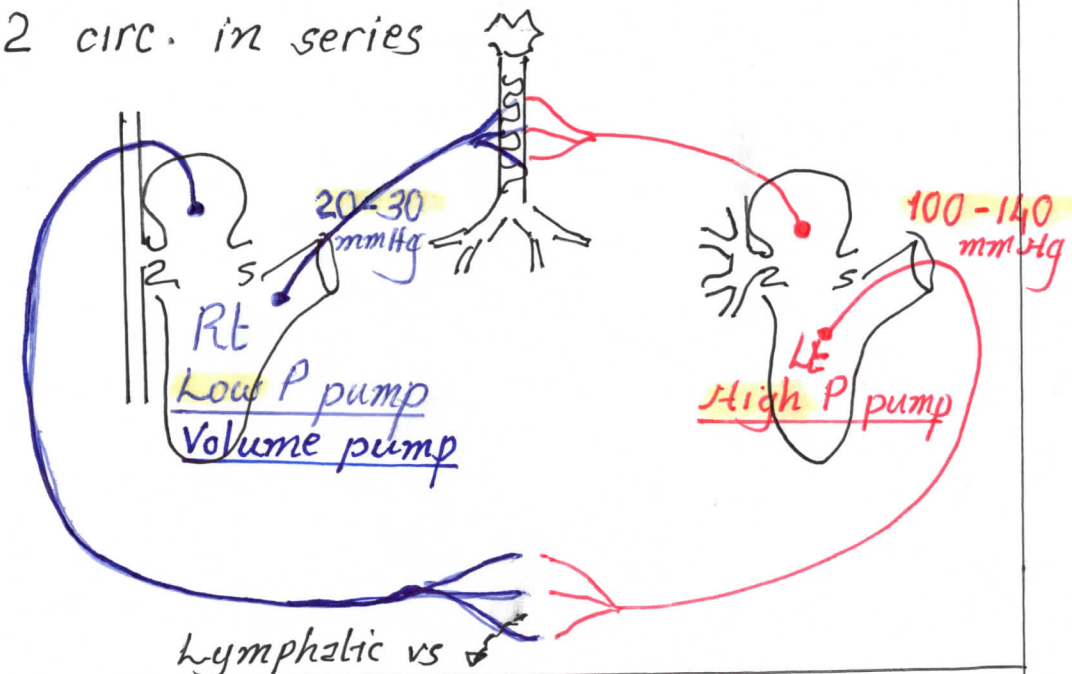
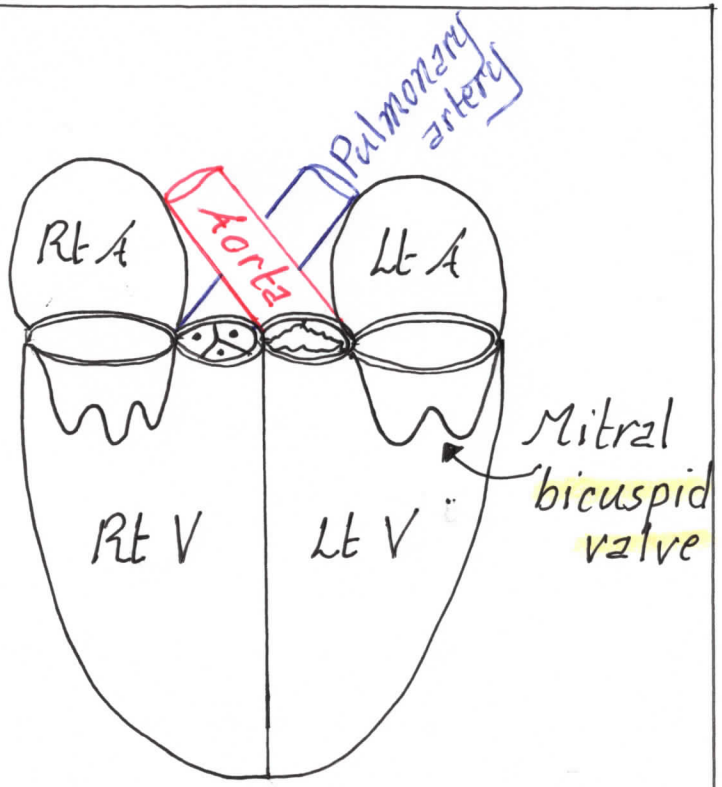
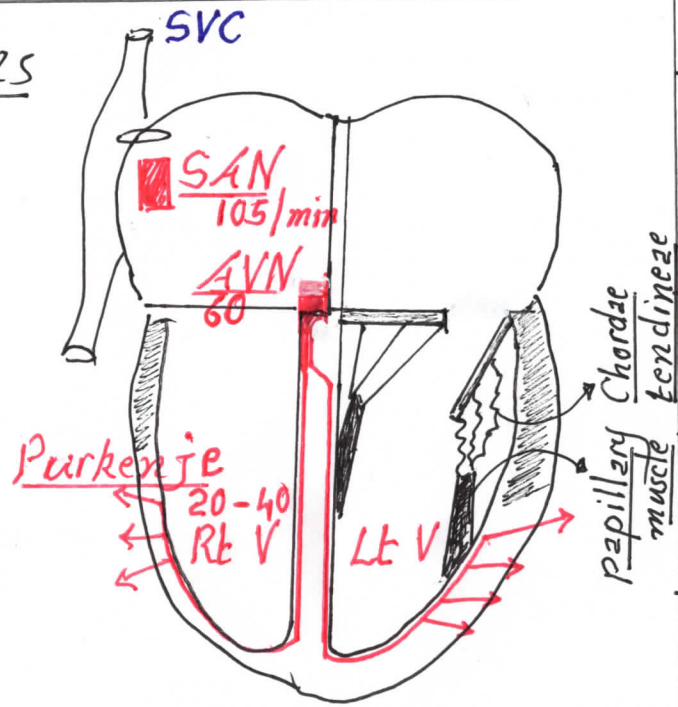
CVS = Heart + bl vessels 2

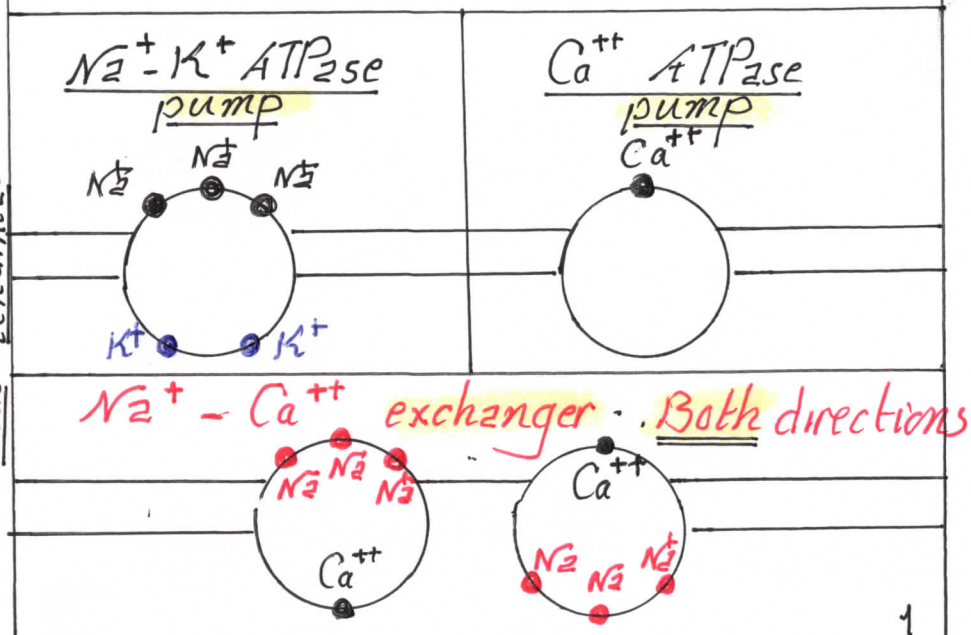
2 circ. in series



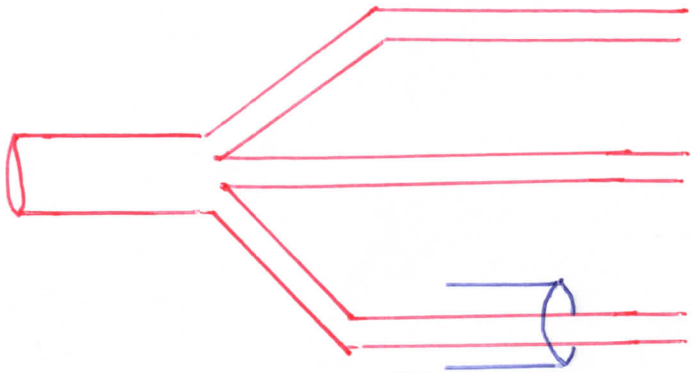


2 circulations
2 Hearts
2 Chambers
2 Types of valves
2 Types of m. fibres
2 Syncitia

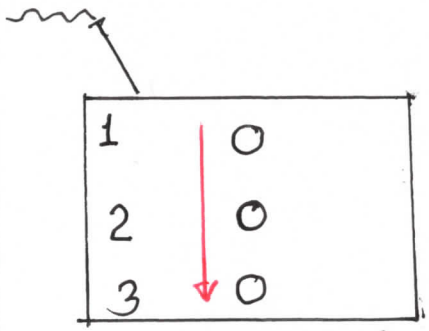




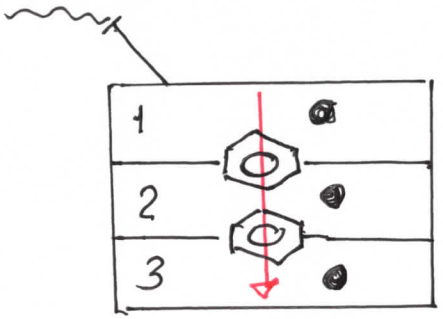
Bl vessels parallel
(not in series)



