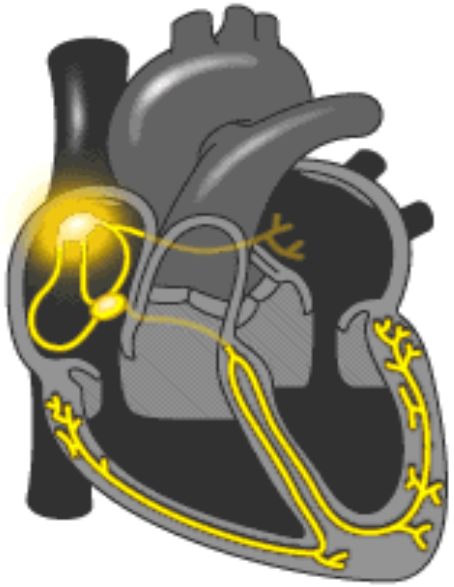
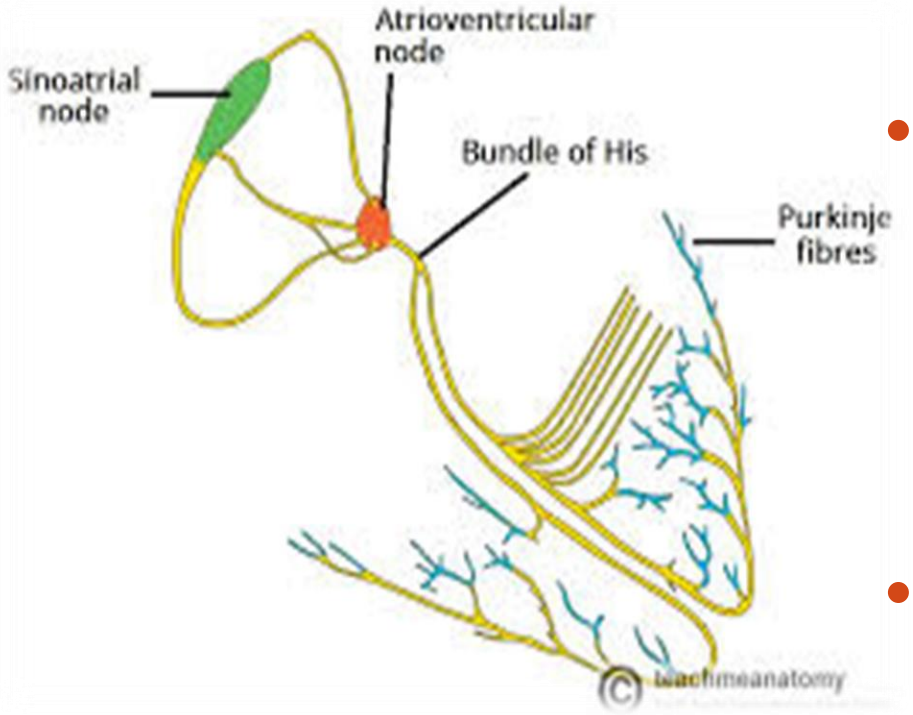
ORIGIN & SPREAD OF CARDIAC IMPULSE



