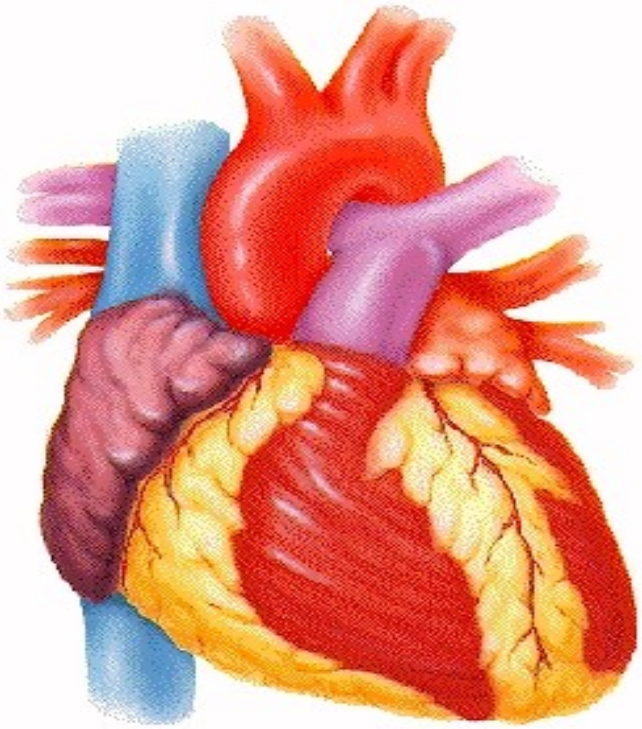
CARDIOVASCULAR SYSTEM

ANATOMY AND PHYSIOLOGY (PART I) OF THE HEART

DR.KRITHIKA KRISHNAN

DR.MADHUR

MODERATOR: DR.JYOTSNA



www.anaesthesia.co.in

anaesthesia.co.in@gmail.com

INTRODUCTION

- Chambers of the heart.
- Flow of blood through the chambers.
- Ventricular structure.
- Coronary circulation.
- Conduction system.
- Cardiac myocyte.

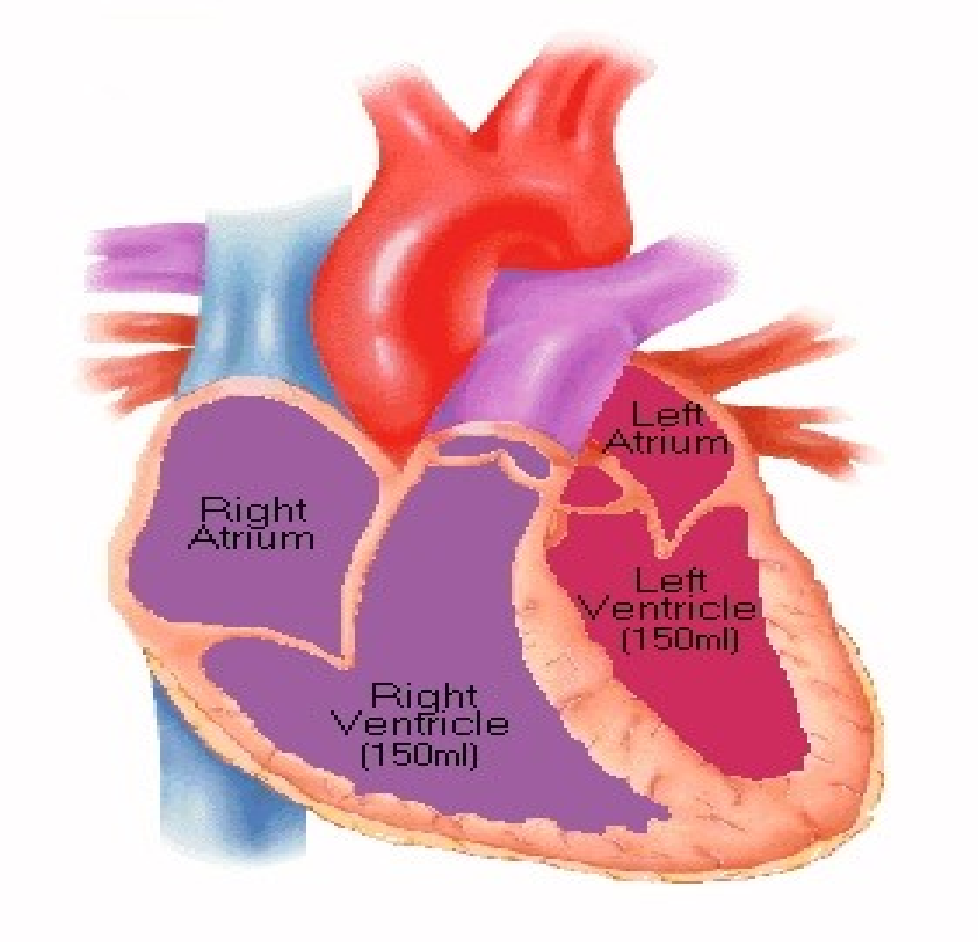
Borders and surfaces

- Right border:- right atrium.
- Inferior border:- right atrium and ventricle and the apex of the heart.
- Left border:- left atrium and ventricle.
- Anterior surface:- right atrium, and ventricle, AV groove, anterior IV groove and left ventricle.
- Inferior surface:- left and right ventricle.
- Base:-left atrium and pulmonary veins.

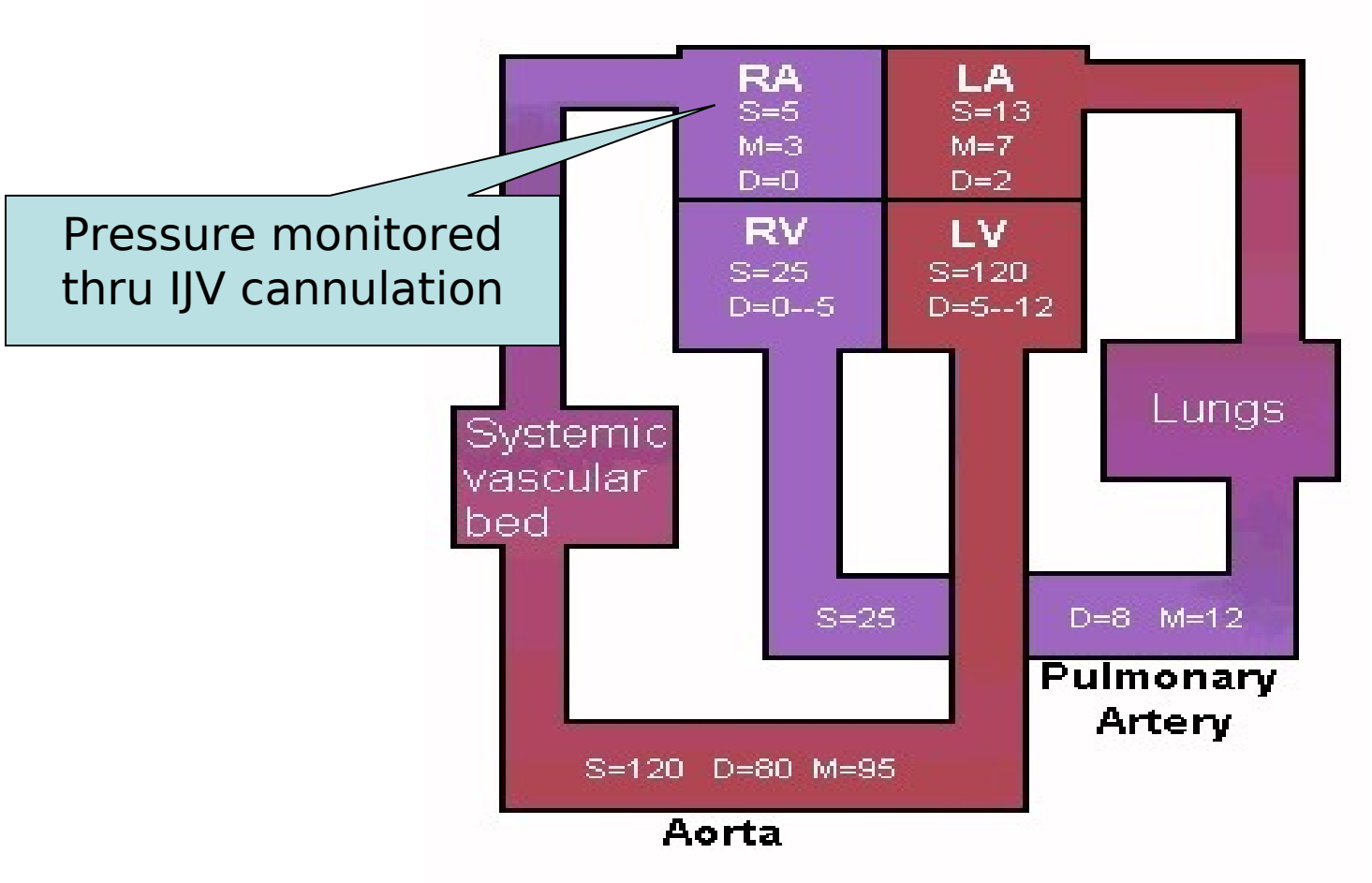
Surface anatomy

- Right border:- from 3rd to the 6th rib 1.25cm to the right side of the sternum.
- Apex:- left 5th intercostal space 9cm from the midline.
- Left border:-from the apex to the 2nd intercostal space 1.25cm lateral to the sternum.

CHAMBERS OF THE HEART



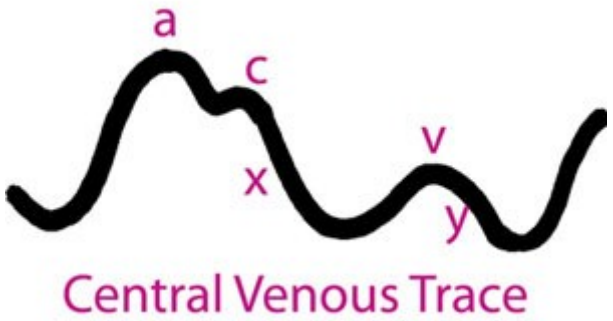
PRESSURES IN THE CHAMBERS



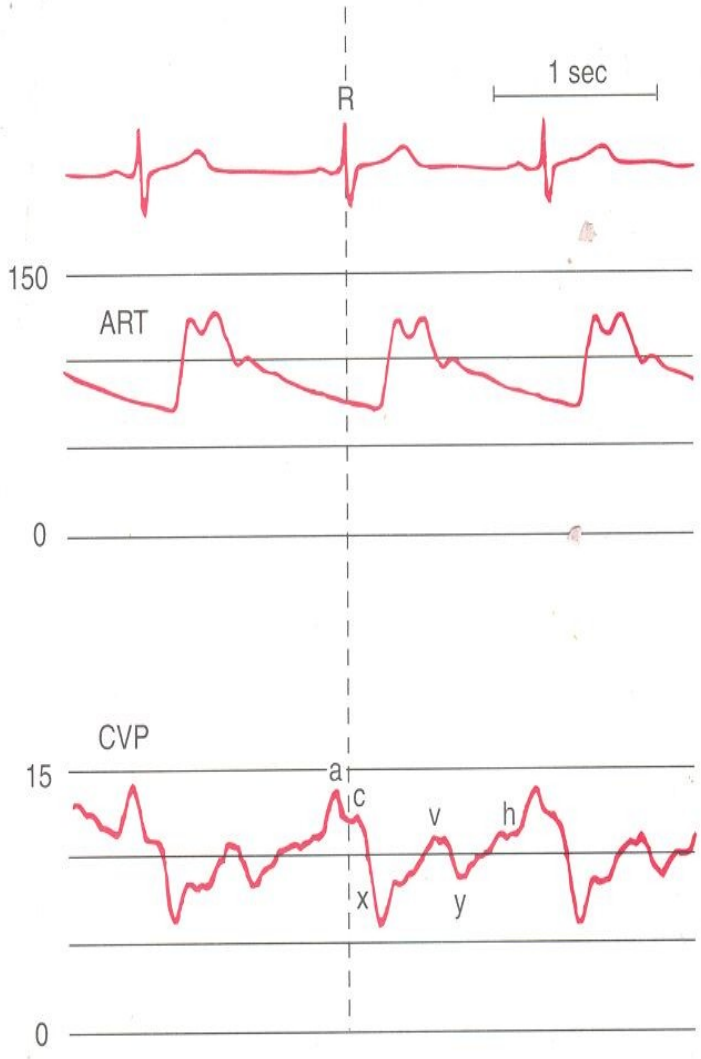
CENTRAL VENOUS PRESSURE

since there are no valves between SVC and RA the RA pressure is identical to the CVP.

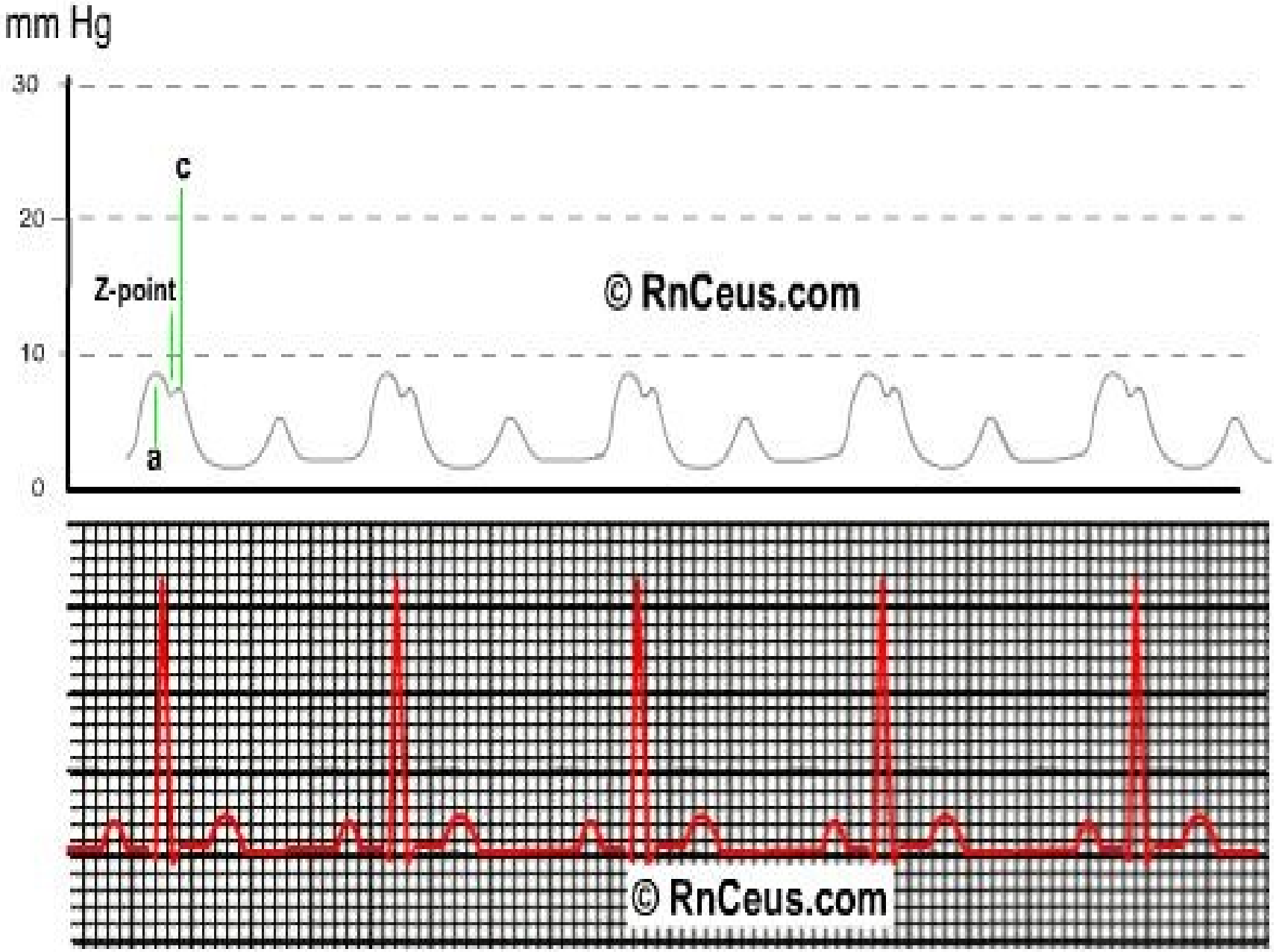
- Appropriateness of the blood volume to the capacity of venous system.
- Functional status of the right heart.