except Liver . Hepatic artery
From Aorta
Portal vein
From GIT



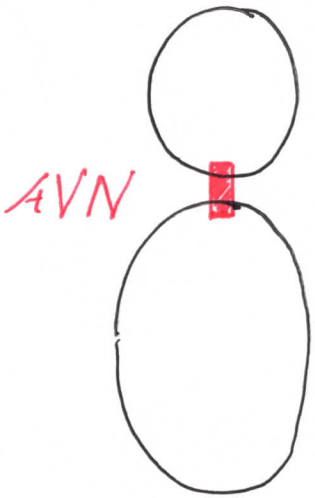
Anatomical



Functional syncitium

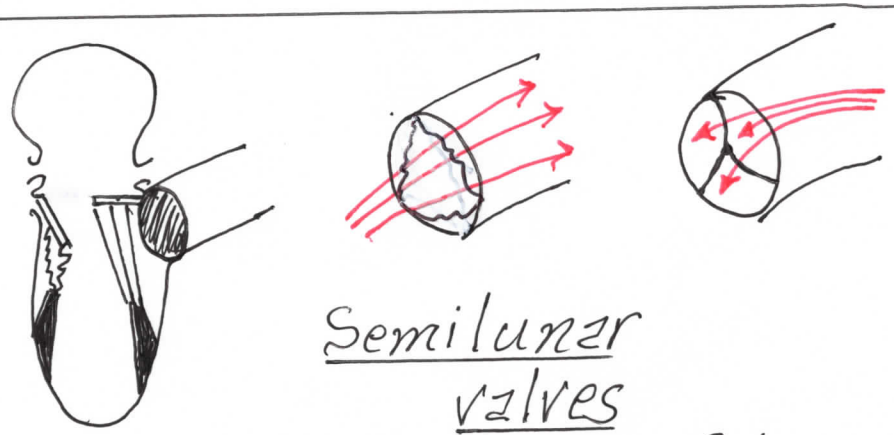
Gap junction
intercalated discs

Heart
2 functional syncitia



Atrial syncitium

Ventricular syncitium



Semilunar valves

Aortic & Pulmonary

Excitability

A potential of atrial or ventricular muscle fibres

Fast response AP

nonpace maker AP

⓪ Rapid Dep

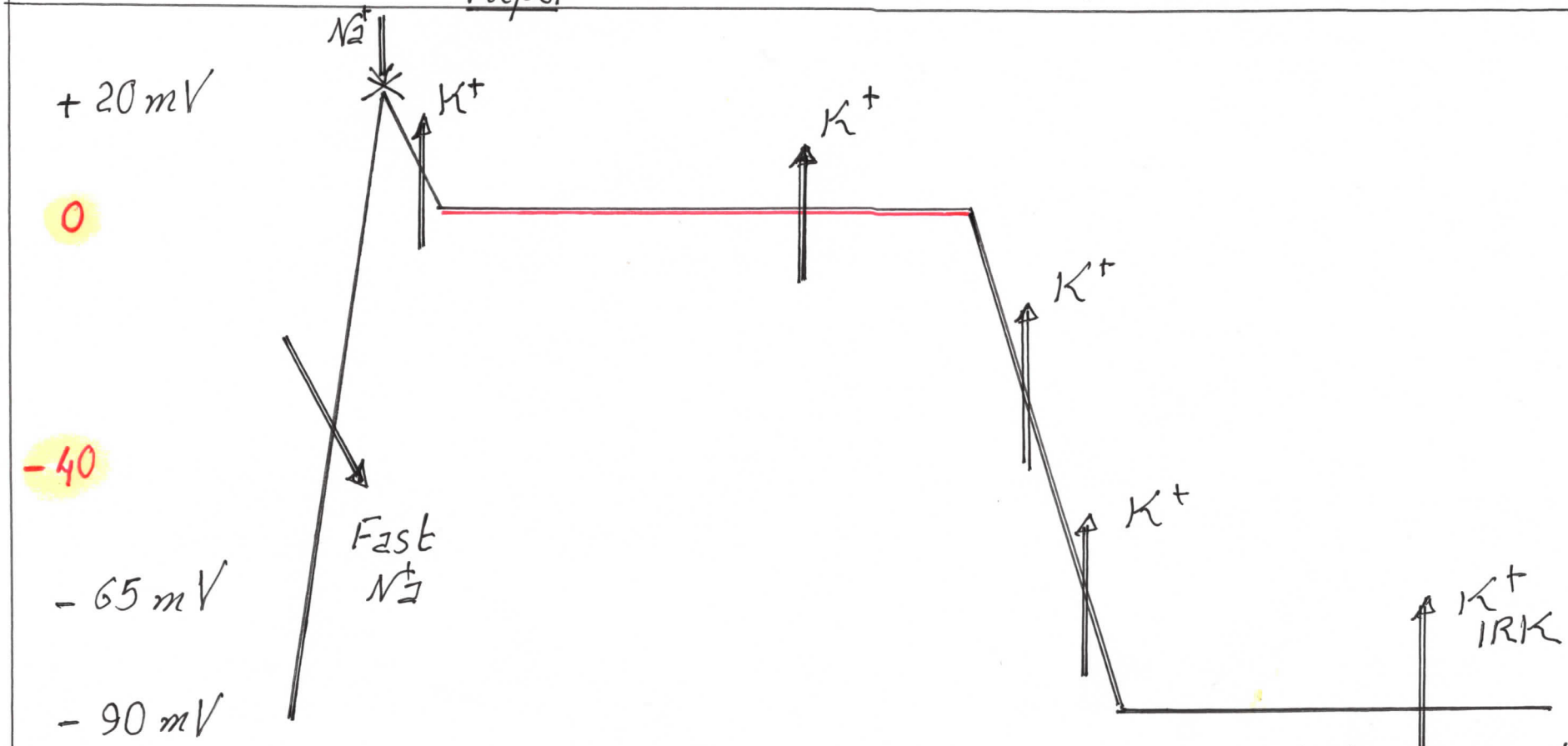
① rapid small initial Repol

② Plateau

③ rapid large Repol

④ RMP

no Hyperpolar



Selective permeability

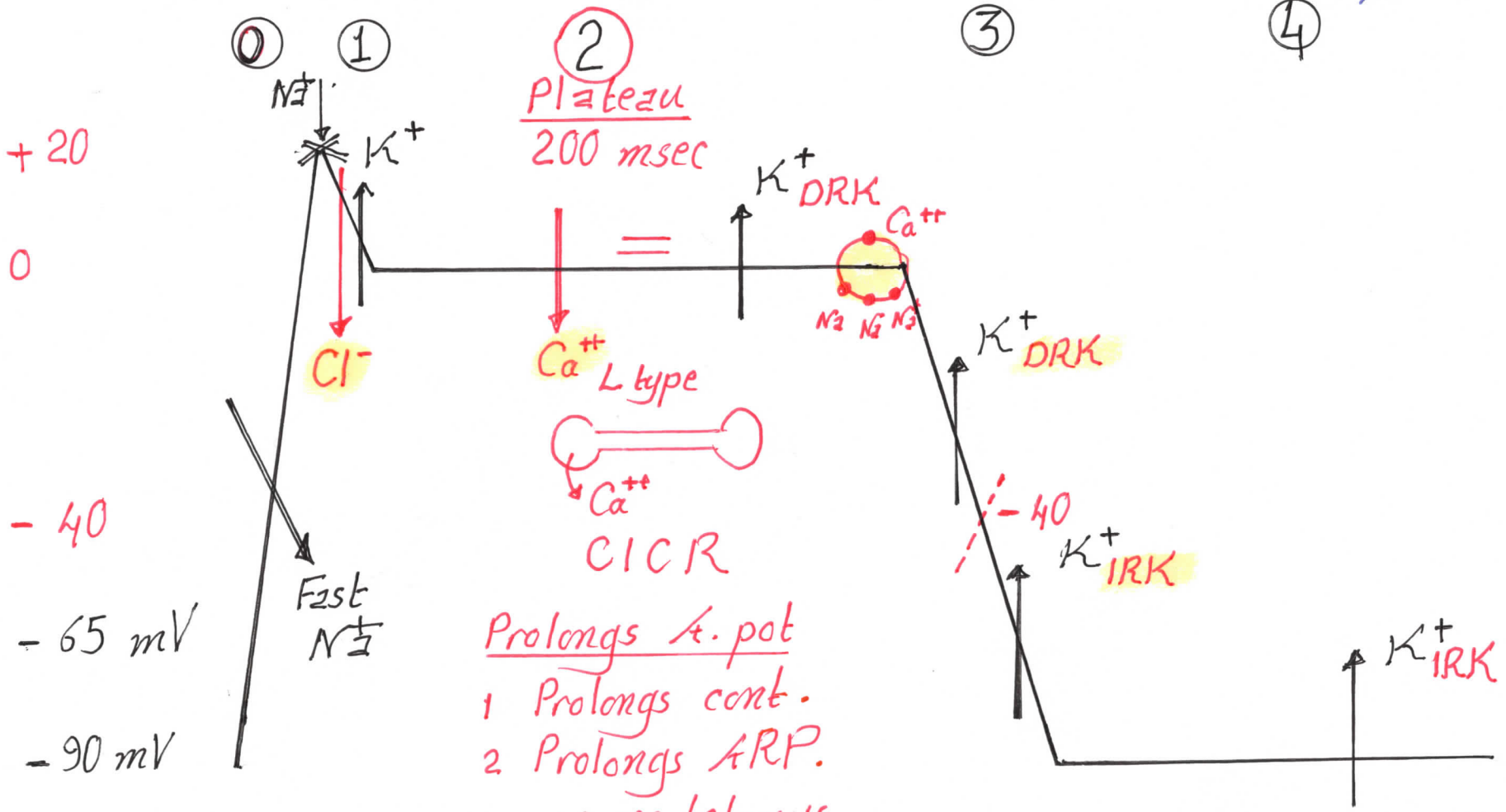
$Na^+ - K^+$ pump

E_K +94 E_{Na} +61

Excitability

A Potential of atrial or ventricular muscle fibres

Fast response A pot



Prolongs A. pot

1 Prolongs cont.

2 Prolongs ARP.

i.e no tetanus

Ca^{++} L type

starts phase 0 -40 mV

Fully active phase 2

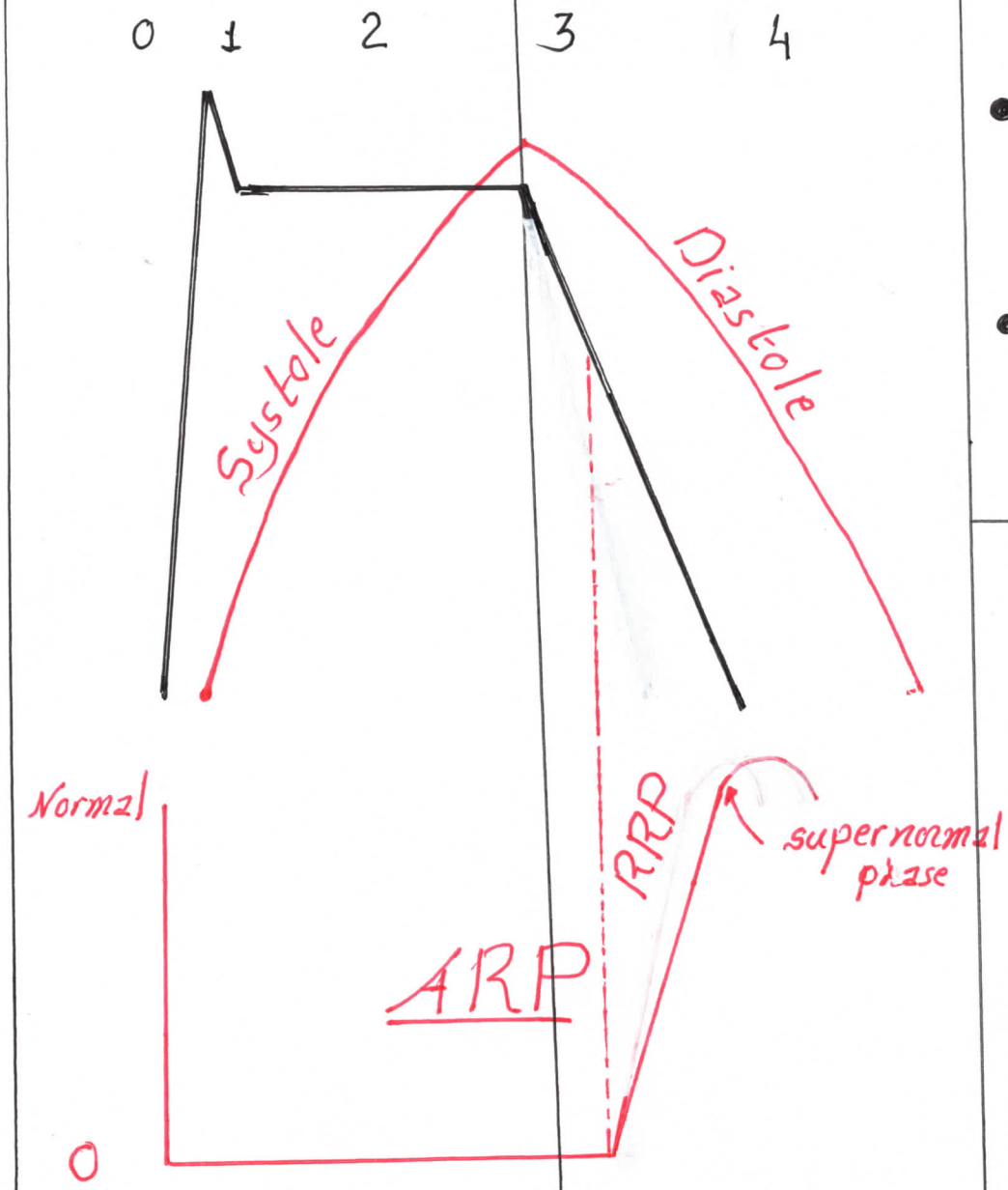
inactivated at end of phase 2

Selective permeability

$Na^+ - K^+$ pump

E_K - 94 mV

E_{Na} + 61 mV



Mechanical changes

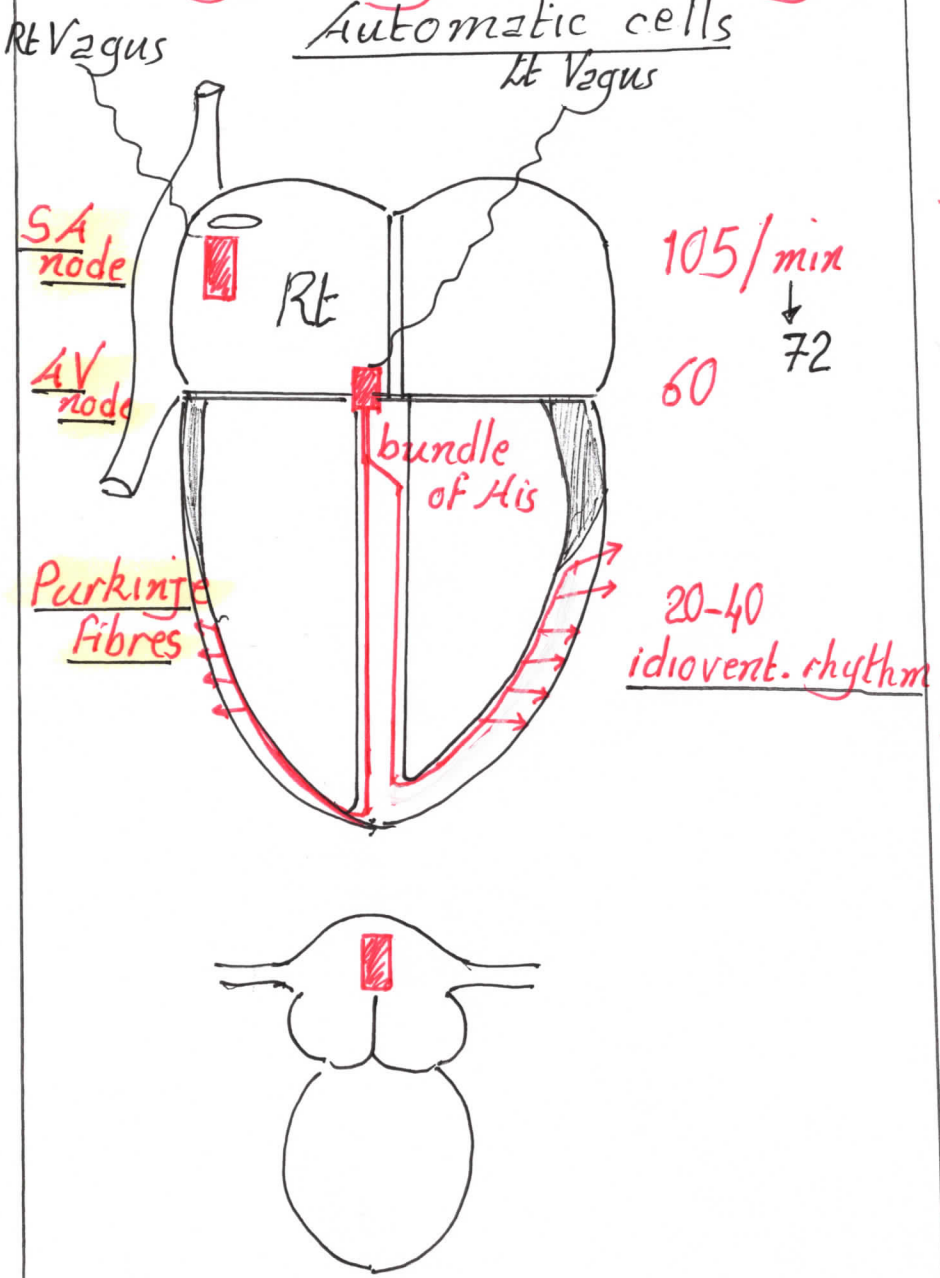
- **Systole**
  - begins immediately after depol
  - ends by end of plateau
- **Diastole** double time of 3rd phase

Mechanical 1½ time Electrical (AP)

Excitability changes

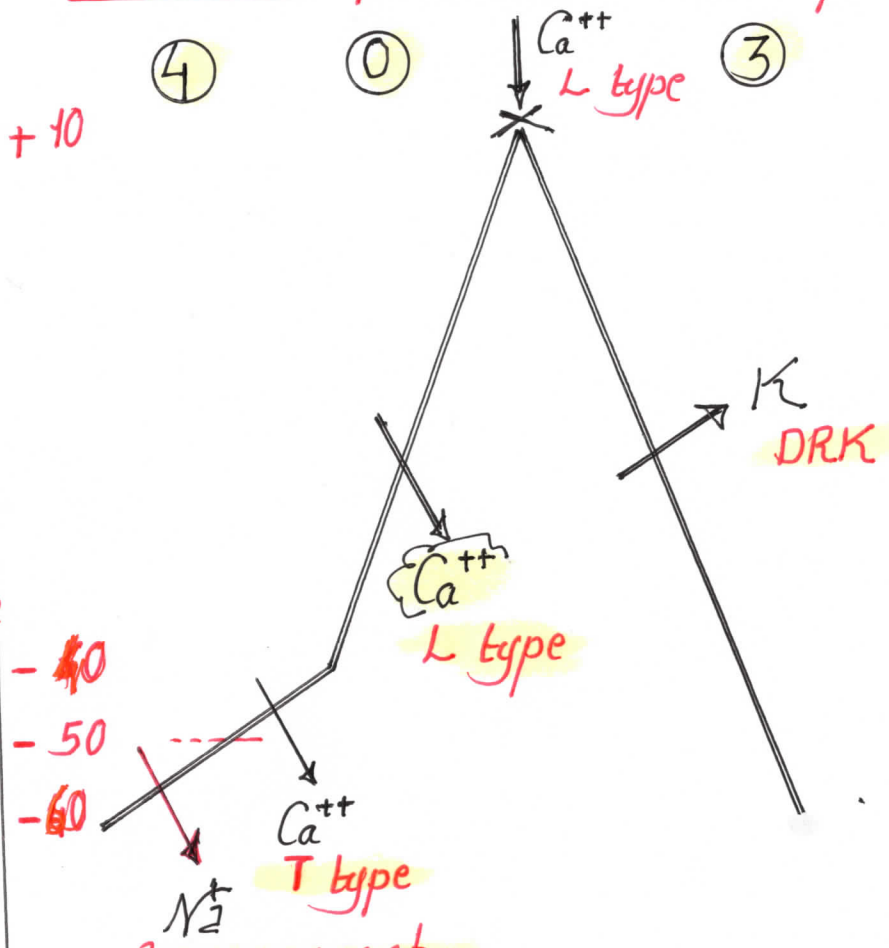
- **ARP** excitability = 0
  - coincides 0, 1, 2 early part 3
  - i.e covers whole period of Systole + early diastole
  - This prevents tetanus (allows filling)
- **RRP** excitability below normal
  - coincides with rest of phase 3
- **Supernormal phase of excitability**
  - vulnerable phase
  - Late part of phase 3

Rhythmicity (Automaticity)



Pacemaker potential

Slow response Apot



prepotential

Spontaneous gradual depol

Spontaneous slow DIASTOLIC depol

notes stable
No RMP
No Plateau

Factors affecting rate of discharge of SAN (rhythmicity or HR):

① Autonomic nerves

Sympathetic ⟶ ++ i.e tachycardia
+ve chronotropy

Mech Noradrenaline (Norepinephrine)
β_1 ⟶ ++ cAMP
++ funny current
++ slope of phase 4
reach threshold " 0
in a shorter time.

Parasympathetic ⟶ -- i.e bradycardia
-ve chronotropy

Mech Acetylcholine
a Muscarinic R ⟶ -- cAMP
b Activates K_{ACh} channels
++ K efflux
Antagonises funny current
-- slope of phase 4

② Catecholamines = Symp. n.s

③ Body temp
1 °C ⟶ 10 beats/min

④ Extracell K
a ↓ K^+ ⟶ ↑ HR ++ slope phase 4
by -- K^+ conductance in SAN
b ↑ K^+ ⟶ ↓ HR

⑤ Calcium channel blocking drugs
↓ HR & ↓ contractility
by inactivating Ca^{++} L type.

Non pacemaker (Atrial & Vent) A potent.

Fast response A pot.

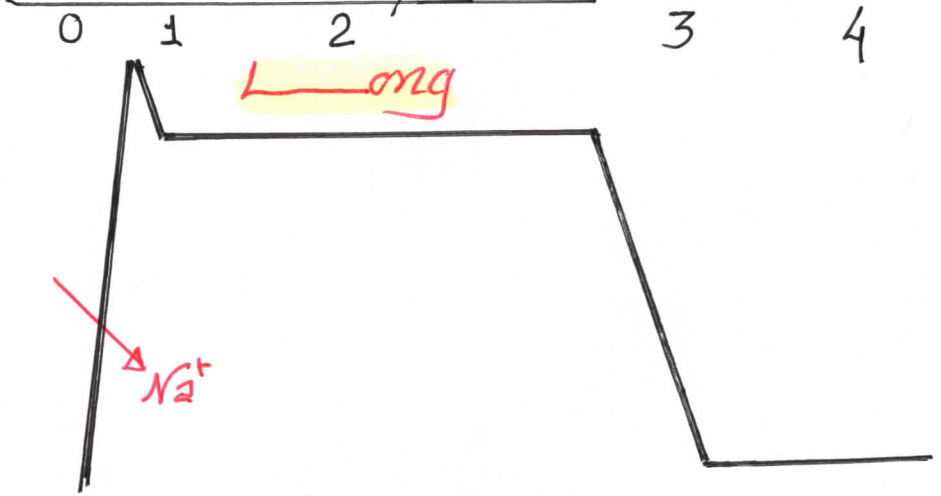
Na^+

Pace maker (SAN & AVN) A. potent

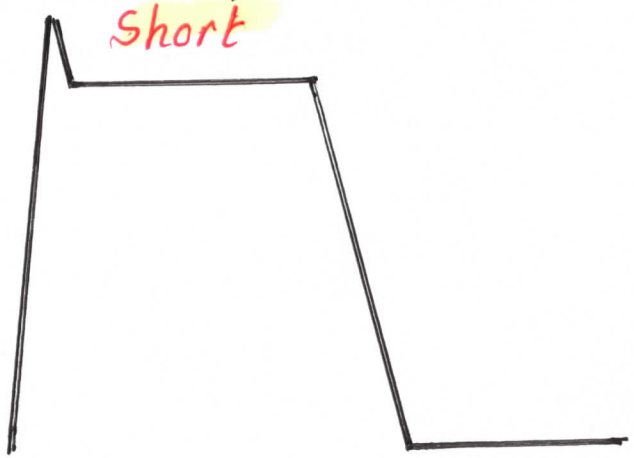
Slow response A pot.

Ca^{++}

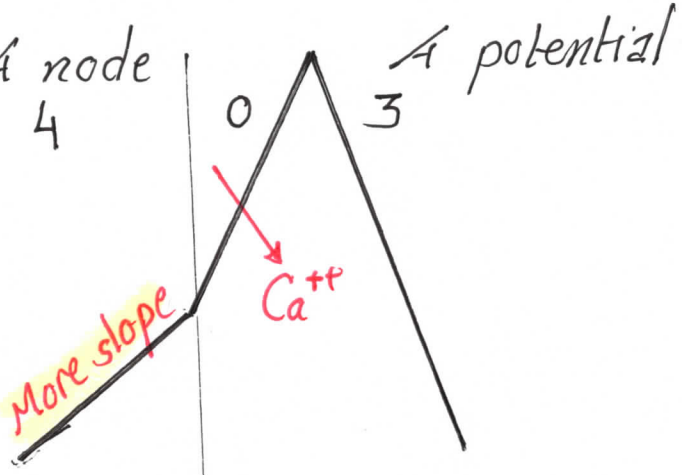
Ventricular A potential



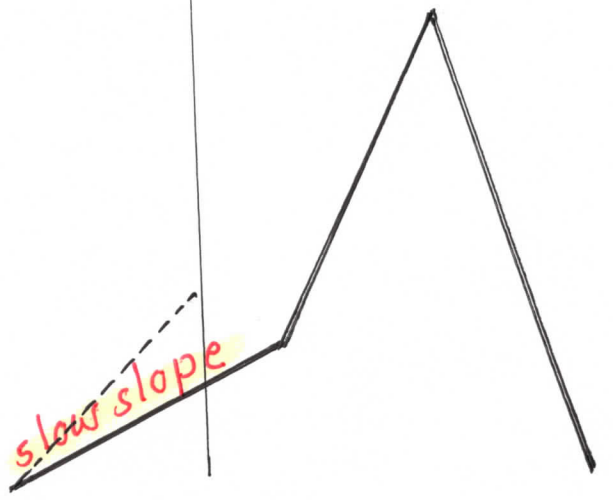
Atrial A potential



SA node A potential



AV node A potential.



Conductivity

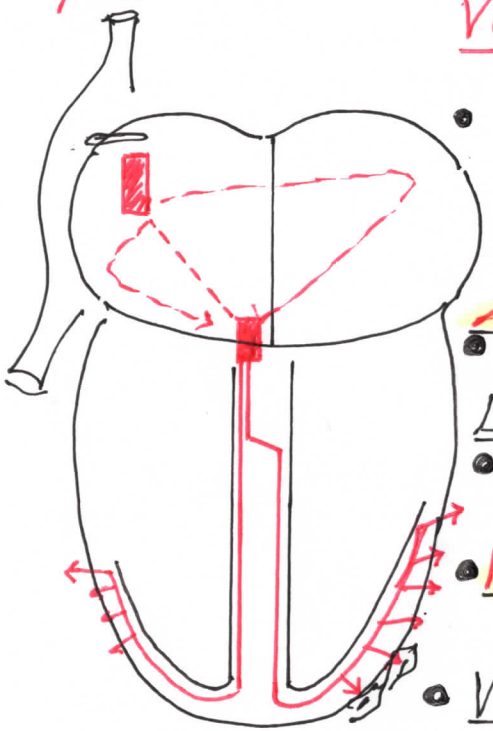
Velocity of conduction depends on :

1. Number of Gap junctions
   - Note Ability to allow current flow
     - is decreased by ↓O_2 & ↑Ca^{++} in myocytes
2. Amplitude & speed of upstroke of A potential

Factors affecting velocity of conduction

1. Autonomic nerves
   - Sympathetic
     - Norepinephrine β_1
     - ++ rate of conduction
     - Mech Faster upstroke
   - Parasymp
     - Acetylcholine Muscarinic R
     - -- rate of conduction
     - Mech Slower upstroke
2. Drugs Digitalis
   - stimulates parasymp.

Propagation of Cardiac impulse



Velocity in meter/sec

- Atrial myocytes 0.5
- Internodal bundles 1
- AV node 0.05
  - Slowest
- Bundle of His. & Rt & Lt bundles 2
- Purkinje F 4
  - Fastest
- Vent myocytes 0.5

AV node	Purkinje Fibres
SLOWES	FASTEST

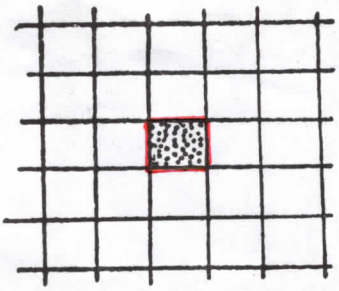
	Gap junctions	
Few	Upstroke of AP	Many
Slow		Rapid

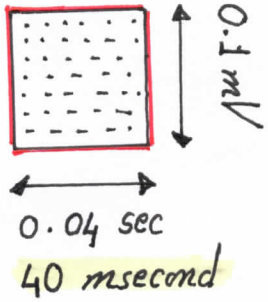
1. Delays vent. cont till end of A emptying
2. Protects vent. against High path. A rhythms

Important: To excite all vent fibres at one time & as one unit → forcible cont

Electro Cardio Gram ECG or EKG

- **Def.:** Record of depol & repol of cardiac myocytes viz 2 skin electrodes i.e **Biphasic A.P.**
- **Recording:**
  - **Apparatus** (1) Monitoring machine: in ICU
    (2) Recording „ : fed to heat stylus pen record. on a moving calibrated strip of paper.

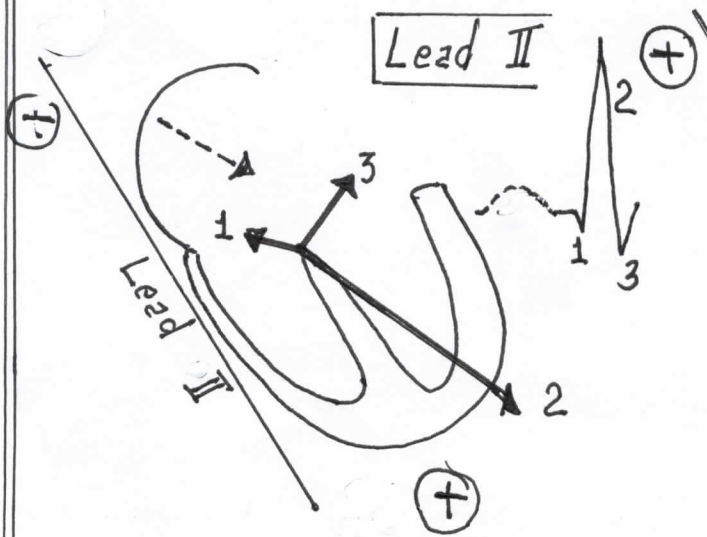


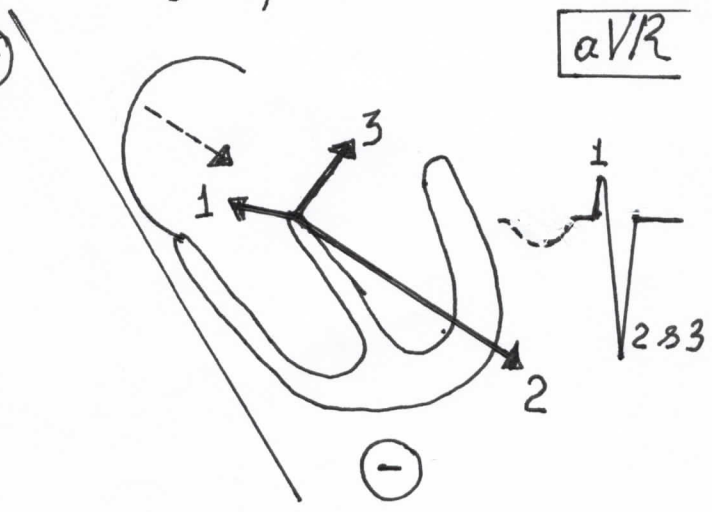


40 msecond

25 small square / second.
1500 „ „ / minute.

- **Vector:** An arrow represents sum of electrical activity.
  - **Mean electrical vector** = Sum of vectors
    - **Atrial** depol. **One** Lt & downwards →
    - **Vent.** depol. **Three** V_1 Lt to Rt Septum
      V_2 Downwards & Lt Lateral wall
      V_3 Upwards & Lt Base of vent.





- Rules :

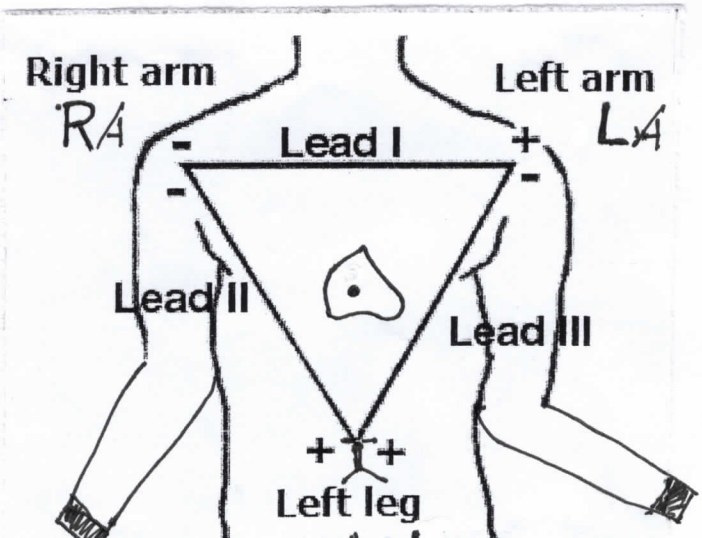
1. Voltage $\propto$ mass of tissue
2. Duration $\propto \frac{1}{\text{velocity of conduction}}$
3. Polarity — Depol is directed towards +ve electrode ⟶ positive wave (upward) & vice versa
   Repol is directed towards +ve electrode ⟶ negative wave (downward) & vice versa
4. Vector is parallel to lead ⟶ maximal recording
   perpendicular to lead ⟶ no (0) recording

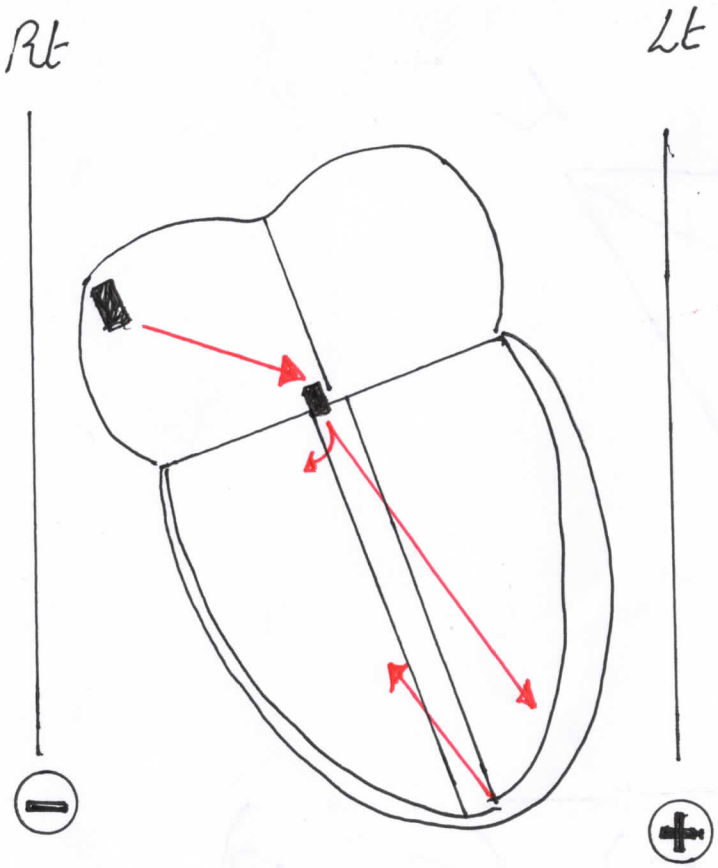
- Lead — Position of the TWO electrodes.
  Actual recording results from this position.

