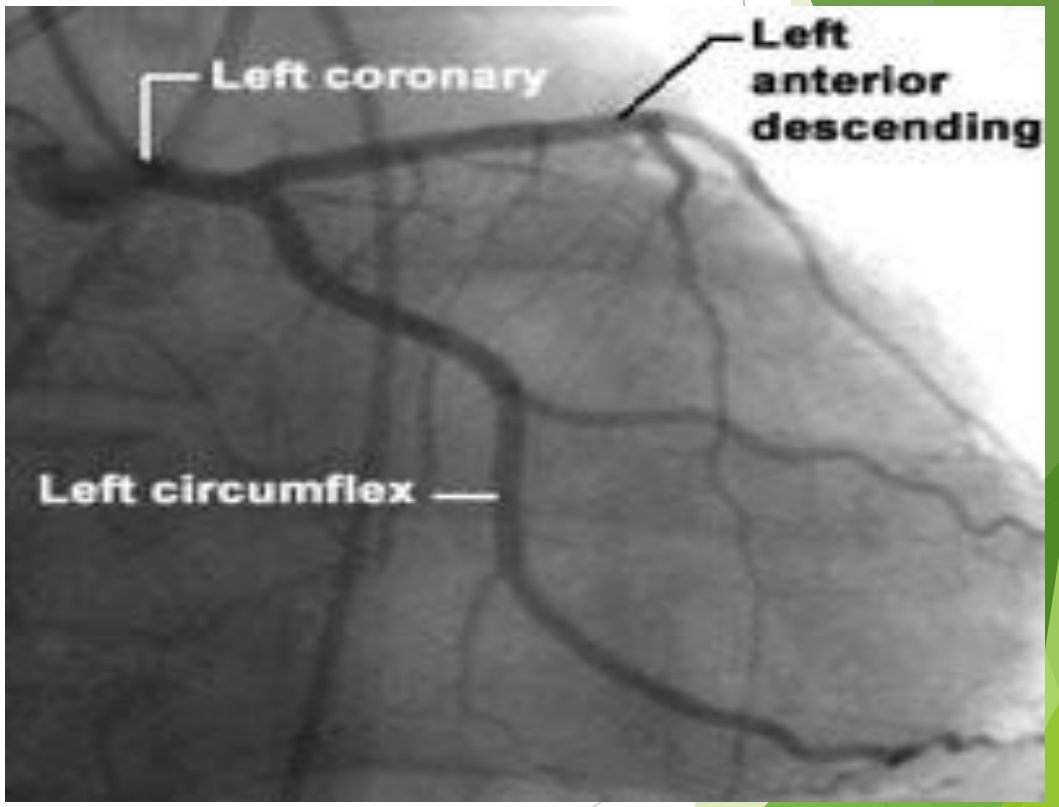
$$\text{CBF} = \frac{\text{N20 uptake/min}}{\text{A-V difference}}$$

- 2)Radionuclide Method- Thallium-201injected iv & uptake measured by radiation detectors over the chest---- uptake is proportional to flow

  Ischemic areas show low uptake

Coronary angiography

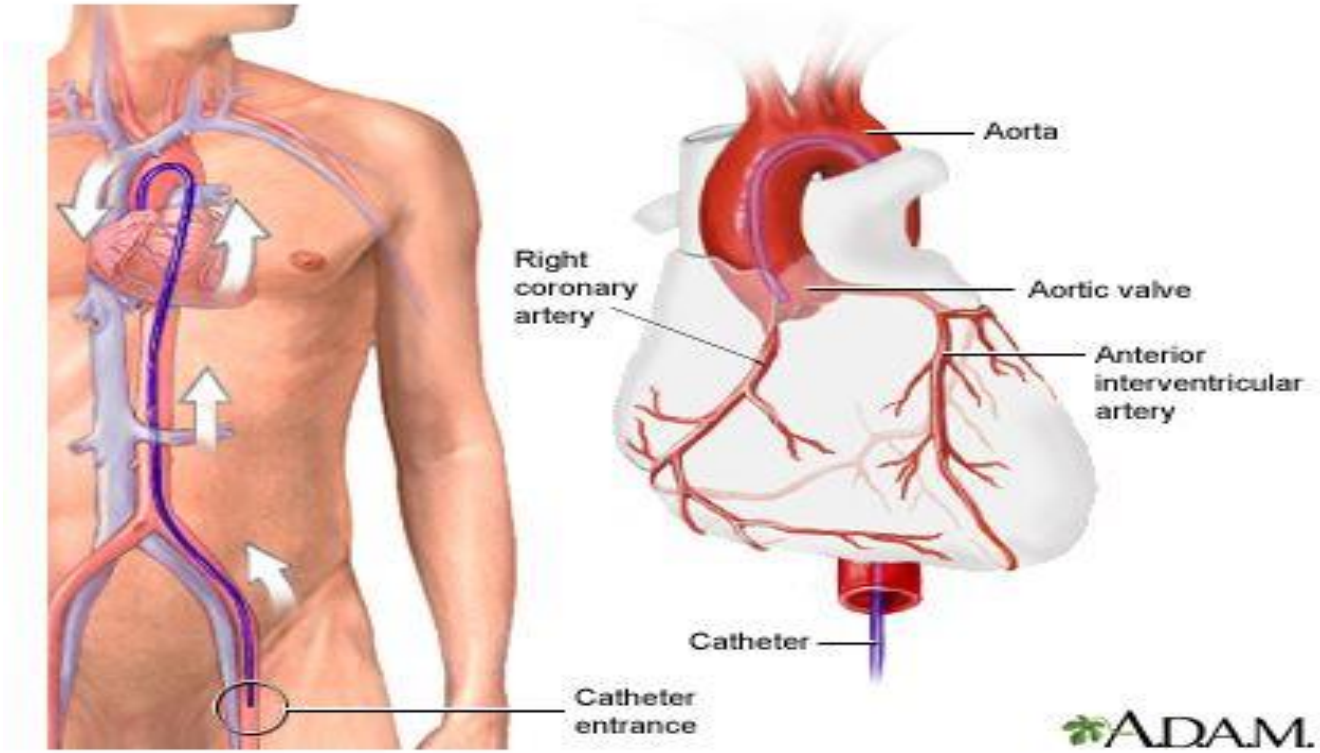




Coronary angiography

Radiopaque contrast medium injected into coronary arteries, and radiographs are used to outline their distribution.