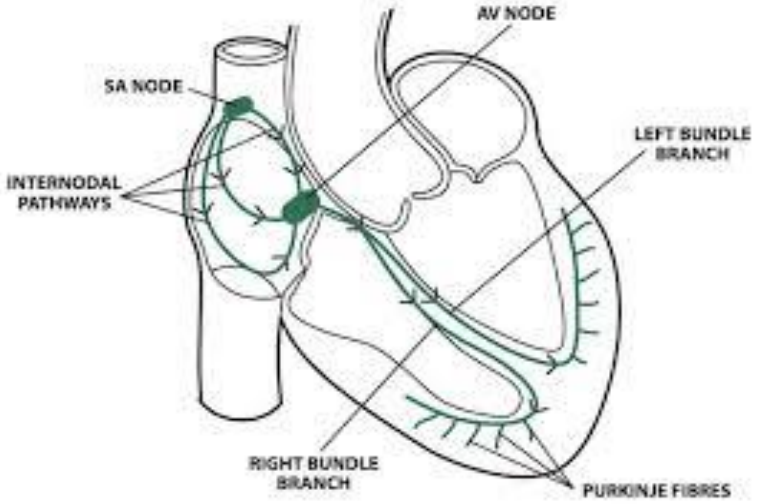
ANUMOL K

CONDUCTING SYSTEM



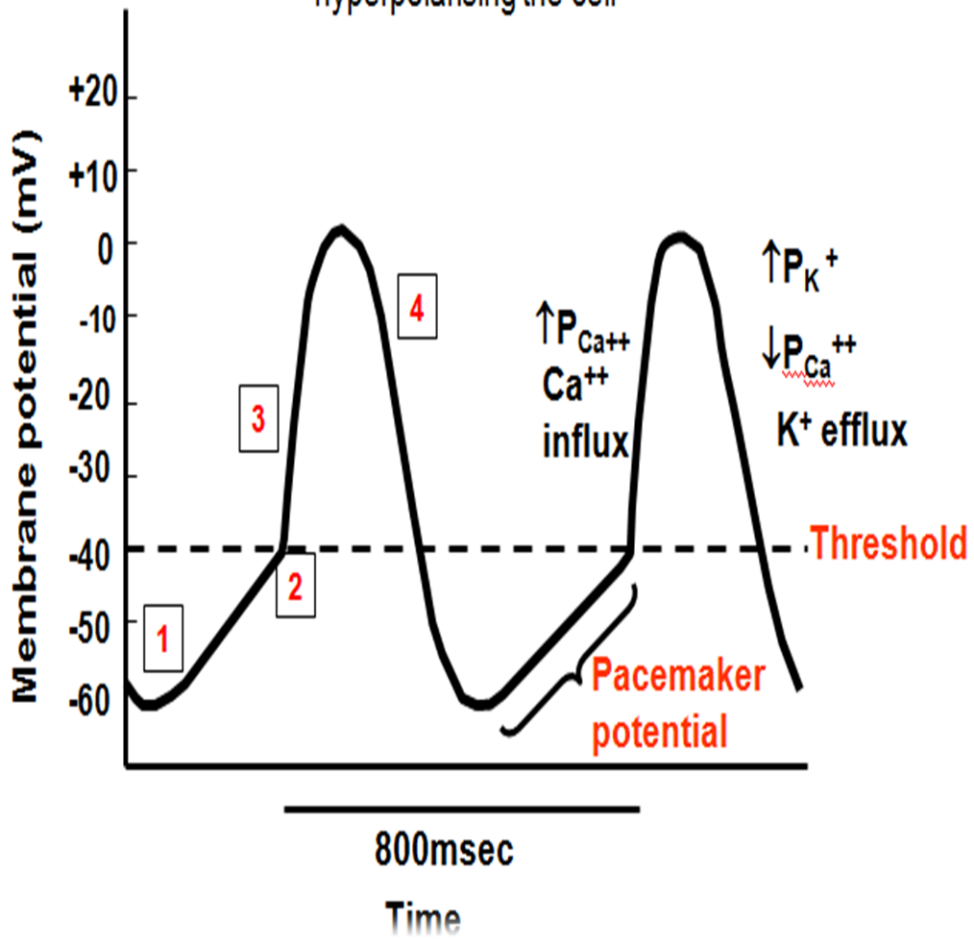
- Heart contains special conducting tissue which generates & conducts its own AP continuously which is responsible for non stop functioning of heart.
- Conducting system of heart is formed by modified cardiac muscle fibers = Junctional tissue.

COMPONENTS		LOCATION	CONDUCTION SPEED (M/SEC)
SINOATRIAL NODE	POSTERIOR WALL OF R.ATRIUM JUST BELOW & RIGHT TO SVC OPENING		0.05
ATRIOVENTRICULAR NODE	ANTERO-INFERIOR PART OF RIGHT SIDE OF INTER ATRIAL SEPTUM		0.05 (SLOWEST)
INTERNODAL TRACTS	ANTERIOR ASPECT OF RIGHT ATRIUM 1. ANTR : BACHMAN 2. POSTR: WENCKEBACH 3. MIDDLE: THOREL		1.0
BUNDLE OF HIS	CONTINUATION OF AV NODE IN INTERVENTRICULAR SEPTUM DIVIDES INTO 1. RIGHT BUNDLE BRANCH (SMALL) 2. LEFT BUNDLE BRANCH (LARGE)		1.0
PURKINJE FIBERS	ORIGINATE FROM TERMINAL DIVISIONS OF BUNDLE OF HIS IT SPREAD ALL OVER VENTRICLES		4.0 (FASTEST)



ORIGIN OF CARDIAC IMPULSE

1. 'Funny' sodium channels (I_f channels) are open ($\uparrow P_{Na}^{+}$); and closing K^+ channels.
2. Transient Ca^{2+} (T-type) channels open, pushing the membrane potential to threshold.
3. Long-lasting Ca^{2+} (L-type) channels open, giving rise to the action potential.
4. Opening of K^+ channels, ($\uparrow P_{K}^{+}$), and closing of Ca^{2+} (L-type) channels, hyperpolarising the cell



- Cardiac impulse is generated & discharged by pacemaker cells of SA node .
- RMP of SA node is -55mv & is unstable because of continuous change in membrane permeability.
- After each AP, the membrane potential declines until firing level is reached.

- RMP that depolarizes is called pre potential as it brings mp to threshold & is due to opening of h/funny sodium channels and transient ca channels
- Once the threshold level of – 40 mv is reached, occurs rapid depolarization due to long lasting ca2+ influx.
- At the end of depolarization k channels open & ca channels close results in repolarization and then to hyperpolarization.
- Later again after hyperpolarization funny channels open which cause pre potential and it repeats.
- An AP is generated which is transmitted all over myocardium via junctional tissue.

SPREAD OF CARDIAC IMPULSE

Mnemonic: SAIL MRS. SAVE APPS

- 1. **S**A node (generates impulse at highest rate, i.e. 70-80/min)
- 2. **A**V node
- 3. **I**nterventricular septum – **L**eft
- 4. **M**idportion **R**ight **S**eptal activation
- 5. **S**eptum (upper to lower) – purkinje system
- 6. **A**pex
- 7. **V**entricular wall – **E**ndocardium to epicardium

  Epicardial surface of right ventricle is depolarized earlier than left because of its thinner wall.
- 8**.** **A**V groove
- 9. **P**osterobasal portion of left ventricle, **P**ulmonary conus and **S**eptum (uppermost portion) – last part of heart to be depolarized