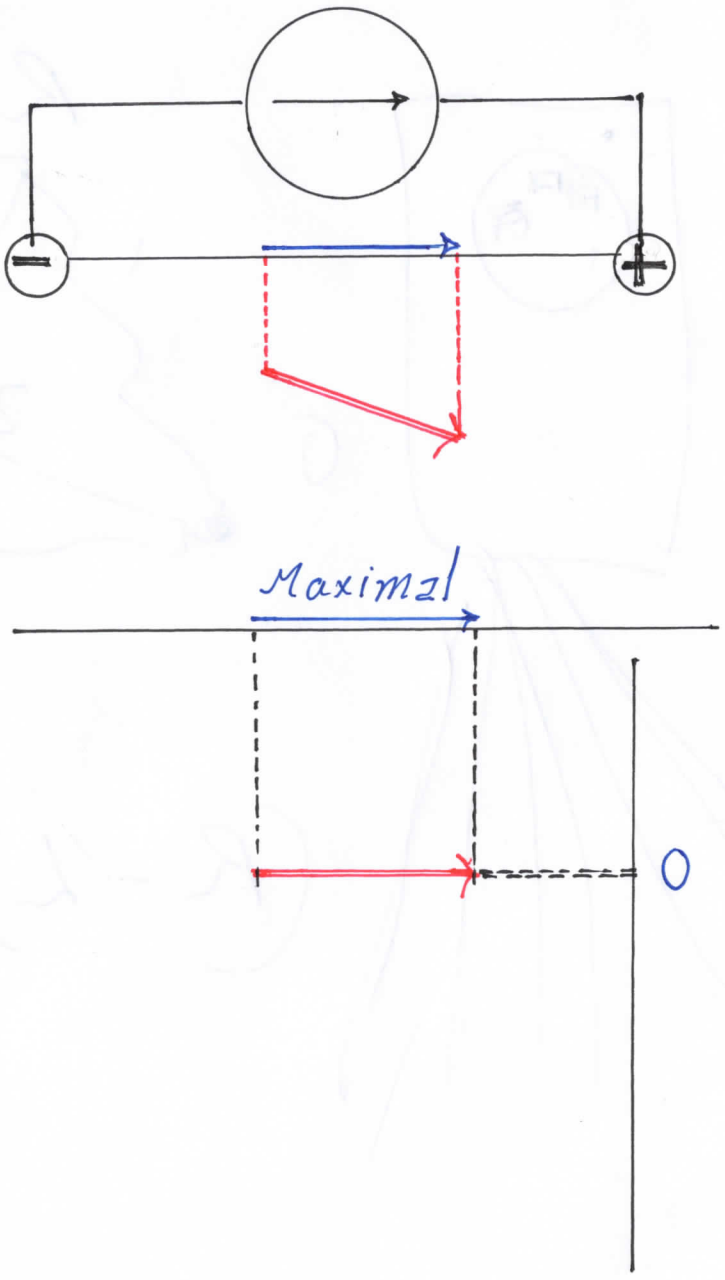
Placement — 4 on 2 arms & 2 legs (Rt leg ground electrode)
6 on defined locations on chest.

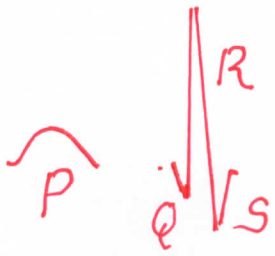
- According to SITE (placement) of electrodes :
  1. Limb leads : Lead I, II & III and aVR, aVL, aVF.
  2. Chest leads : V_1, V_2, V_3, V_4, V_5 & V_6.
- According to TYPE of electrodes :
  1. Bipolar lead : TWO electrodes exploring (+ve & -ve)
  2. Unipolar lead : One exploring (+ve) & One indifferent (0 -ve)

Standard limb leads & Einthoven's triangle



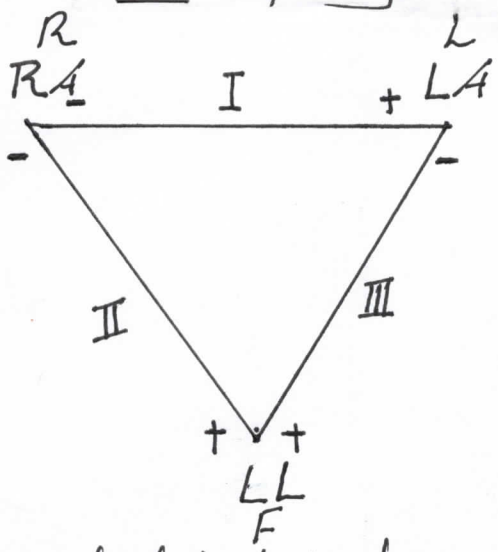

Rt
Lt


Maximal
0


P
Q
R
S

Bipolar limb leads

TWO exploring elect.

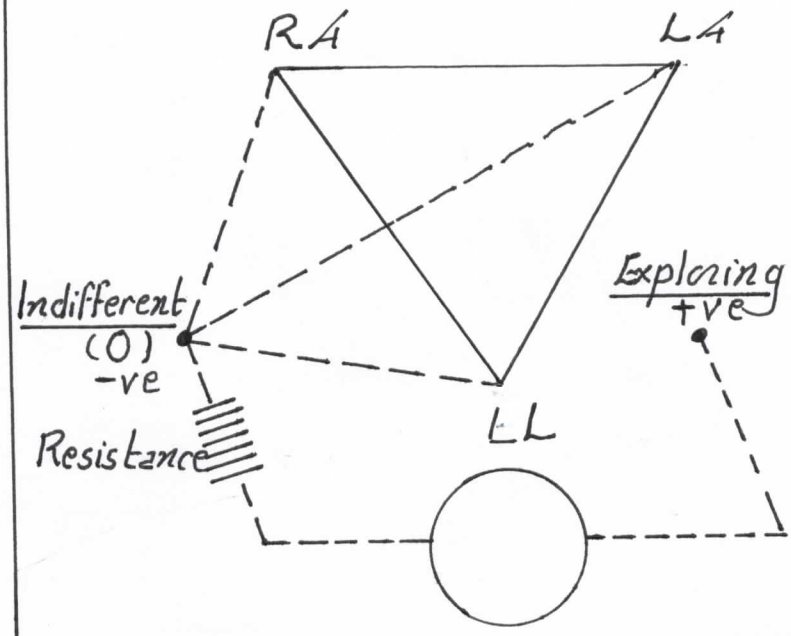


Standard limb leads

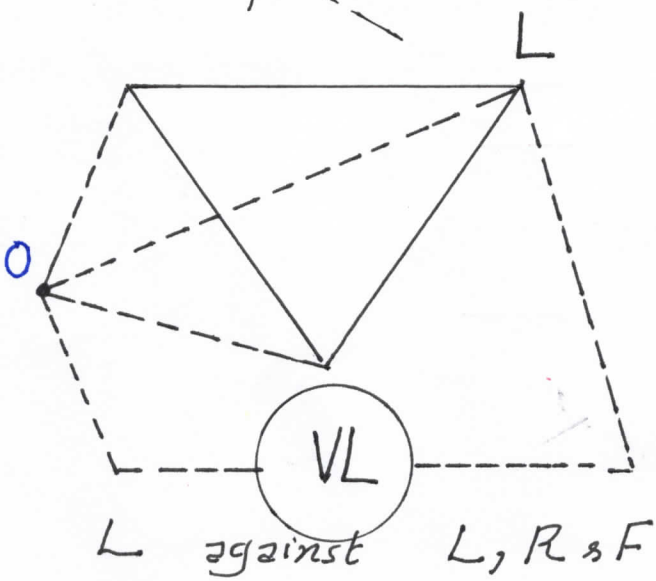
I , II, III

Unipolar leads

ONE exploring (+ve) elect.

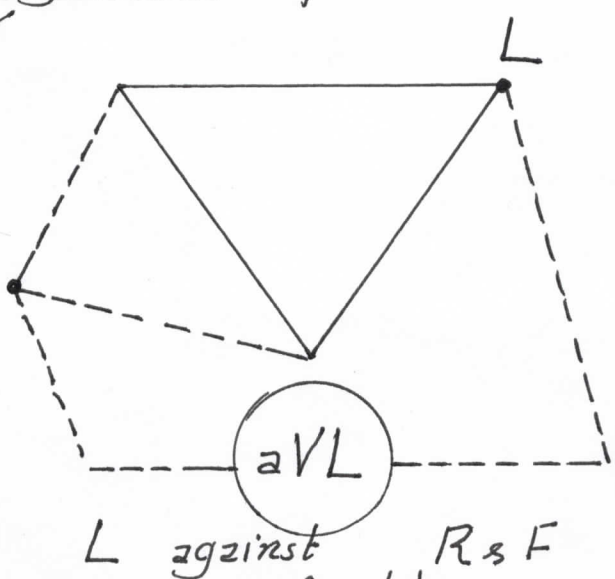


unipolar limb lead



L against L, R & F

Augmented unipolar limb lead

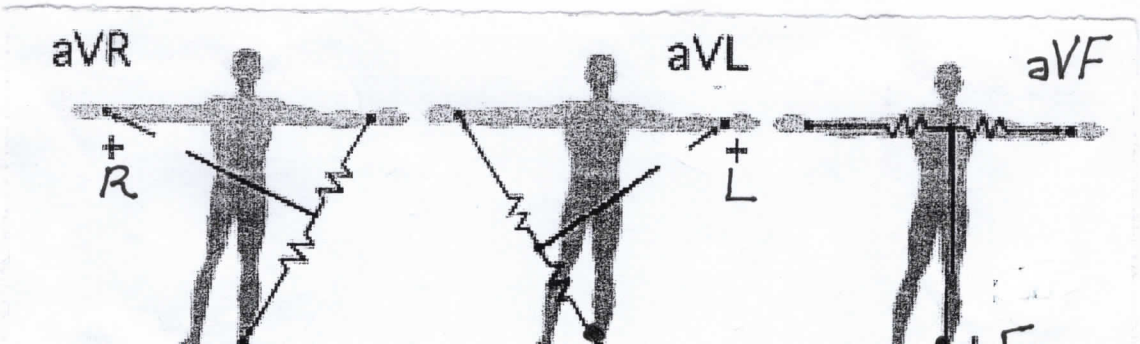


L against R & F

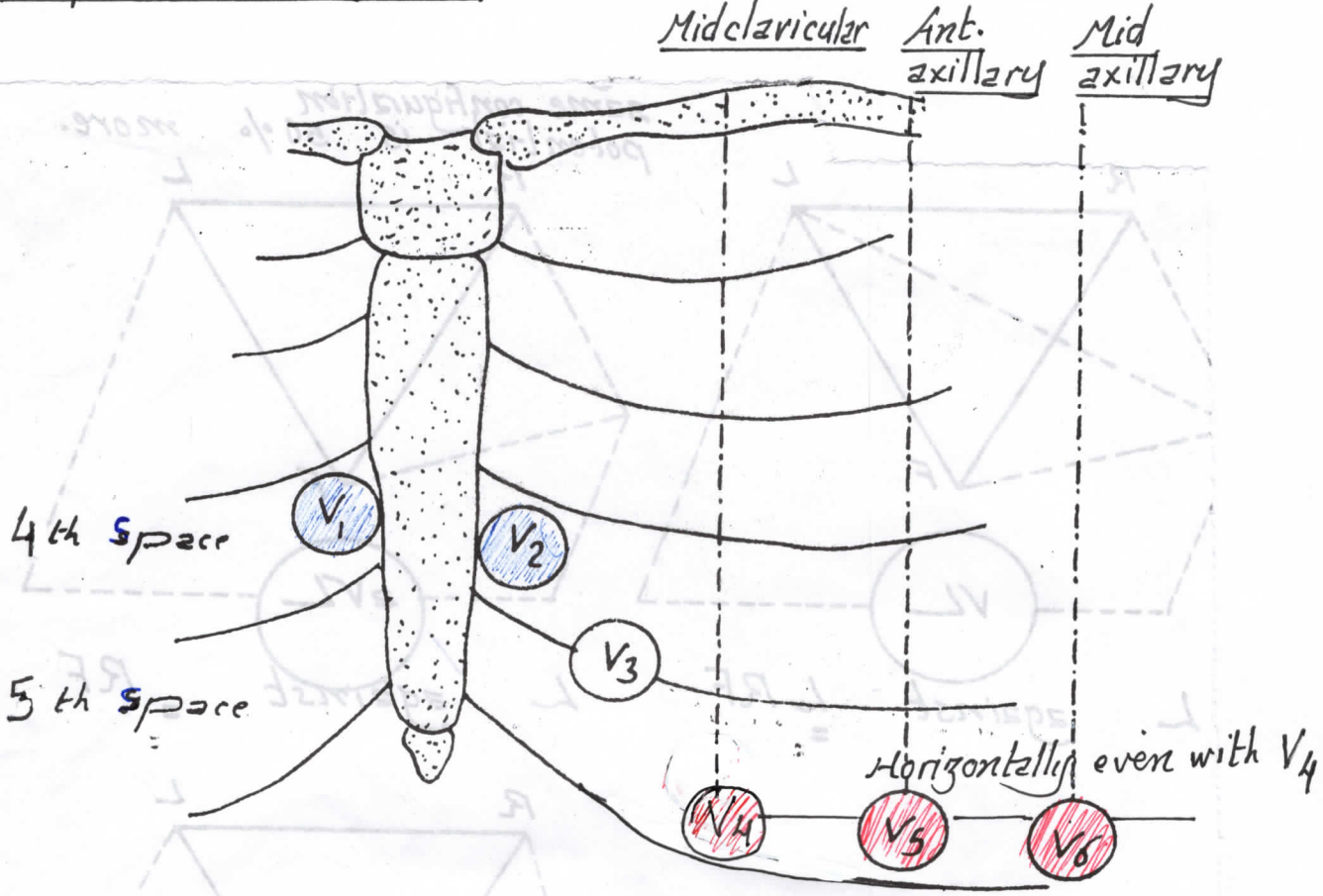
Same configulation

Potential 50% more

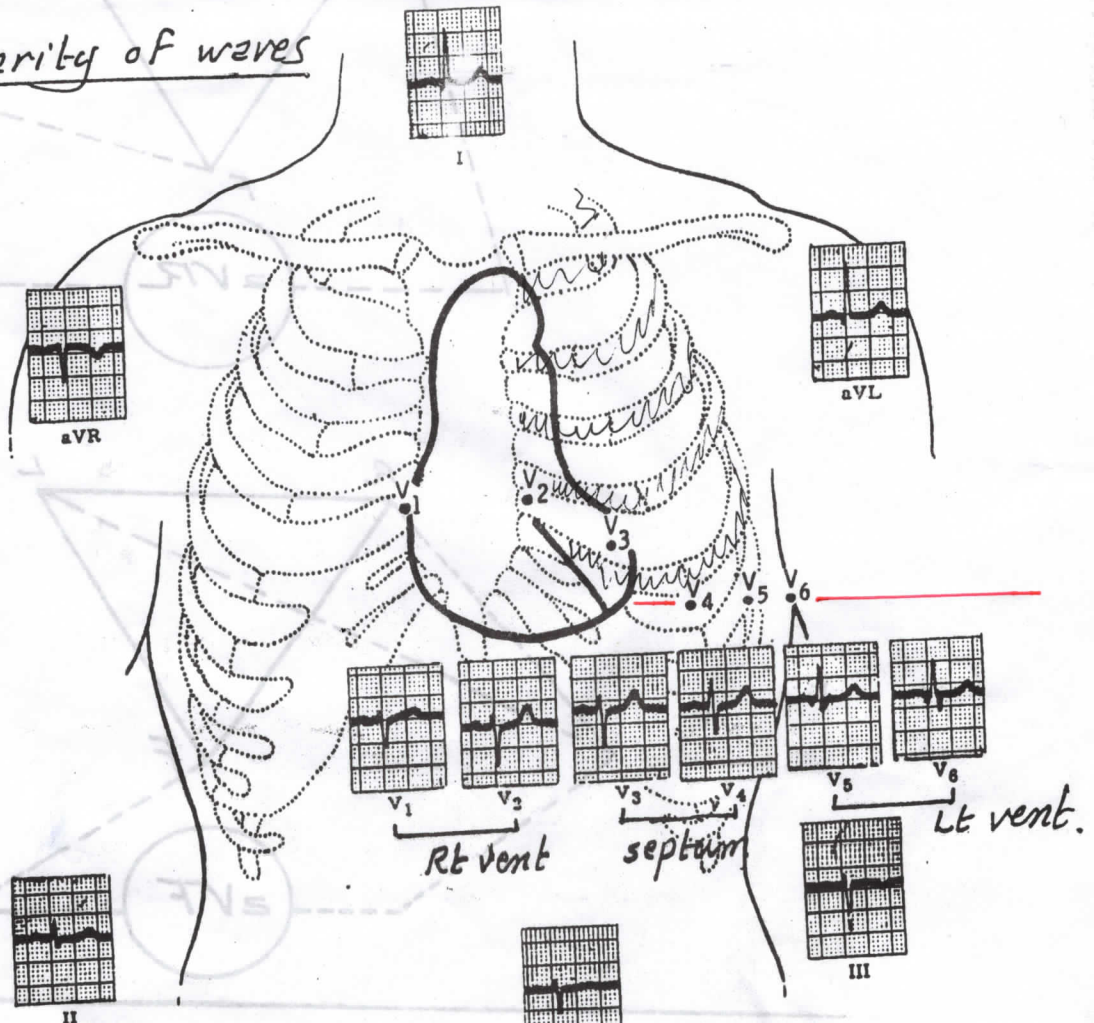
augmented unipolar limb leads



Unipolar chest leads

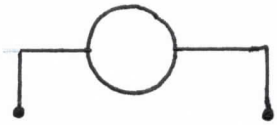


Polarity of waves



Leads

Position of 2 electrodes



Bipolar limb leads :

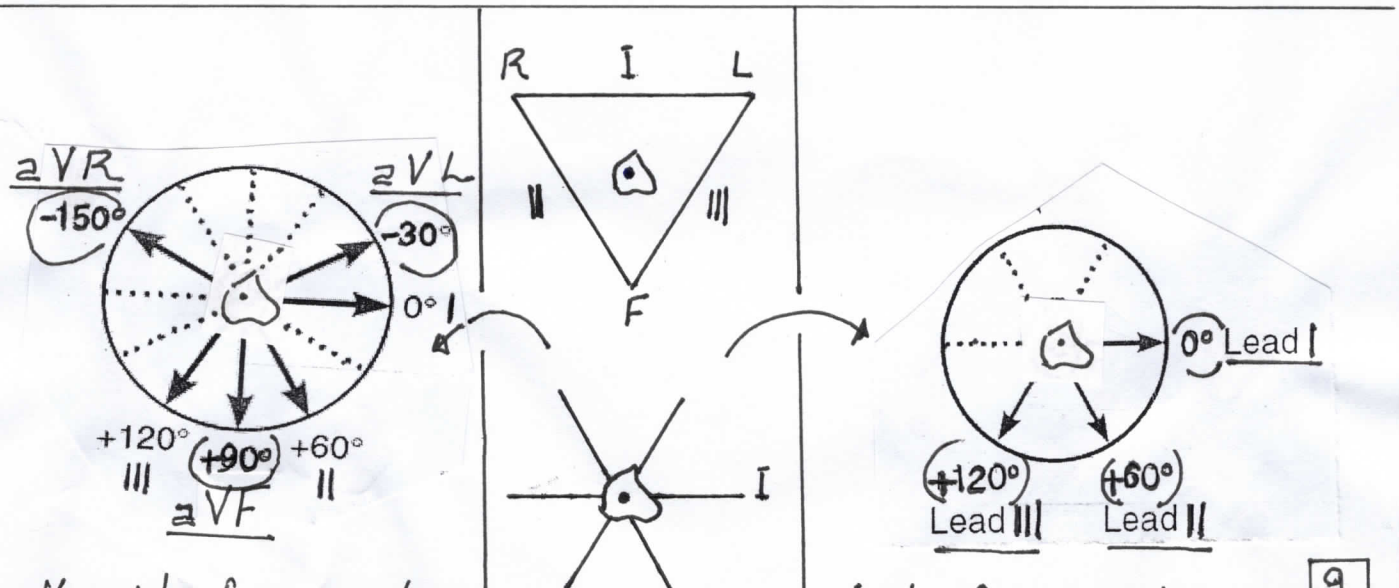
		Exp.	Exp.	
Frontal	I	R	L	+ve
	II	R	F	+ve
	III	L	F	+ve

Augmented unipolar limb leads :

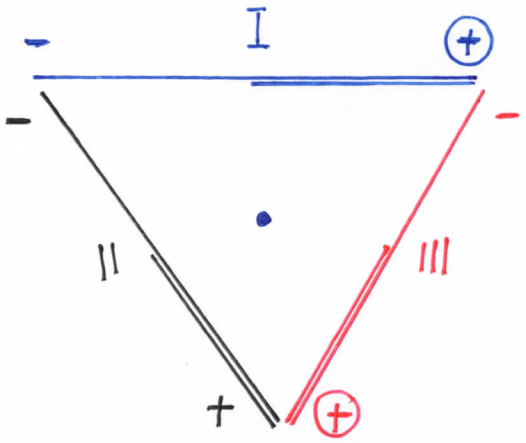
		Indifferent	Exp. +ve
Frontal	aVL	RF	L
	aVR	LF	R
	aVF	RL	F

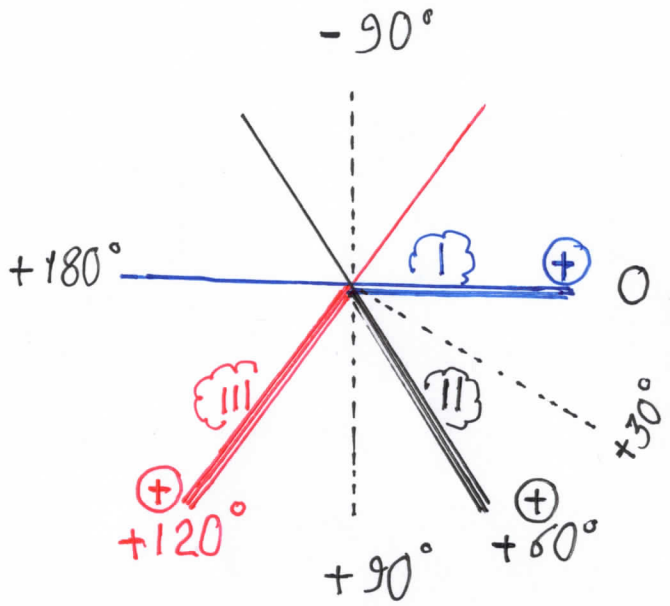
Unipolar chest leads :

			Indiff.	Exp. +ve		
RV	Septum	V_1	RLF	Rt	4th space	parasternal
		V_2	RLF	t	4th space	parasternal
		V_3	RLF	t	Between V_2 & V_4	
		V_4	RLF	t	5th space	midclavicular line
LV	Horizontal	V_5	RLF	t	Horizontally even with V_4	ant. axillary line
		V_6	RLF	t	Horizontally even with V_4	mid axillary line