Coronary angiography

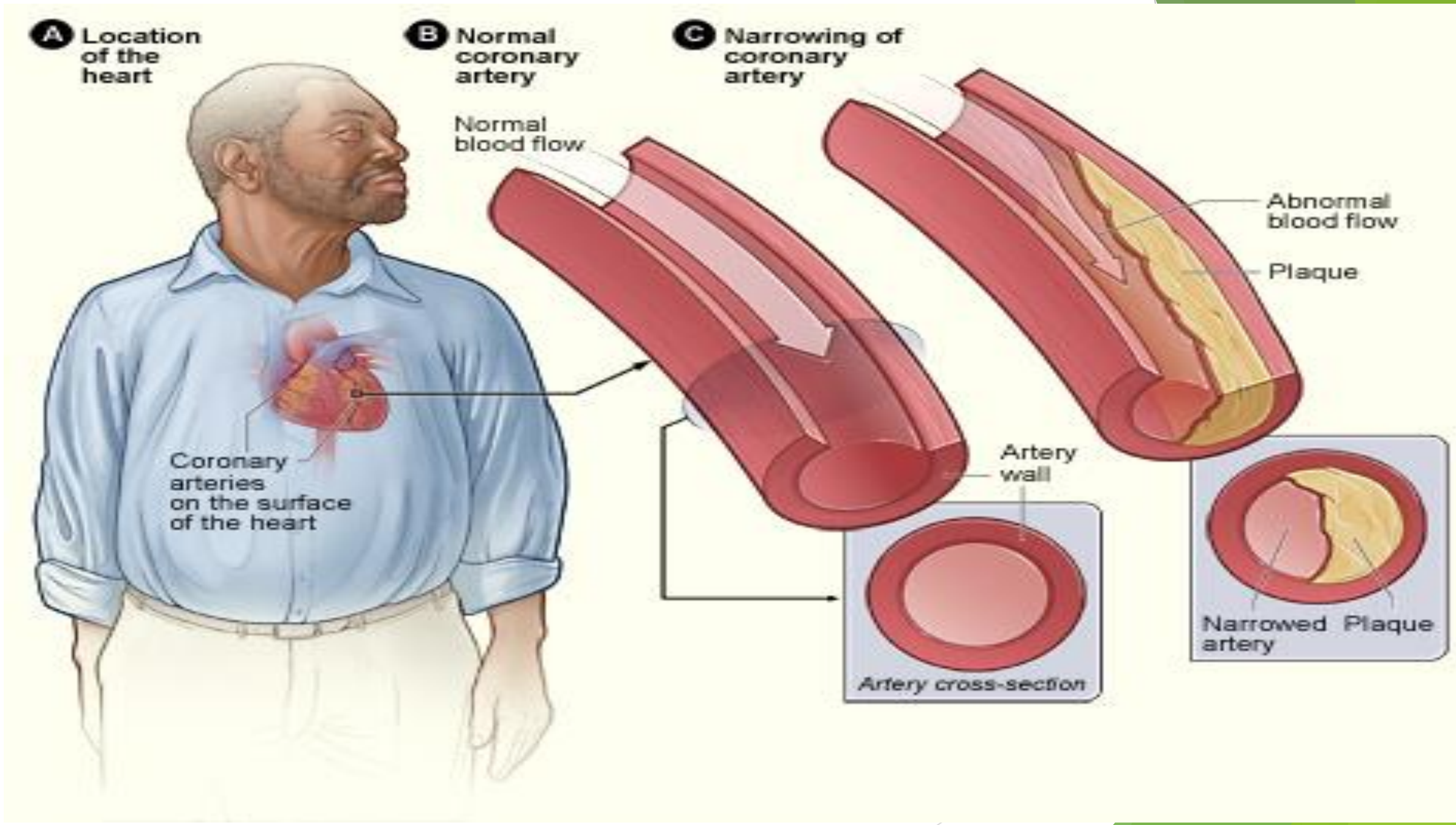


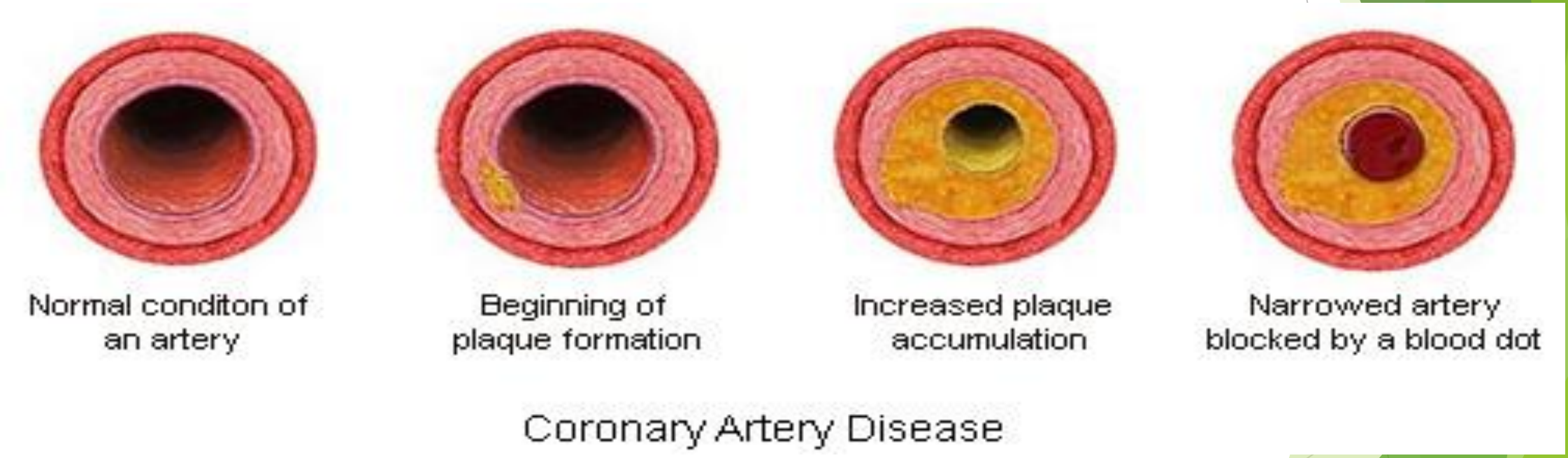
Coronary Artery Disease/ **Ischemic Heart Disease**

- Insufficient coronary blood flow –due to atherosclerotic narrowing of coronary Blood vessel
- Atherosclerosis in Coronary arteries---hardening of arterial wall, narrowing of lumen due to plaque formation

Risk factors-

- family history,
- age (M>60,F>65),
- Diabetes,
- Hypercholesterolemia, Hypertension,
- Smoking,
- Obesity, sedentary lifestyle


A Location of the heart
B Normal coronary artery
C Narrowing of coronary artery
Normal blood flow
Abnormal blood flow
Plaque
Coronary arteries on the surface of the heart
Artery wall
Artery cross-section
Narrowed artery
Plaque


Normal conditon of an artery
Beginning of plaque formation
Increased plaque accumulation
Narrowed artery blocked by a blood dot
Coronary Artery Disease