CVP wave form

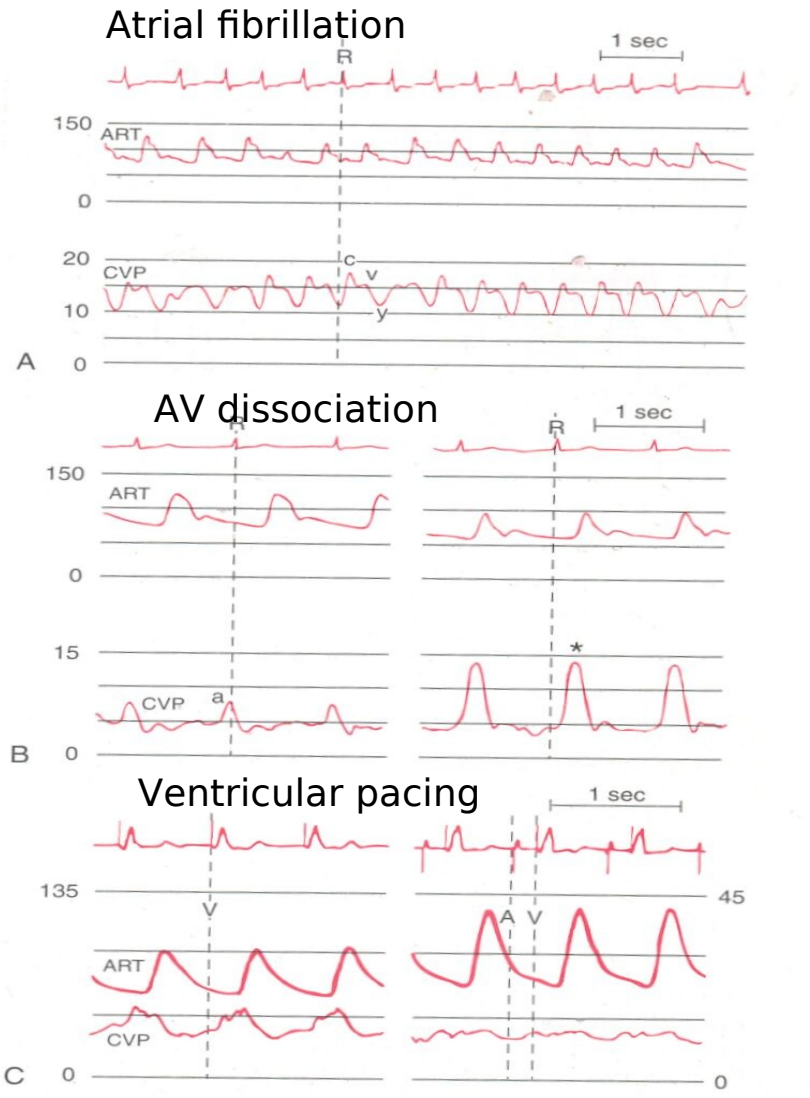


- A wave – first positive wave of atrial pressure (follows the P wave on the ECG), end diastolic,atrial contraction.
- C wave – second positive wave ,early systolic, isovolumetric ventricular contraction, carotid impact, (after R wave on the ECG).
- X descent – first negative wave after the A wave (represents atrial relaxation),mid systole.
- V wave – third positive wave represents the low pressure in the atrial rising because of venous filling (pooling), late systole.
- Y descent – early atrial emptying, early diastole.


mm Hg
30
20
10
0
c
Z-point
a
© RnCeus.com
© RnCeus.com

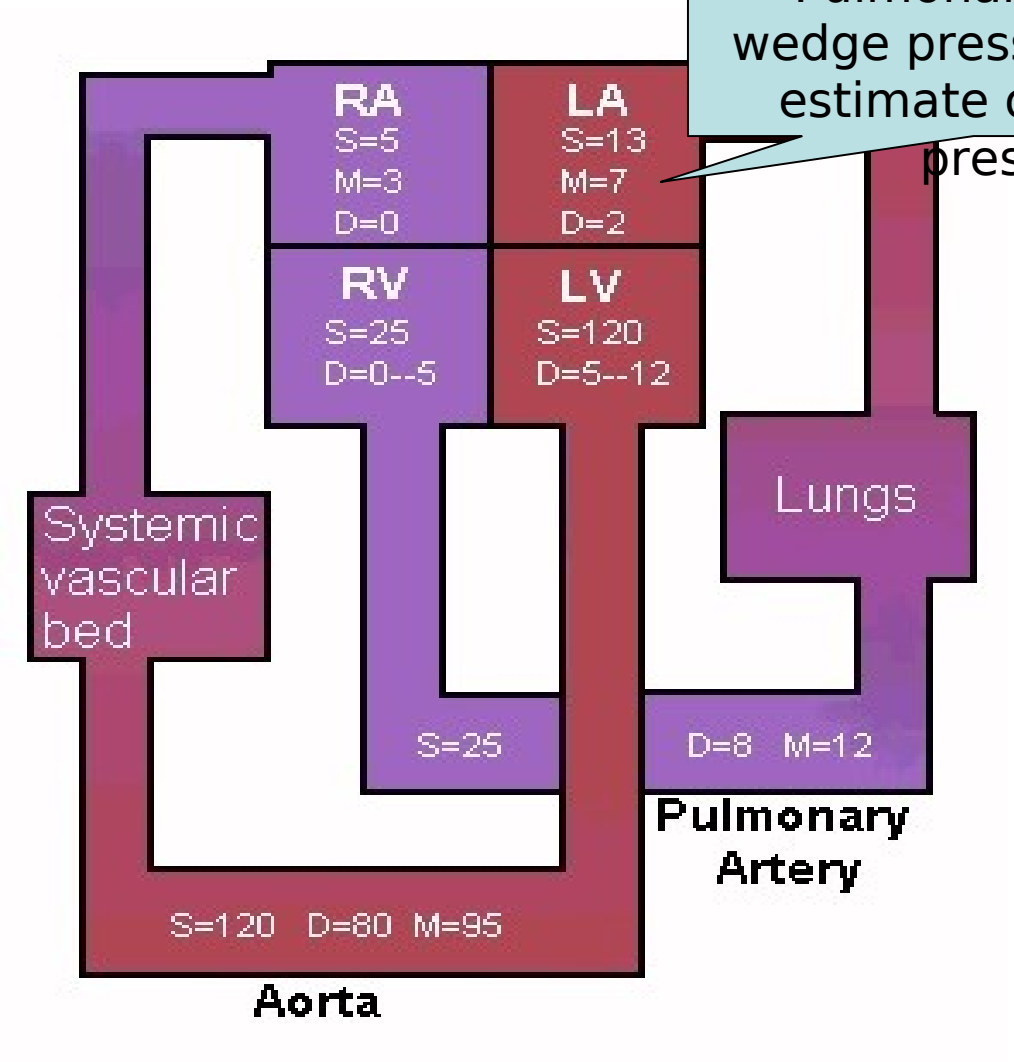
ABNORMAL CVP WAVE FORMS

ATRIAL FIBRILLATION	Loss of a waves, prominent c waves
A-V DISSOCIATION	Cannon a waves
TRICUSPID STENOSIS	Tall a wave, accentuation of y descent.
TRICUSPID REGURGITATION	Tall systolic cv waves, loss of x descent.
CONSTRICTIVE PERICARDITIS, RV ISCHEMIA	Tall a and v waves, steep x and y descent.
CARDIAC TAMPONADE	Dominant x descent, attenuated y descent.



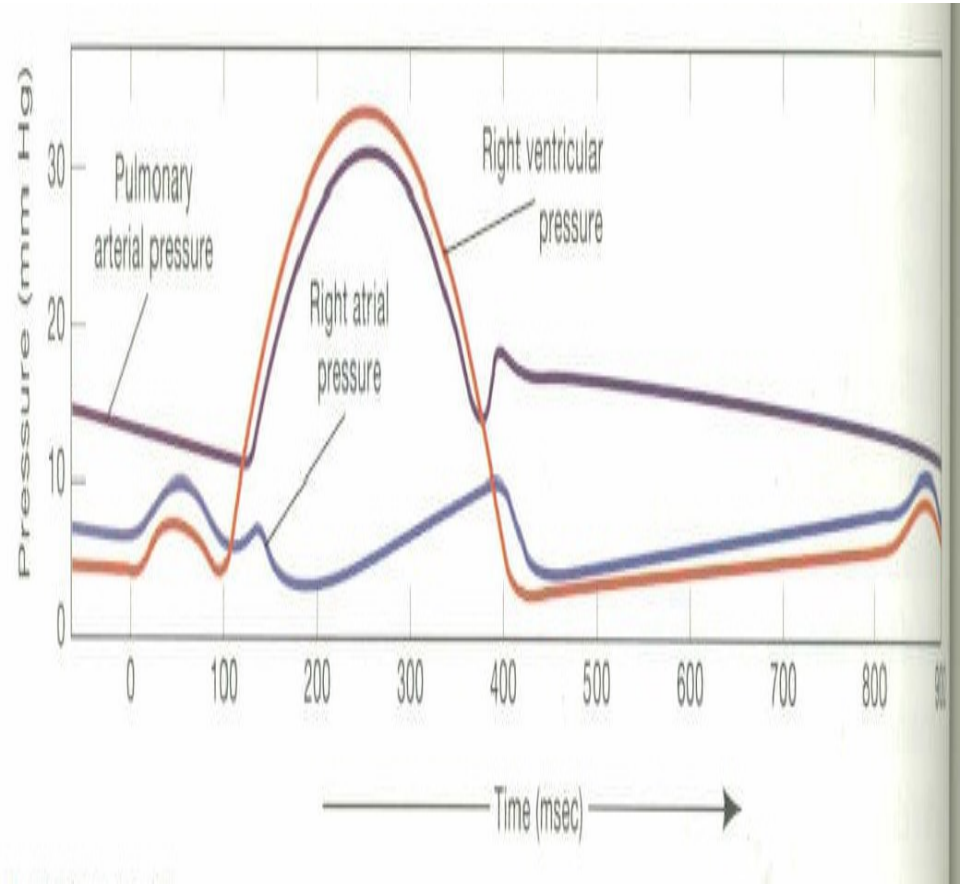
PRESSURES IN THE CHAMBERS

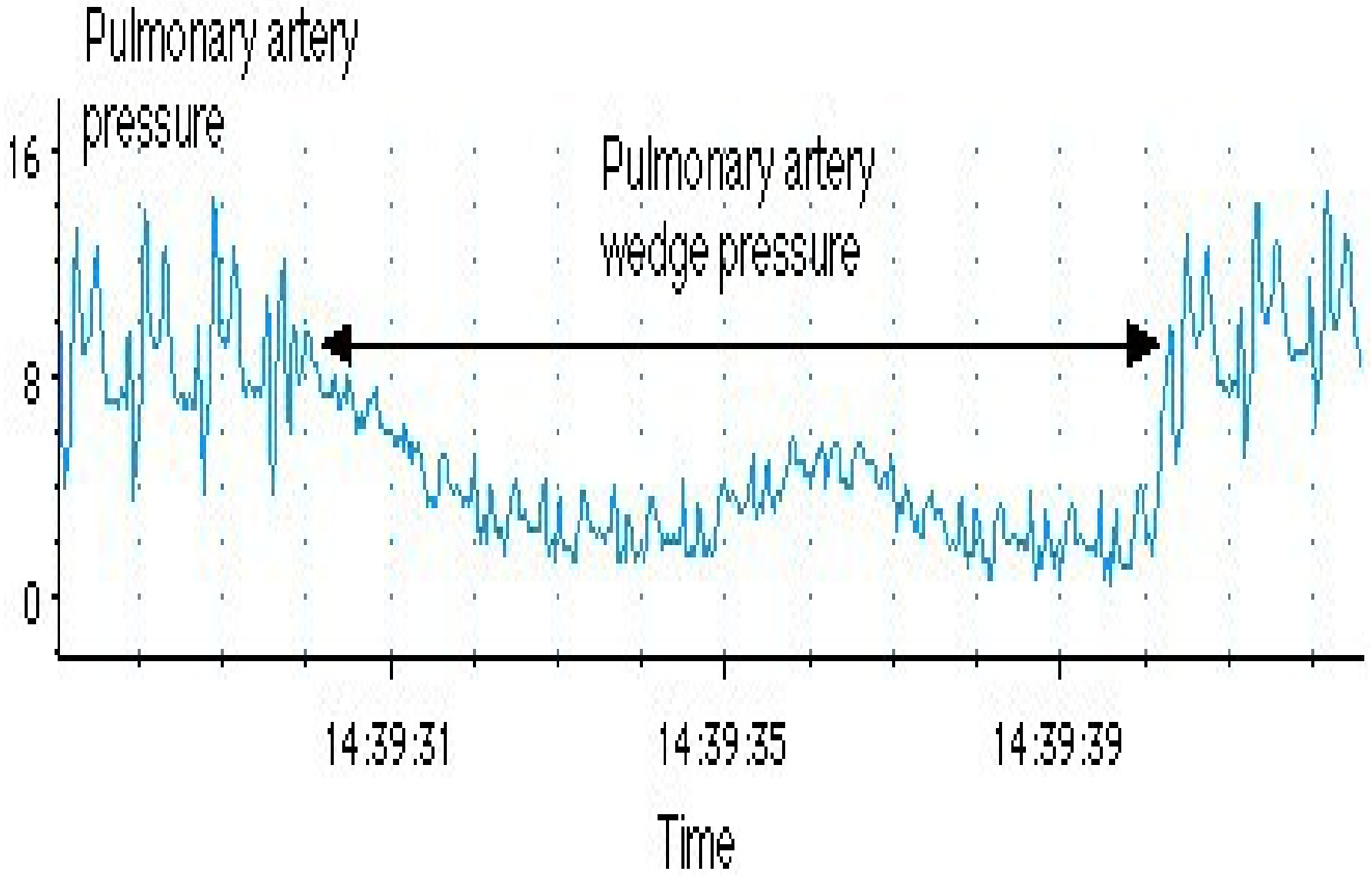
Pulmonary capillary wedge pressure gives an estimate of left atrial pressure.