ATRIAL ACTIVATION

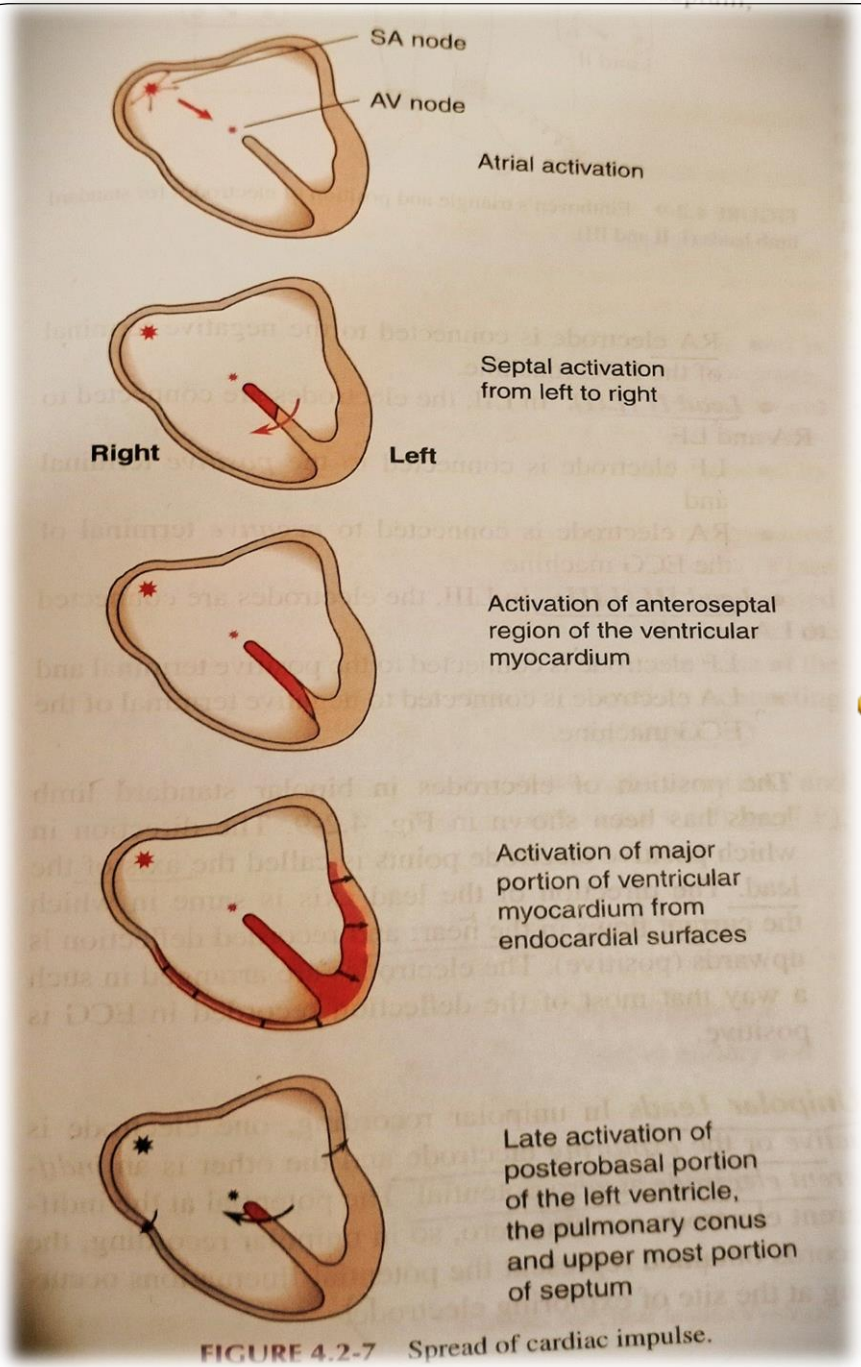


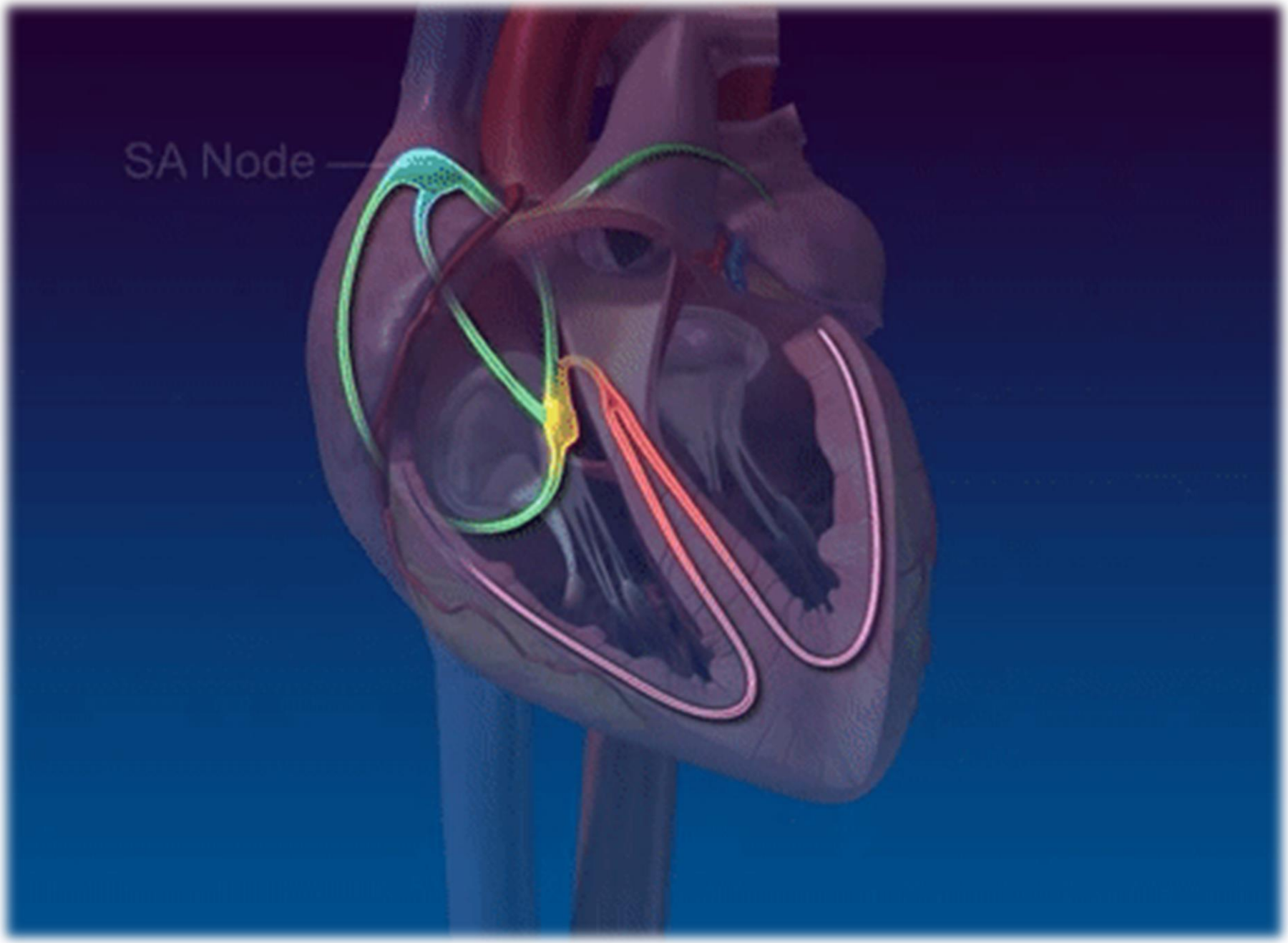
FIGURE 4.2-7 Spread of cardiac impulse.