Smoking
Heredity
Age
Inactive Lifestyle
Obesity
Buzzle.com

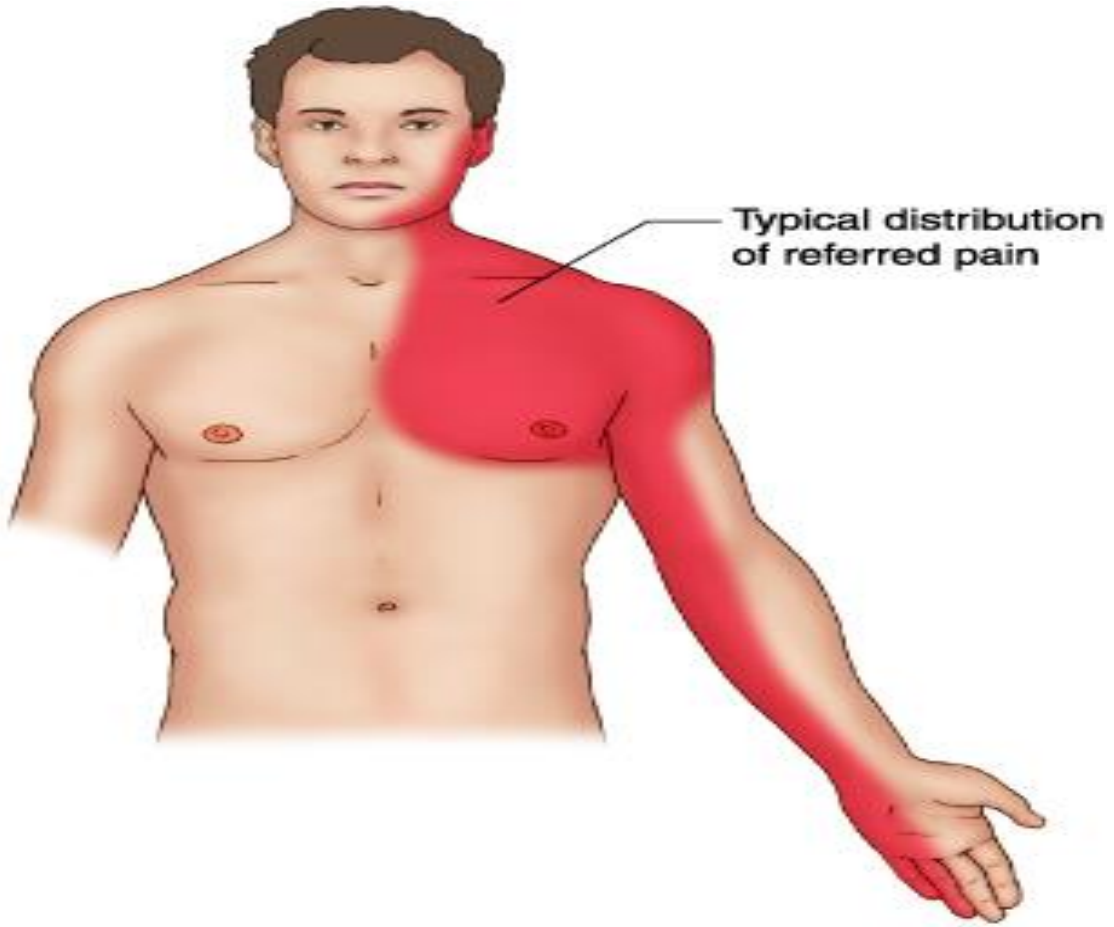
Angina pectoris

Coronary artery occlusion (vasospasm)