LEFT ATRIUM
LEFT ATRIUM AND THE PCWP

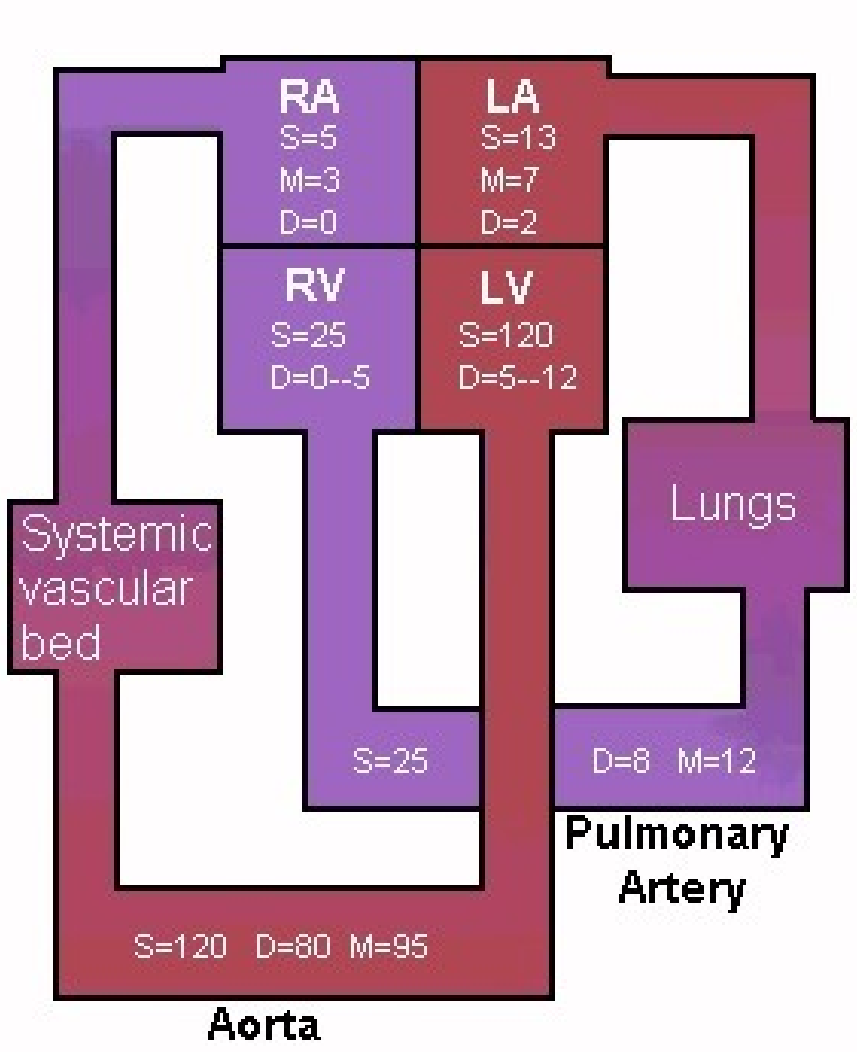
- Indirect measurement of the LAP. Measured by floating an end hole balloon catheter out through the pulmonary artery until the catheter occludes a small branch




Pulmonary artery pressure
Pulmonary artery wedge pressure
16
8
0
14:39:31
14:39:35
14:39:39
Time

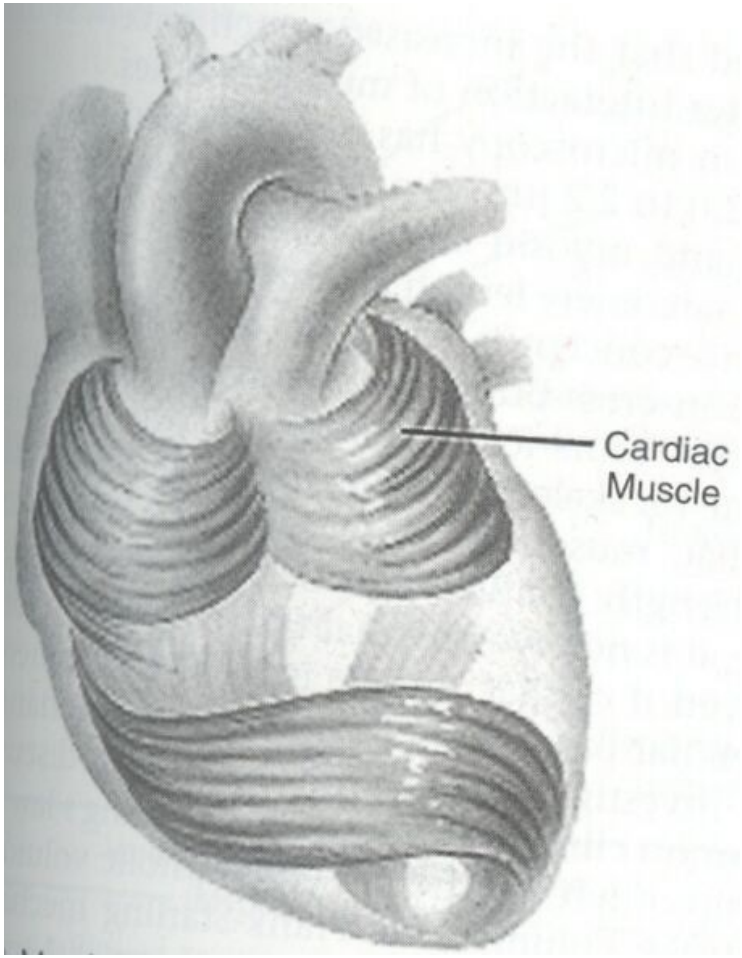
VENTRICLES

- RV:LV- in fetal life it is 1:1, at birth with fall in pulmonary artery pressure, it acquires an adult value of 1:2 by the first month of life.



Ventricular structure

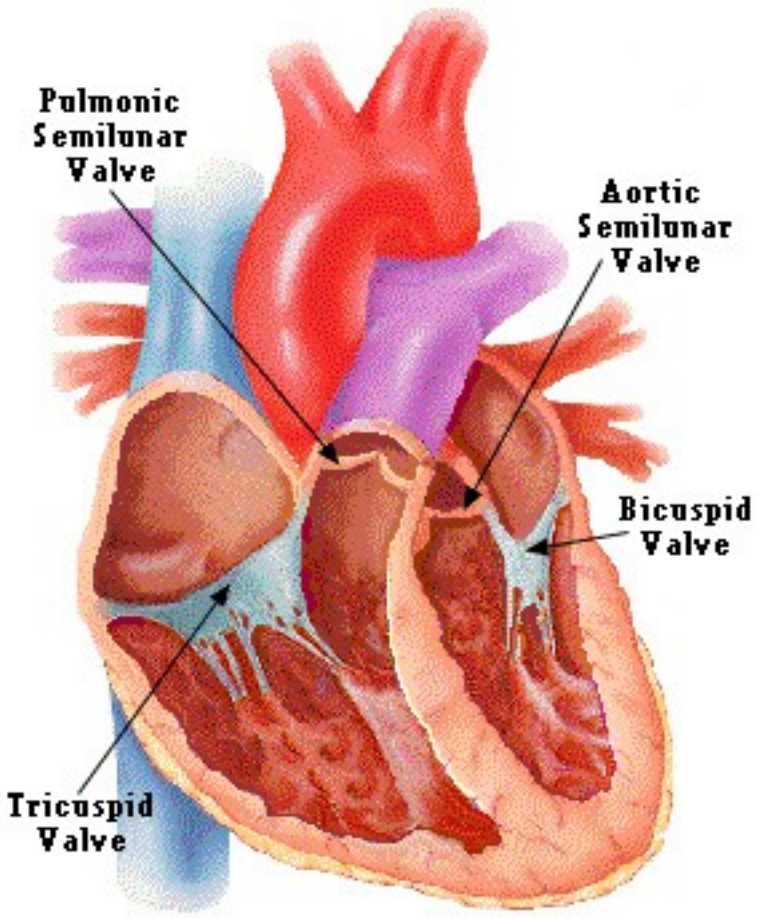
- LV ellipsoidal in shape – least wall stress.
- Laminar arrangement of the cardiac muscles.
- RV is crescent shaped, force generated thru the LV based septum.



VALVES OF THE HEART

Tricuspid valve:

- anterior, middle and posterior leaflets.
- 8-11 cm^2.
- Through chordae tendinae attached to papillary muscles which prevents prolapse of the valve into the RA.



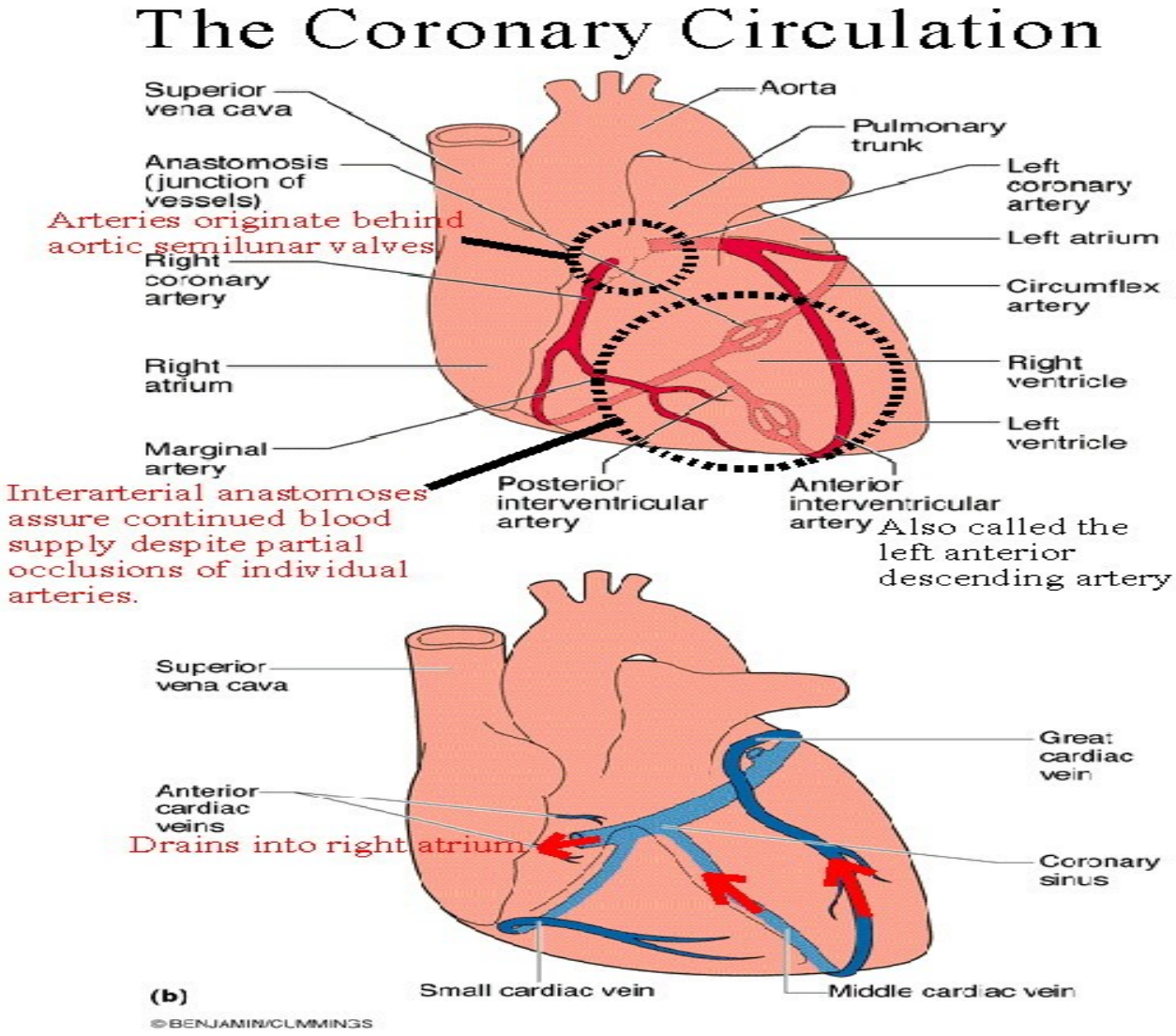
Mitral valve:

- Anterior and posterior leaflets.
- 4-6cm^2.
- Normal gradient:- <2mmHg.
- Flow:- 150-200 ml/sec/diastole.
- LVEDP<5mmHg.

Semilunar valves.

- Pulmonic valve: 4 cm^2, anterior, right and left cusps.
- Aortic valve: 3-4 cm^2, posterior, right and left cusps.
- Ensures unidirectional flow.
- Behind the cusps of the aorta are the sinuses of valsalva in which eddy currents are produced which prevents occlusion of the coronary ostia.

CORONARY CIRCULATION



SA node:-59% RCA.
38% LCA
AV node:- 90% RCA
10% LCA

DISTRIBUTION OF CORONARY CIRCULATION

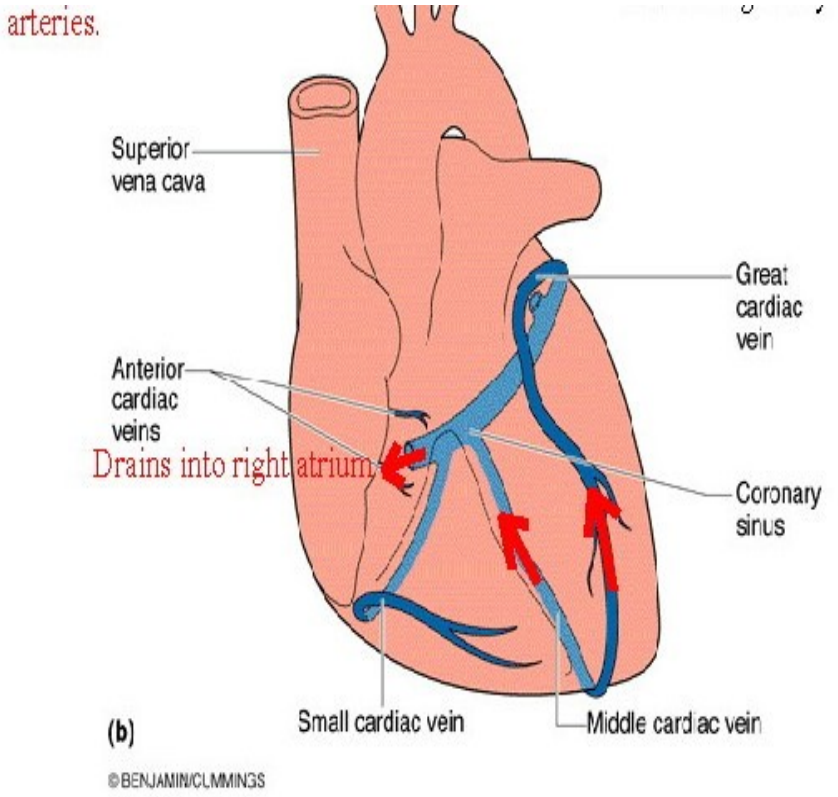
LEFT CORONARY ARTERY	• Ant descending branch. • Right bundle branch. • Left bundle branch. • Ant & post papillary muscle. • Ant lat left ventricle.
CIRCUMFLEX BRANCH	Lateral left ventricle.
RIGHT CORONARY ARTERY	• SA and AV nodes. • RA and RV • Post interventricular septum. • Inter atrial septum. • Post fascicle of LB

Occlusion in the….