- Impulse generates in the SA node travel to the myocardium in a radial direction like ripple in a pond
- Via common musculature , impulse spreads to left atrium & the conduction velocity in the atrium is 1 m/sec.
- Impulse reaches the AV node via inter nodal tracts at a speed of 0.05 m/ sec. (Time: 0.03 sec after its origin)

VENTRICULAR ACTIVATION

- Impulse from AV node travels through Bundle of His, its branches & Purkinje fibers & stimulates ventricles
- 1 st region get stimulated: Lft. Interventricular septum- mid portion.
- Moves first to the right across midportion of septum

  Wave of depolarization then spreads down the septum to (R) apex & then (L) apex
- Then it return along the ventricular walls to AV groove, proceeding from endocardial to epicardial surface
- Last regions to get stimulated: pulmonary conus, upper portion of interventricular septum & finally the postero-basal region of (L) ventricle


SA Node

TOTAL TIME FOR CONDUCTION

SA NODE

0.03 SEC

AV NODE

0.13 SEC

BUNDLE BRANCHES

0.03 SEC

PURKINJE FIBRES

0.03 SEC

ENDOCARDIAL & EPICARDIAL SURFACE OF VENTRICLES

AV NODAL DELAY

- It is the delay in transmission of impulse arriving from SA node to ventricles
- It is usually about 0.1 sec.
- Maximum delay occurs in the A- N region & N region of AV node & is usually inversely proportional to HR.

Reasons

- Small & primitive: transitional fibers that connect internodal tracts & AV node are small
- Much more negative RMP than other cardiac tissue
- Fewer gap junctions connecting successive fibers in the pathway.

Detrimental contraction: Ability of AV node to slow & block the rapid impulse

- AV nodal delay – shortened by sympathetic stimulation & lengthened by vagal stimulation

Importance of AV nodal delay

- It allows atrial systole to complete before ventricles get excited.
- During atrial systole, ventricles will be in the diastole which helps in filling.
- Beneficial in pathological conditions (atrial fibrillation), in which atrial depolarization is very high.