myocardial ischemia

Accumulation of P factor in myocardial tissue

Substernal pain radiating to inner border of left arm


Typical distribution
of referred pain

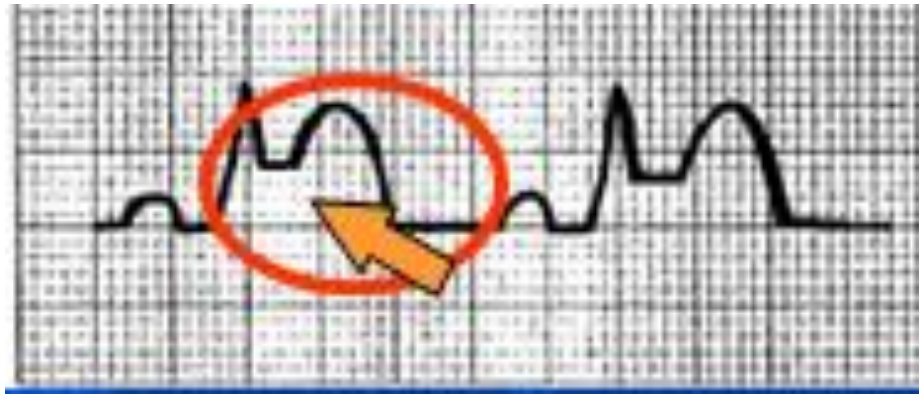
Prolonged myocardial ischemia

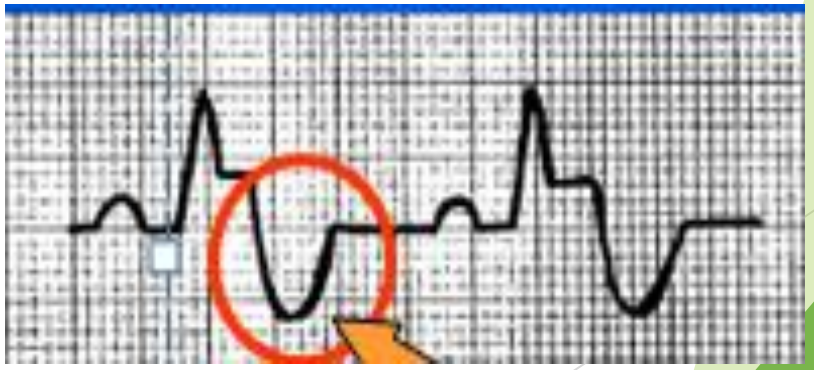
↓

Irreversible damage &Death of myocardial cells

↓

Myocardial infarction



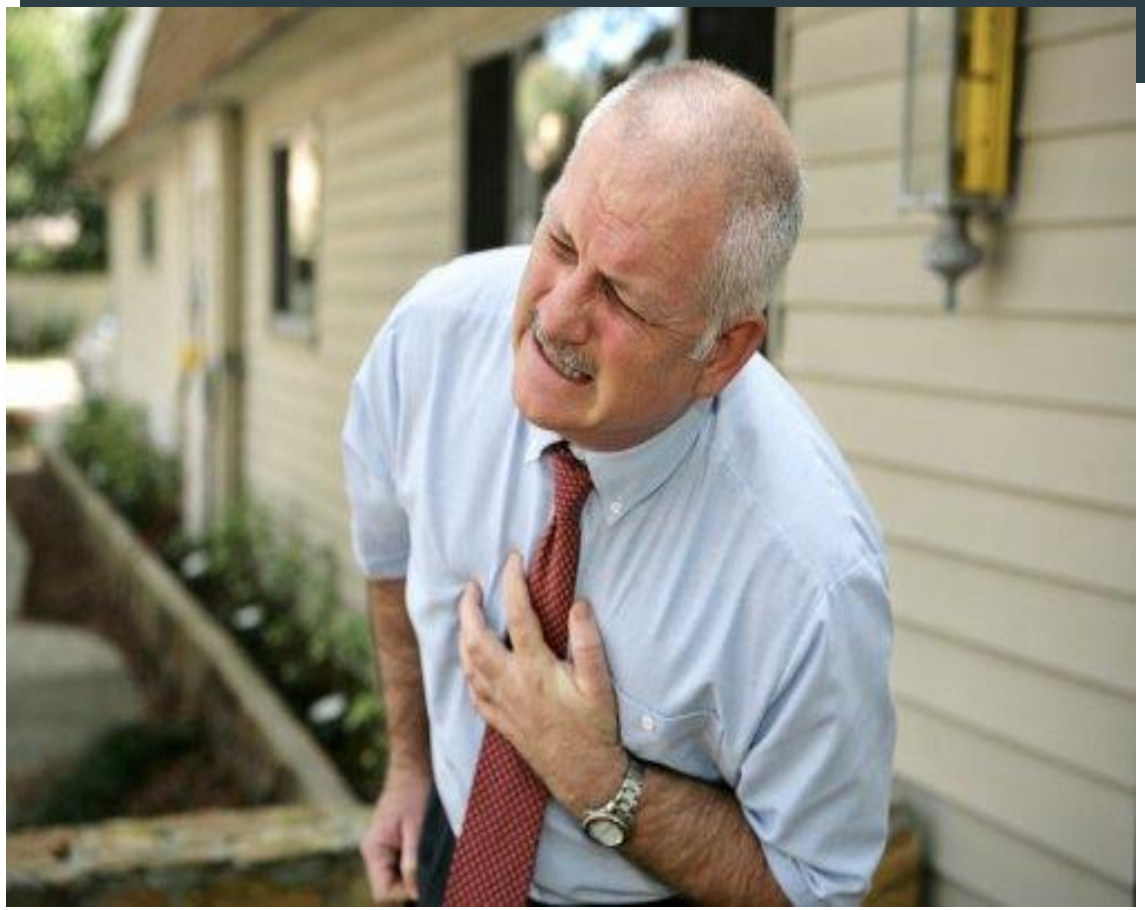


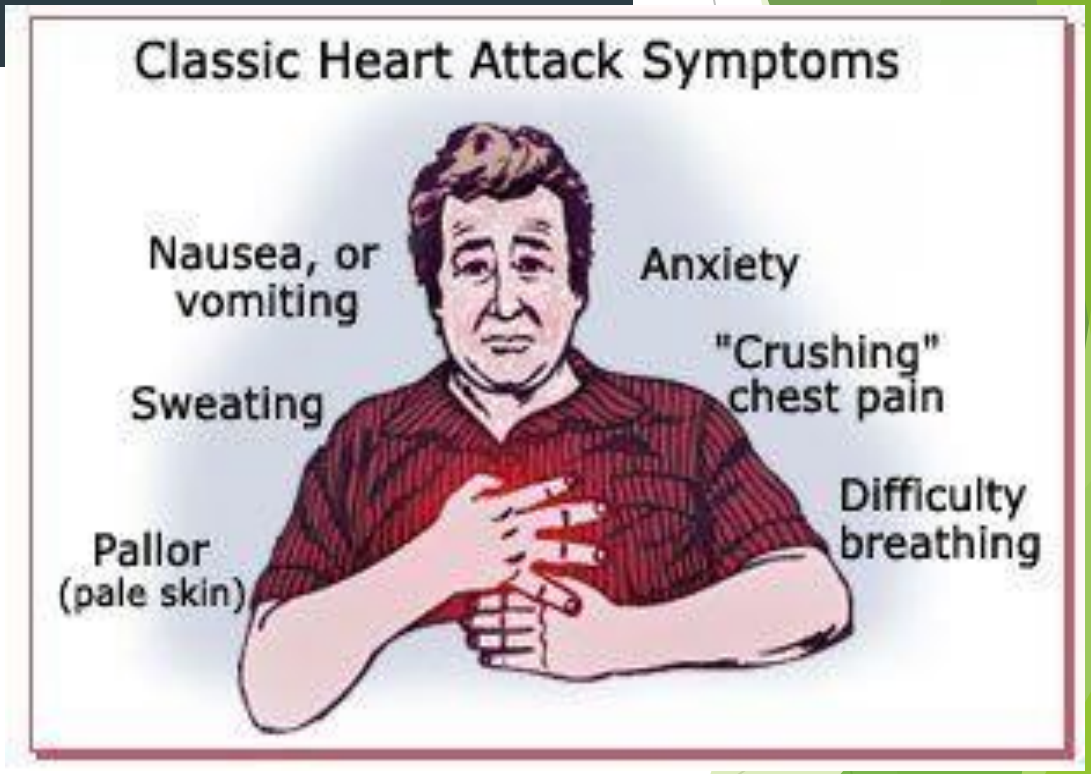
MI

Causes

- When a coronary artery which is narrowed by atherosclerosis is further constricted by vasospasm
- rupture of an **atherosclerotic plaque-** formation of a coronary-occluding blood clot

Myocardial infarction





Cardiac biomarkers- enzymes & proteins leak into circulation from damaged myocardial cells

- Troponin T, Troponin I- released 2–4 h

  after MI. Peak levels are seen after 12 h persist upto 7days
- Creatine Kinase (CK-MB) -*increase within*

  4–6 h persist upto 2-3 days

- lactate dehydrogenase (LDH)

ECG in MI- initial stage

- ST Segment elevation in leads overlying the area of infarct & also T wave inversion

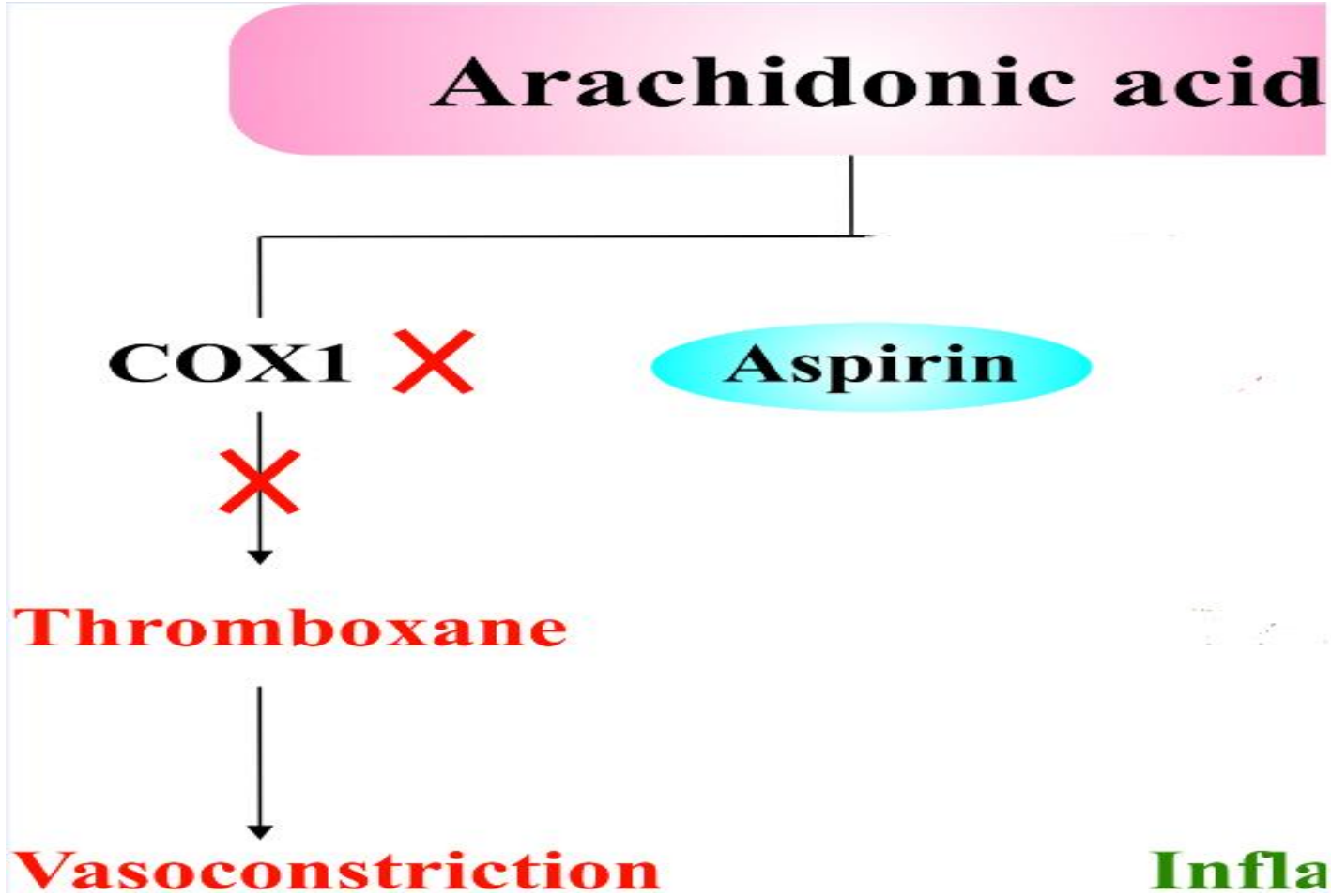
Management

- Vasodilators -Nitrates
- Thrombolytic agents (streptokinase)-conversion of plasminogen to plasmin---cause fibrinolysis.