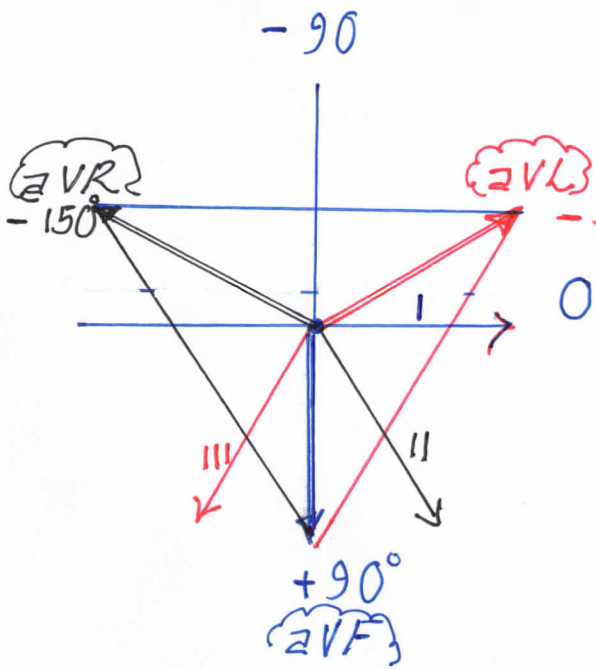


Hexaxial reference system





Axial
reference system

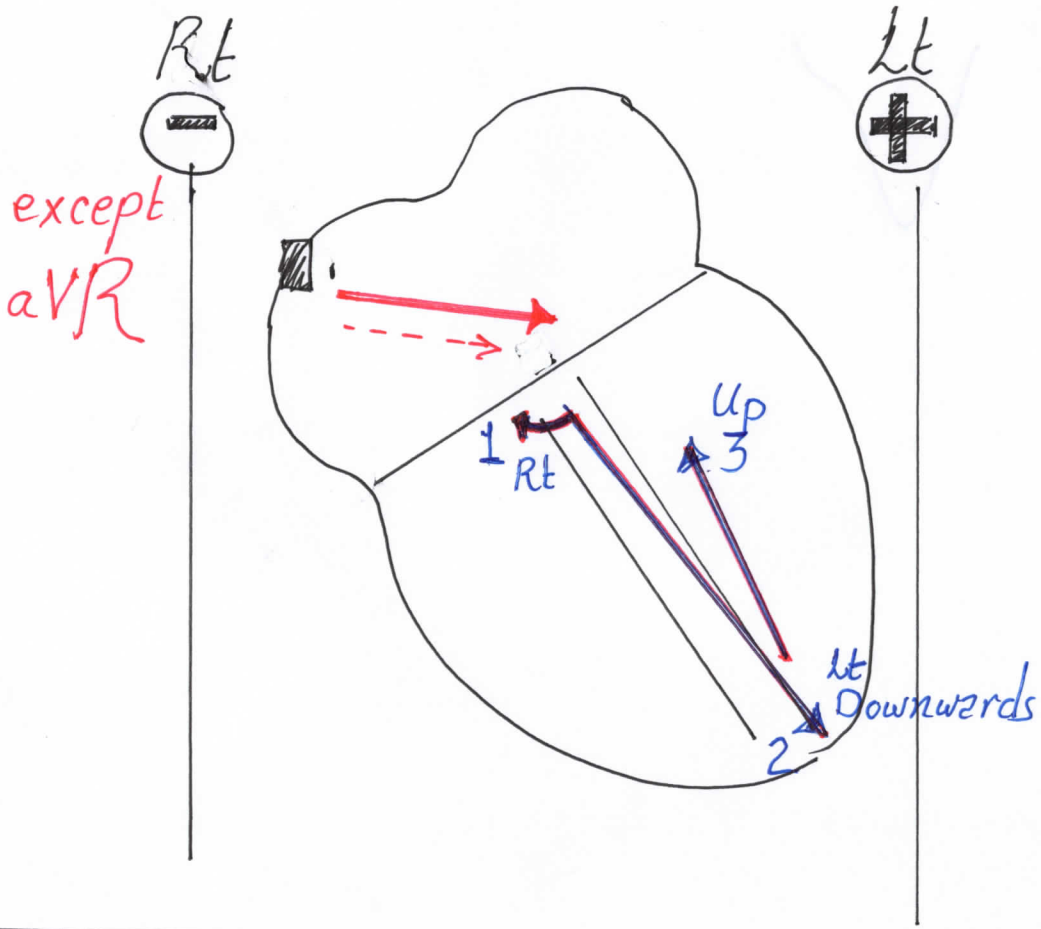


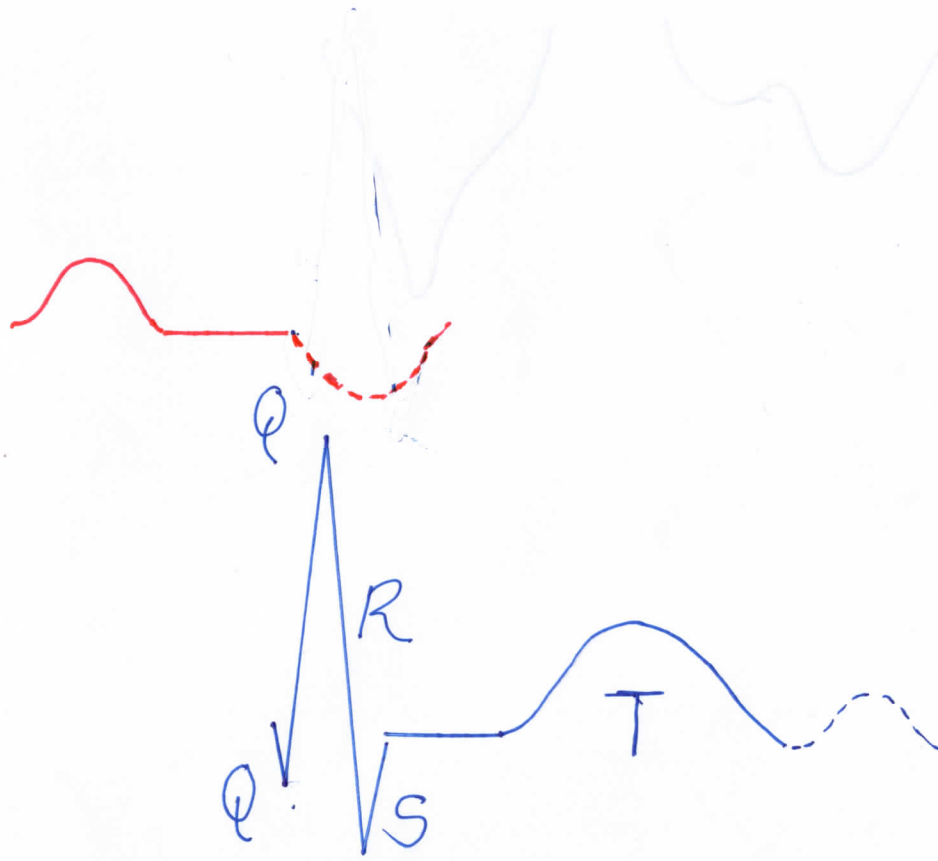
Hexaxial
reference system

ECG

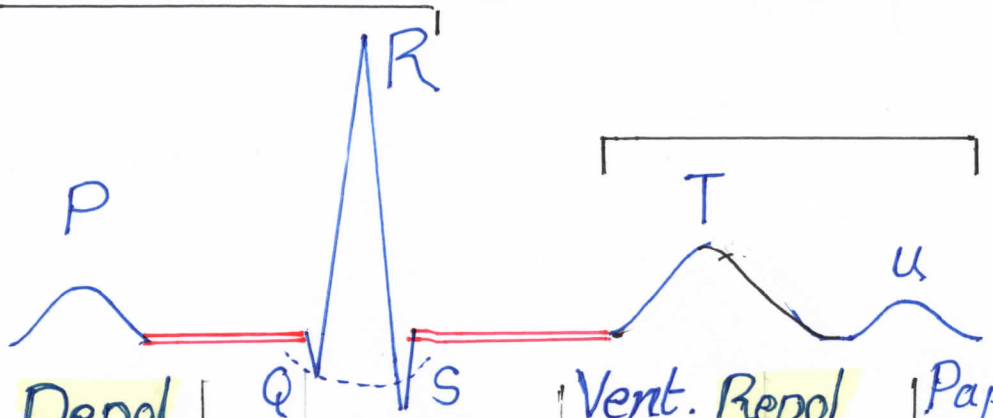
Two Electrical activity
- Depol
- Repol

Two syncitia
- Atria
- Ventricles





Normal ECG : Waves + Segments = Intervals



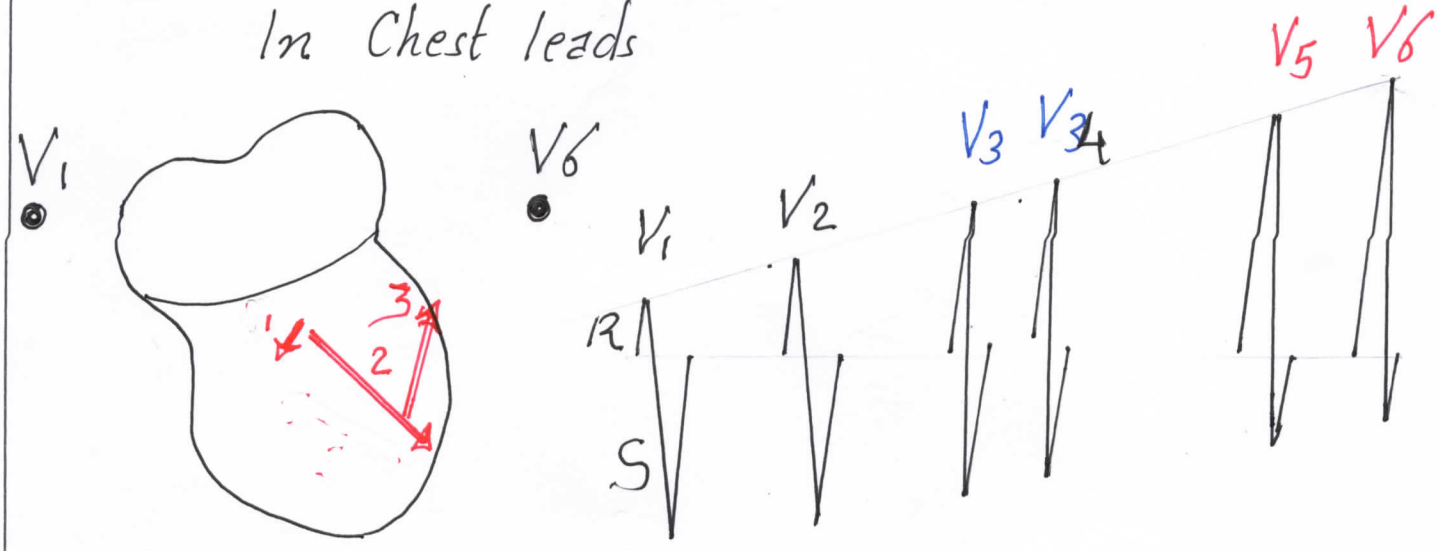
Waves

	P	QRS	T	U
Represents	Atrial Depol	Vent. Depol	Vent. Repol	Papillary ms Repol
Duration	< 80 ms = 0.08 s	80 ms	160 ms	0.05 ms.
Voltage (Heigh) Limb leads	¼ mV 0.25 mV	1 mV	< ½ mV	Usually not recorded.
Chest leads		3-4 mV		
Shape	1st half Rt atrium 2nd half Lt atrium	V_1 & V_2 small R large S V_5 V_6 large R small S	Slightly rounded	
Direction	Upright in most leads Inverted in aVR	R always +ve Q -ve before, S -ve after R	Asymmetrical Inverted in aVR	
Clinical	Absent in A fibrillation Long in A enlargment If inverted in other leads ectopic A pacemaker	> 120 ms BBB, vent ectopic focus, ↑ K^+ or TCA ovendose tall Unusually tall Lt vent enlarg Very low pericardial effusion infiltrative myo disease	Inverted sign of myocard ischemia Peaked sign of ↑ K^+ or early myo-infarction Flat ↓ K^+	Very prominen ↓ K^+, ↑ Ca^{++} hyperthyr

- A repol not recorded — Masked by QRS complex; Very low voltage
- SAN, AVN & Purkinje depol not recorded. Small mass of tissue.

QRS complex — R 1st +ve wave; Q -ve before; S -ve after R

In Chest leads

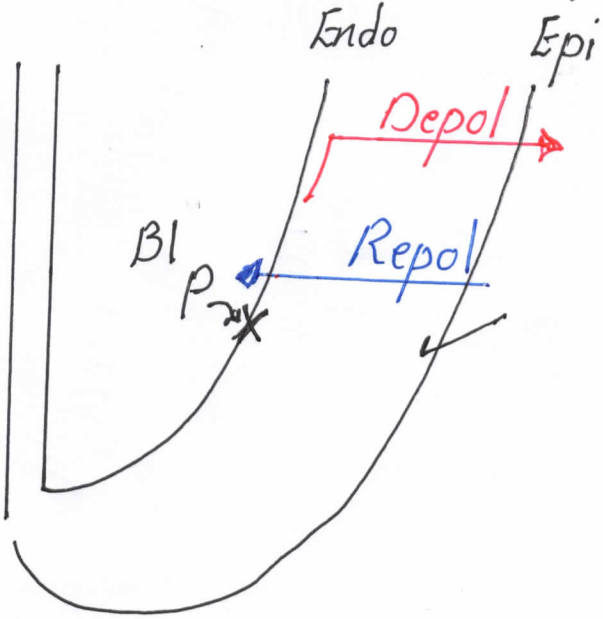


T vent repol. in same direction R vent depol !

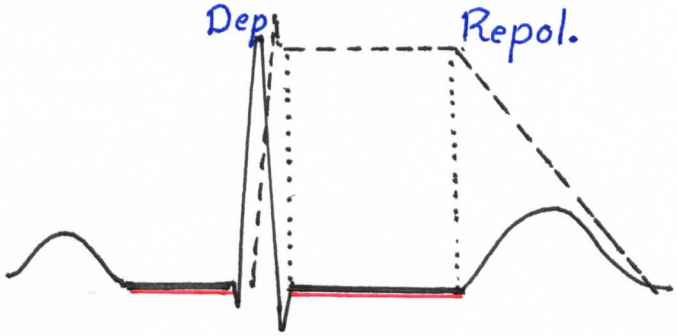
1st part to be depol
last part to be repol

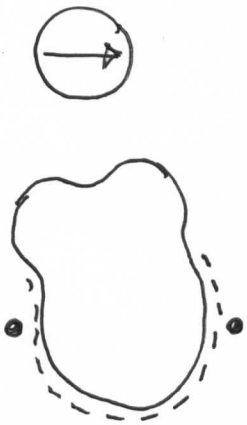
or

1st part to be depol
last part to be repol



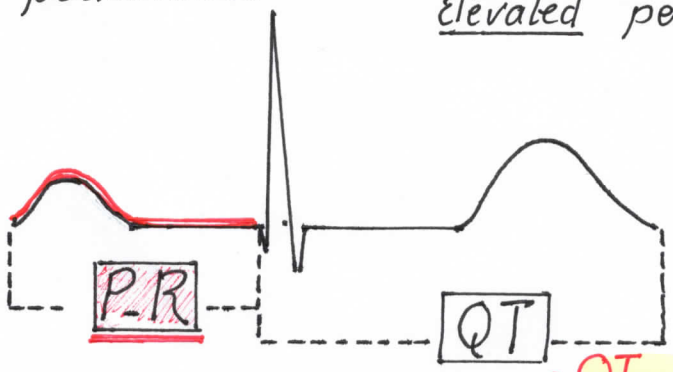
Segments





Measurement	end of P to begining of QRS	end of QRS to beginning of T
Importance	Conduction AVN 0.06 ~ 0.1s Typically flat IF depressed in pericarditis	Isoelectric segment Corresponds to plateau of Vent. AP ↑ Elevated or depressed in MI & ischem Depressed LVH or digoxin drug Elevated pericarditis.

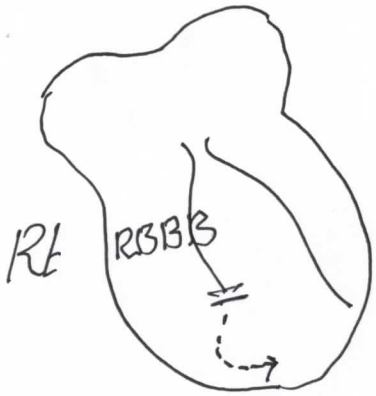
Interval

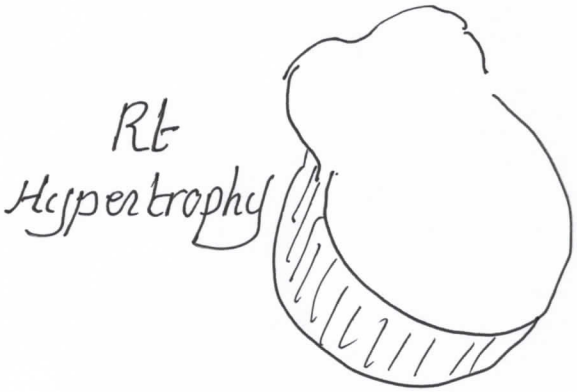


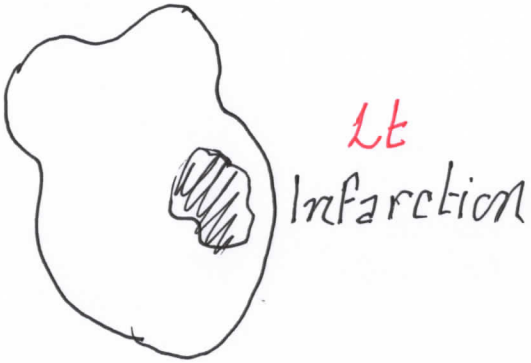
& QT_c ie corrected QT

Measurement	beginning of P wave to beginning of QRS	beginning of QRS to END of T wave
Represents	A depol + AVN cond.	V. depol. plus V. repol.
Normal	0.12 to 0.20 second.	0.2 - 0.4 second
Prolonged	A-V block 1st degree Heart block Atrial enlargement Vagal stim.	-- HR Prolonged QT_c ($QT/\sqrt{PR}$) risk factor for Vent tachyarrhythmia & sudden death (genetic - medication)
Shortened	A-V nodal rhythm Wolf Parkinson-White syndrome (by passing AV node)	++ HR Severe hypocalcemia