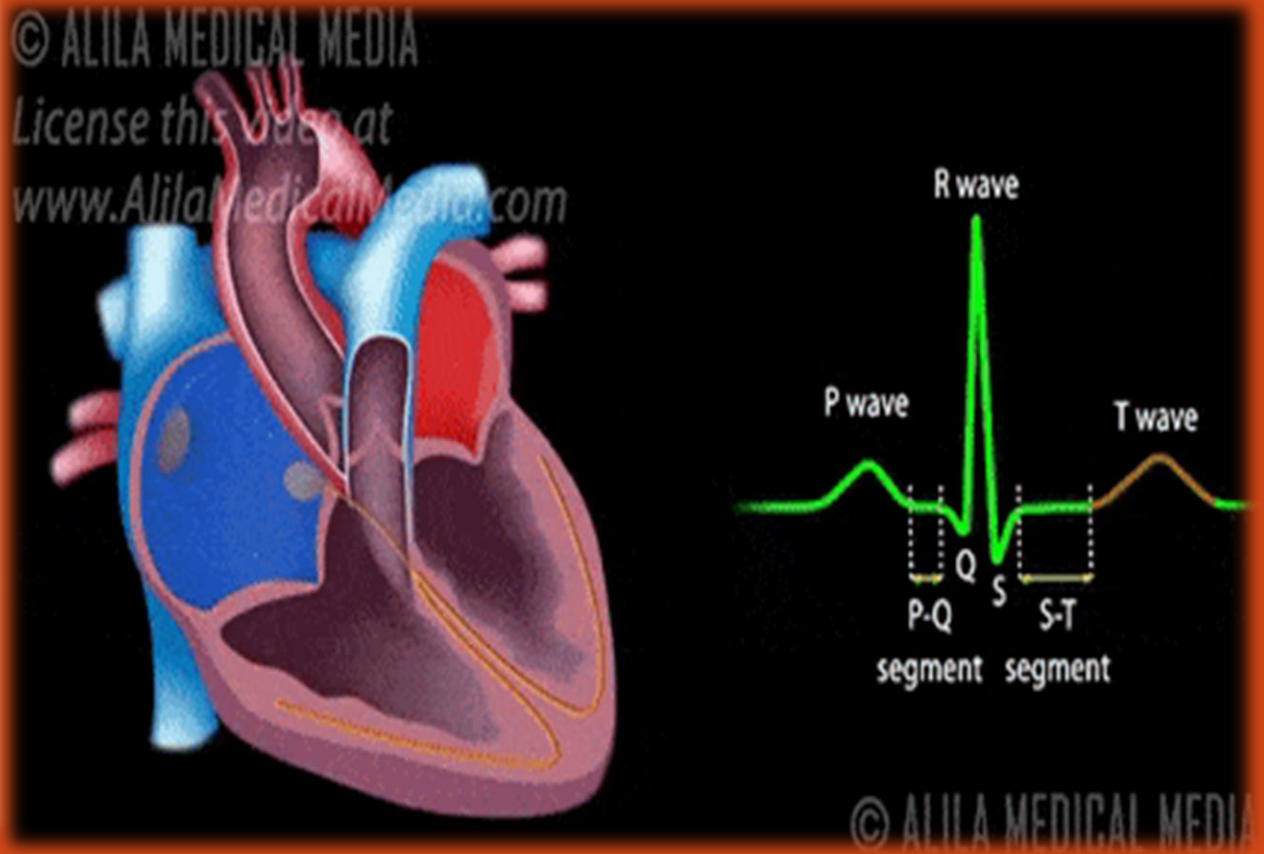
CLINICAL SIGNIFICANCE

- Digitalis, beta blockers reduce hr by promoting AV nodal delay

Pathology

- Av block: failure of impulse to pass normally from atria to ventricle through AV nodal & bundle system.
- Wolff- Parkinson white syndrome : Abnormal atrioventricular transmission of cardiac impulse through an aberrant pathway (additional pathway (bundle of kent) doesn't have the property of AV delay)

CARDIAC CYCLE



CARDIAC CYCLE

- A series of events should occur regularly & systematically in the heart in a particular sequence for accomplishing the proper pumping action of the heart

DEFINITION

- Cardiac cycle is defined as the sequence of electrical & mechanical events occurring in the heart cyclically, from the beginning of one heart beat to the beginning of the next.