  tissue plasminogen activator
- Aspirin –inhibit platelet aggregation

Coronary angioplasty

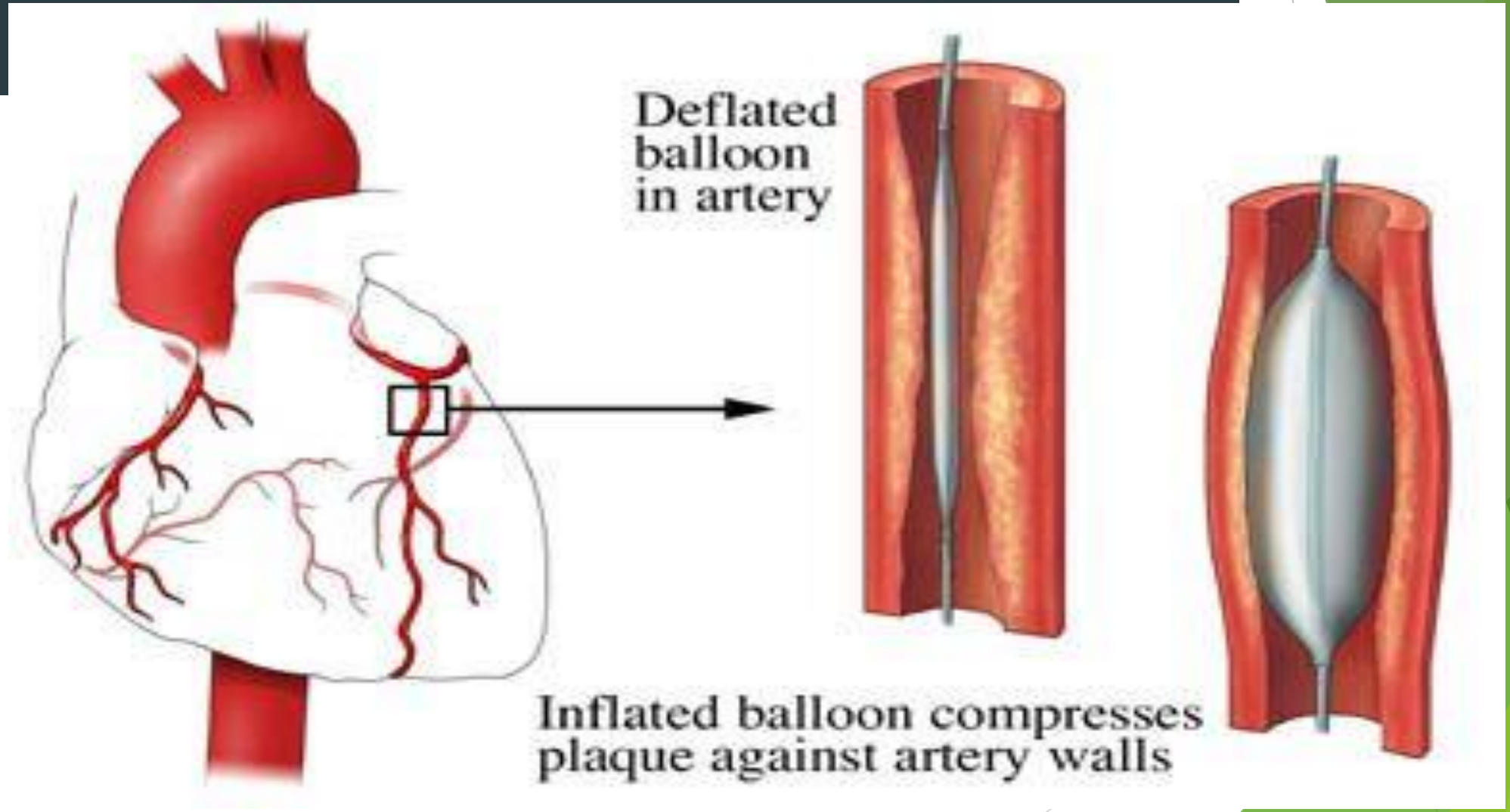
Coronary bypass surgery


Arachidonic acid
COX1
Aspirin
Thromboxane
Vasoconstriction
Infla

Coronary angioplasty

- Inflatable balloon tipped catheter is inserted into coronary artery,on reaching the narrowing site it is inflated to dilate constricted artery

Coronary angioplasty



Coronary bypass surgery

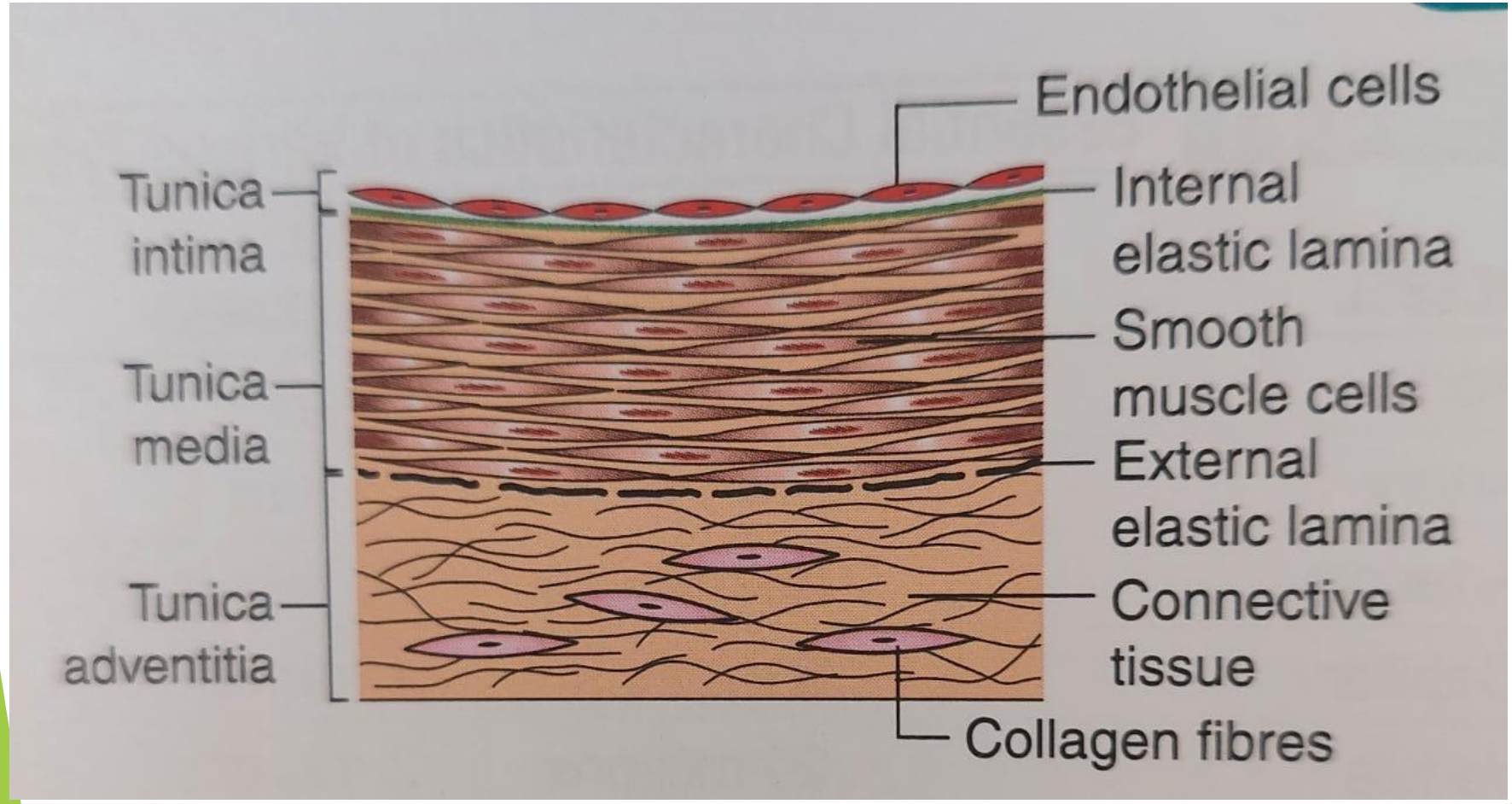
- CABG-coronary artery distal to the obstruction is anastomosed with aorta using a suitable vessel (saphenous vein,internal mammary artery as graft)

BLOOD VESSELS

- ANGIOGENESIS: Formation of new blood vessels

STRUCTURE OF BLOOD VESSELS

- Blood vessels except Capillaries have 3 layers
- TUNICA INTIMA
- TUNICA MEDIA
- TUNICA ADVENTITIA


Endothelial cells
Tunica intima
Internal elastic lamina
Tunica media
Smooth muscle cells
External elastic lamina
Tunica adventitia
Connective tissue
Collagen fibres