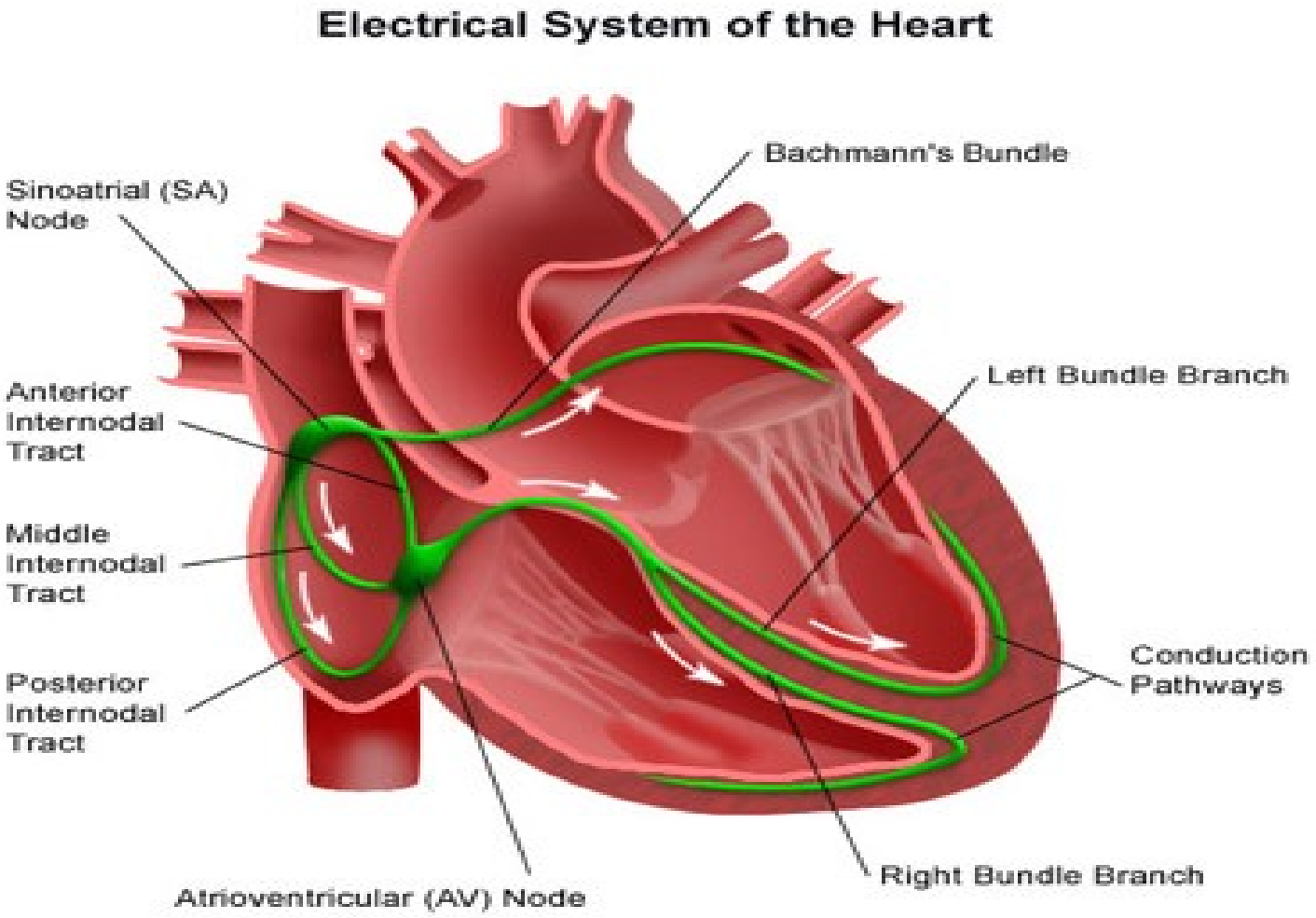
- Anterior descending artery: leads V_{3-5}.
- Left circumflex artery: leads I and aVl.
- Right coronary artery: leads II, III and aVF.

Venous drainage of the heart

- Coronary sinus:- drains the great cardiac vein, middle cardiac vein and the posterior cardiac vein.
- Anterior cardiac veins.
- Direct:- arterioluminal, arteriosinusoidal and thebasian veins.



Conduction system



Conduction speeds

Tissue	Conduction rate (m/s)
SA node	.05
Atrial pathways	1
AV node	.05
Bundle of His	1
Purkinje	4
Ventricular muscle	1

CARDIAC MYOCYTE

STRUCTURAL UNIT:-
SARCOMERE

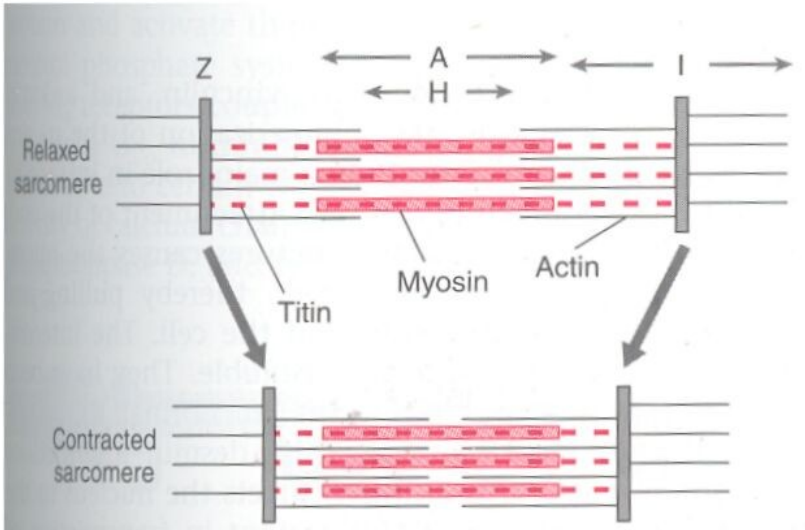
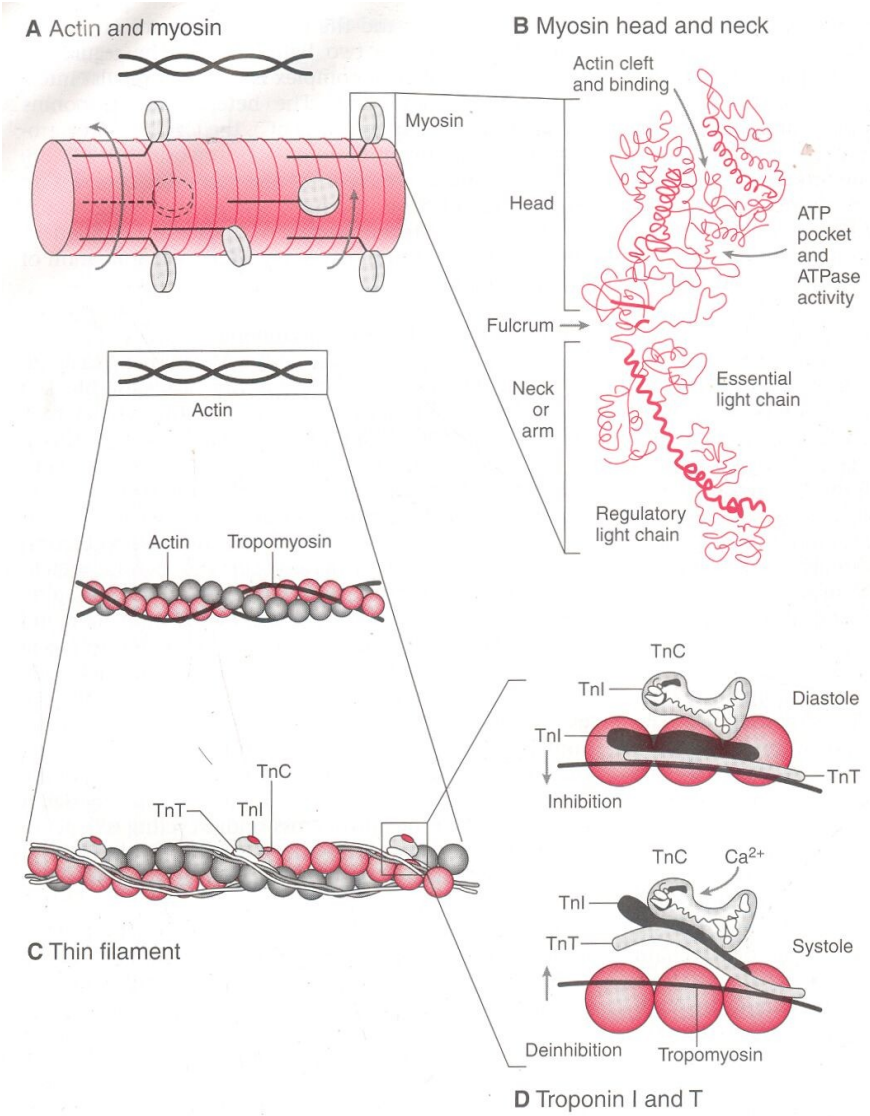


Figure 18–13 The basic unit for contraction is the sarcomere. A contracted and a relaxed sarcomere is depicted. Z lines are located at the ends of the sarcomere. The A band is the site of overlap between myosin and actin filaments. The I band is located on either side of the A band and contains only actin filament. The H zone is located in the center of the A band, and only myosin is present.

Contractile elements



- Thin filament:- actin.
- Thick filament:- myosin.

Cardiac myonecrosis and cardiac enzymes

Creatinephosphokinase (CPK):-

- It is first elevated 4-6 hours after symptom onset, peaks at 24 hours, and returns to baseline 48-72 hours.
- Lacks specificity for STEMI as increased in muscle trauma as well.
- CPK MB isoenzyme has the advantage of being more specific.
- ***MB index %*** (MB divided by total CK x 100) - greater than 2.5% is suggestive but not diagnostic of AMI - both MB and total CK must both be elevated for an MB index to be considered elevated.

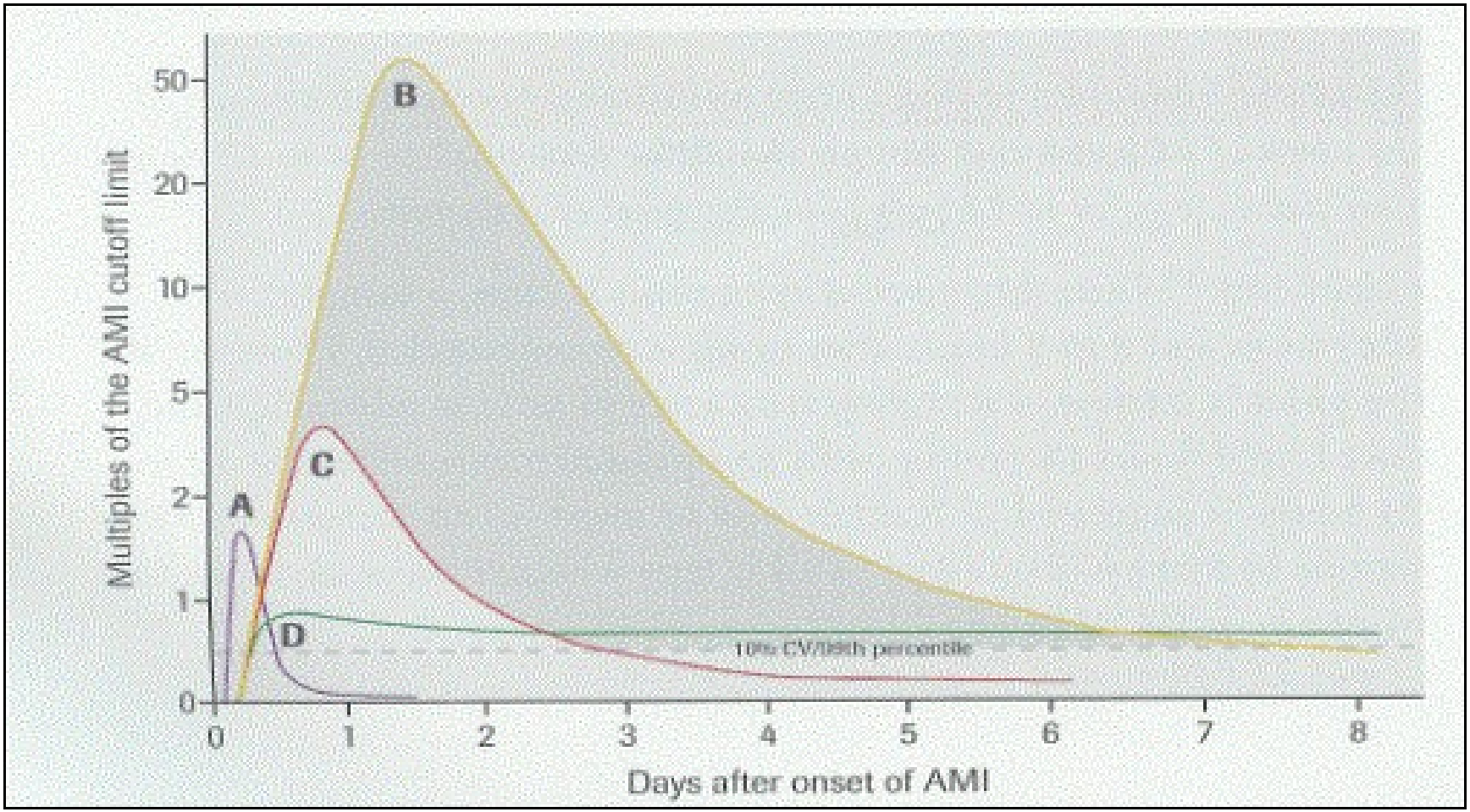
Troponin

- There are 3 subforms (TnI, TnT, TnC) of this molecule that are all muscle components – two have distinctly cardiac forms (cTnI, cTnT). Cardiac troponins are more sensitive and can detect myocardial damage even if CK and MB isoenzymes are not elevated.

- The bulk of troponin is released from muscle during myocardial necrosis. Troponins are also partially dissolved in the cytosol of the myocardial cell (2% of total) and can leak very small amounts even with reversible damage (ischemia) to the cell like ischemia. In infarction, levels rise within 6-8 hours and stay elevated for up to 10 days.

- ***Myoglobin*** is a storage molecule of oxygen in muscle. It, is the earliest marker of MI and the first marker to clear.
- Rises within 2-4 hours of infarction, peaks at 6-12 hours, returns to normal within in 24-36 hours
- LDH on the other hand appears 12 hours after the infarct and peaks 2 days later to last 14 days.