DURATION

Duration of 1 cardiac cycle when HR= 72bpm

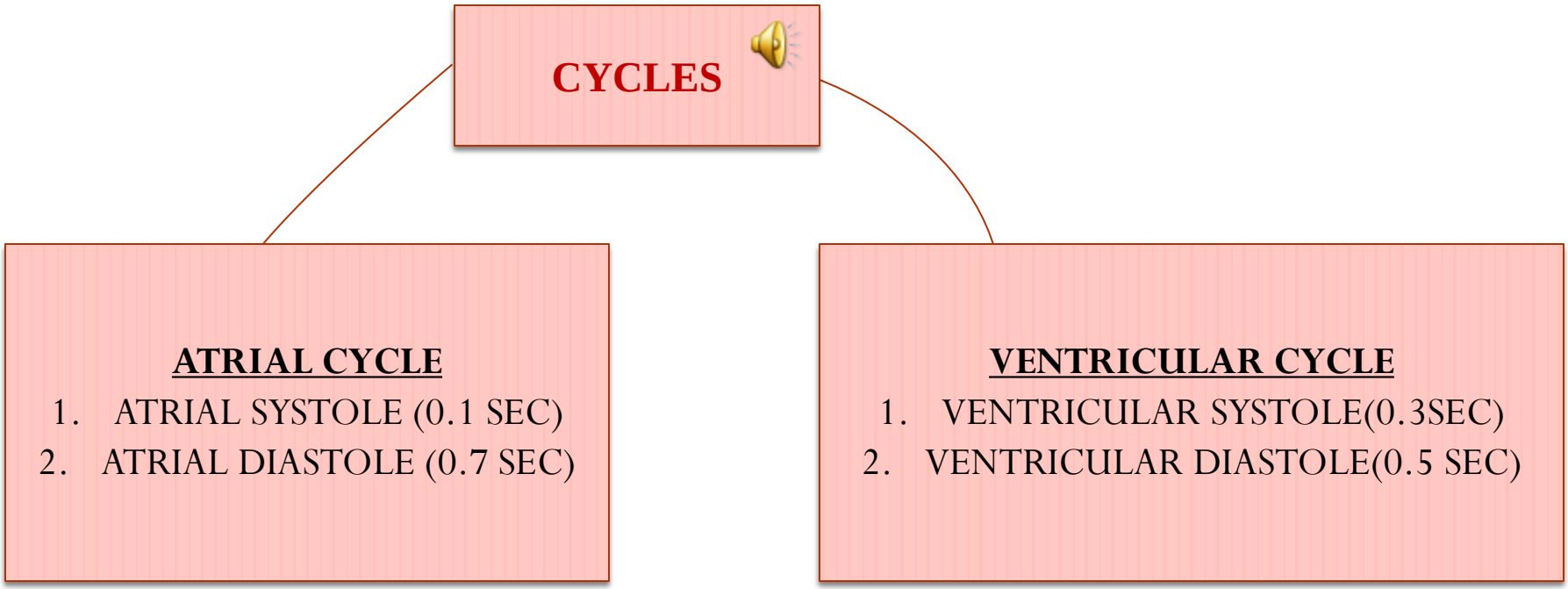
60/72= 0.8 sec

PHASES

Cardiac Cycle Consists Of 2 Main Phases

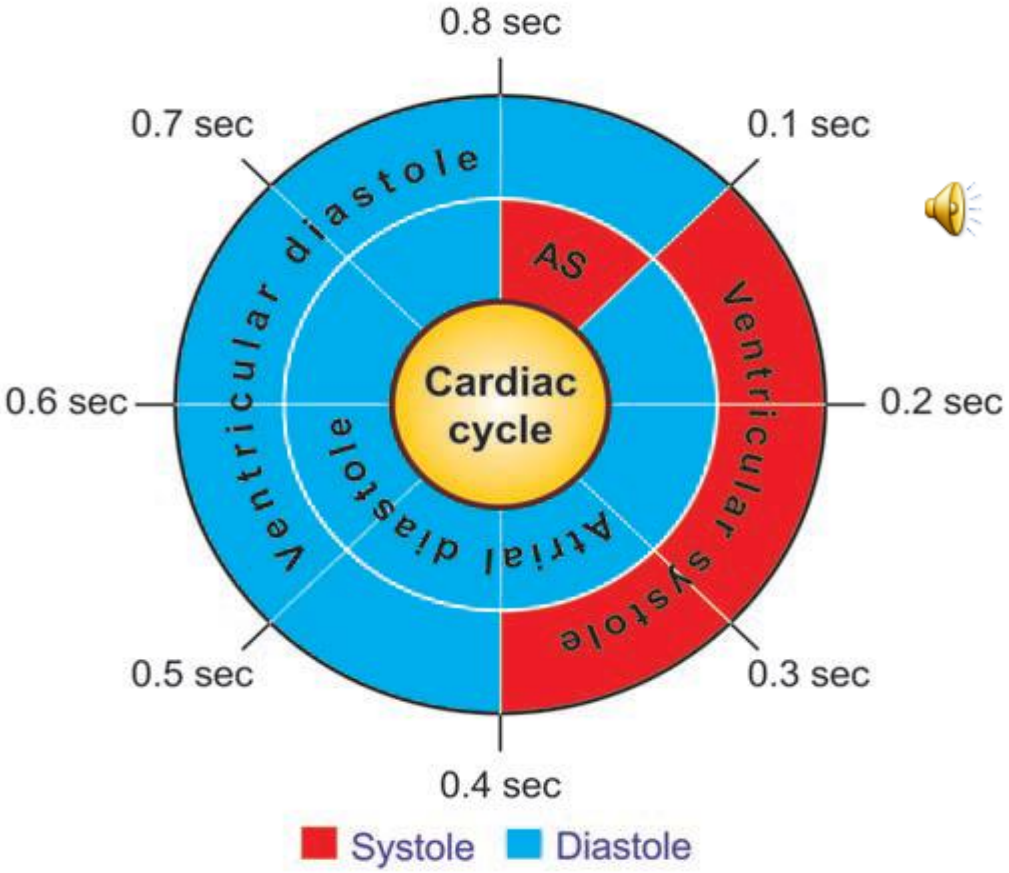
1.Systole : Period Of Contraction Of Heart

2. Diastole : Period Of Relaxation Of Heart



PHASES

- 1. ATRIAL SYSTOLE
- 2. ATRIAL DIASTOLE



1. VENTRICULAR SYSTOLE

a) ISOVOLUMETRIC CONTRACTION

b) RAPID EJECTION

c) SLOW EJECTION

2. VENTRICULAR DIASTOLE

a) PROTO DIASTOLE

b) ISOVOLUMETRIC RELAXATION PHASE

c) INITIAL RAPID FILLING

d) DIASTASIS

e) LAST RAPID FILLING- FOLLOWED ATRIAL SYSTOLE

EVENTS OF CARDIAC CYCLE

MECANICAL EVENTS

Study of mechanical events of heart- cardio dynamics

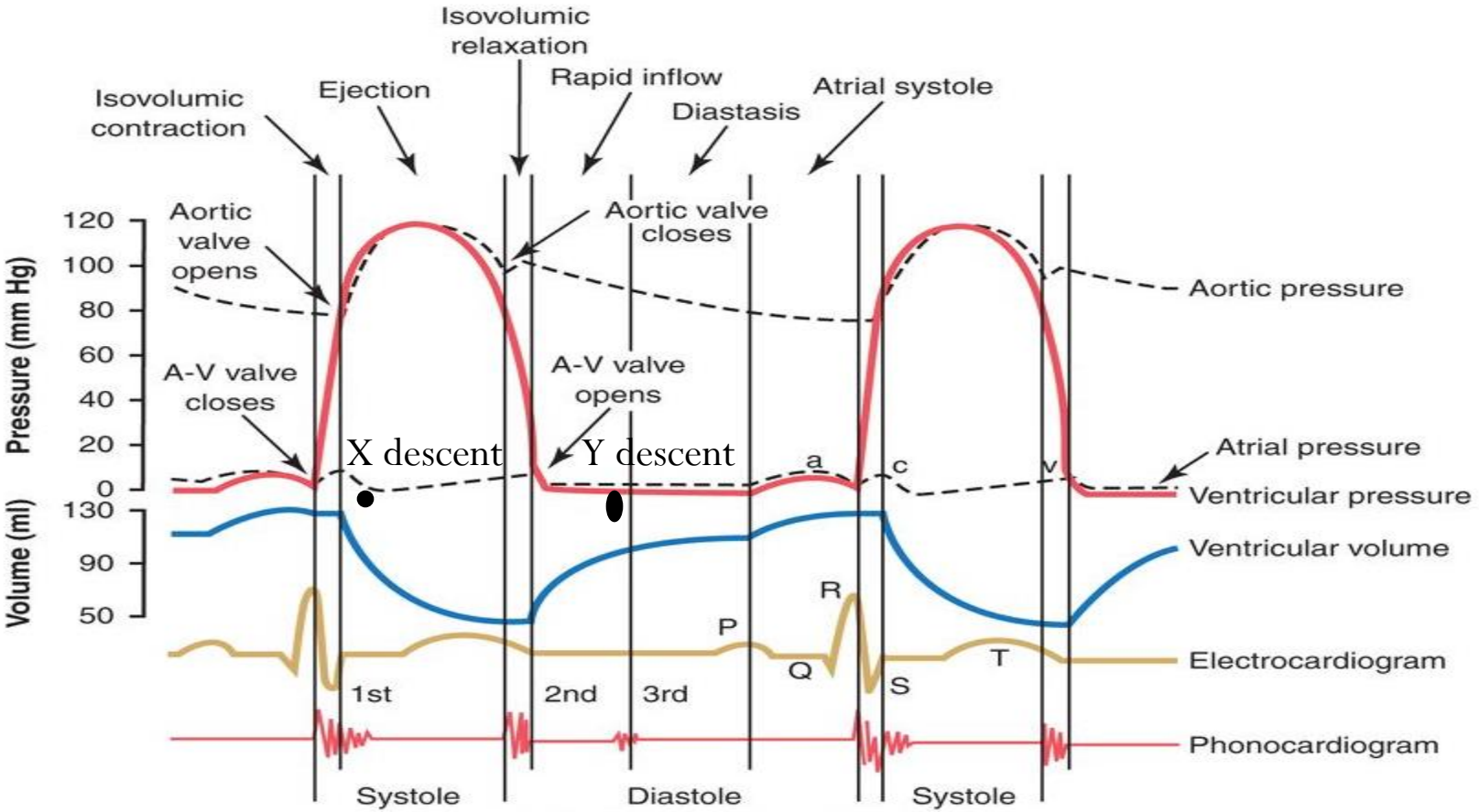
1. Pressure Changes In Atria, Ventricles, aorta
2. Volume Changes In Ventricles
3. Valvular Events – Heart Sound