- TUNICA INTIMA:
- Innermost coat of vessel wall,consists of (inside to outside)

1. Endothelial lining-single layer cells,contact with blood
2. Basal lamina-glycoprotein layer
3. Subendothelial connective tissue
4. Internal elastic lamina-elastic fibres

- TUNICA MEDIA:
- Middle thickest layer
- Consists of smooth muscles & elastic tissues
- Ratio of two tissues varies from vessel to vessel
- Outside tunica media ,a layer formed by elastic fibres called external elastic lamina

- TUNICA ADVENTITIA:
- Outermost layer composed of connective tissue
- Collagen fibres are prominent
- Prevents undue stretching or distension of blood vessel

TYPES OF BLOOD VESSELS

- AORTA----ARTERIES----arterioles& metaarterioles----Capillaries------Venules-----Veins

CLASSIFICATION of BLOOD VESSELS

- **1.WINDKESSEL VESSELS:**

  Large elastic arteries---Aorta &its branches
- T.Media –dominant elastic tissue ---provides the property of distensibility during systole to accommodate blood & elastic recoil during diastole
- Recoil effect is windkessel effect
- Windkessel means elastic reservoir

- **DISTRIBUTION VESSELS:** **arte**ries like radial,ulnar,popliteal
- Elastic tissue in tunica intima & media is much less, smooth muscle increases

RESISTANCE VESSELS:

Arterioles,metaarterioles

- **Arterioles –seat of peripheral resistance**
- Contain more smooth muscles &less or no elastic fibres in T.media
- Have relatively narrow lumen—less radius
- SYMpathetic stimulation produces smooth muscle contraction & Vasoconstriction

SPHINCTER VESSELS:

- Precapillary sphincters
- Regulate the opening of capilliaries & blood flow through capilliaries

EXCHANGE VESSELS

- **capilliaries**
- Allow easy exchange of gases & nutritive substances across them
- Have single layer of endothelial cells.
- No T. Media & T.Adventitia
- Total cross sectional area of capilliary bed is 1000 times that of aorta

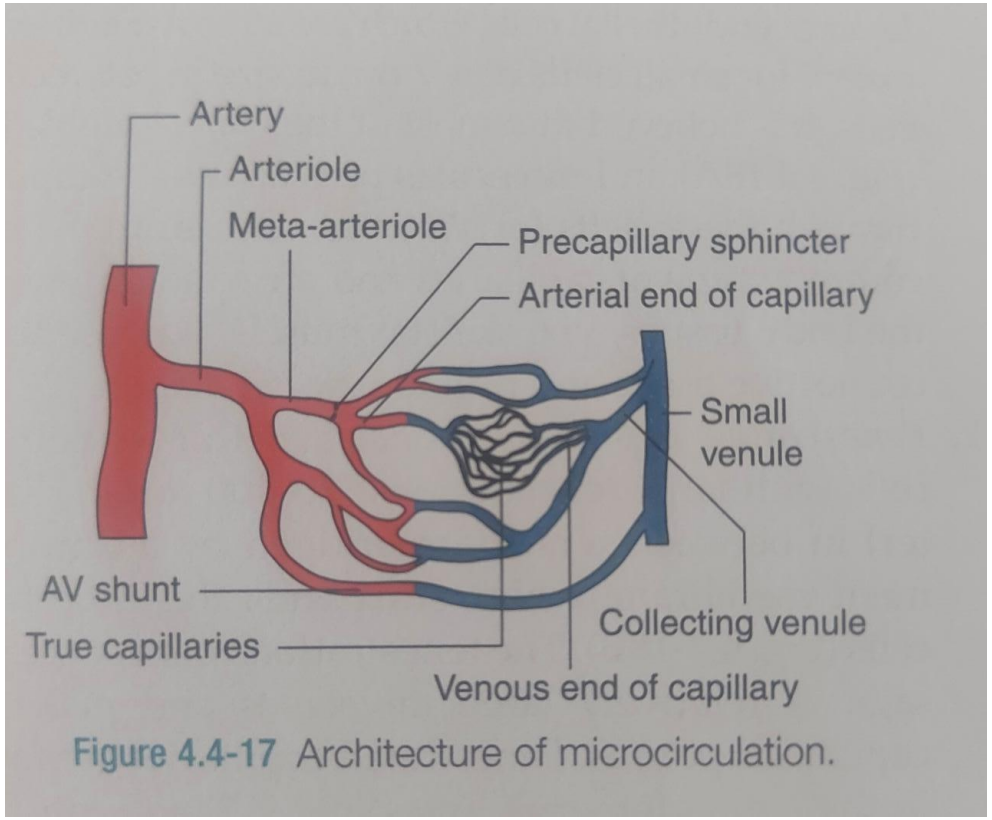


Figure 4.4-17 Architecture of microcirculation.

CAPACITANCE vessels

VEINS

- Vessels can change their luminal shape & can accommodate large volume of blood /unit length without increasing pressure
- Valves present prevents backflow &direct blood flow to heart

- SHUNT VESSELS
- Vessels bypass the capilliaries
- Vessels directly connect meta arterioles with venules as Arteriovenous shunts /AV anastomoses
- Role in thermoregulation
- Permit rapid flow of blood

CROSSSECTIONAL AREA OF BLD VESSELS

- AORTA<ARTERY<ARTERIOLE<CAPILLIARY
- Velocity inversely proportional to C.AREA

Table 4.4-1 Essential Characteristics of Various Types of Blood Vessels

VESSEL	LUMEN DIAMETER	WALL THICKNESS	TOTAL APPROXIMATE CROSS-SECTIONAL AREA (cm^2) OF ALL VESSELS OF EACH TYPE	PERCENTAGE OF BLOOD VOLUME CONTAINED
Aorta	2.5 cm	02 mm	4.5	2
Artery	0.4 cm	01 mm	20	8
Arteriole	30 microns	20 microns	400	1
Capillary	5 microns	1 micron	4500	5
Venule	20 microns	2 microns	4000	54
Vein	0.5 cm	0.5 mm	40	
Vena cava	3 cm	1.5 mm	18	
Heart	–	–	–	12
Pulmonary circulation	–	–	–	18

MICROCIRCULATION

- Involves a meshwork of vessels less than 100 microns in diameter
- Includes small arterioles,meta arterioles,capillaries,venules & arteriovenous shunts