Cardiac enzyme release pattern. (A = myoglobin, B = troponin after STEMI, C = CK-MB, D = troponin after NSTEMI)



Cardiac Physiology -I

Physiology of Cardiac Contraction

& Cardiac Cycle

Pressure Volume Loop

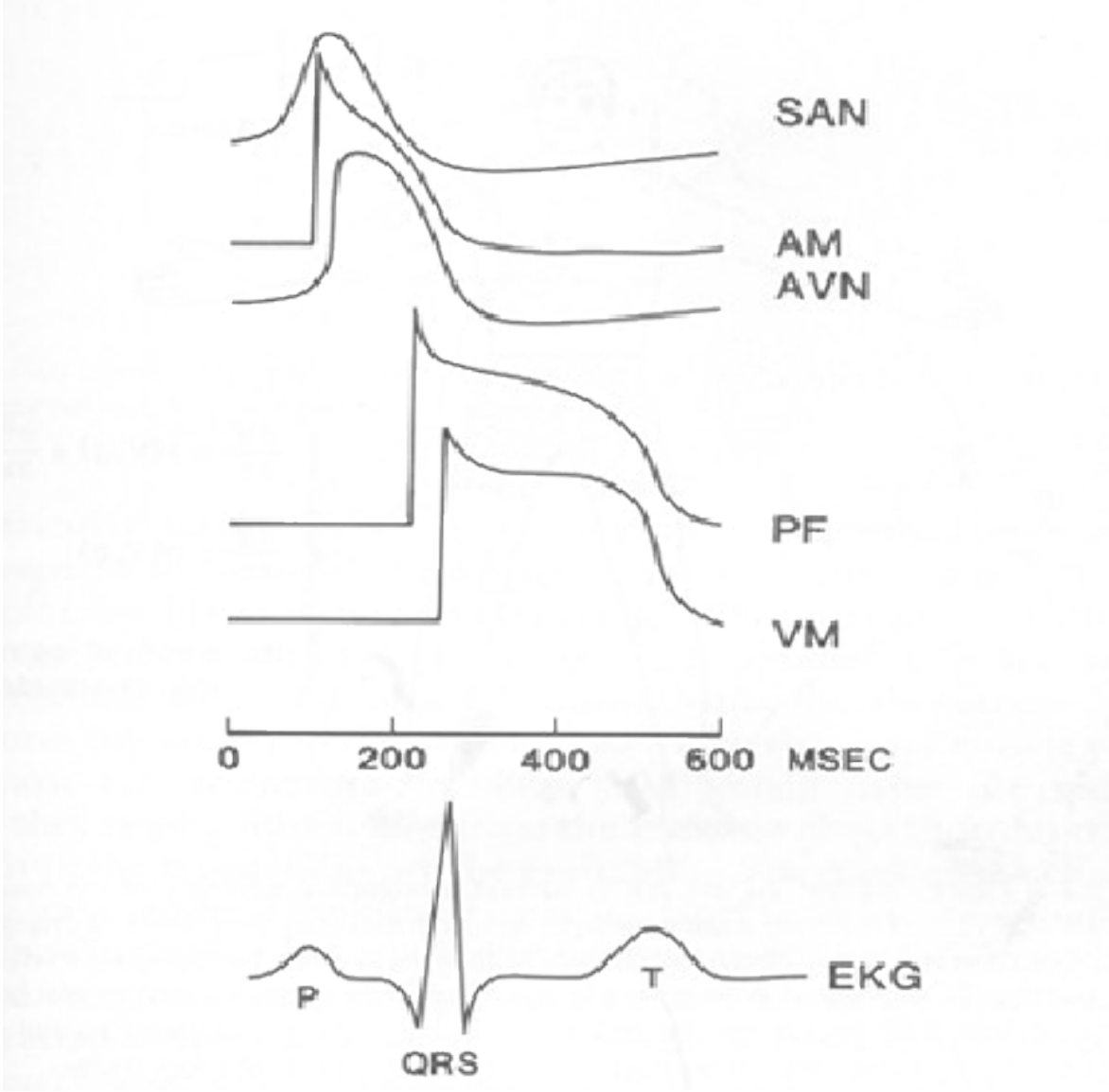
Determinants of Cardiac Output

Frank Starling Law

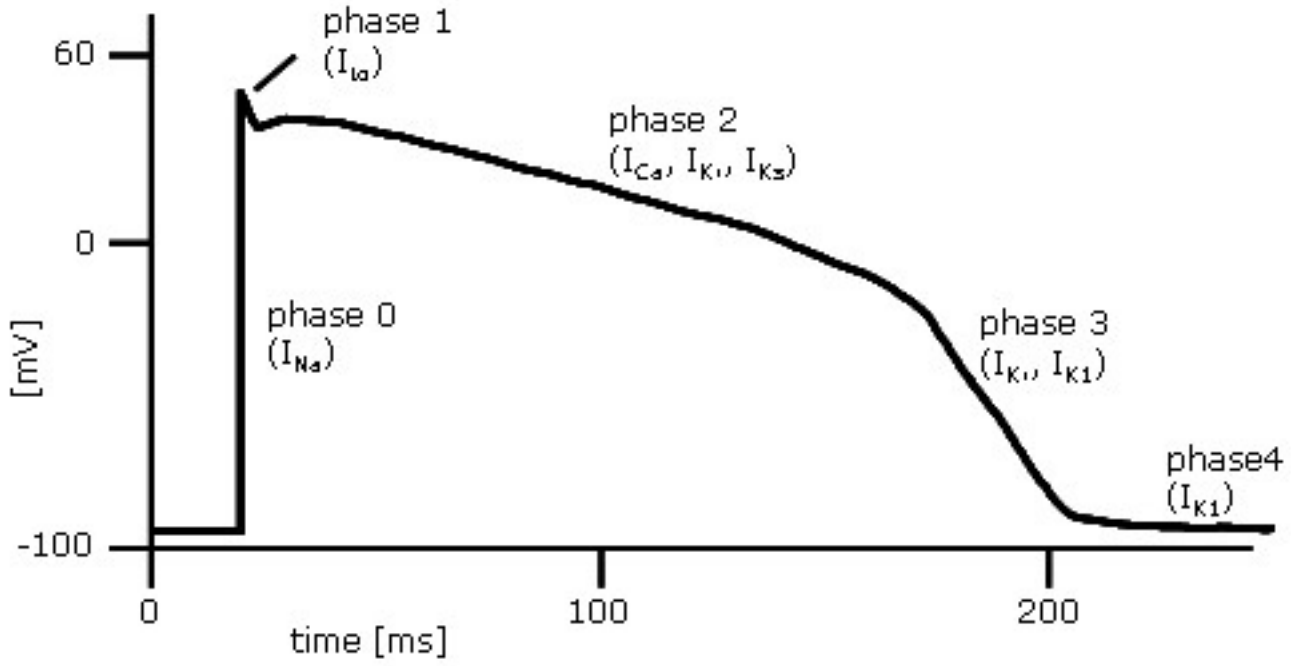
Heart as a PUMP

- Excitability (BATHMOTROPIC)
- Conductivity (DROMOTROPIC)
- Rate & Rythmicity
  (CHRONOTROPIC)
- Contractility (INOTROPIC)
- Relaxation (LUSITROPIC)