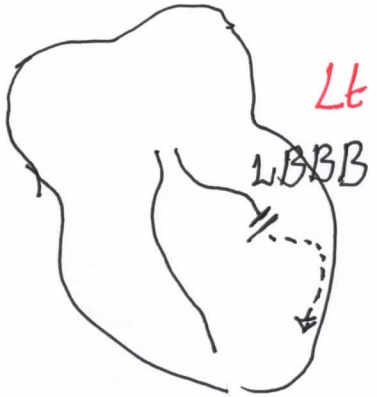
Rt axis deviation

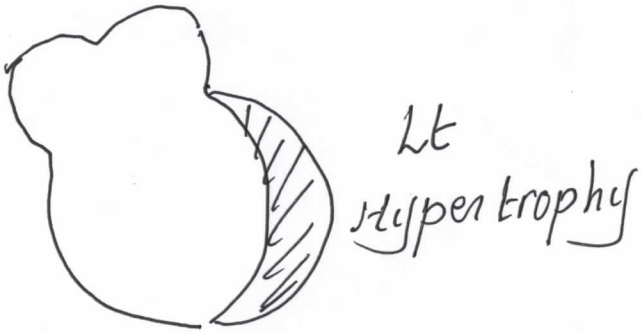




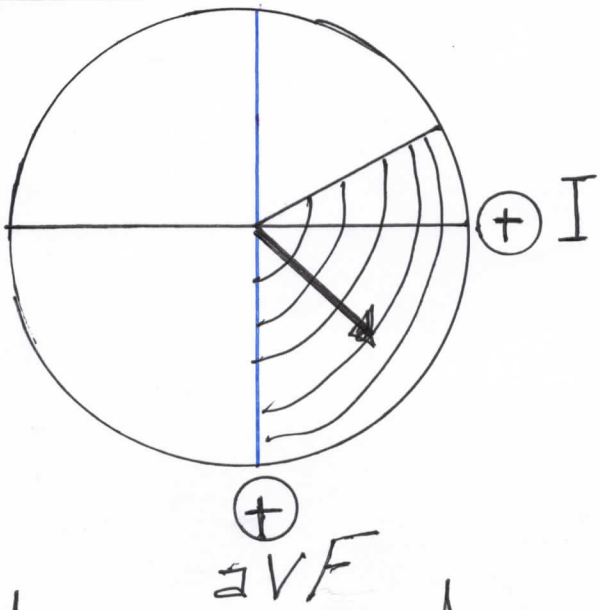


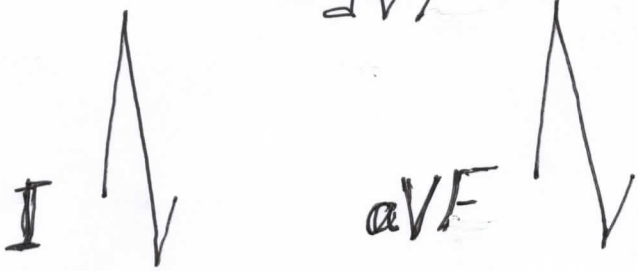
Lt axis deviation



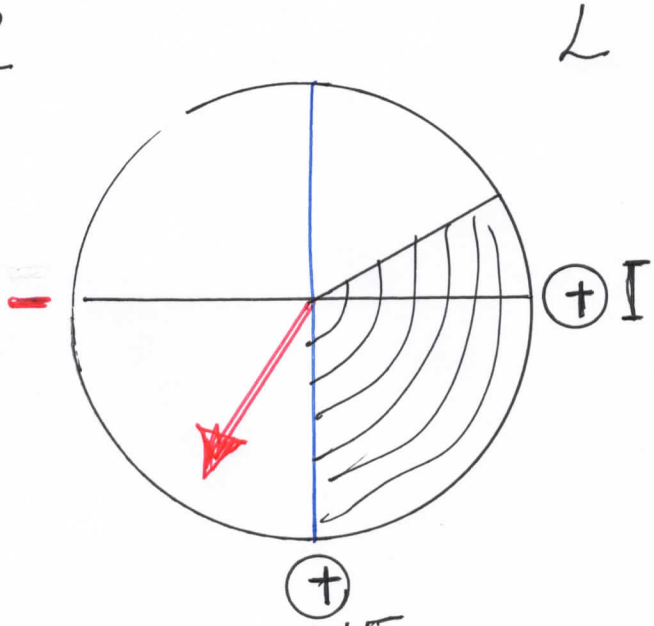


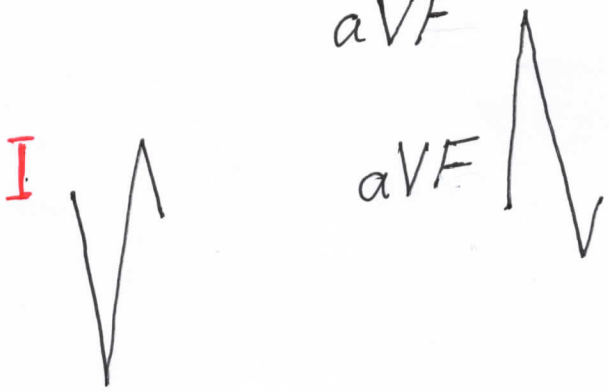
Normal



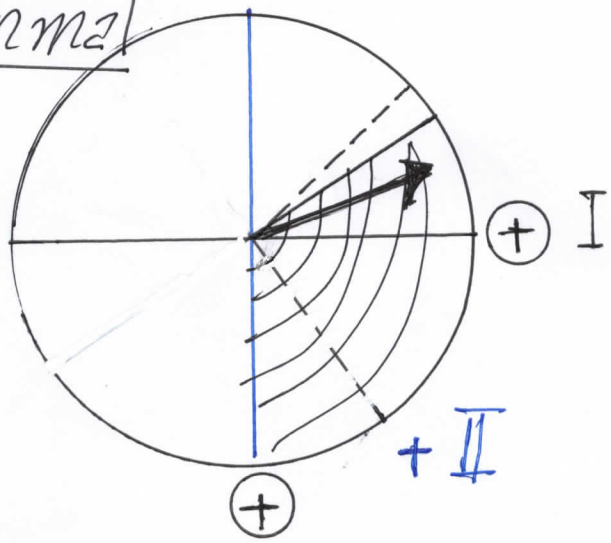


Rt axis deviation

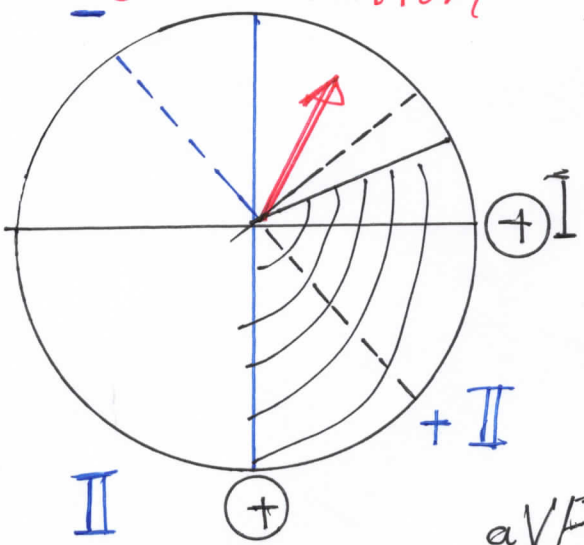


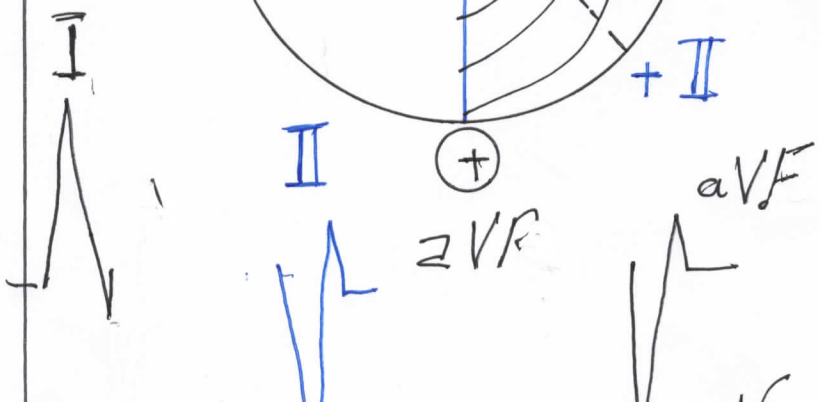


Normal



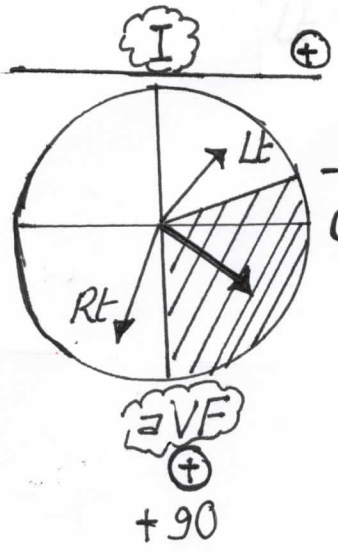
Lt axis deviation





Electrical Axis of the heart — Mean QRS Cardiac Vector

- **Def** Mean Value of V. depol. QRS
- **Represented by** Vector (Direction & Amplitude)
- **Normal direction** $-30°$ to $+90°$



- **Rt axis deviation**

  QRS -ve in Lead I
  +ve in aVF

  Axis More than $+90°$

  Physiol. Long selender

  Path. RVH
  RBBB

  Lt ventricular infarction

- **Lt axis deviation**

  QRS +ve in lead I
  -ve in aVF & lead II

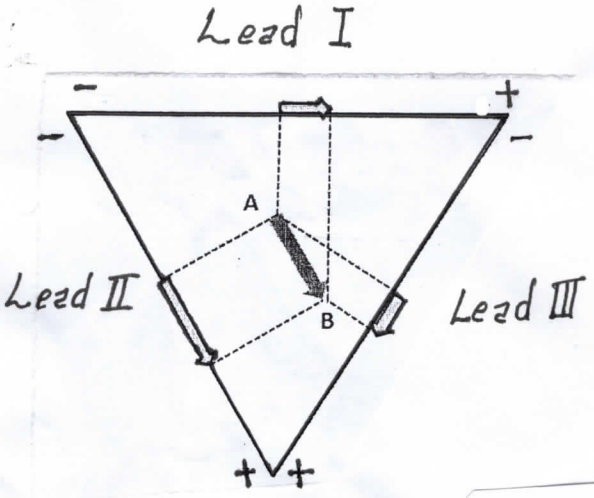
  Axis Less than $-30°$

  Short stunted person.
  Full term pregnancy
  LVH
  LBBB

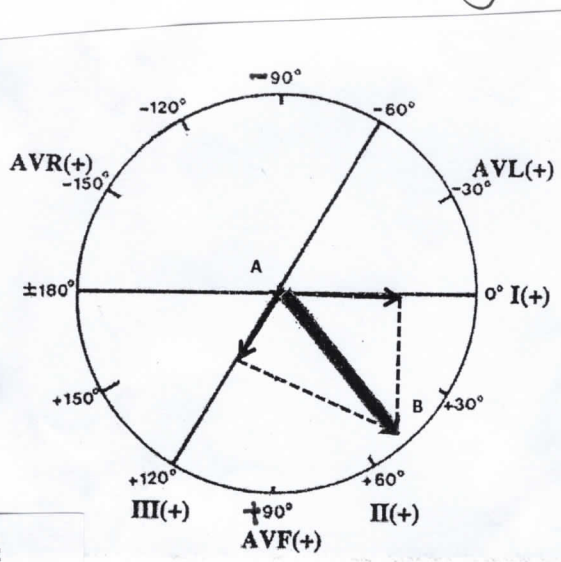
  Rt ventricular infarction

- **Determination of electrical axis :**

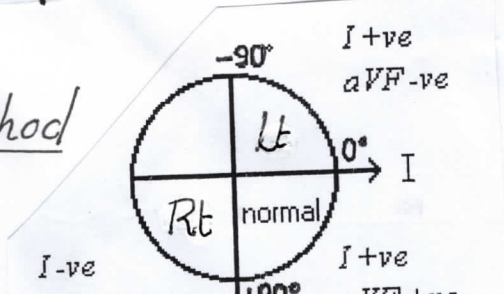
1 Standard limb leads and Einthoven's tringle

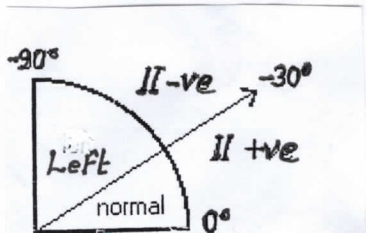


2 Lead I and lead III in hexaxial reference system



3 Quick method





Effect of myocardial ischemia on ECG

A less severe or short duration ischemia : **ST segment** Elevated or Depressed.

Injury current from ischemic depol area to surrounding normal area

Due to 1 Membrane depol caused by ++ K^+ in ECF

-- ATP < opening of K ATP channels
-- Na^+- K^+ pump

2 -- slope of phase 0 & -- conduction velocity.

a Transmural ischemia

Elevation

Away from overlying electrode

ST segment

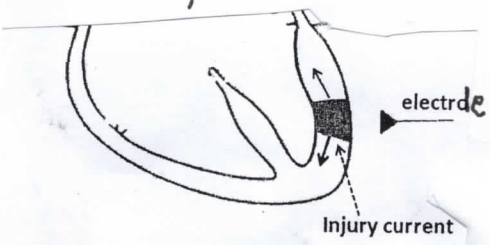
Injury current

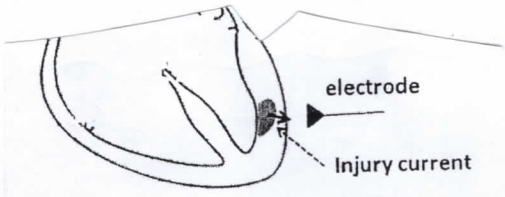
b Subendocardial ischemia

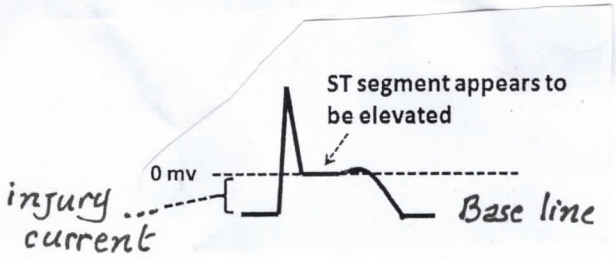
Depression

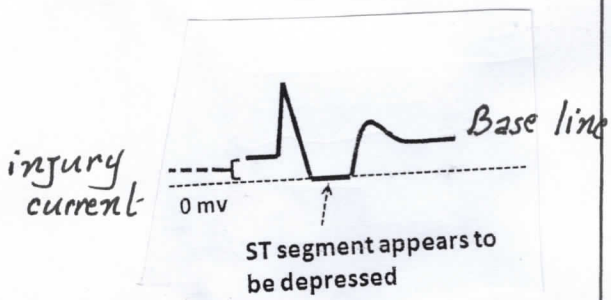
Towards overlying electrode

Depression — Base line — Elevation





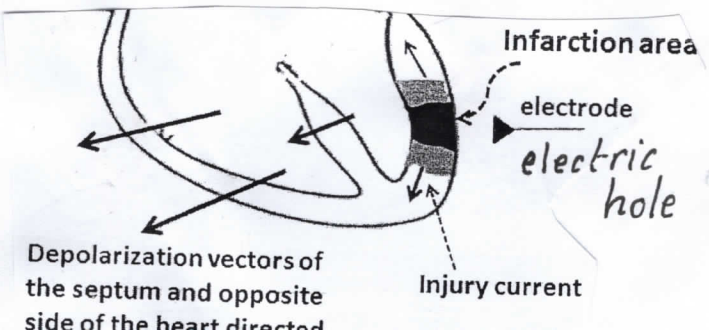


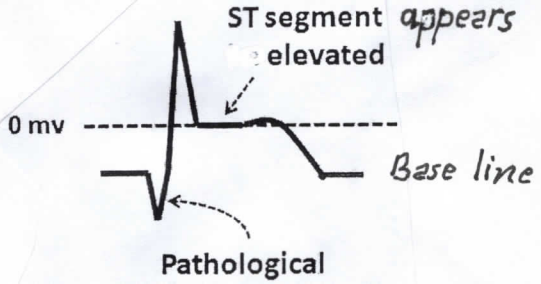


B Severe & prolonged ischemia : **Pathological Q**

Pathological Q i.e larger & longer i.e > 25% of its R

> 0.04 sec





Cardiac cycle

75 min 0.8 second.

0.1 0.3 0.5 0.7

Vent

Atria

Systole

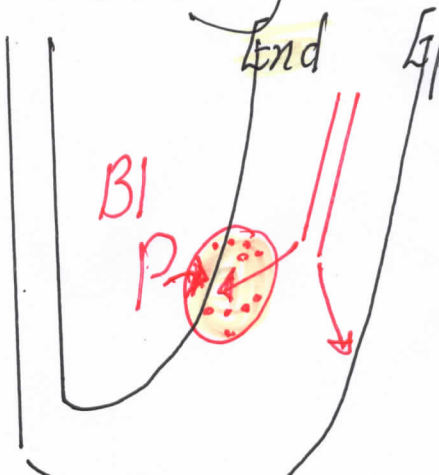
Pump

Diastole مهم جدا MCQ

Fill

Rest

Coronary Bl Flow



Phases

1 Atrial systole

2 Isometric cont.

3 Maximum ejection
rapid ejection

4 Reduced ejection

Vent. Systole

5 Protodiastolic

6 Isometric relax

7 Maximum Filling
rapid Filling

8 Reduced Filling

Ventricular systole

2 isometric

3 & 4 isotonic

Ventricular diastole

5, 6 & 7 Early

8 Mid

1 Late

Cardiac cycle

8 phases 8 changes

1 Atrial systole.

2 Isovolumetric contraction.

3 Maximal (rapid) ejection.

4 Reduced ejection.

5 Protodiastolic.

6 Isovolumetric relaxation.

7 Maximal (rapid) filling.

8 Reduced filling.

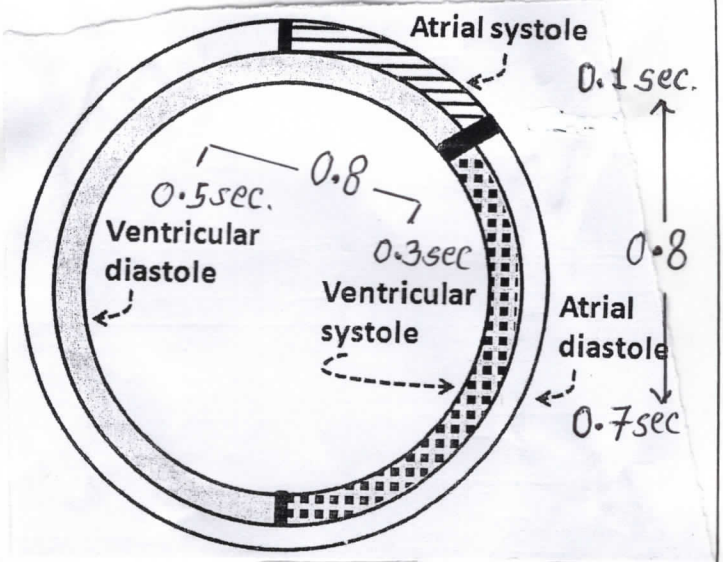
- Ventricular systole 0.3 sec.

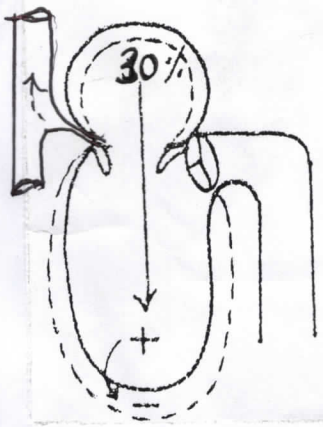
  phases 2, 3 & 4

- Ventricular diastole 0.5 sec.

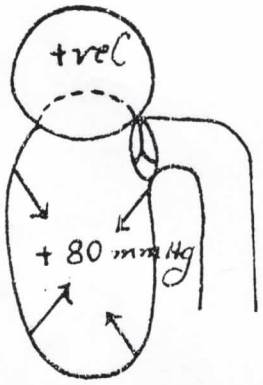
Early 5, 6 & 7 — Mid 8 — Late 1 Atrial systole

++ HR ⟶ more -- in diastole

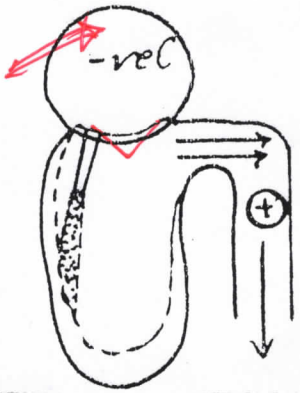




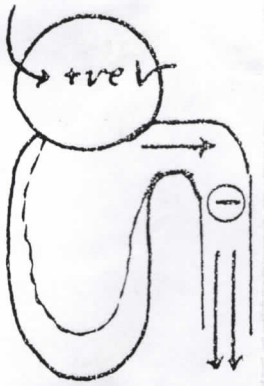
A. systole



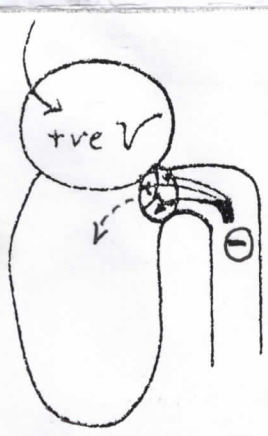
Isometric cont.