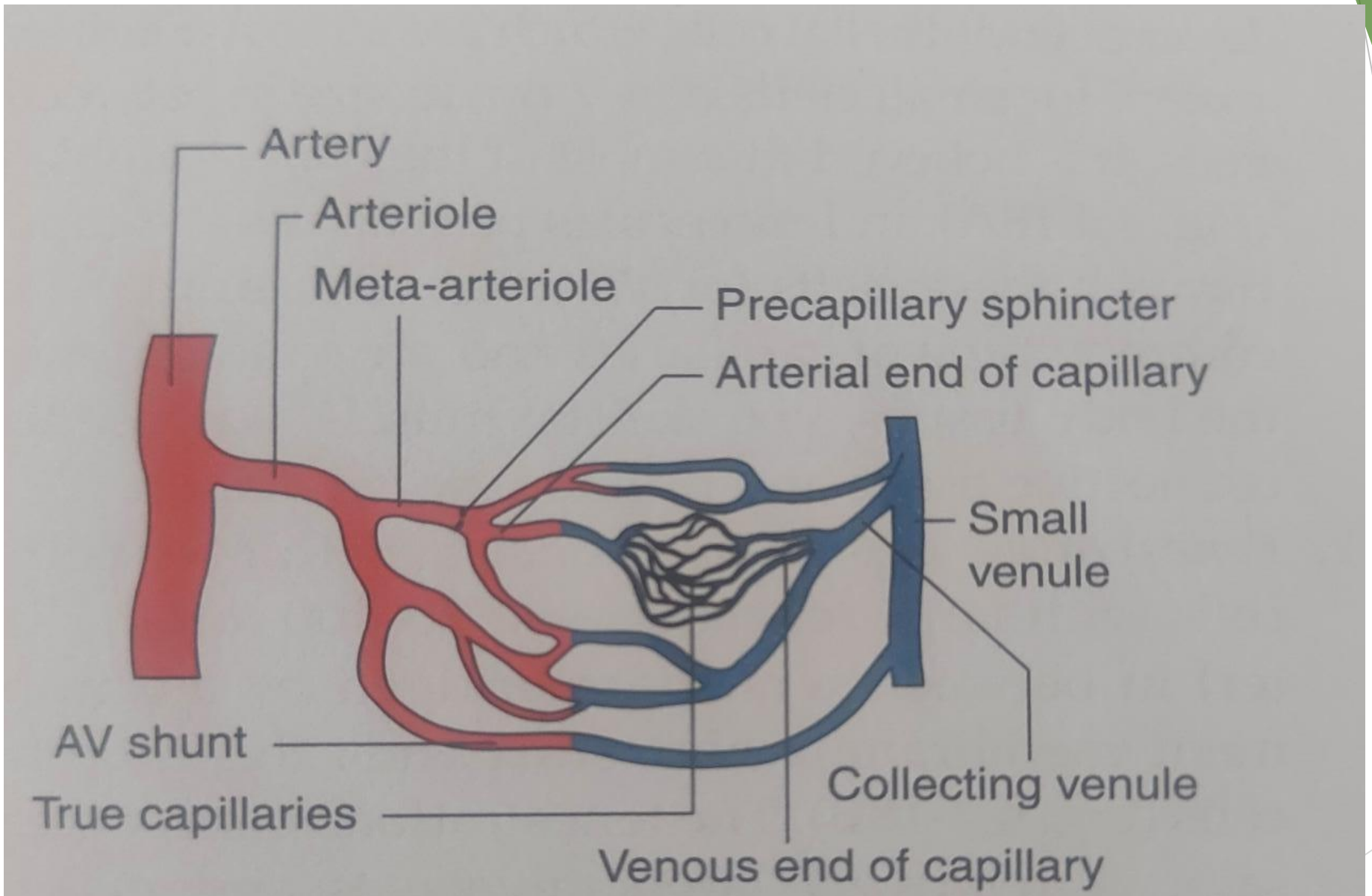


Figure 4.4-17 Architecture of microcirculation.

CAPILLARY CIRCULATION

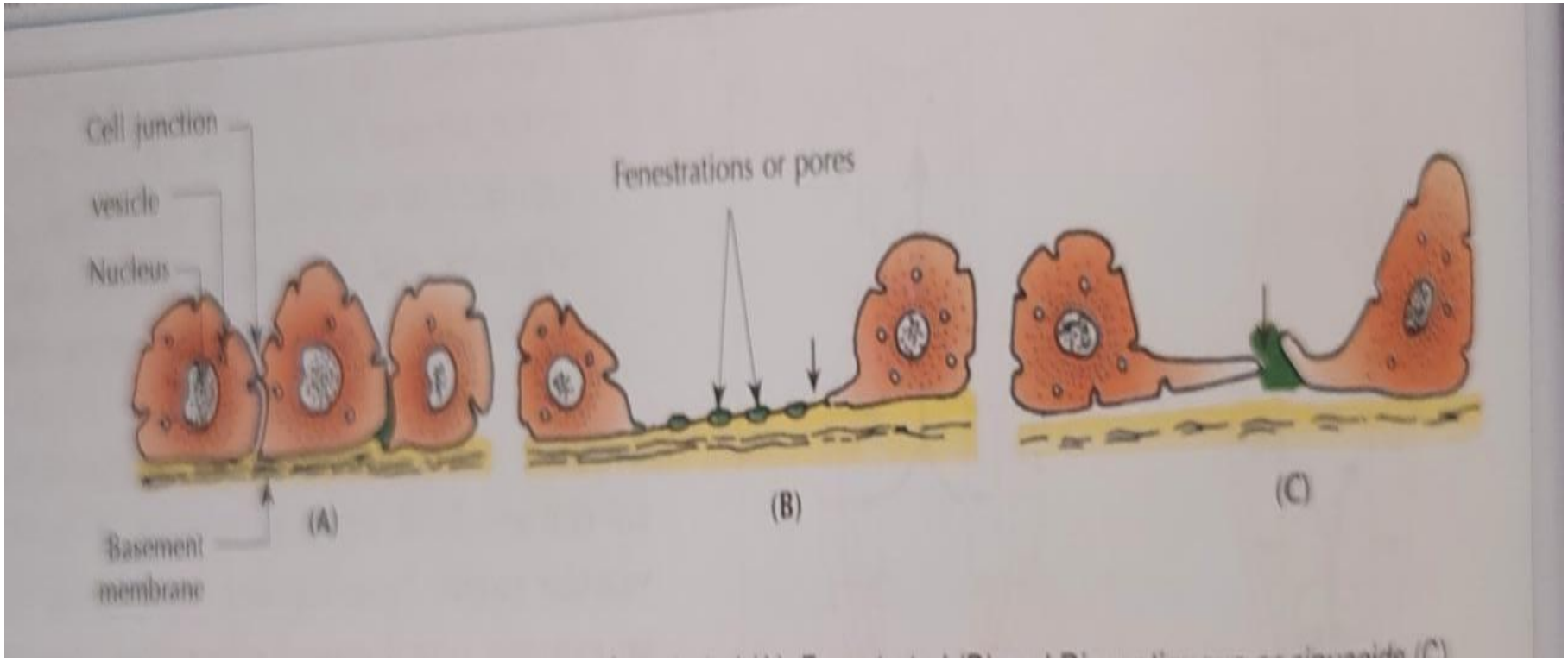
- CAPILLARIES
- Single layer of endothelial cells
- Capillaries not controlled by nervous & metabolic factors
- Controlled by precapilliary sphincters
- Endothelial structure of capillaries varies in different organs depending on the function of particular tissue

TYPES of CAPILLIARIES

- CONTINOUS:
- Endothelial cells almost continous except for small clefts /small pores
- Molecules & water soluble ions can pass across the capillary through these pores
- Most commonest type
- Present in skeletal muscle,adipose tissue,connective tissue, pulmonary circulation

- FENESTRATED:
- Thin endothelial cells with large fenestrations surrounded by basement membrane
- Fenestrations permit large molecules
- Found in renal glomeruli, intestinal villi,most endocrinal glands

- DISCONTINOUS CAPILLIARIES:
- Large gaps between endothelial cells,not closed by basement membrane
- Called sinusoids
- Found in bonemarrow,liver,spleen


Cell junction
vesicle
Nucleus
Fenestrations or pores
Basement membrane
(A)
(B)
(C)

ACTIVE & INACTIVE CAPILLIARIES

- IN resting tissues:most of the capilliaries are collapsed(inactive)----blood bypasses them ---- flow to venules through thoroughfare vessels
- IN ACTIVE TISSUES:metaarterioles & precapilliary sphincters dilate & blood flows through capilliaries ,more &more capilliaries become patent---recuritment of closed capilliaries--- controlled by local metabolic vasodilators &sym system

FUNCTIONS OF CAPILLIARIES

- Transport nutrients to the tissue & remove waste products that produced by tissues
- Cells of the different tissues close to capilliaries---easy transport/exchange of substances occur between them---so called exchange vessels

MECHANISM OF TRANSPORT OF SUBSTANCES IN CAPILLIARIES

- FILTRATION
- DIFFUSION
- VESICULAR TRANSPORT

1) **Filtration** : In accordance with Starlings hypothesis
   - At the arterial end of capillary fluid moves into the tissue spaces(Hydrostatic pressure exceeds oncotic pressure).
   - At the venous end fluid moves into the capillary.(oncotic pressure more than hydrostatic pressure)
   - In filtration molecules move through the pores in between the endothelial cells (Not through the body of endothelial cells)