ELECTRICAL EVENTS

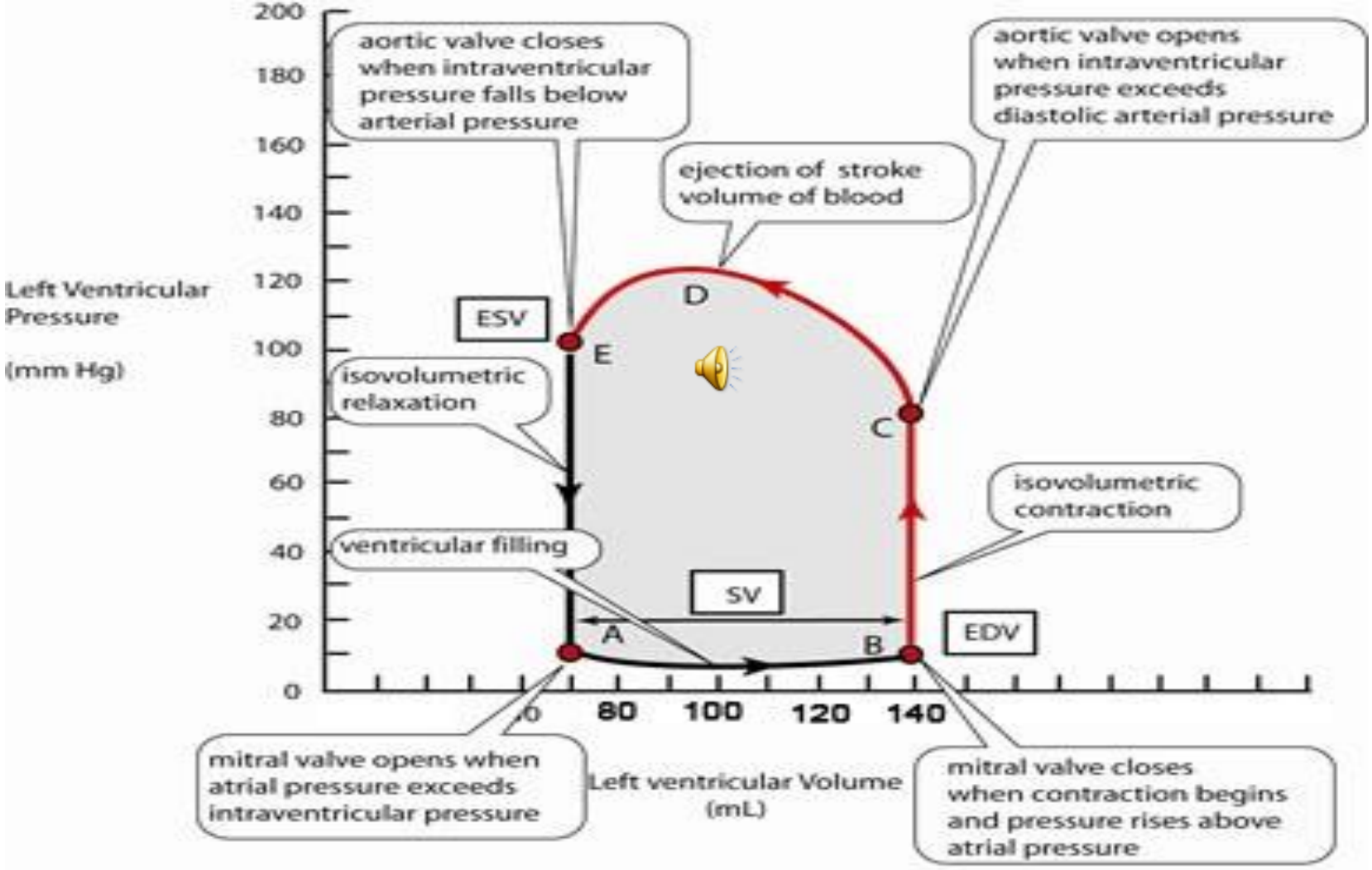
- Can be depicted using ECG
- Electrical events are followed by mechanical events

WIGGER'S DIAGRAM

- Events of cardiac cycle can be expressed in simple diagram which includes ECG changes, pressure-volume changes, heart sounds= Wigger's Diagram



Pressure Volume Loop- Ventricle



ATRIAL SYSTOLE

PRESSURE CHANGES IN ATRIA

1. ***Before onset of systole*** **:**
   - Intra atrial pressure slightly above zero & Slightly greater than intraventricular pressure
2. ***During systole:***
   - Sharp rise in atrial pressure by 4-6 mmHg in RA
   - 7-8 mmHg in LA
   - Causes a pressure wave **'a'**
   - **(atrial systole)** wave from the jugular vein
3. ***Immediately after systole***:
   - Atrial pressure falls down.