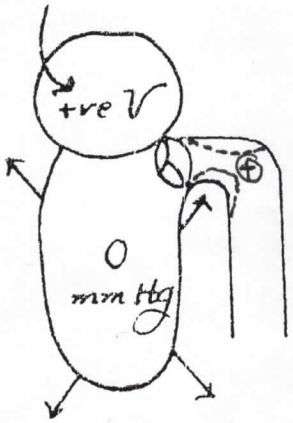


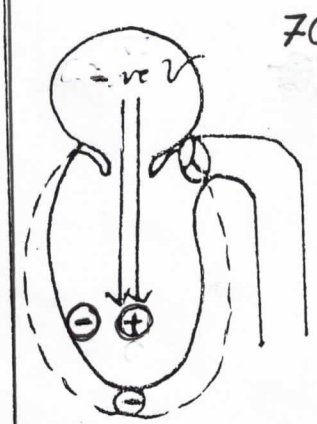
Rapid ejection

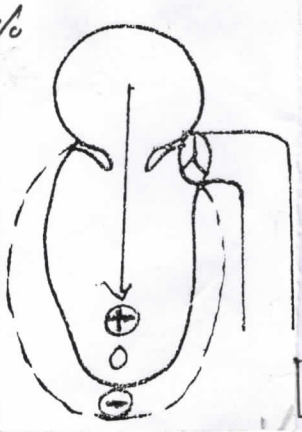


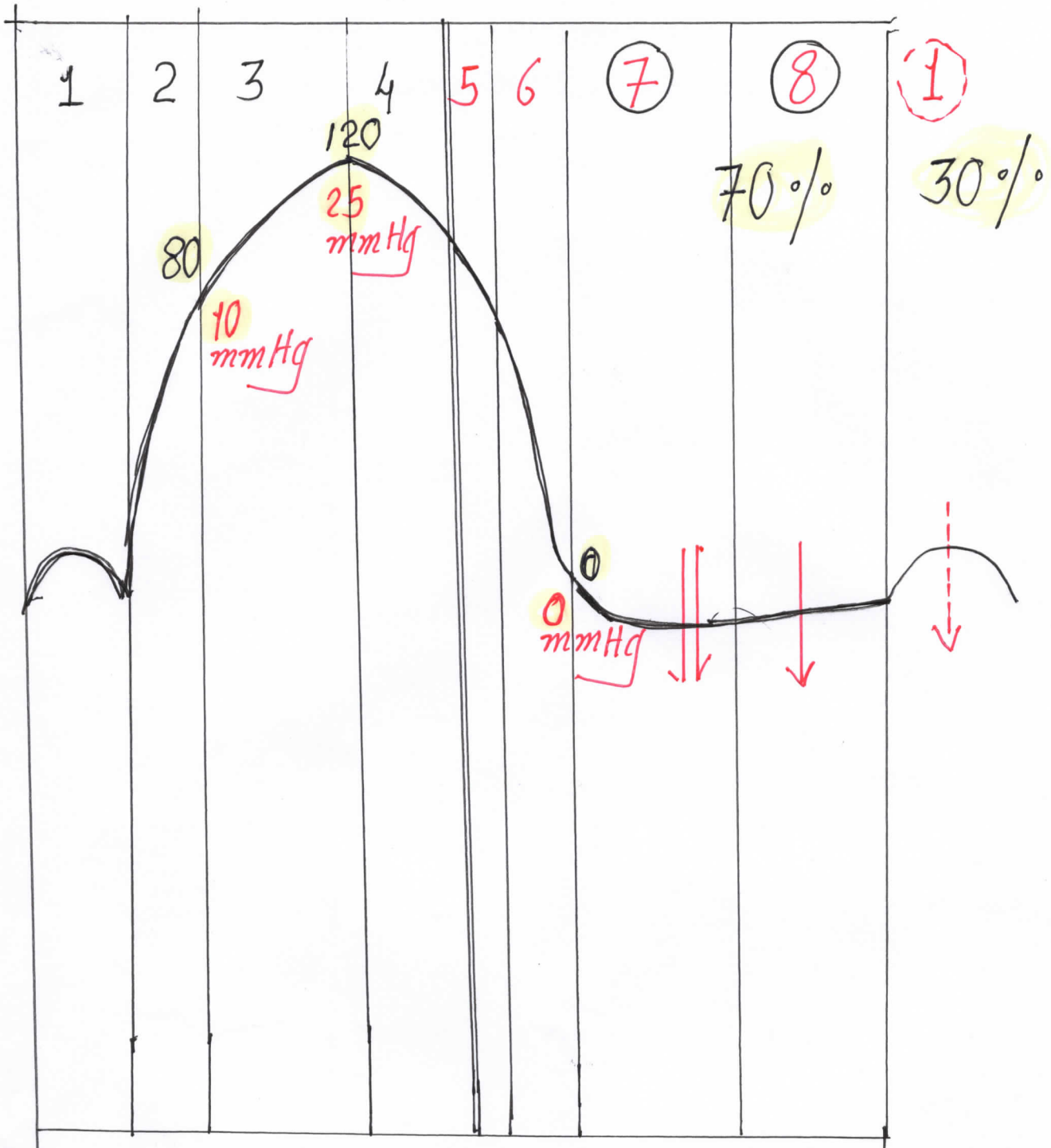
Reduced ejection

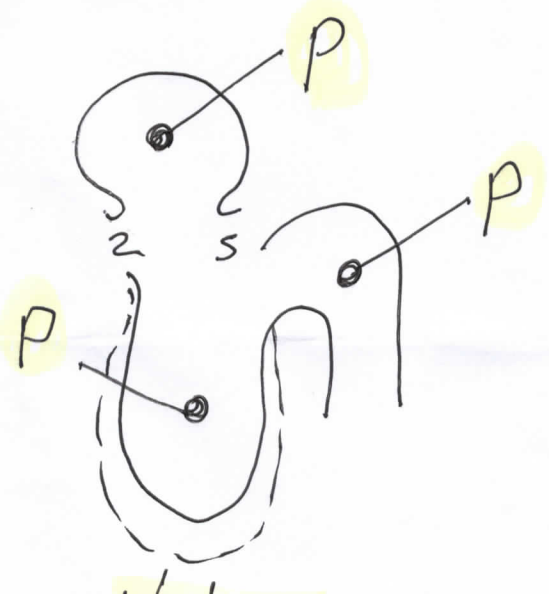






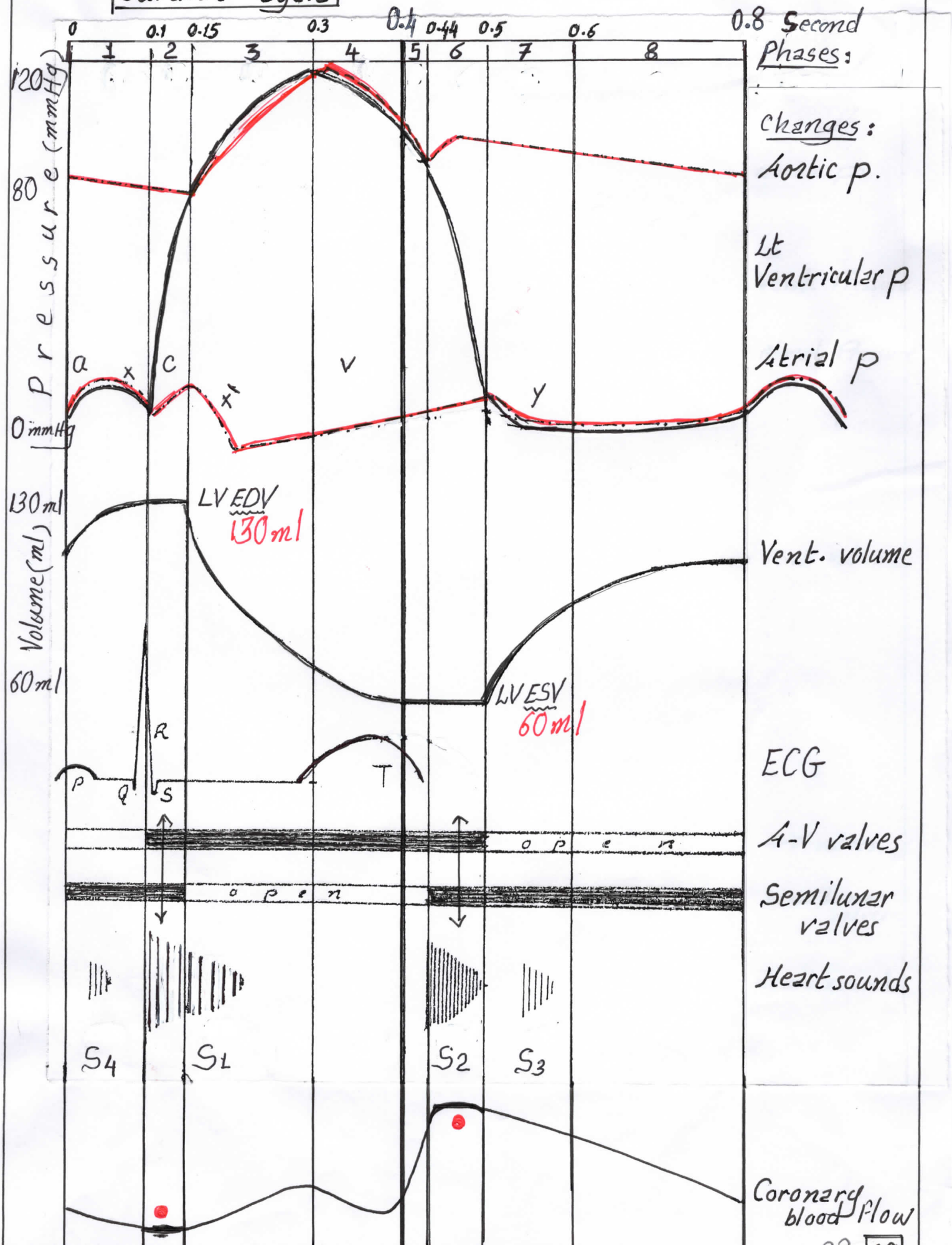




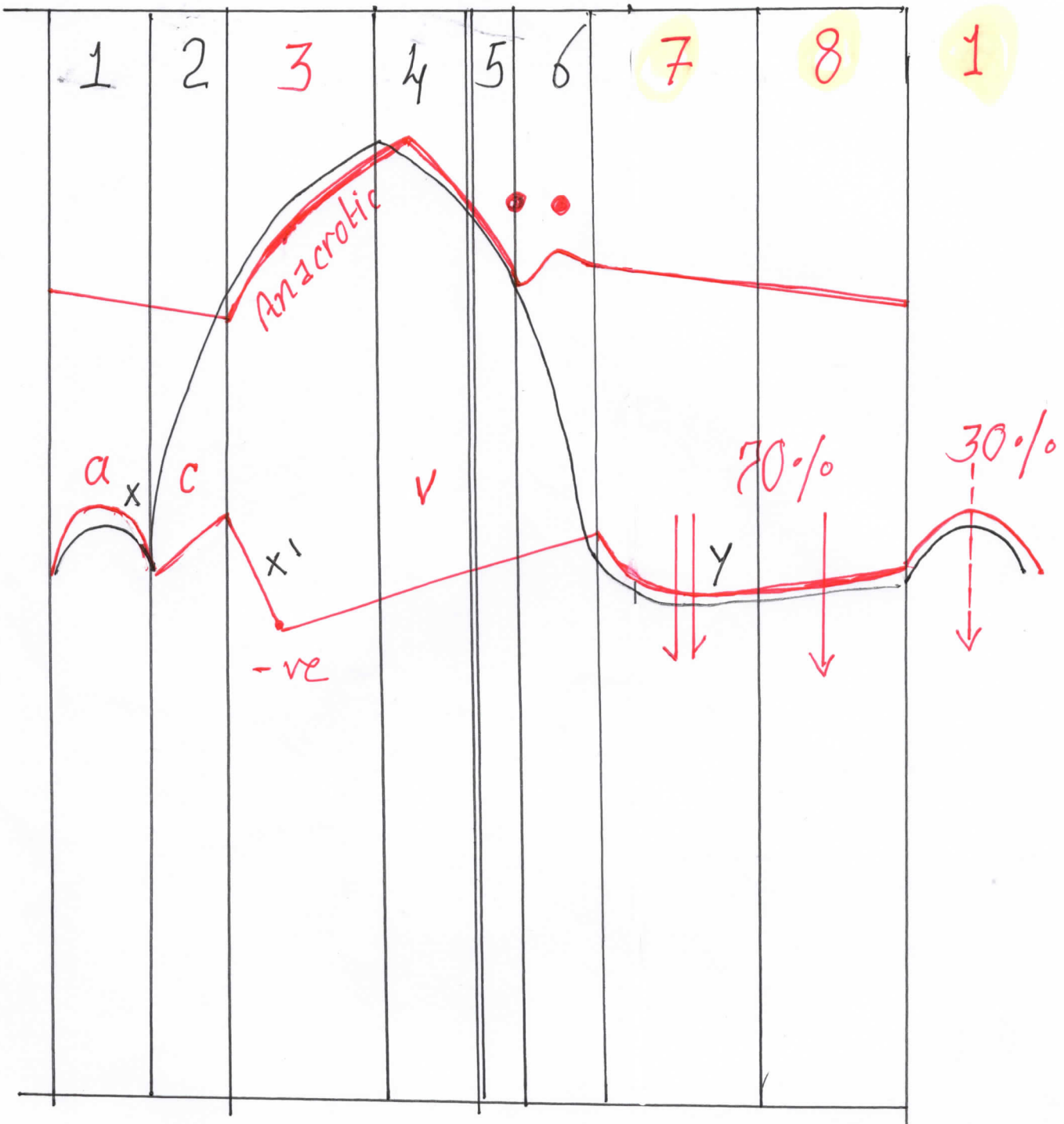


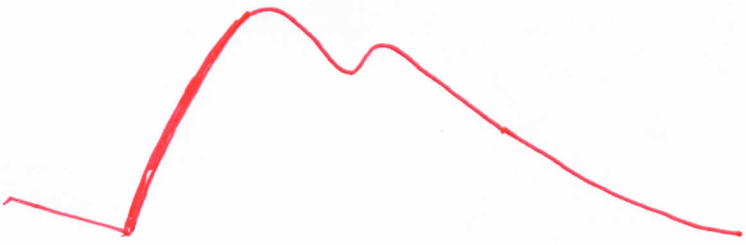
5 Valves
6 HS
7 CBF
8 ECG

Cardiac cycle



Phase / Change	Late diastole	Ventricular Systole			Early diastole			Mid dias
	1 A. systole	2 Isometric (isovol) cont.	3 Maximum (rapid) ejection	4 Reduced ejection	5 Proto diastolic	6 Isometric (isovol.) relax.	7 Maximum (Rapid) filling	8 Redu Fillin
	0.1 sec.	0.05 sec.	0.15 sec.	0.1 sec.	0.04 sec.	0.06 sec.	0.1 sec.	0.2 sec
1 Ventricular Pressure	(++) due to A. systole then (--) due to V. diastole	(++) to 80 mmHg Lt V. 10 mmHg Rt V. ends by opening of semilunar valves	(++) to maxim. 120 mmHg Lt V. 25 mmHg Rt V	(--)	(--)	(--) to 0 mmHg Lt. V. 0 mmHg Rt V	- Early (--) V. relax > V. Filling - Then (++) Vent. filling > V. relax Rate of vent. fill Rapid	(+ Sl
2 Ventricular Volume	Increased 30% EDV 130 ml	Constant	Decreased rapidly	slowly ESV 60 ml	Constant		Increase rapidly 70%	slo
3 Aoric Pressure	Catacrotic Blood leaves aorta Aortic p. drops to minimum	80 mmHg by end of this phase	Ascending Anacrotic Bl. ejected in aorta > Bl leaving aorta 120 mmHg	(Des Bl enters aorta < Bl leaves » aortic p. exceeds vent. p.	cendi Dicrotic notch (incisura) sudden closure of aortic valve	wave ++ elastic recoil of aorta	ng) Blood leaves ao to peripheral vess	
4 Atrial Pressure	a +ve Atrial systole	X -ve Bl leaves atria to vent.	C +ve Bulge A-V cusps	X -ve Decline	V +ve Accumulation of VR in atrium		Y -ve Passage of VR from atria to vent	
5 A-V valves	open	C	L	O	S	E	D	open
Semilunar »	C l o s e d		open	open	open	C l o s e d		
6 Heart Sounds	4th HS (S_4) vent. filling by A. systole	1st HS (S_1) Sudden closure of A-V valves Long Low pitch LUB				2nd HS (S_2) closure of aortic & pulm. valves	3rd HS S_3 rapid vent. filling	
7 ECG	P starts 0.02s before A cont	QRS starts 0.02s before V. cont		T wave begins				
8 Coronary bl. Flow		MINIMUM	Slight ++ with ++ aortic p	Slight -- with -- aortic p		MAXIMUM		


1
2
3
4
5
6
7
8
1
Anacrotic
a
x
c
x1
v
-ve
y
70%
30%




a
x
c
x1
v
y

Rt atrial pressure (Jugular venous pulse)

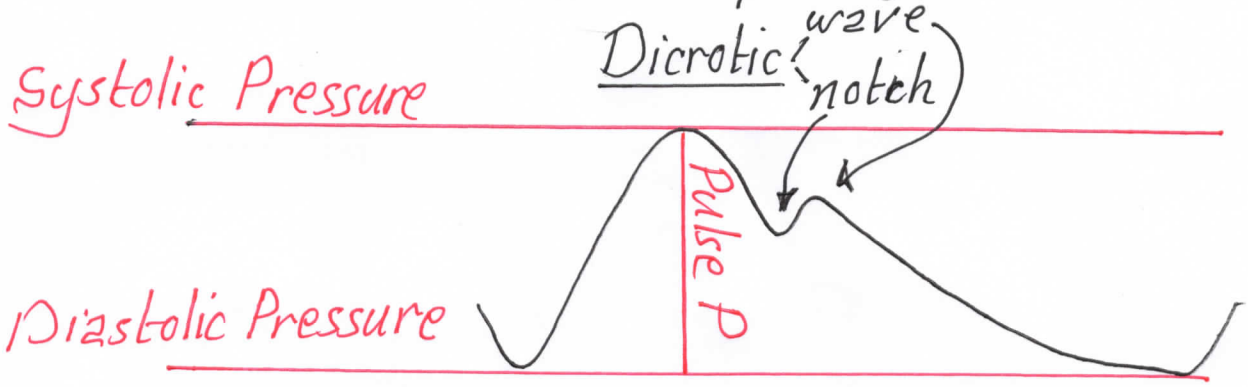
Clinical importance

1 No a ∴ A fibrillation

2 Cannon a wave ∴ A-V dissociation
e.g 3rd degree heart block
V Tachycardia

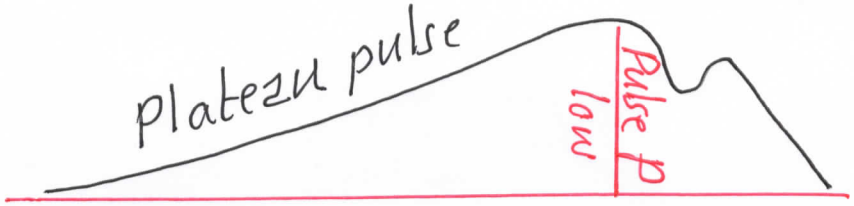
3 a wave represents atrial cont
c wave represents vent cont
So, a-c interval = P-R interval in ECG

Aortic pressure (carotic pulse) curve.

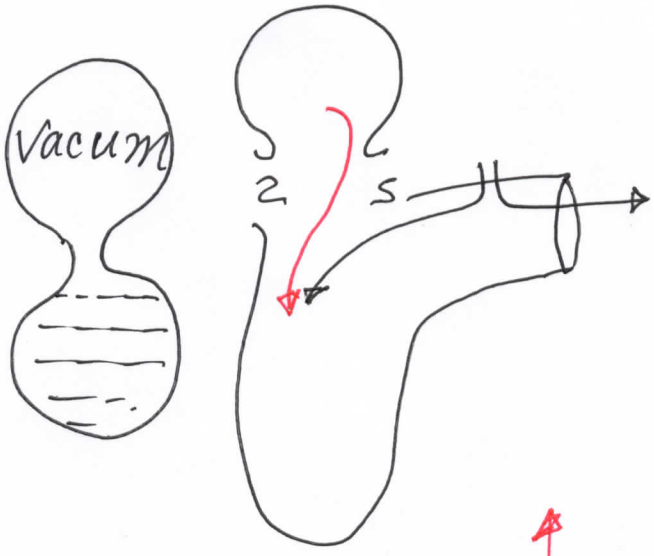


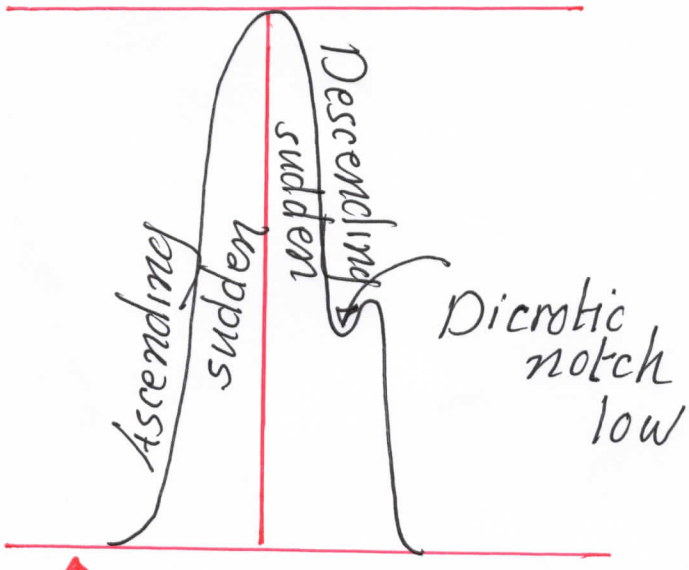
Clinical importance

1 Aortic stenosis



2 - Aortic regurgitation Water Hammer pulse





PP = S - D

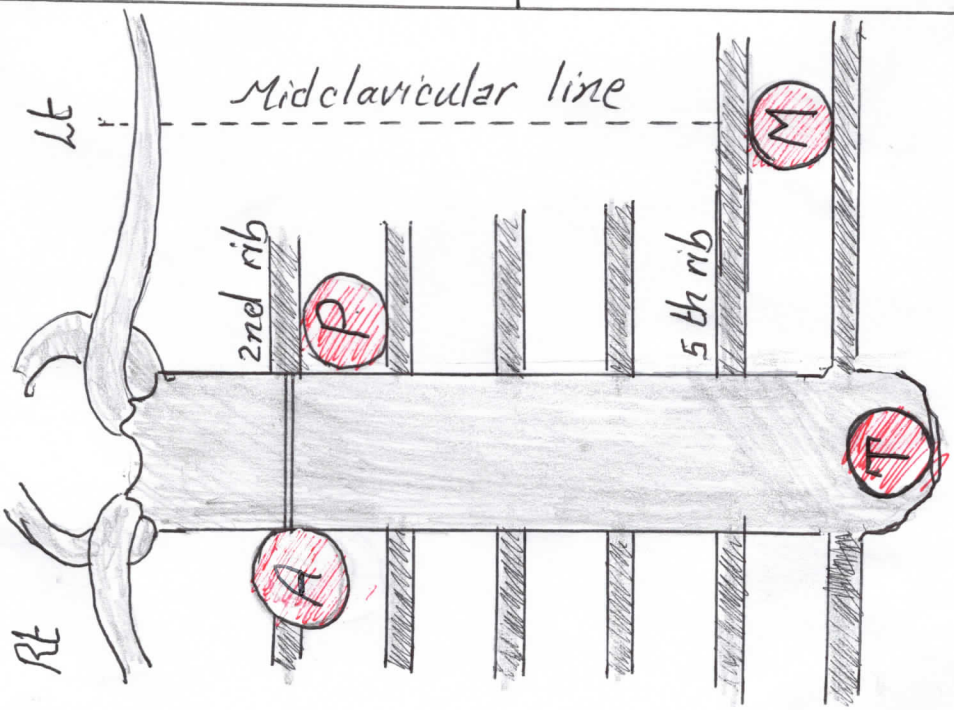
Heart sounds

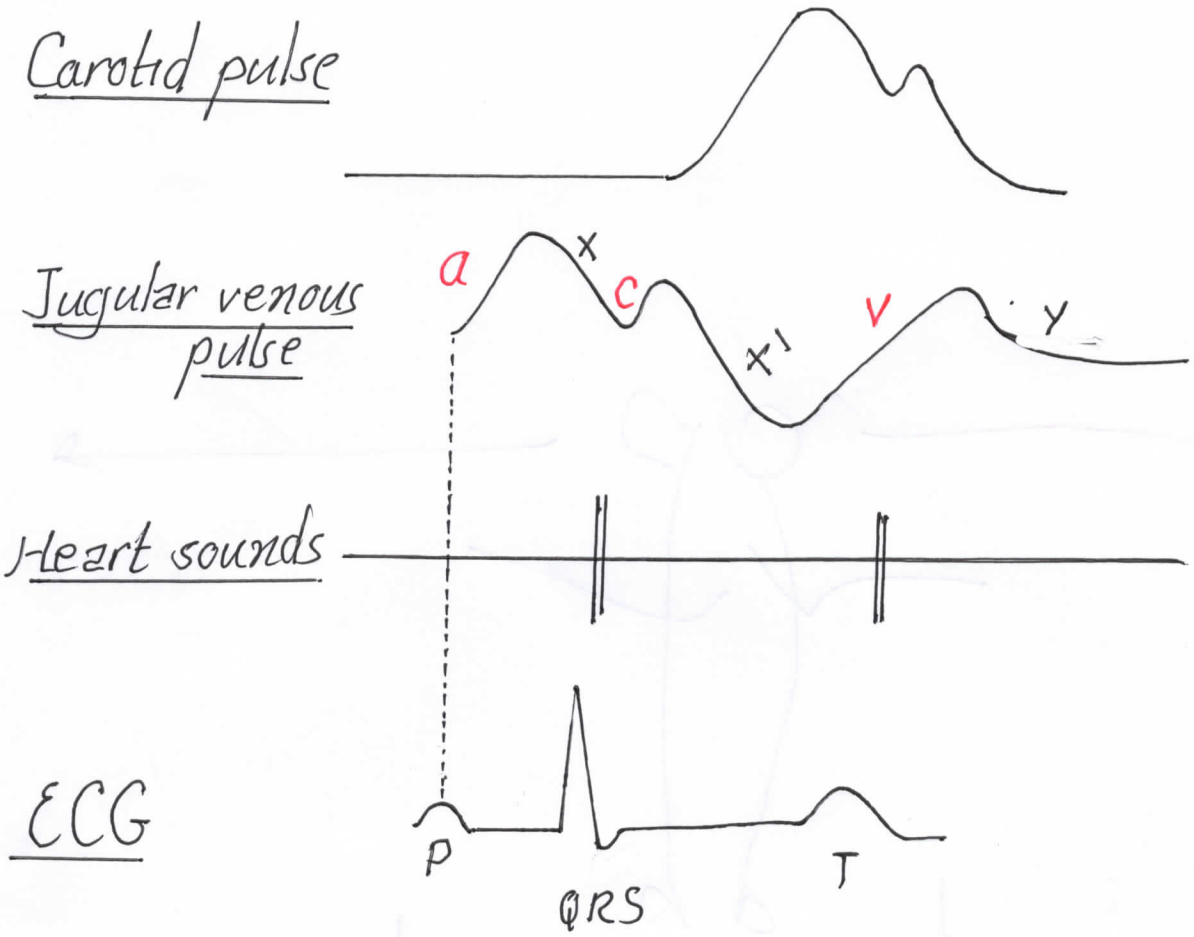
Stetho scope

Phono cardio gram

First heart sound (S_1)	Second heart sound (S_2)
A-V valves Diastole end (Start of systole) Apex of heart (mitral) lower end of sternum (tricuspid) Long isovol. cont rapid erect 0.15 s Low pitch 30 Hz LUB	Semilunar valves Systole end (beginning of diastole) Second intercostal Aortic (Rt) Pulm (Lt) Short isovol. relax. 0.12 s High pitch 50 Hz Dup (dub)

S_3	S_4
Rapid flow of blood from atria to ventricle	
Rapid filling phase	Atrial systole phase
Normally Audible in children, athletes or may in pregnancy	Non audible.
Abnormal CHF	Failing of Hypertrophic Lt vent.