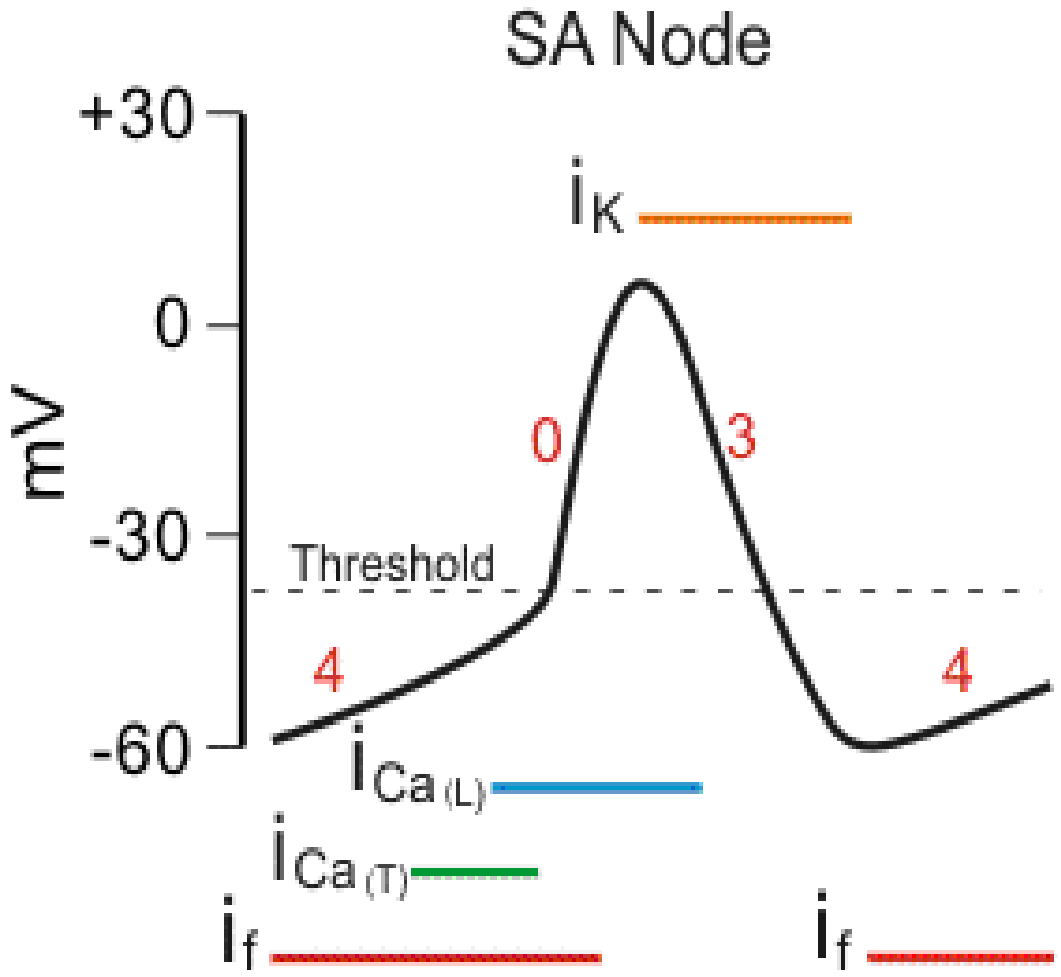
Action Potentials



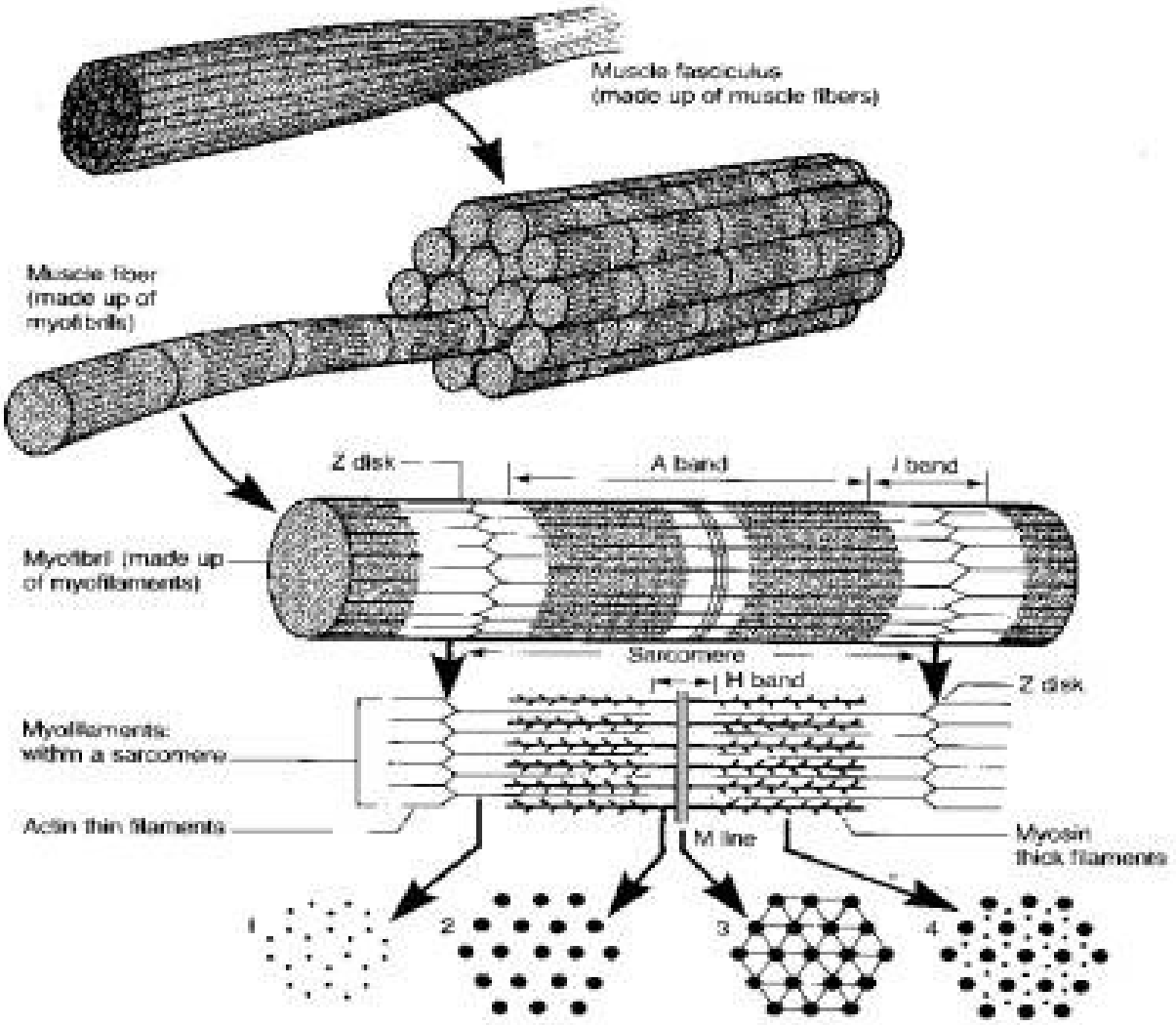
Cardiac Myocyte Action Potential



Pacemaker Action Potential



Cardiac Ultrastructure



Excitation Contraction Coupling

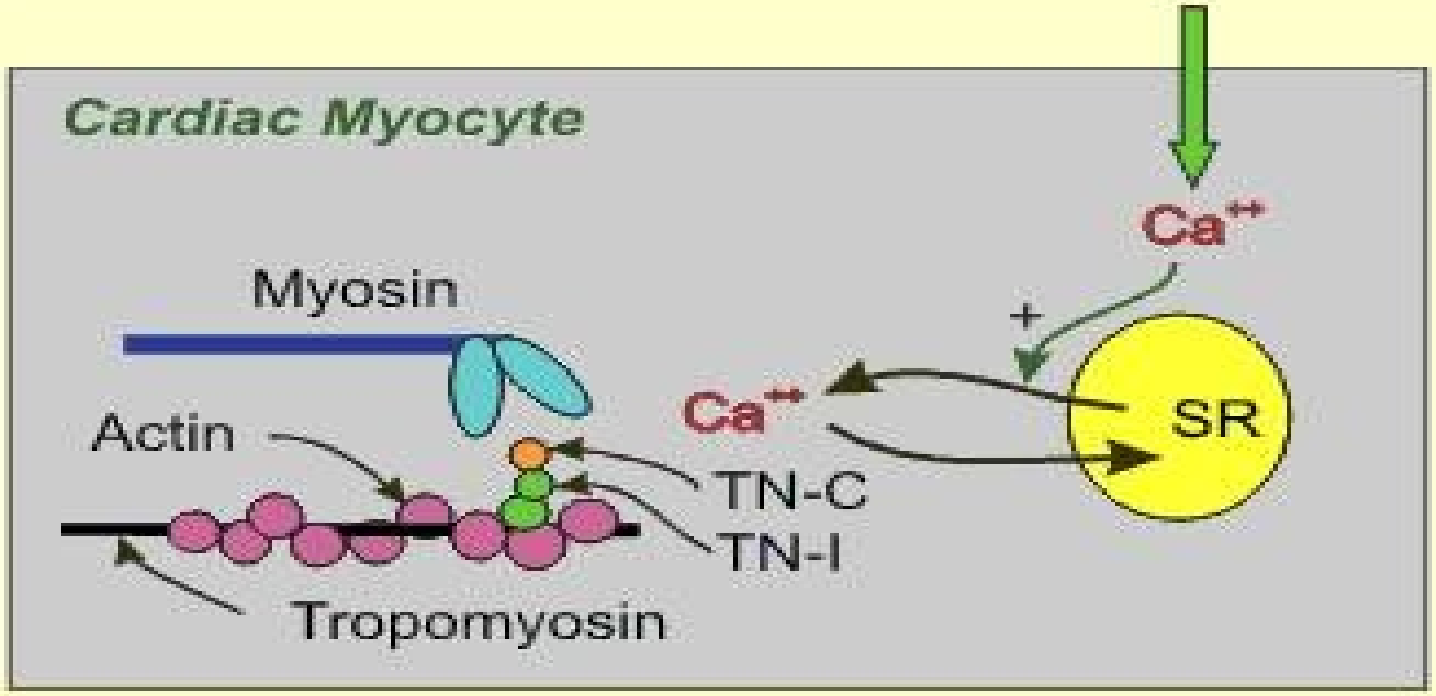
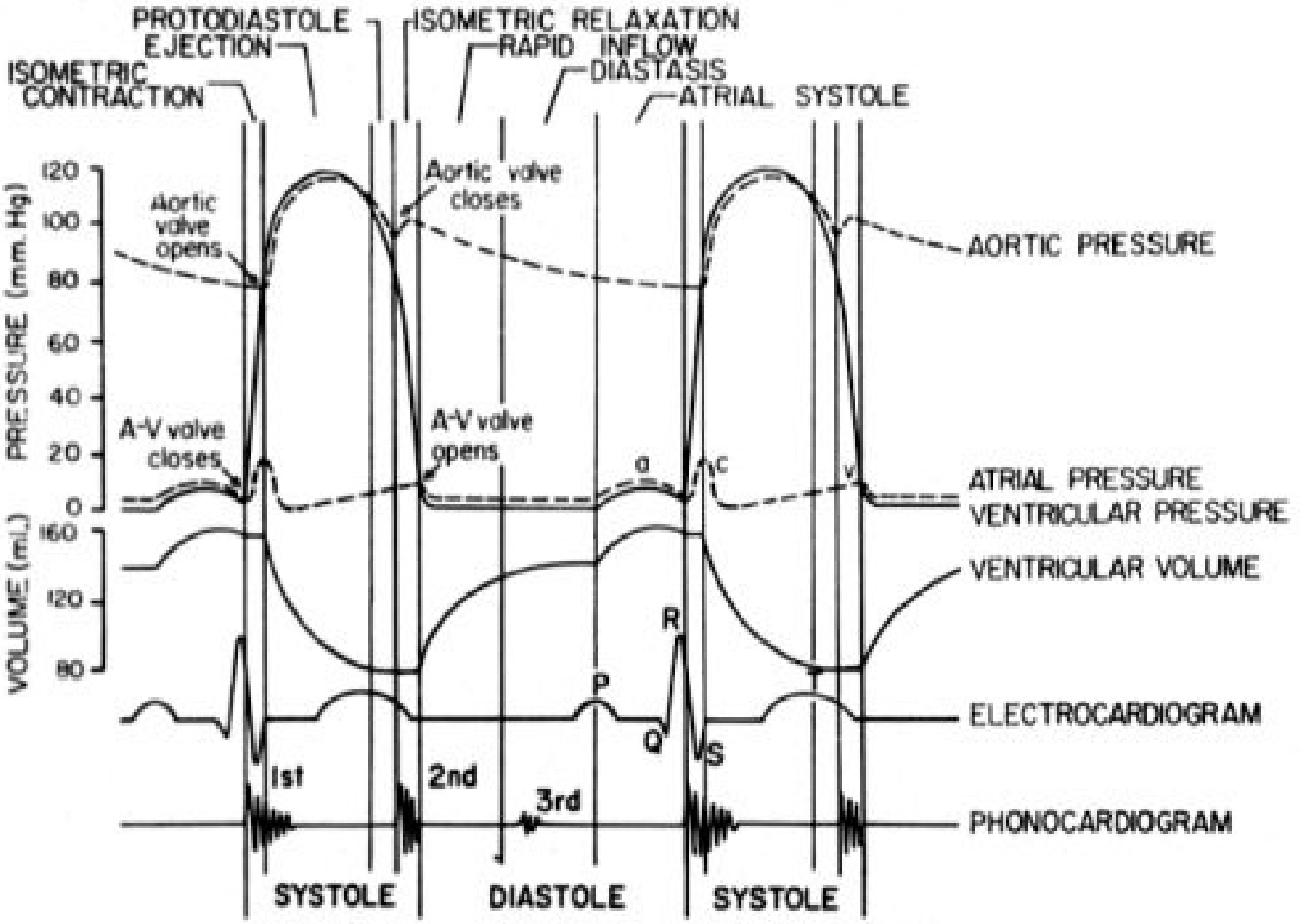
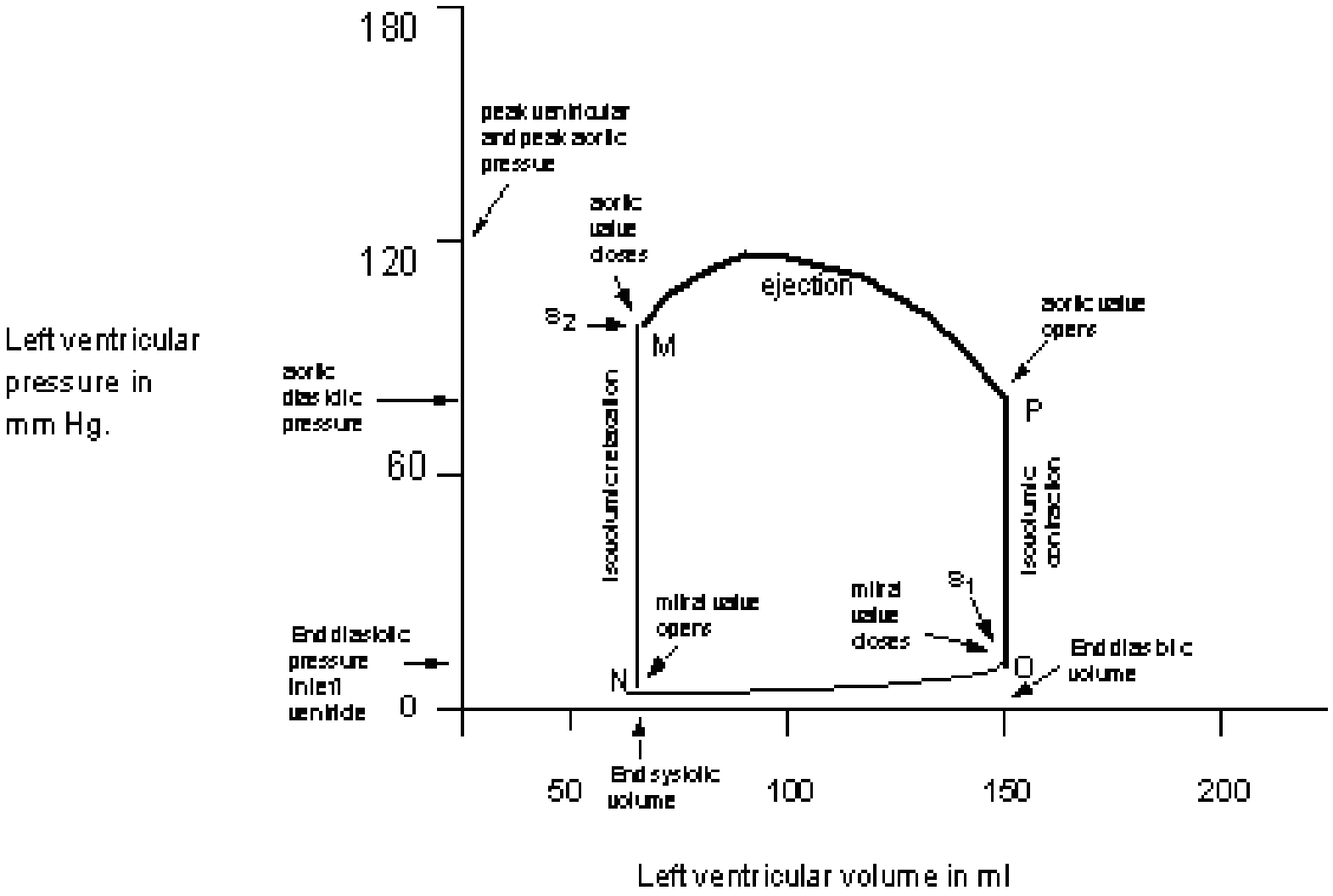


Figure 2. Cardiac myofilaments. Myosin (thick filament) contains two heads having ATPase activity. Thin filament is made up of actin, tropomyosin, and troponin (TN). TN-C binds Ca^{++} released by the sarcoplasmic reticulum (SR). TN-I inhibits actin-myosin binding until Ca^{++} binds to TN-C.

CARDIAC CYCLE



Pressure Volume Loop



End Diastolic PV Relationship

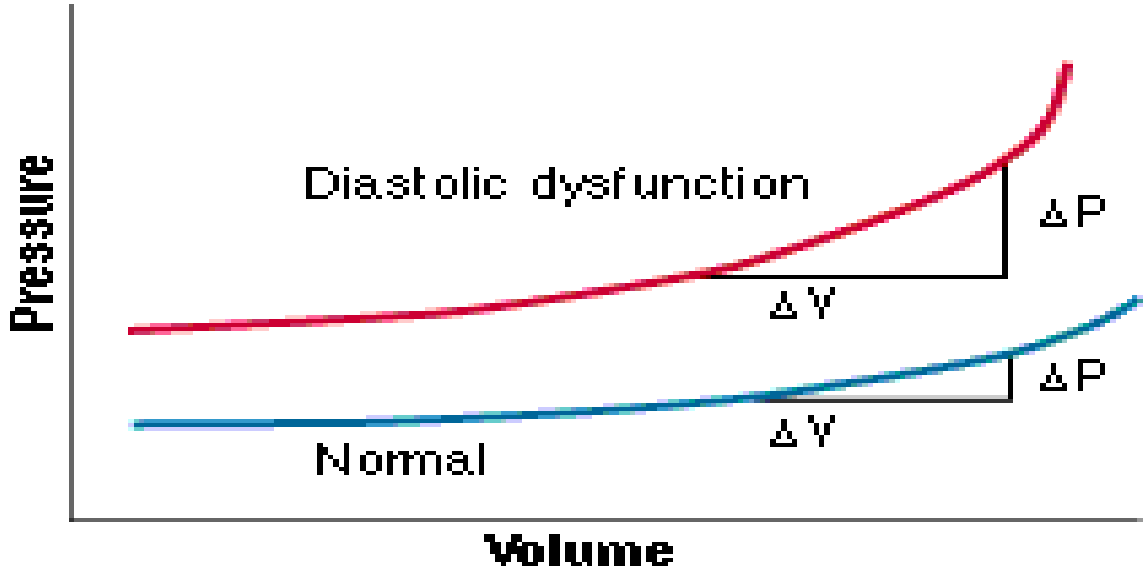
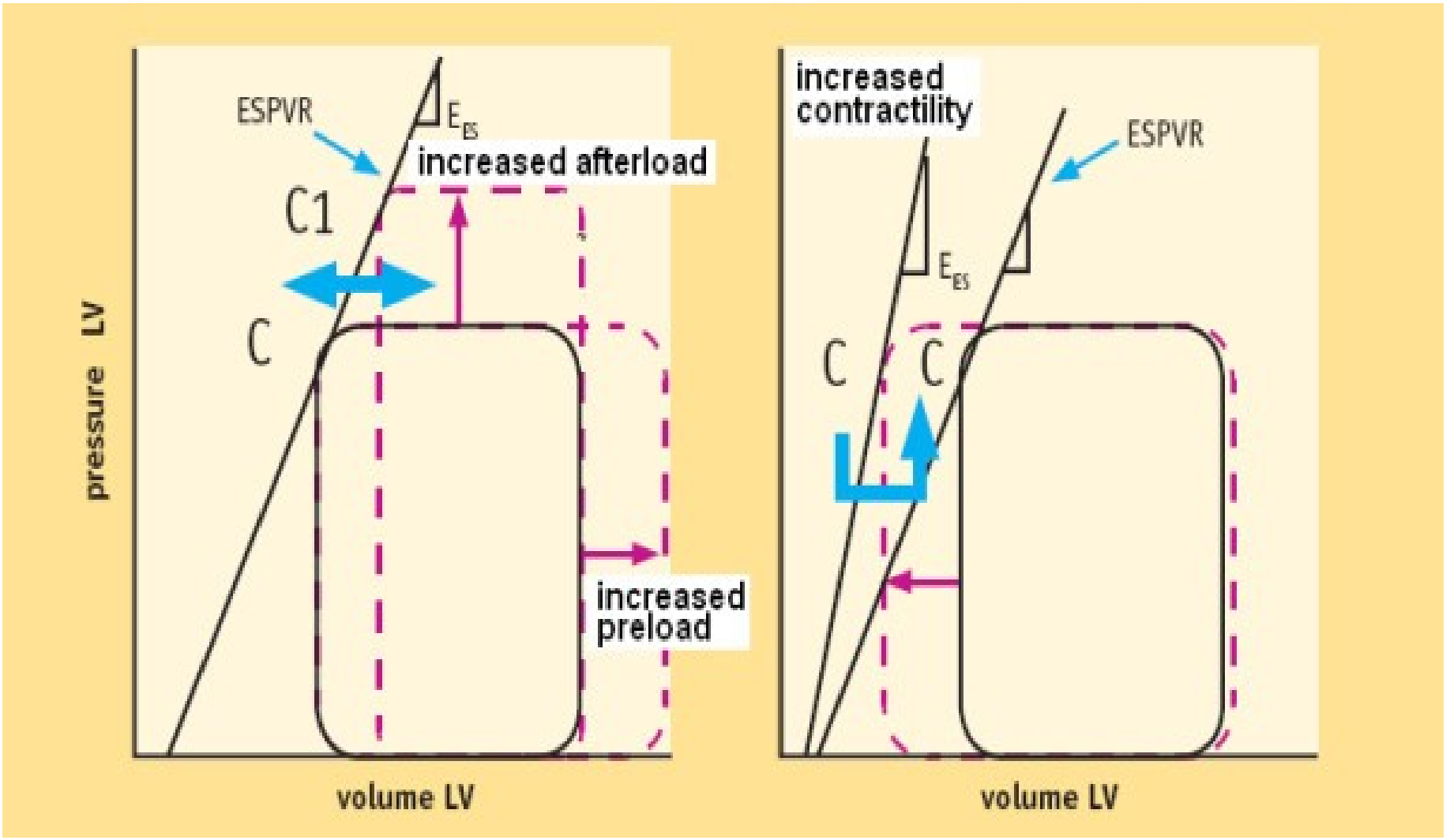


Figure 1. Left ventricular diastolic pressure-volume relationship. Upper curve represents diastolic filling with abnormal diastolic function. Note that for a given left ventricular volume, diastolic pressure is always higher. In addition, the change in pressure for a given change in volume is much greater. The symbol Δ P means pressure change; Δ V means volume change.