- DIFFUSION:passage of molecules from higher concentration to lower concentration without expenditure of energy
- FACTORS AFFECTING
- 1.LIPID SOLUBILITY:can easily pass through entire wall
- 2.lipid insoluble diffuse through intracellular pores through channels

- 3.SIZE OF THE MOLECULE:permeability decreases with increase in M W
- 4.capilliary wall surface area for diffusion determined by precapiliary sphinctors
- 5.velocity of flow of blood:

  Velocity of flow decreases diffusion decreases

- VESICULAR TRANSPORT:
- Large molecules
- Small vesicles in endothelial cells fuse with cell membranes &discharge their contents into /out of cell

Factors Governing Blood Flow in Capillaries

INCREASE THE FLOW/VASODILATORS/

- Hypoxia,acidosis,hypercapnia
- Histamine,bradykinin,&acetylcholine
- Increase in temp,extreme cold
- Sympathetic cholinergic stimulation

- DECREASE IN BLOOD FLOW/VASOCONSTRICTORS
- Catecholamines,vasopressin
- Decreasein temp
- Sym(adrenergic stimulation)

CEREBRAL CIRCULATION

- ARTERIAL SUPPLY:
- Principal arterial supply to the brain in humans by
- 2 internal carotid artery &

  basilar artery formed 2 vertebral arteries

- INTERNAL CAROTID ARTERY divides into anterior & middle cerebral arteries near optic chiasma
- Basilar artery divides into 2 posterior cerebral arteries
- ANTERIOR,MIDDLE,POSTERIOR cerebral arteries of both sides united by communicating arteries to form a vascular ring called CIRCLE OF WILLIS

- VENOUS DRAINAGE:
- Through superficial & deep veins draines to dural sinuses finally reaches internal jugular veins
- Small amount drained into ophthalmic & pterigoid venous plexuses

INNERVATION of cerebral blood vessels

- SYMPATHETIC—through superior cervical ganglion—large A---causes vasoconstriction
- PARASYMPATHETIC---sphenopalatine ganglion---large A---causes vasodilators
- SENSORY FIBRES----Trigeminal ganglion---to small vessels----vasodilation
- TOUCHING /PULLING in the cerebral vessels causes Pain

MEASUREMENT OF CEREBRAL BLOOD FLOW

- KETY METHOD using Ficks principle –N20 used
- --only knows average flow of perfused brain
- RADIOACTIVE Xe133&I123-SPECT
- Regional blood flow can be detected
- PET scan

FEATURES OF CEREBRAL CIRCULATION

- 1.Cerebral blood flow is 15% of resting CO
- 2.Average blood flow is 54ml/100g/min. whole brain 756 ml/minute
- 3.Regional variation of blood flow to the brain.Grey matter-70ml/1oog/min, white matter-28ml/100g/min

- 4.Marked variation in local blood flow with brain activity.

Increase in blood flow with increase in physical/mental activity

Epileptic foci-increase blood flow

Alzheimers –decreased flow in superior parietal &frontal regions

- 5.NO crossing over between arteries of both hemisphere because pressure is equal in both sides
- Even when pressures are not equal,anastomotic channels of circle of wills do not permit very large flow
- So subjected injected into one side distributed only on one side

- 6.Insufficient anastomosis & collateral circulation to maintain the normal circulation &prevent infarction when unilateral arterial occulsion

- 7.Density of capillaries is great because of high metabolic needs
- 8.Cerebral capillaries are non fenestrated ,tight junctions between cells,limits passage of substances,also capilliaries surrounded by endfeet of astrocytes forming BBB.

- 9.Brain highly sensitive to changes in extracellular environment. BBB prevents toxic substances from entering brain
- 10.AUTO REGULATION:cerebral blow shows autoregulation between 60 to 140mm of Hg
- 11.CNS ischemic response-if BP below 60mmHg,CBF decreased ,cerebral ischemia causes CNS Ischemic response to increase BP to maintain blood flow

- 12.O2 consumption of brain is 20% of total body resting O2 consumption of whole body

- 13.Brain highly sensitive to hypoxia.

  O2 lack causes unconsciousness,long period O2 lack causes irreversible damage

- 14.Glucose is the major source of energy for the brain,so brain is sensitive to hypoglycemia
- 15.Effect of gravity on cerebral circulation

When decrease/increase in arterial pressure corresponding de/increase in venous pressure also----EPP same---brain will get adequate perfusion

- 16.When increase in intracranial pressure , cerebral vessels compressed,cerebral blood flow decreases ----overcome by Cushing reflex

REGULATION OF CEREBRAL BLOOD FLOW

- FACTORS AFFECTING Cerebral BF
- 1.Intracranial Pressure
- 2.Mean Arterial pressure of brain
- 3.Mean Venous Pressure of brain
- 4.Local factors:chemical &Autoregulation
- 5.nervous regulation
- 6.viscosity

1.INTRACRANIAL PRESSURE

- MUNRO-KELLIE DOCTRINE:
- The volume of brain,CSF& blood is constant
- Any increase in brain volume,cerebral blood vessels are compressed
- ICP rises above 33mmHg,the person lose consciousness
- ICP rises it initiate cushing reflex----increase in ICP----- compression of Blood vessels----decrease blood flow----- hypoxia,hypercapnea------stimulation of VMC-----increase in BP &reflex baroreceptor stimulation causes decrease in HR

- ICP continues to increase for long time ,ICP exceeds arterial pressure--- cerebral circulation stops

MEAN ARTERIAL PRESSURE

- MAP increases ,cerebral blood flow increases

MEAN VENOUS PRESSURE

- Increase mean venous pressure ----decrease effective perfusion pressure(EPP= mean pressure in cerebral artery –mean pressure in cerebral veins)-----decrease in cerebral blood flow
- MECHANISM of decrease blood flow
- 1.compression of blood vessels due increase in ICP
- 2.decrease in EPP

VISCOSITY

- Increase in viscosity causes increase cerebral vaso resistance ---decrease in blood flow eg polycythemia
- Decrease in viscosity causes increase in flow eg.anemia

METABOLIC FACTORS

- Increase in PCO2 upto 80mmHg ---vasodilation---- increase in cerebral blood flow
- Decrease in pCO2 upto20mmHg--- vasoconstriction----decrease in flow

- Increase H + concentration of blood—causes increase in Pco2—diffuse in CSF ---causes vasodilation

- Hyoxia ---vasodilation---increase in flow
- Hyperbaric O2 therapy causes decrease in flow due to disruption of neuronal metabolism

AUTOREGULATION

- Cerebral blood flow shows autoregulation between 60-140mmhg---cerebral flow remains constant—MYOGENIC HYPOTHESIS
- Increase in pressure results in vasoconstriction
- Decrease cause vasodilation
- Below 60—syncope
- Above 140mm cause disruption BBB-odema

NERVOUS REGULATION

- Parasympathetic stimulation causes vasodilation
- Sympathetic causes vasoconstriction
- IN severe hypertension---SYM stimulation---vasoconstriction----prevents cerebral hge &stroke & disruption of BBB

STROKE

- Blood supply to a part of brain is interrupted-----damages /kills neuronal cells-----stroke
- 2 types

1.hemorrahagic—cerebral artery /arteriole ruptures

2 Ischemic—flow in a vessel is compromised by atherosclerotic plaque/thrombi from other parts

- Ischemia ----decrease glutamate uptake by astrocytes---excitoxic damage to neurons---death of neurons

management

- Determine if a stroke is hgic/thrombotic
- If thrombotic,clot lysis---tpA
- Drugs to reduce glutamate