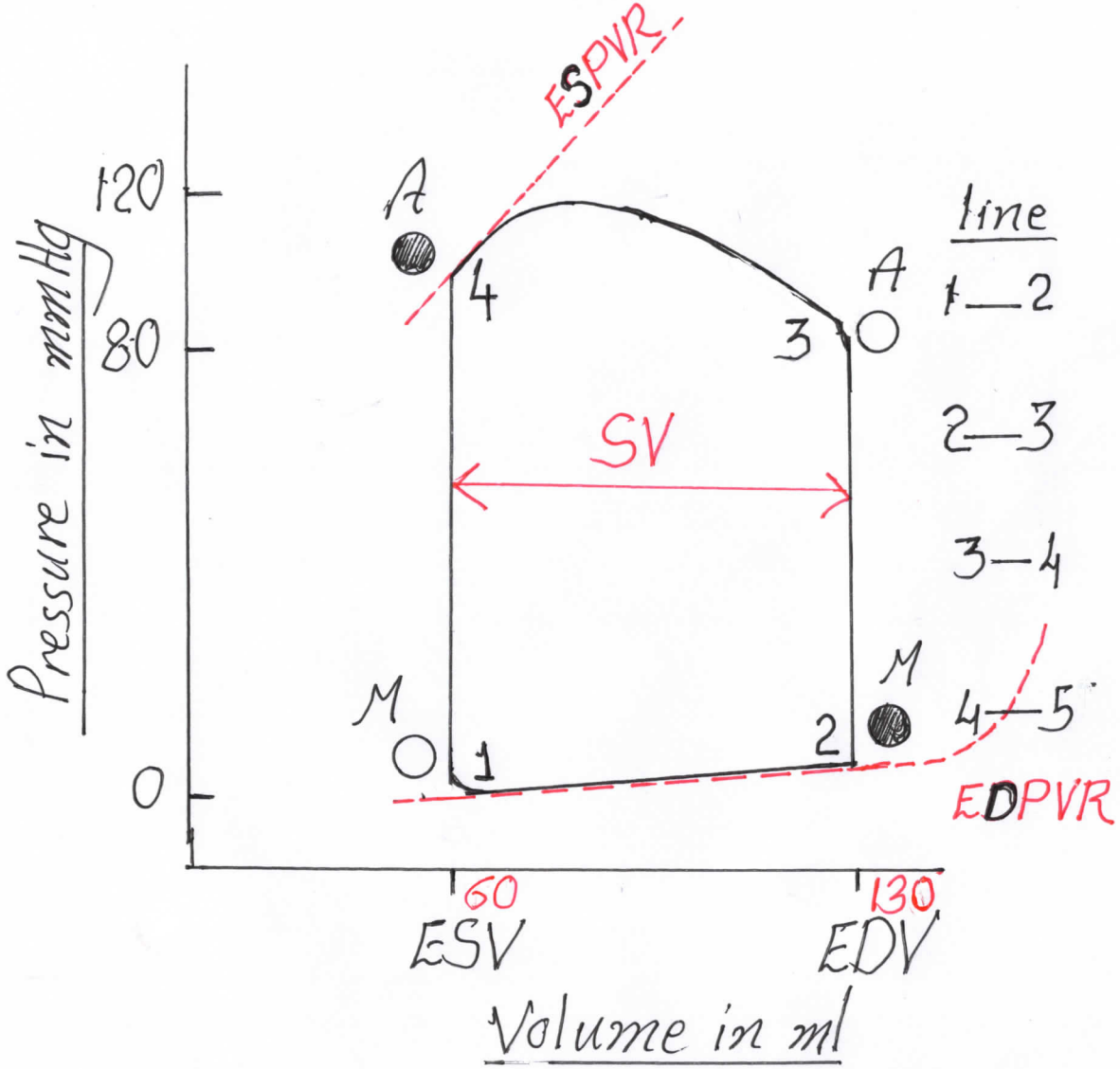




- a wave — atrial cont. follows P wave of ECG
- c wave — vent. cont. follows QRS complex of ECG

- x` wave occurs with carotid pulse.
  i.e normally jugular vein collapses with carotid pulsation
- v wave occurs with descending limb of carotid pulse.
  i.e with collapsing of carotid pulse

Left ventricle Pressure Volume loop



4 phases

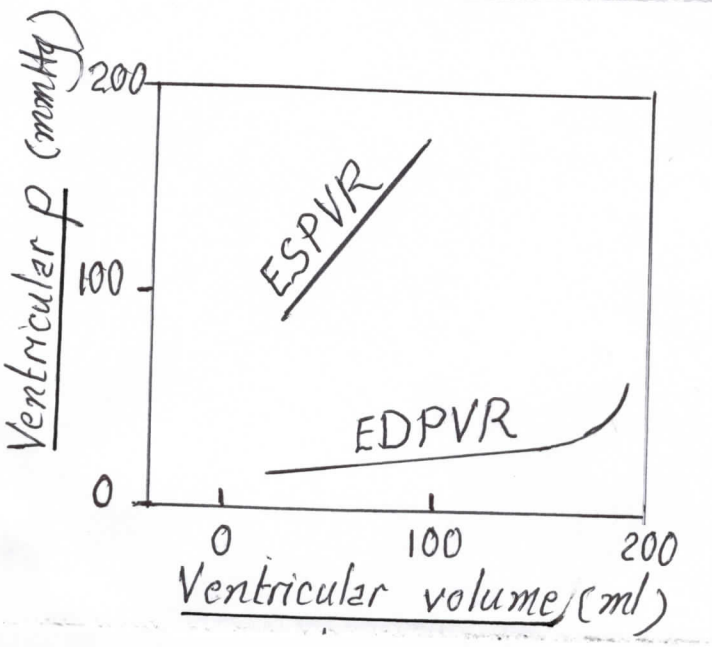
Line	Represents	Vent volume	Vent P
1—2	V filling phases	++ from ESV to EDV	slightly
2—3	Isovol. cont. phase ±		++ to 8
3—4	Ejection phases	-- from EDV to ESV	++ to 12 then
4—5	Isovol relax phase ±		-- to 0

Point	
1	Mitral valve opens
2	" " closes
3	Aortic valve open
4	" " closes

Ventricular Pressure Volume Relationship

Modified length-tension relation.

Volume represents length. Pressure represents tension.



EDPVR

++ EDV ⟶ ++ vent. p.

= passive length tension relation.

ESPVR

++ EDV ⟶ ++ ESP (aortic clamp)

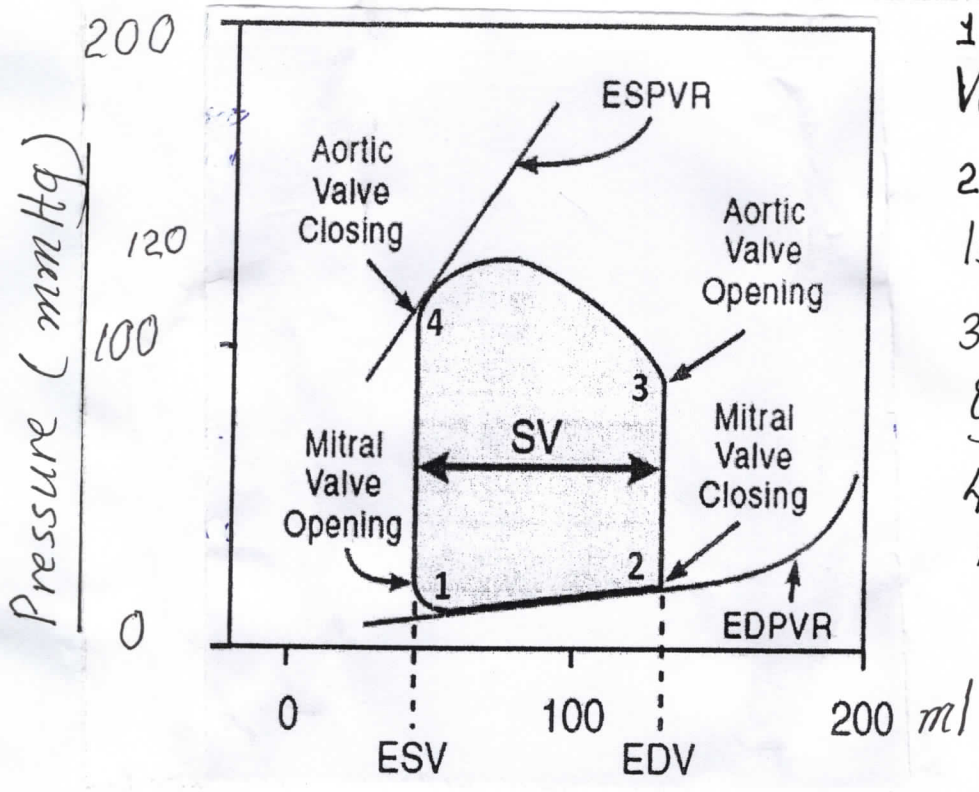
= active length tension relation.

its slope depends on inotropic state.

Pressure - Volume loop (Lt vent. pressure - volume loop)

Area ∝ Vent. work

4 phases



1 ——— 2
Vent. filling phase

2 ——— 3
Isometric cont. phase

3 ——— 4
Ejection phases

4 ——— 5
Isometric relax. phase

1 Mitral opens
2 Mitral closes
3 Aortic opens

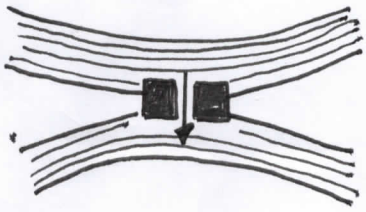
Myocardial properties

- Structure of cardiac myocyte (muscle fibre)
  Length 100 μ & diameter 25 μ.

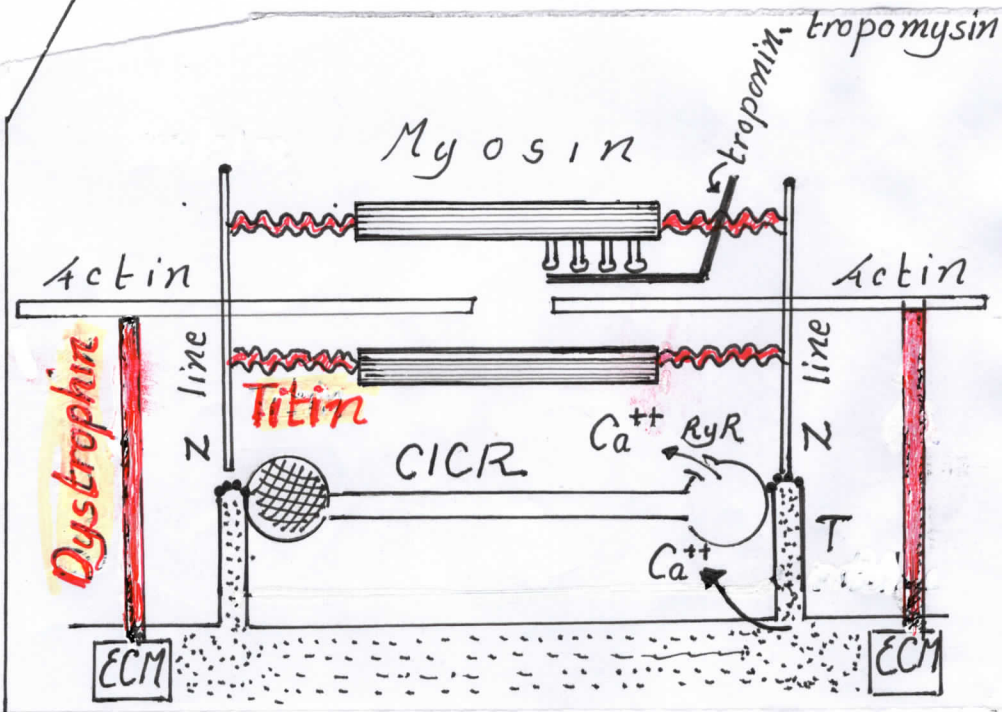
Resembles

Smooth muscle — Syncitium

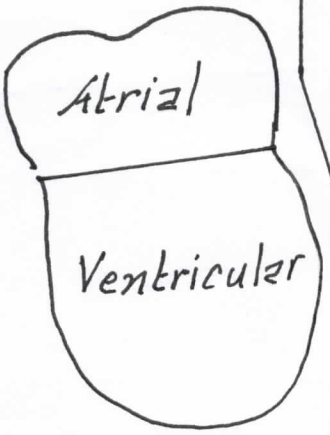
Skeletal muscle — Straited



Intercalated disc
Gap junction



2 functional syncitia



- T tubule at Z line

(connectin)

- Titin Giant filamentous elastic ptn
  Connects myocin to Z line.
  Elasticity of myocytes ↷ passive mechan. properties
- Dystrophin Rod shaped Ptn.
  Connects actin to ECM.
  Stability of myocytes

Excitation Contraction Coupling like sk ms

3 differences

SERCA Sarco-Endoplasmic Reticulum Ca^{++} ATPase.

(1) Contraction Ca^{++} **TWO** sources

a ECF via L type Ca channels — Sarcolemma, T tubules
small amount

b SR via RyR Ca^{++} channels
LARGE amount
CICR

[2] Relaxation Ca^{++} removal **THREE** mechanisms

a SR via ATP dependent Ca^{++} pump (SERCA)

b, c] OUT of myocytes via sarcolemma by [ATP dependent Ca^{++} pump; Na^{+}-Ca^{++} exchanger

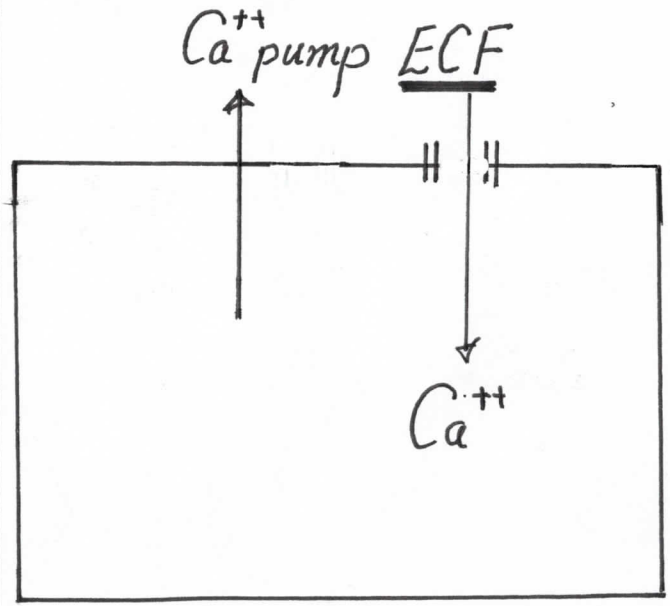
[3] Ca^{++} plays MAIN ROLE in determining force of contraction.

. ++ Ca^{++} ⟶ +ve inotropic

15

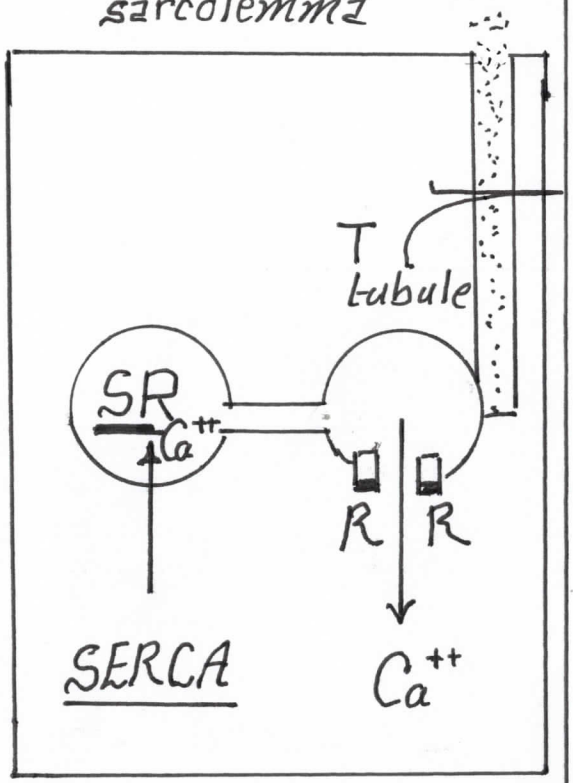
2

Smooth ms (Slow)



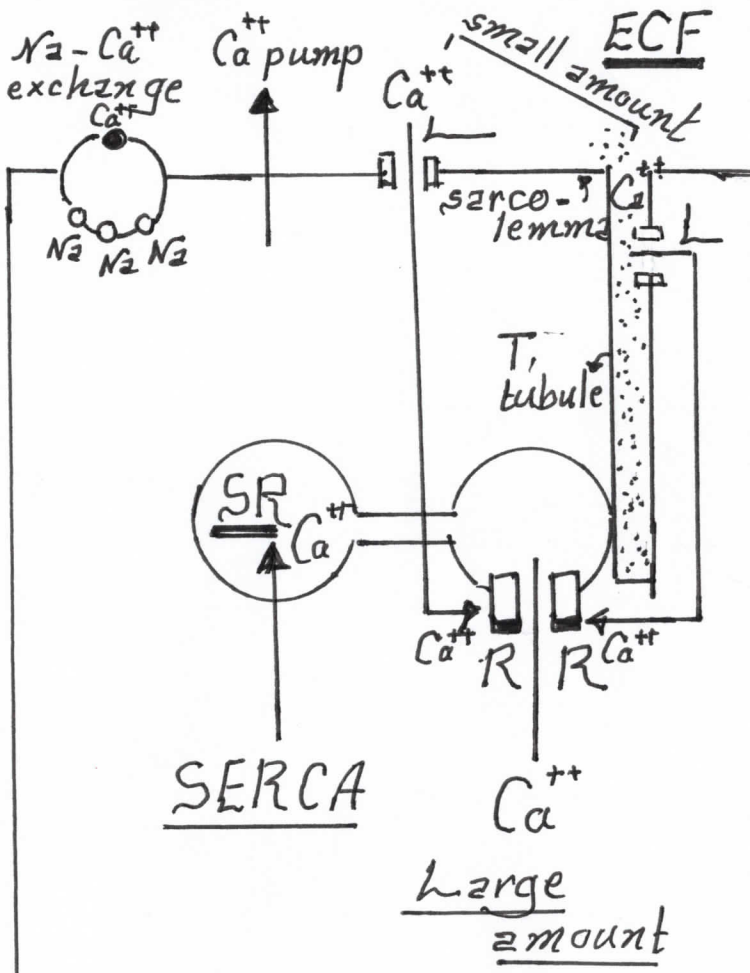
Mainly ECF

Skeletal ms (Fast)



SR

Cardiac ms



CICR

ECF & SR (large amo

Regulation of contractility (Inotropic state):

Positive inotropic

1 β adrenergic receptors
++ cAMP ⟶ ++ Ca^{++}.
2 Glucagon ++ cAMP.
3 ++ Ca^{++} in ECF.
4 Drugs:
a Digitalis ⟶ ++ Ca^{++}.
b Xanthine ++ cAMP.

Negative

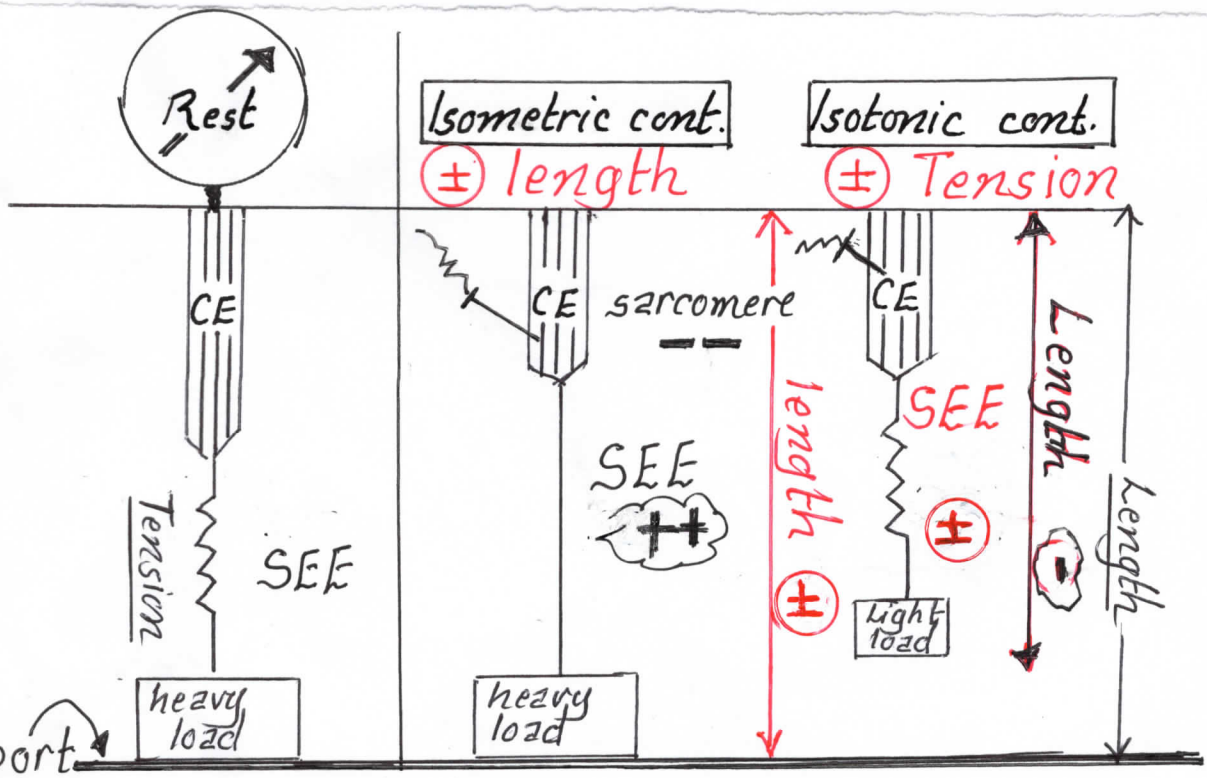
1 Muscarinic (M_2) receptors
-- cAMP.
2 Adenosine -- cAMP.
3 Hypoxia -- ATP.
4 Drugs:
a Ca^{++} channels blockers.
b Anesthetic drugs.

Regulation of relaxation:

(1) β adrenergic receptors
++ cAMP ⟵ activates SERCA pump.
-- binding of troponin C to Ca^{++}.

(2) Myocardial ischemia
++ Ca^{++} ⟶ inhibits relaxation.