End Systolic PV Relationship



Parameters Derived

- Stroke Volume (SV)
- Cardiac Output = SV x HR
- Cardiac Index = CO/BSA
- Cardiac Reserve = $CO_{exercise} - CO_{rest}$
- Stroke Index = SV/BSA
- Ejection Fraction = EDV-ESV/EDV x 100 (%)

- Ventricular Stroke Work(gm.mtr) = SV x MAP
- Ventricle Stroke Power(watt)= SV x MAP x 100/Duration of systole.
- Mean Ejection Rate = SV/ Dur’n of systole

Determinants of Cardiac Output

- Heart Rate
- Stroke Volume

-Preload

-Afterload

-Contractility

-Wall motion abnormalities

-Valvular dysfunction

HEART RATE

- Heart rate = 118(beats/min) -0.57 x Age(yr)
- Normal HR = 60-100 /min
- Cardiac Output is directly proportional to Heart Rate
- Heart Rate may affect stroke volume

PRELOAD

Factors Affecting Preload

a) Venous return

Venous tone , valves

Blood volume

Posturing

Intrathoracic pressure

Heart Rate

Rhythm

- b) Ventricular Compliance

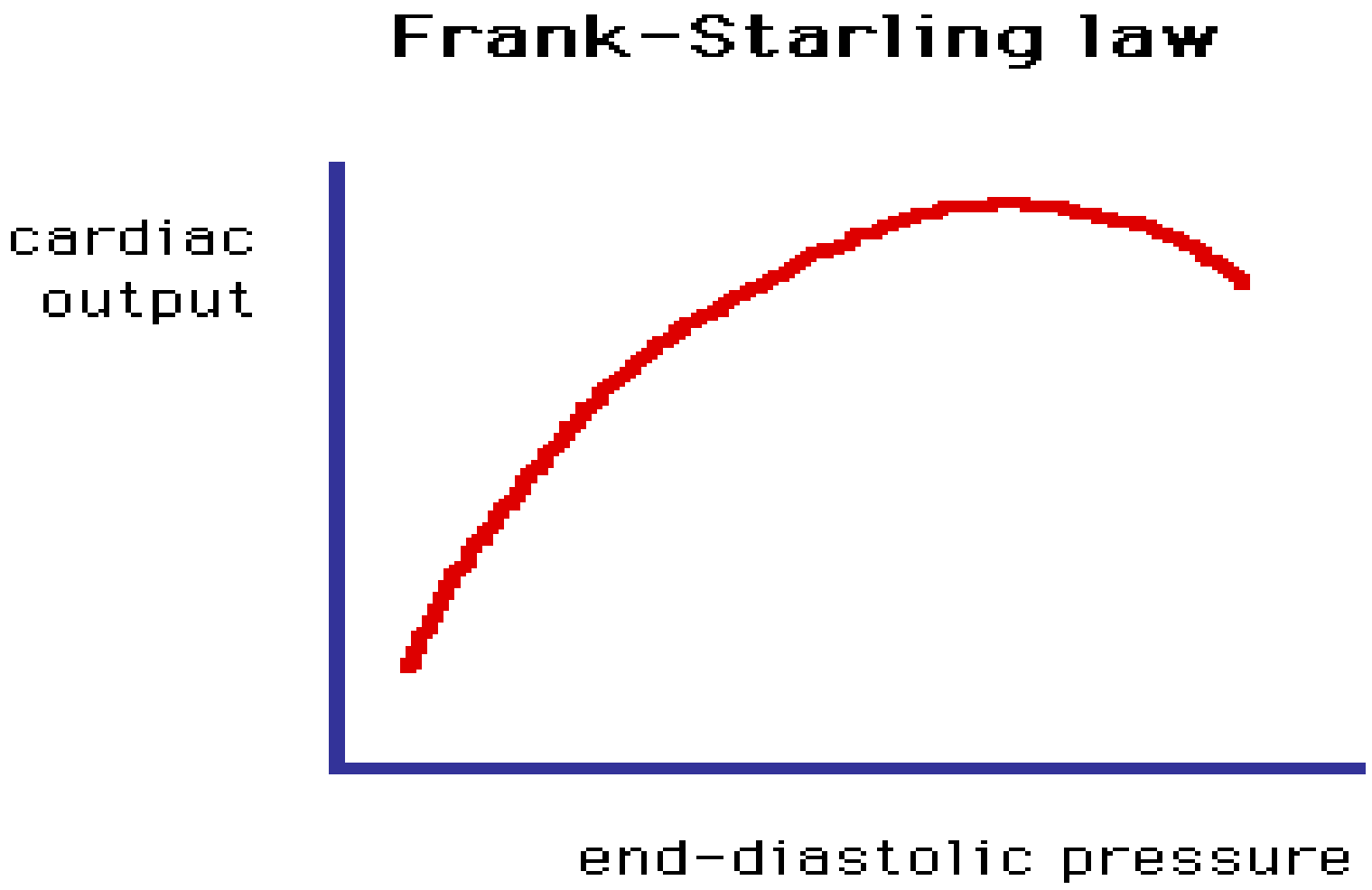
Hypertrophy

Myocardial Infarction

Extrinsic Compression

Restrictive Cardiomyopathy

PRELOAD



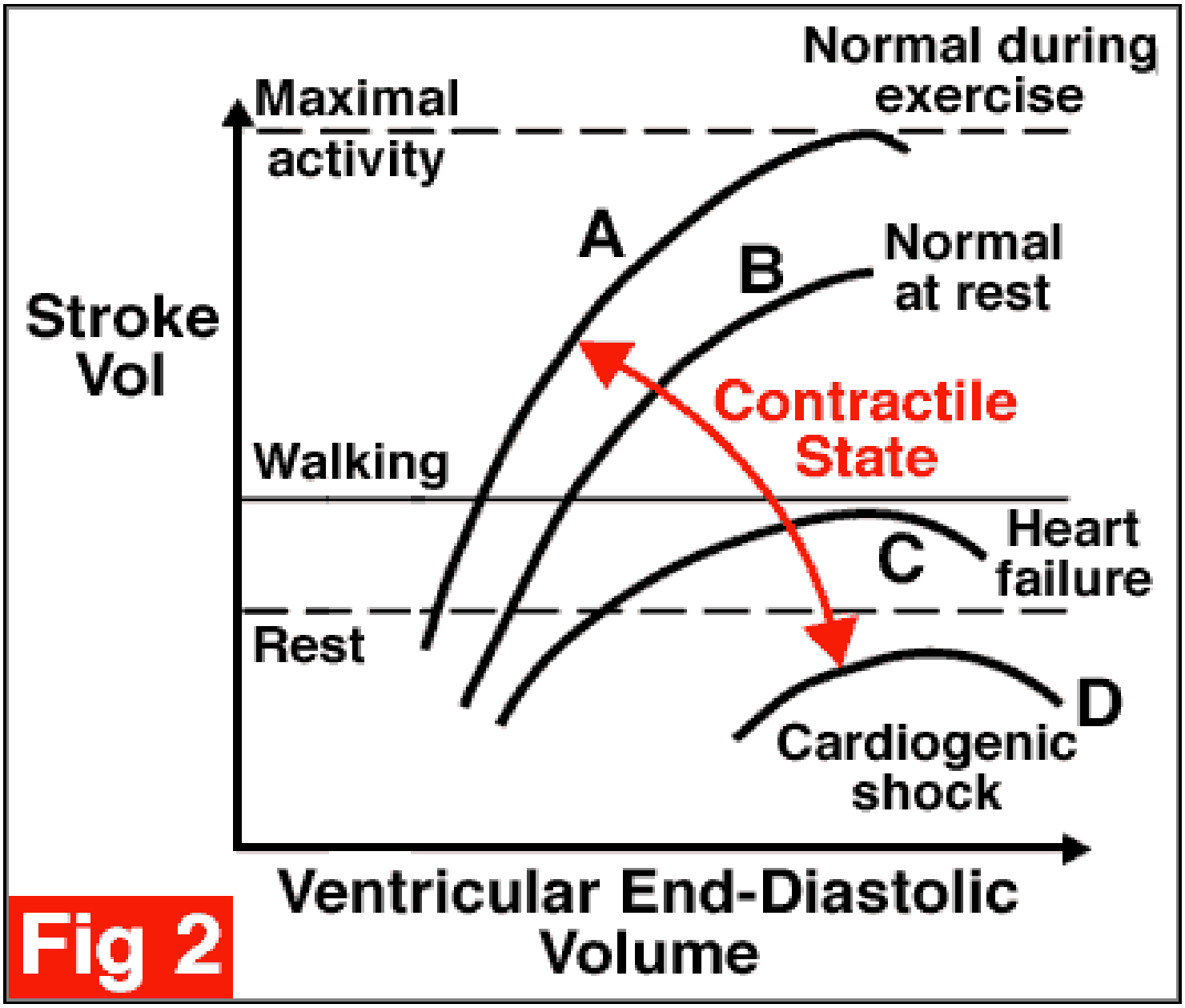
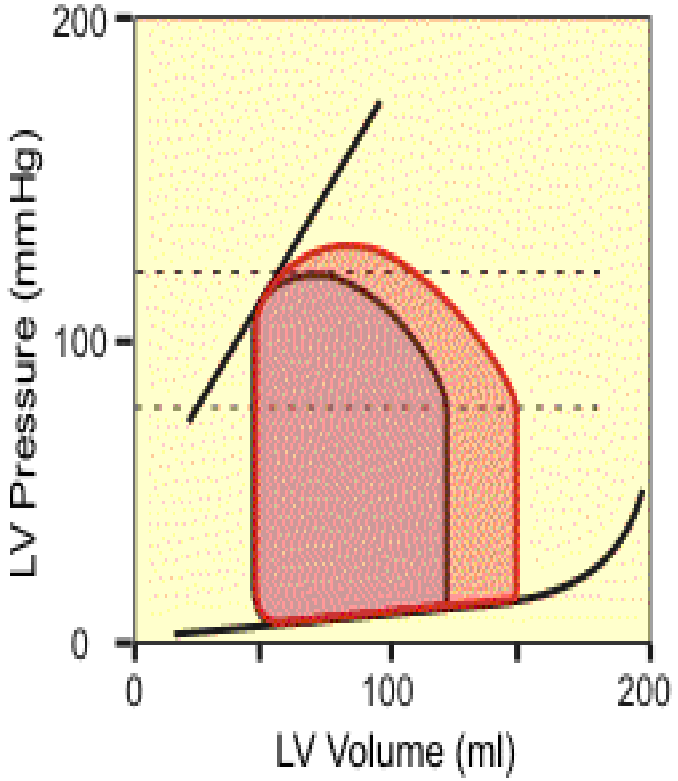
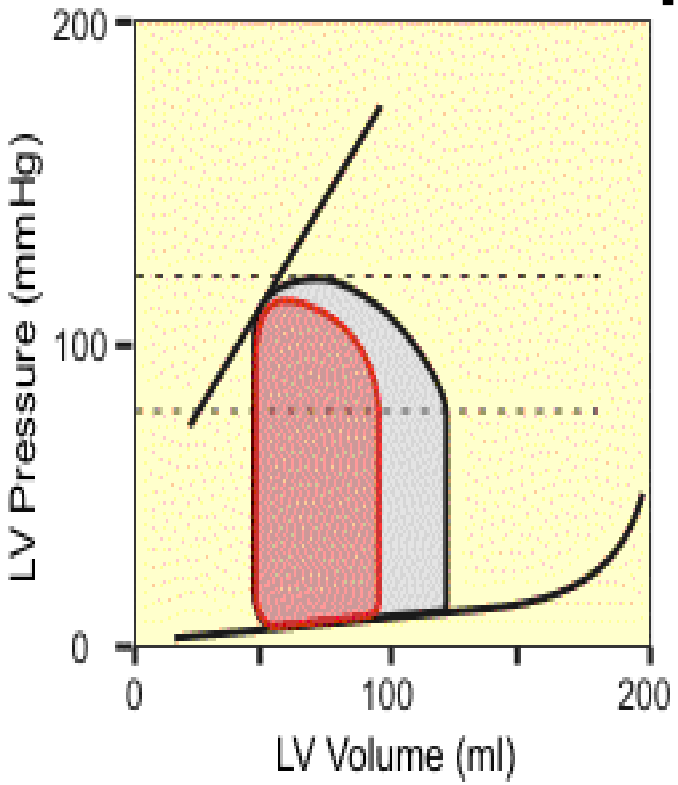

Normal during exercise
Maximal activity
A
B
Normal at rest
Stroke Vol
Contractile State
Walking
Heart failure
C
Rest
D
Cardiogenic shock
Ventricular End-Diastolic Volume

Fig 2

Effect of Preload on PV loop



Increased preload (increased EDV; red loop) at constant aortic diastolic pressure and inotropy. SV increases and ESV remains unchanged; EF increases slightly. Dashed lines represent normal aortic systolic and diastolic pressures.



Decreased preload (decreased EDV; red loop) at constant aortic diastolic pressure and inotropy. SV decreases and ESV remains unchanged; EF decreases slightly. Dashed lines represent normal aortic systolic and diastolic pressures.