PRESSURE CHANGES IN VENTRICLES

1. Before onset of systole **:**

- Ventricular pressure is little above zero

2. During systole:

- There occurs a slight increase in IVP.
- Pumping of blood in ventricle cause
- RV : 6-7 mmHg
- LV : 7-8 mmHg
- **Segment AB** in pressure volume loop curve denote these pressure changes
- A- Atrial systole
- B- Closure of AV valves

PRESSURE CHANGES IN AORTA

1. Pressure in the aorta during atrial systole is about 80mmHg.

VENTRICULAR SYSTOLE

1. ISOVOLUMETRIC CONTRACTION

PRESSURE CHANGES IN ATRIA

- Due to contraction , sharp rise in IVP,
- AV valves close & bulge into atria.
- Small but sharp rise in atrial pressure
- Produce **'c' wave** (contraction of ventricle in wiggers diagram.

PRESSURE CHANGES IN VENTRICLES

- Ventricles contracts as a closed chamber(AVV Closed; SLV not yet opened).
- Intraventricular pressure increases rapidly
- Segment BC (PV Loop)
  - B- Closure of AVV
  - C- Opening of SVV & Commencement of ventricular ejection phase

PRESSURE CHANGES IN AORTA

- unaltered

VENTRICULAR SYSTOLE

11. RAPID & SLOW EJECTION PHASE

PRESSURE CHANGES IN ATRIA

- Intra atrial pressure drops sharply
- Produces "X descent"
- It occurs due to relaxation of atria & downward pulling of AV ring
- Atrial Pressure continues to rise as it get filled with the blood draining from great veins

PRESSURE CHANGES IN VENTRICLES

- Ventricles contract @ greater than rate at which blood ejected causes great increase in P.
- P rises maximum in LV : 120 mmHg& RV:- 25 mmHg.
- Phase represented by CD Segment where D – Peak point of IVP (P_V Loop)

IN AORTA

- When aortic valves open, blood starts flowing from ventricle to aorta.
- Aortic Pressure rises reaches maximum of 120 mmHg at the end of rapid ejection.
- But it remains slightly < IVP
- later on it declines to fall on 80mm Hg

VENTRICULAR DIASTOLE- PROTO DIASTOLE & ISOVOLUMETRIC RELAXATION

P CHANGES IN ATRIA

- Atrial pressure continuous to rise till the end of these phases
- 3 rd wave " V" wave – Venous filling-

P CHANGES IN VENTRICLES

PROTO DIASTOLE

- IVP starts decline
- When IVP< P of aorta 7 Pulm.Artery; SVV closes due to backflow of blood.
- Represented by Segment EF

ISO VOLUMETRIC

•Ventricles relax as closed chamber

•Rapid fall in IVP from 80 to 2-3 mmHg in LV

•Represented by "Segment FG"

• G- opening of AVV.

VENTRICULAR DIASTOLE-
RAPID &REDUCED PASSIVE FILLING

P CHANGES IN ATRIA

- In Rapid Phase, AVV open
- Rapid flow of blood to ventricles
- Atrial P falls sharply a little above zero
- This fall produces “Y descent”

P CHANGES IN VENTRICLES

- IVP falls further in rapid filling phase because ventricles are relaxing.
- In Reduced Filling Phase, no turbulence
- IVP remains little above zero.

VENTRICULAR DIASTOLE- PROTO DIASTOLE & REMAINING PHASES

Pressure changes in Aorta

- Aortic Pressure slightly higher than that in LV
- Causes backward flow of blood
- Sudden Closure of SVV- sharp rise in aortic pressure = Incisura
- During rest of the phases, aortic p remains to about 80 mmHg

Volume changes

- Atrial capacities are slightly greater than corresponding ventricles
- Atria act as reservoirs.
- Total diastolic volume of heart- 540ml
- During systole- 140 ml ejected by each ventricle

Atrial Volume Changes

During atrial systole,

- flow is zero due to back pressure into the great veinsAVV closed, ; and major part of atrial filling occurs.

Ventricular systole

- Atrioventricular ring moves down during V. systole so the volume in the atria increases

In ventricular diastole,

considerable flow occurs into the atrium.

VENTRICULAR VOLUME CHANGES

During Atrial Systole,

- It coincides with the last filling phase of ventricular diastole
- when atrial contraction begins, 75% (105 ml) blood has already flown into the ventricles.

During Ventricular systole, -

1. Isovolumetric contraction

   no change since all the valves are closed.

2. Maxim & reduced Ejection Phase

- Volume decreases rapidly
- About 80 ml is ejected by each ventricle= stroke volume
- About 50 ml blood remain in ventricle at the end of V.Systole= End systolic volume

Ventricular Volume changes

Proto diastole & Isovolumetric relaxation

- No change

Rapid and slow filling phase

- volume changes rapidly and slowly
- 75% filling occurs at this phase
- The amount of blood in the ventricle at the end of each diastole = End diastolic volume.

VALVULAR & ELECTRICAL EVENTS

Heart Sound	Occurs during:	Associated with:	Correlation with ECG
S1	Isovolumetric contraction	Closure of mitral and tricuspid valves	Coincides with peak of R wave
S2	Isovolumetric relaxation	Closure of aortic and pulmonic valves	Coincide with end of T wave
S3	Early ventricular filling	Normal in children; in adults, associated with ventricular dilation (e.g. ventricular systolic failure)	Appears between T & next P wave
S4	Atrial contraction	Associated with stiff, low compliant ventricle (e.g., ventricular hypertrophy; ischemic ventricle)	Interval between end of P wave & onset of Q wave

The most fundamental heart sounds are the first and second sounds, usually abbreviated as S_1 and S_2. **Phonocardiography** is the recording of all the sounds made by the heart during a cardiac cycle.

APPLIED PHYSIOLOGY

- Valvular diseases (stenosis, incompetency etc.
- Cardiac murmurs

QUESTIONS

1. Define cardiac cycle with diagram, Explain MECHANICAL EVENTS
2. Phases of cardiac cycle
3. Left ventricular pressure changes
4. Pressure volume changes
5. Av nodal delay

THANK YOU