AFTERLOAD

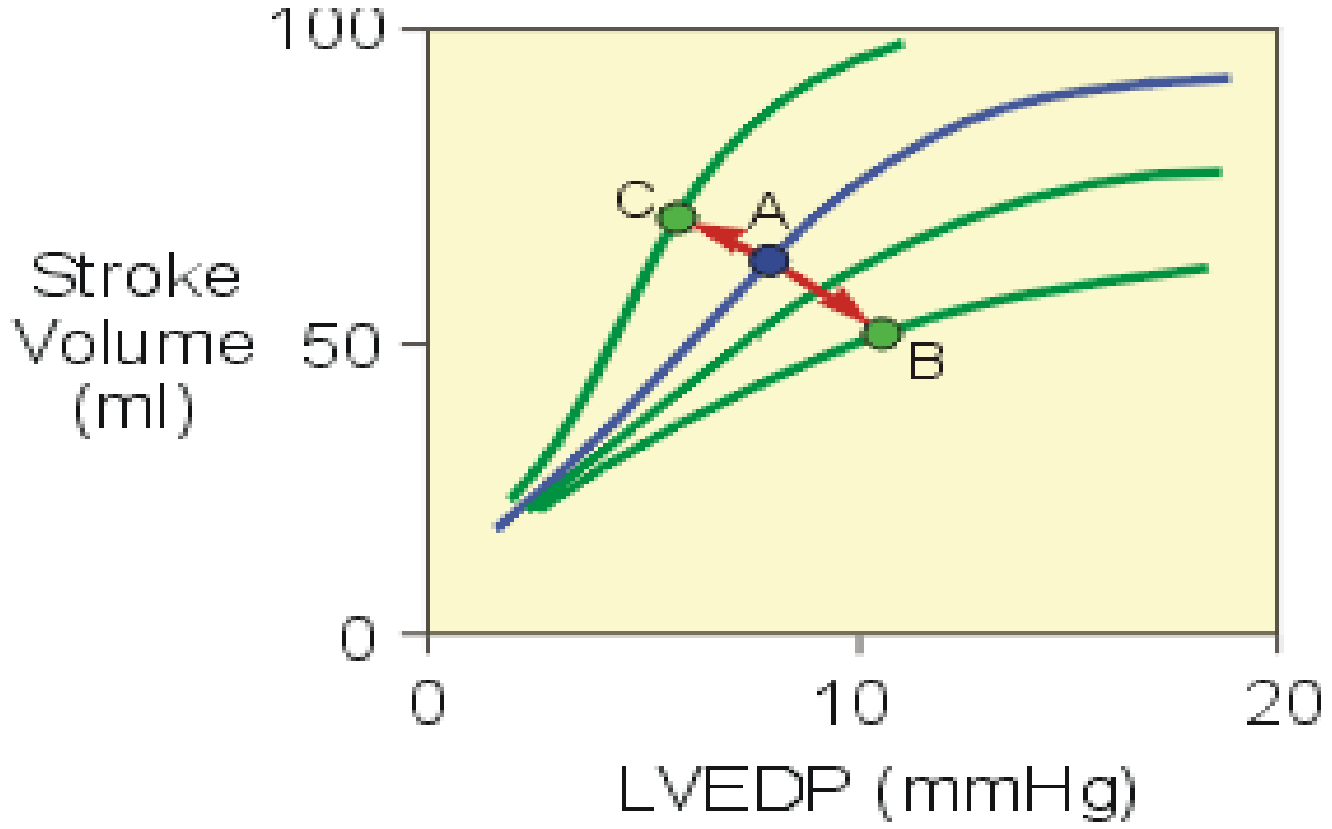
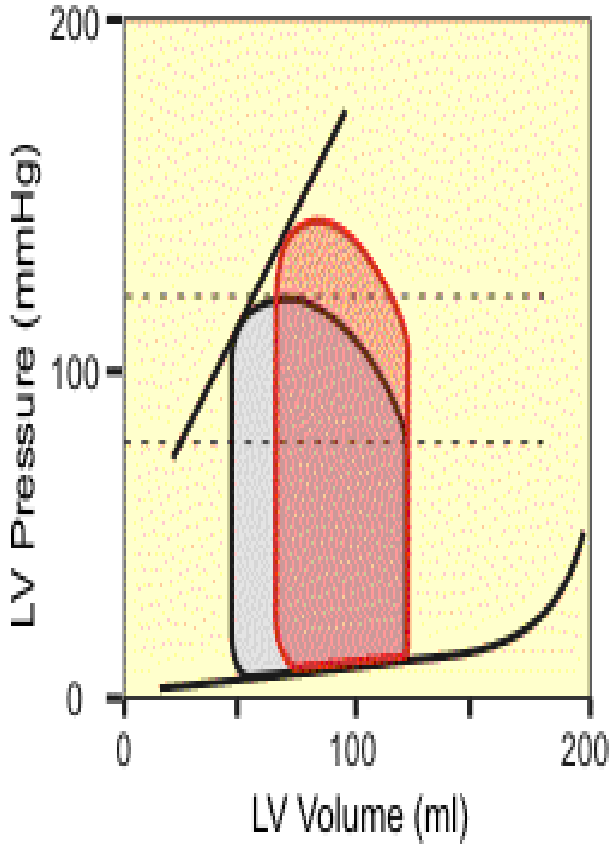
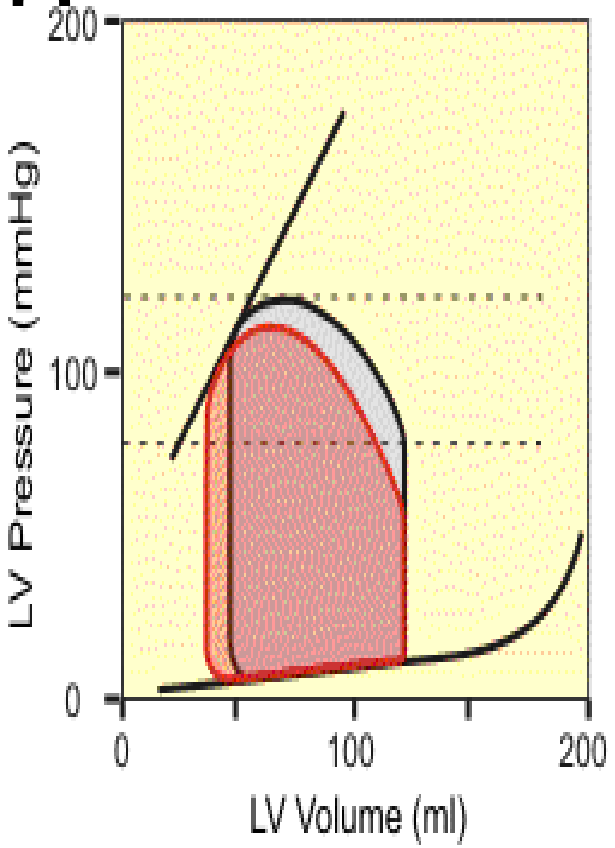


Figure 1. Effects of changes in afterload on Frank-Starling curves. A shift from A to B occurs with increased afterload, and from A to C with decreased afterload.

Effect of Afterload on PV loop



Increased afterload (increased aortic pressure; red loop) at constant preload (EDV) & inotropy. SV decreases and ESV increases; EF decreases. Dashed lines represent normal aortic systolic and diastolic pressures.



Decreased afterload (decreased aortic pressure; red loop) at constant preload (EDV) & inotropy. SV increases and ESV decreases; EF increases. Dashed lines represent normal aortic systolic and diastolic pressures.

CONTRACTILITY

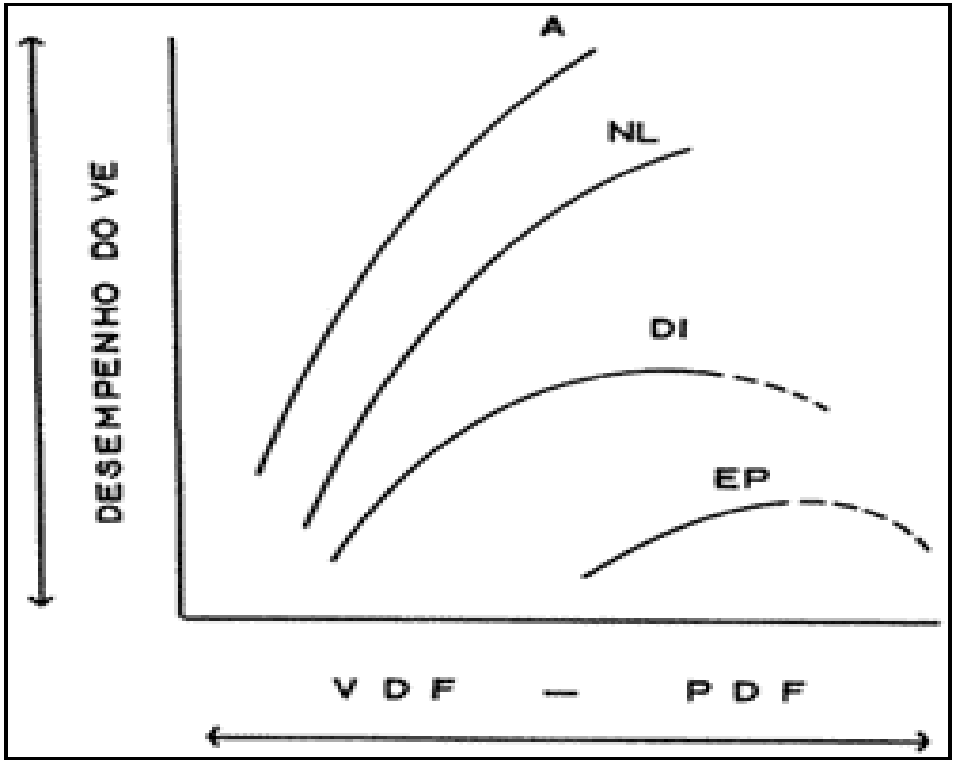
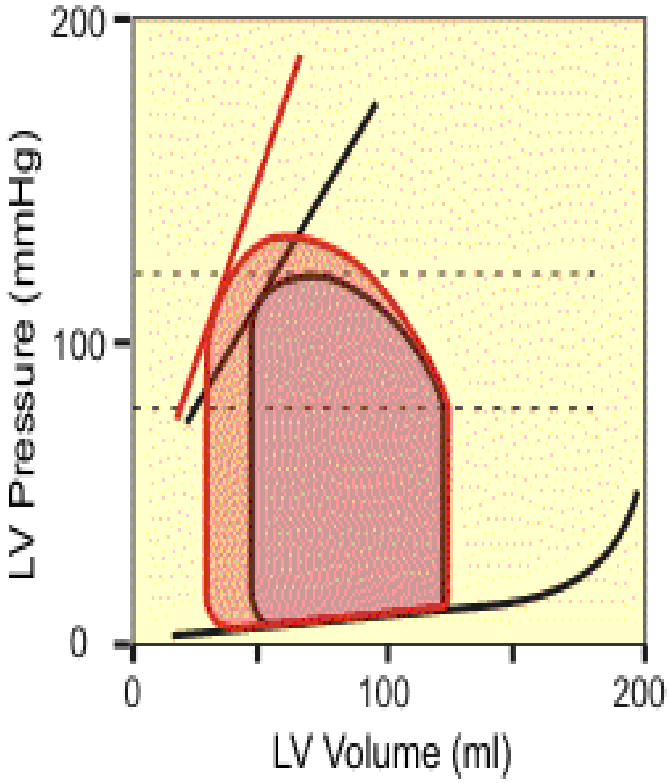
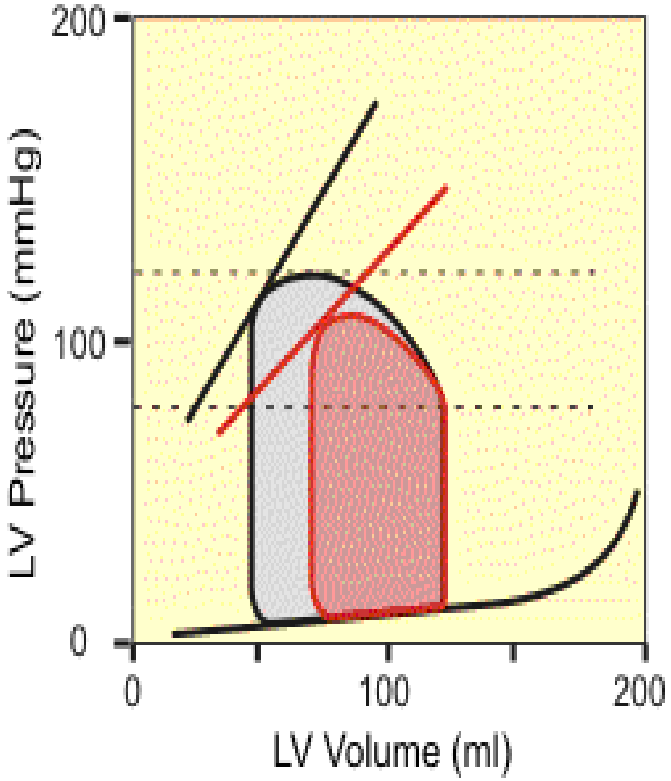


Fig. 4 - Ventricular function curves relate cardiac performance (stroke volume, cardiac output, systolic ventricular work) with the distension of the myocardium (end diastolic volume - VDF, end diastolic pressure - PDF or Pd_2). Increase in contractility (A) means a curve deviation to the left of normal curve (NL), while decrease in contractility (DI) means a deviation to the right. An extremely depressed left ventricular function means a poor performance with a very large distension (dilation), producing pulmonary edema (EP). Delineated lines represent a possible descending phase of Frank-Starling's curve [1].

Effect of Inotropy on PV loop



Increased inotropy (red loop) at constant preload (EDV) & aortic diastolic pressure. SV increases and ESV decreases; EF increases. Dashed lines represent normal aortic systolic and diastolic pressures.



Decreased inotropy (red loop) at constant preload (EDV) & aortic diastolic pressure. SV decreases and ESV increases; EF decreases. Dashed lines represent normal aortic systolic and diastolic pressures.

Wall Motion Abnormality

- Hypokinesis
- Akinesis
- Dyskinesis

Valvular Dysfunction

- Stenosis of mitral /tricuspid valve→

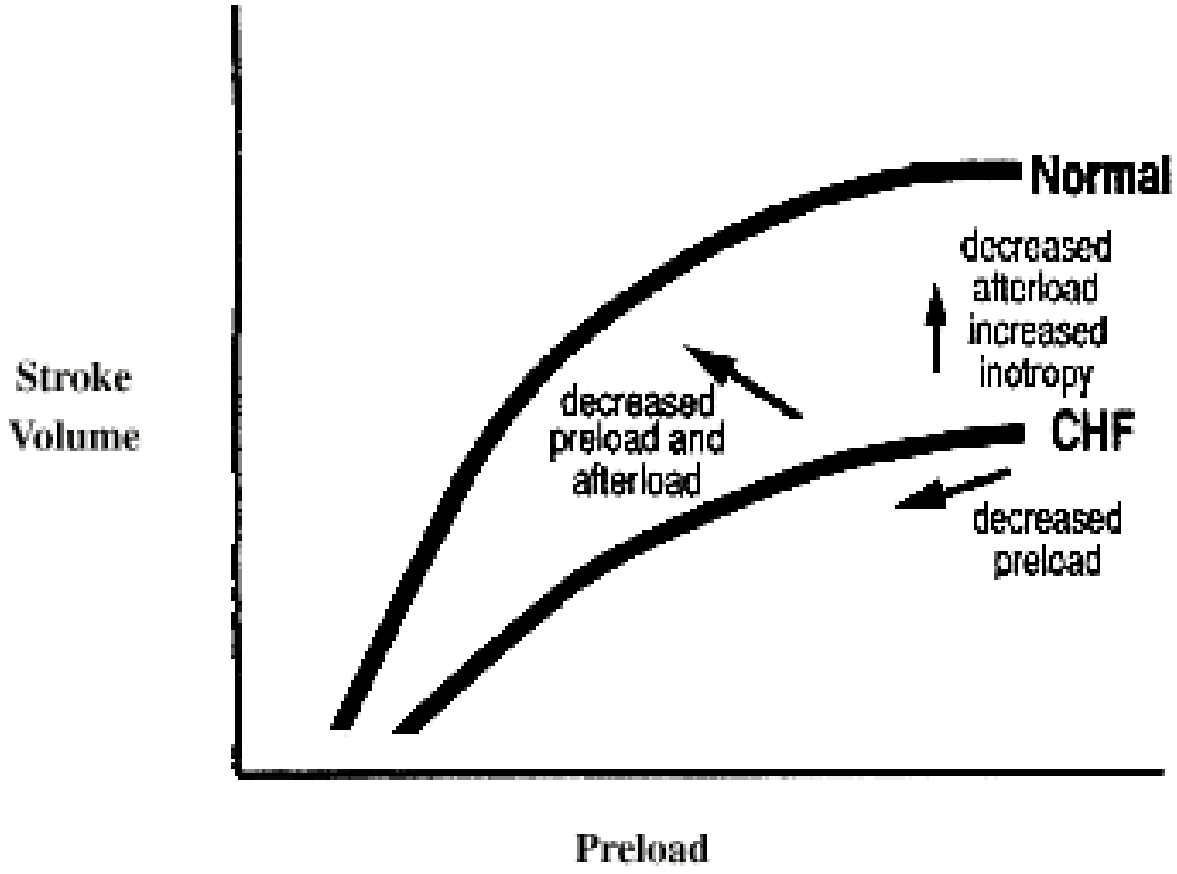
decreased preload

- Stenosis of Aortic / Pulmonary valve→

Increases afterload

- Regurgitant lesions → inadequate forward flow
- Decrease in stroke volume

Conclusion


Stroke Volume
Normal
decreased afterload increased inotropy
decreased preload and afterload
CHF
decreased preload
Preload



www.anaesthesia.co.in

anaesthesia.co.in@gmail.com