Isometric and isotonic contraction of isolated Cardiac ms



c AMP —Activate→ ptn kinase A → phosphorylation

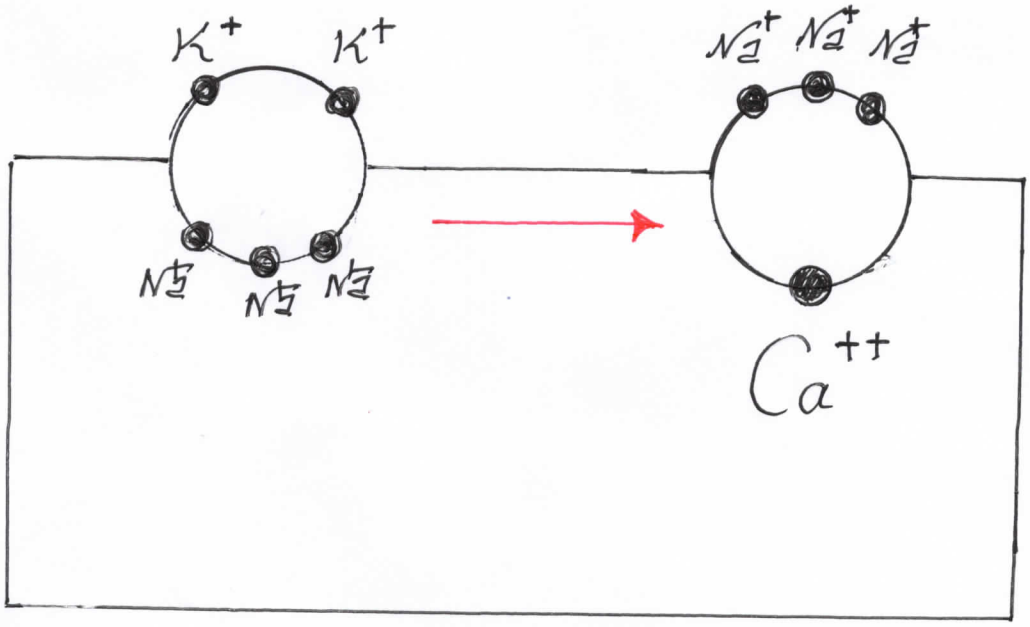
L type Ca^{++} ch. — Open longer time

Certain site in SR — Release more Ca^{++}

Digitalis

inhibits

modulates



Preload degree of stretch → **Before** muscle contracts

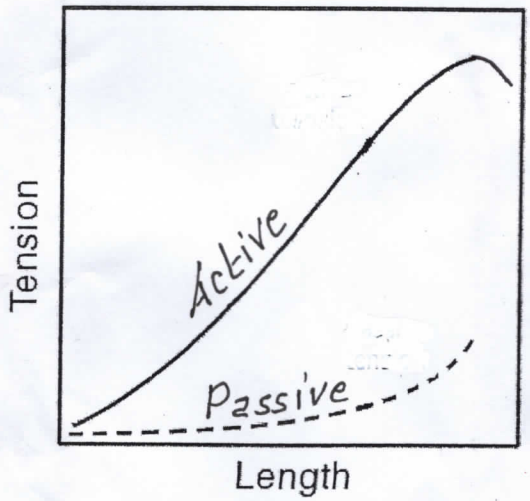
→ **Passive** tension $\xrightarrow{\text{measures of}}$ Preload

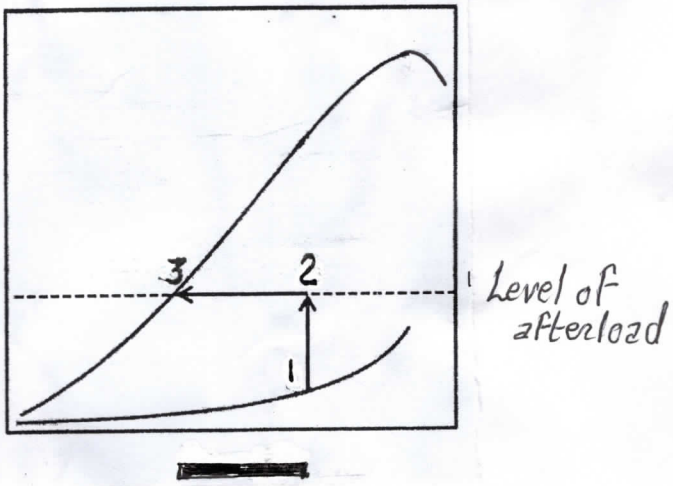
Afterload load against which muscle contracts.

→ **Active** tension $\xrightarrow{\text{measure of}}$ Afterload

Indicators of contraction

Isotonic cont.	Isometric cont
Degree of shortening	Degree of active tension
Velocity of shortening	





degree of shortening during cont. (represented by line 2-3)

Length-Tension relationship

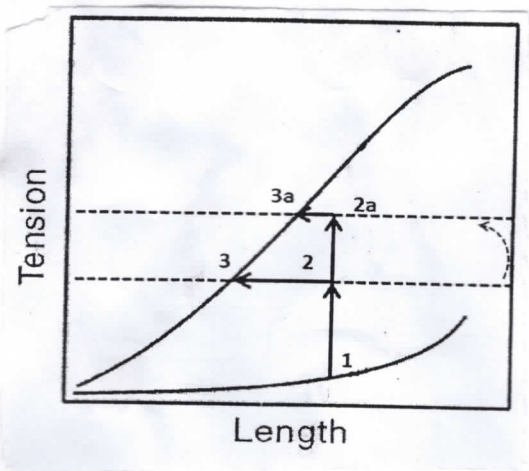
Four major factors affecting performance of isolated C. muscle

1. Preload ++ → ++ within limits
2. Afterload ++ → −−
3. Inotropic state +ve → ++
4. Frequency ++ → ++ then ±

1 Afterload

a Degree of shortening $\propto \frac{1}{\text{Afterload}}$ length tension curve

b Velocity of shortening $\propto \frac{1}{\text{Afterload}}$ load velocity curve



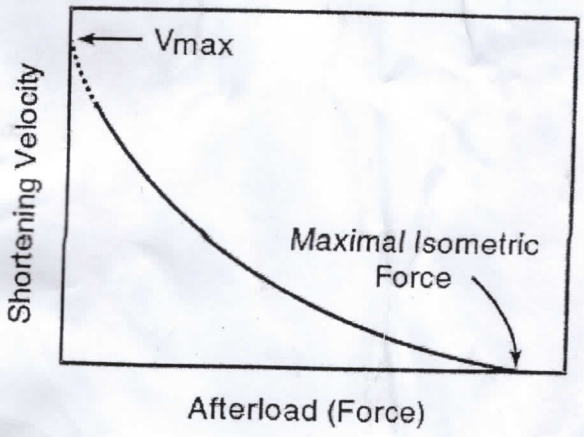
Higher afterload
Level of afterload

1 isometric cont → 2a

2a isotonic cont → 3a

2a Degree of shortening → 3a
Smaller

Effect of increasing afterload on cardiac muscle shortening



Velocity

0 Load > max: tension

V max. Load 0

extrapolated point

Effect of increasing afterload on velocity of shortening of cardiac m

---

2 Preload

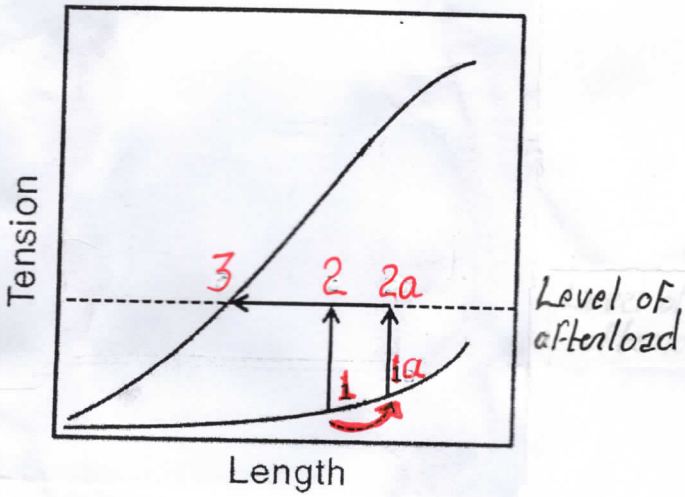
a Degree of shortening ∝ Preload within limit

b Velocity of shortening ∝ Preload within limit

++ preload a ++ velocity curve shifts upwards & to Rt

b V max is not changed.

2 Preload



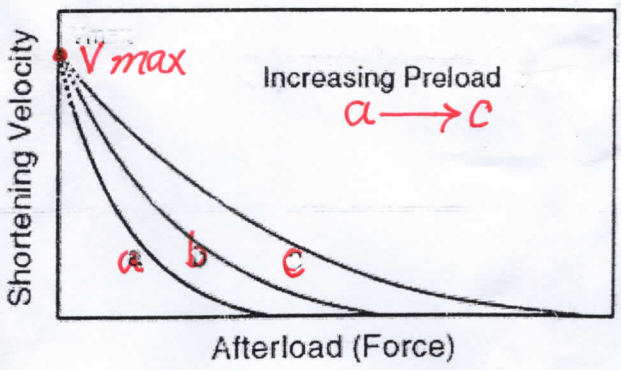
Effect of increasing preload on cardiac ms shortening

1a → isometric cont → 2a

2a → isotonic cont → 3

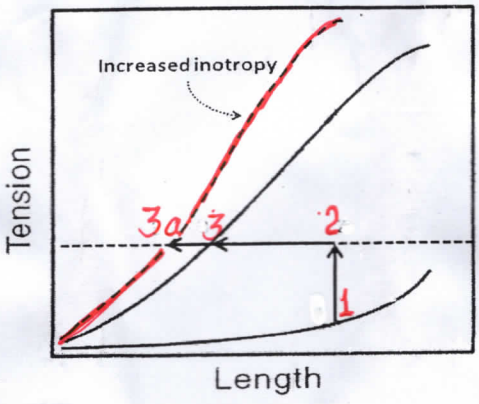
2a → degree of shorten. → 3

Greater



Effect of increasing preload on velocity of shortening of cardiac ms. (V max constant)

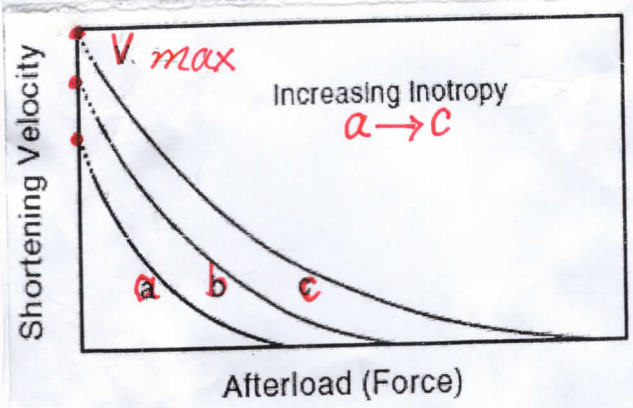
3 +ve Inotropic



Effect of increased inotropy on degree of shortening of cardiac muscle

2 → degree of shorten. → 3a

Greater



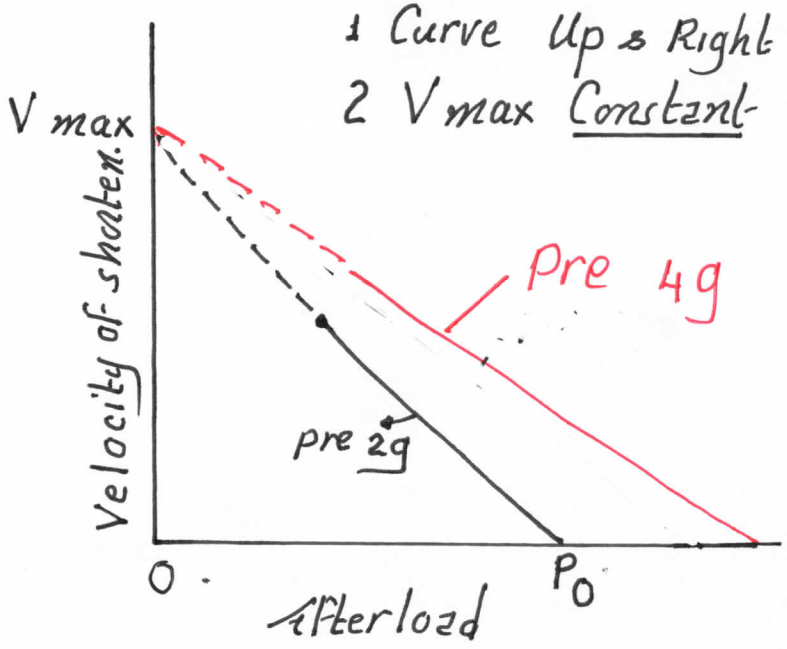
Effect of increased inotropy on velocity of shortening of cardiac ms (Vmax increased)

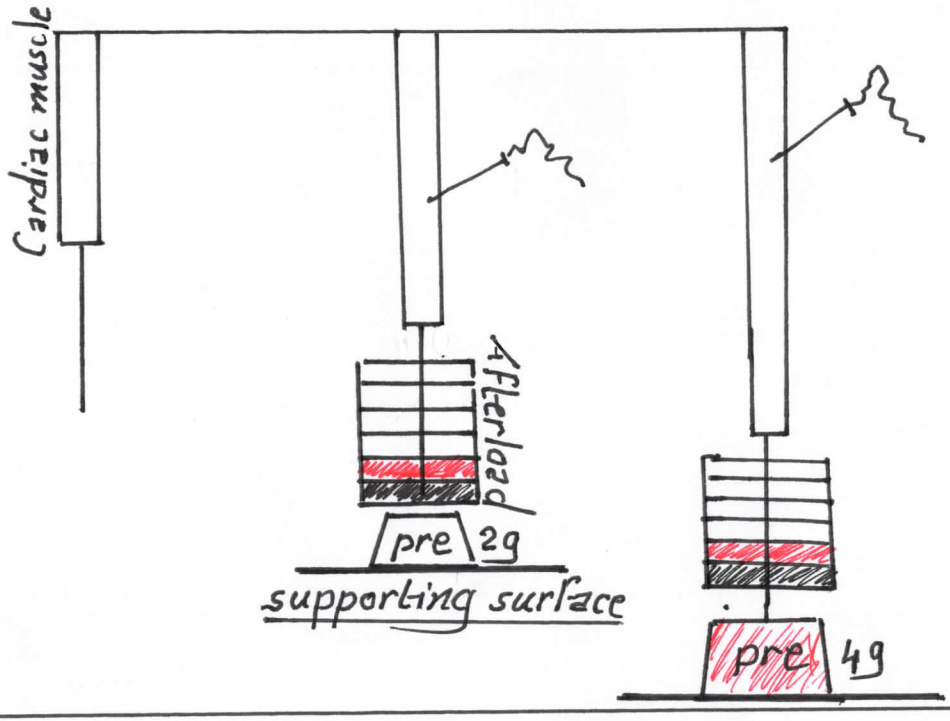
Increased velocity of shortening i.e Curve shifts upwards ⇒ to Rt

++ preload

1 Curve Up & Right

2 V max Constant

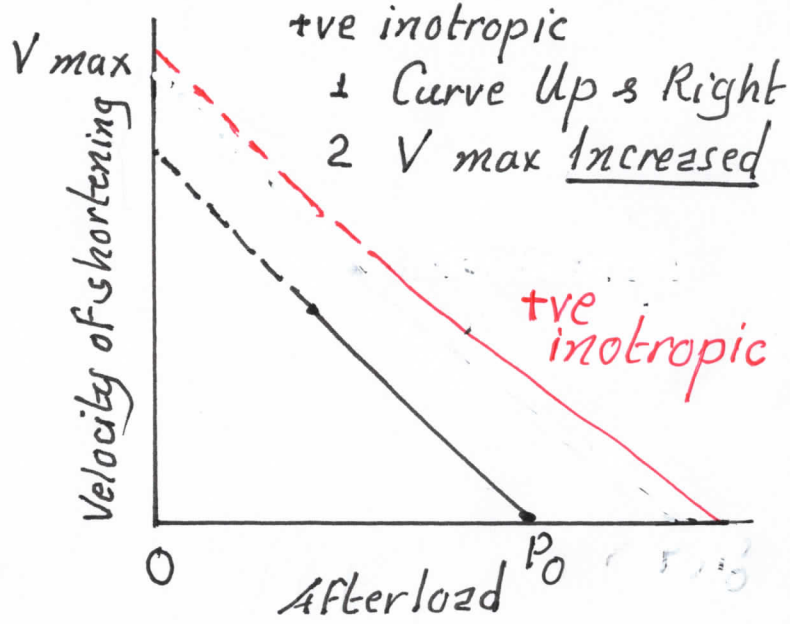


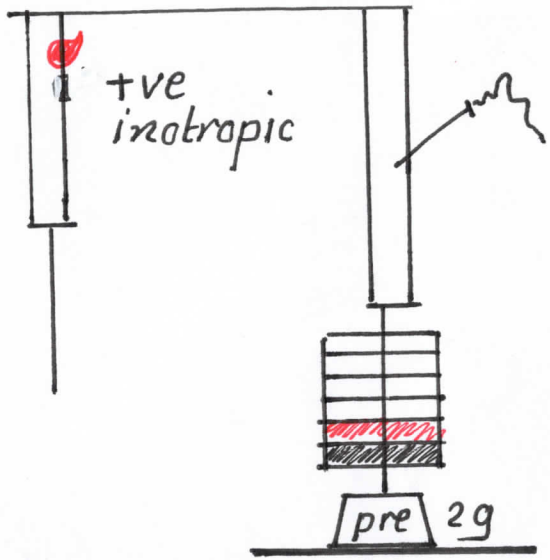


+ve inotropic

1 Curve Up & Right

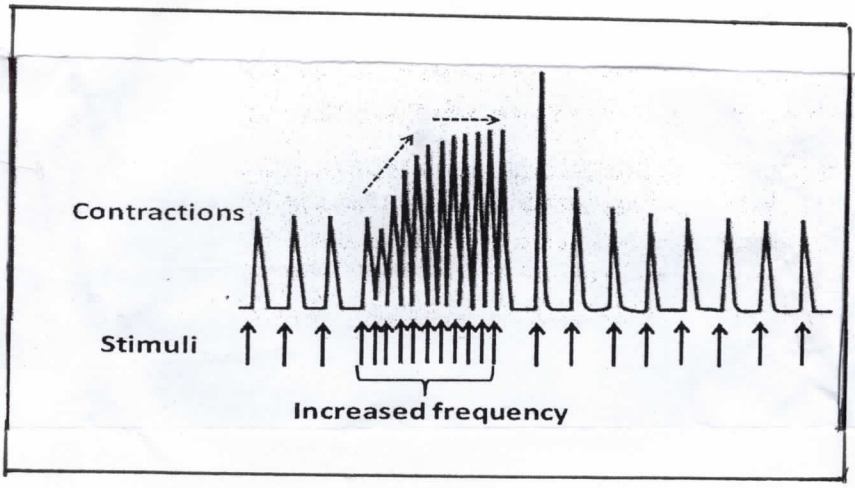
2 V max Increased





4 Frequency

++ Frequency ⟶ gradual ++ in Force
then higher steady state.

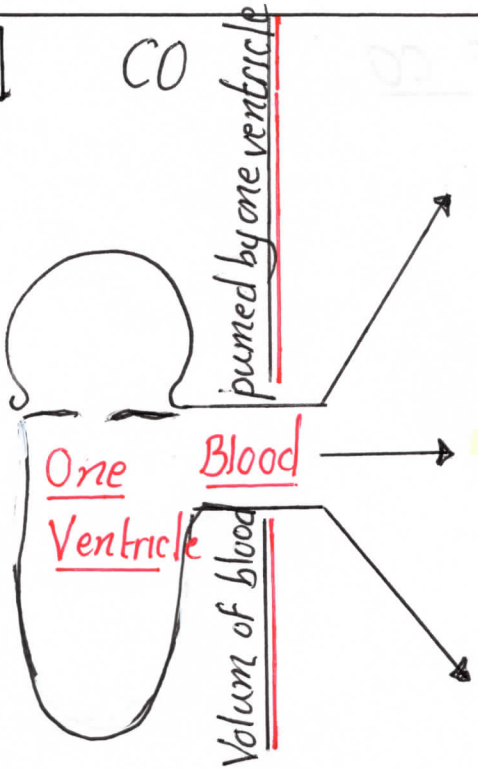


Staircase (Treppe) phenomenon.

<u>Cause</u> ++ Ca^{++} concentration.
no enough time for complete removal of
released Ca^{++} ⟶ ++ Ca^{++} conc. in myocytes.

Cardiac output CO

- **Definitions:**
  - **Stroke volume (SV)**
  - **Cardiac output (CO)** (Minute volume)
  - **Cardiac index (CI)**



Beat

$SV = EDV - ESV$

130 - 60

Minute

$CO = SV \times HR$

70 x 70

Minute / sq. m.

5 / 1.7

3.2 L / m^2

- **Various conditions that affect CO:**

Increased:		Decreased:	No change:
• Excitement & anxiety	up to 100%	• Sitting or Standing from lying 30%	• Sleep
• Exercise	up to 700%	• Rapid arrhythmia	• Moderate change in temp
• Eating	30%	• Heart diseases	
• Exposure to high temp.			
• Epinephrine			
• End of pregnancy			

- **Determination of CO:** $= SV \times HR$

  $SV = EDV - ESV$ measured by echocardiogram

- **Control of CO:** refer to performance

  (1) HR More important

  (2) SV ++ by 1 ++ preload affects EDV

  2 -- afterload " ESV

1 Heart rate

3

60 ← HR alone → 150

CO: Decreased | constant | Decreased

= ↓SV × ↓HR

= ↑SV × ↓HR

= ↓SV × ↑HR

= ↑SV × ↑HR

Exercise

physiol. condition

Increased

2 Stroke volume

3

1 Preload = Degree of stretch of C. myocytes before cont

Volume

Measured by

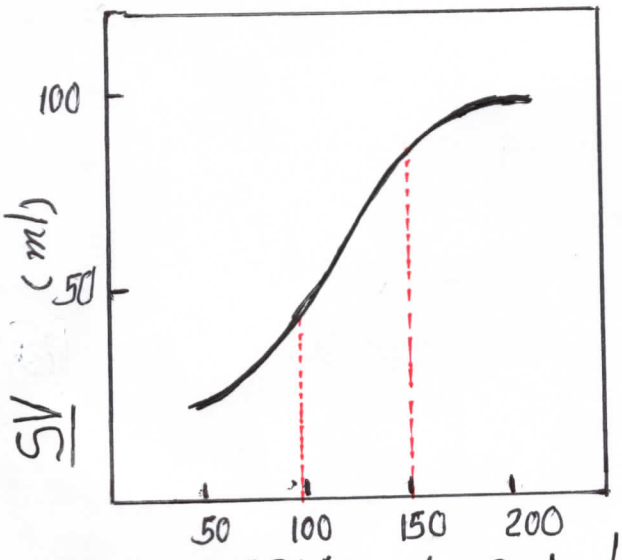
Sarcomere length / Myocyte

EDV (intact heart)

Depends on VR (RAP)

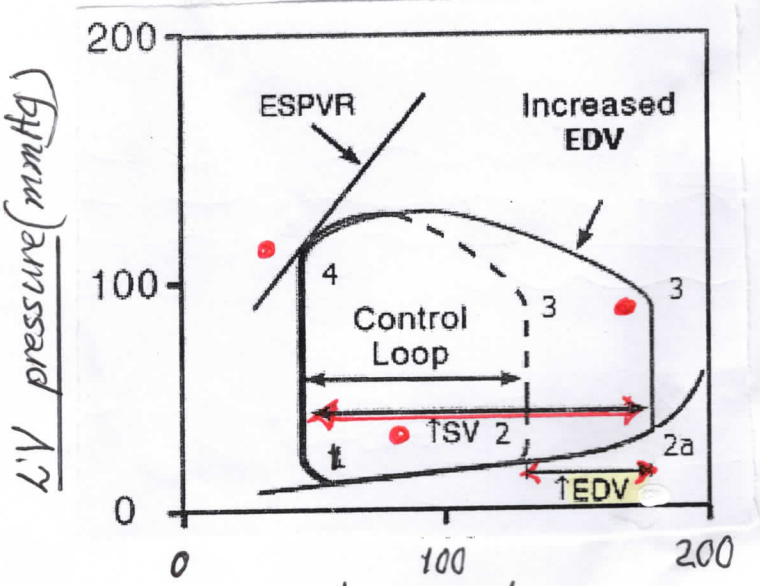
i.e Starling law

Heterometric autoregulatory mech.



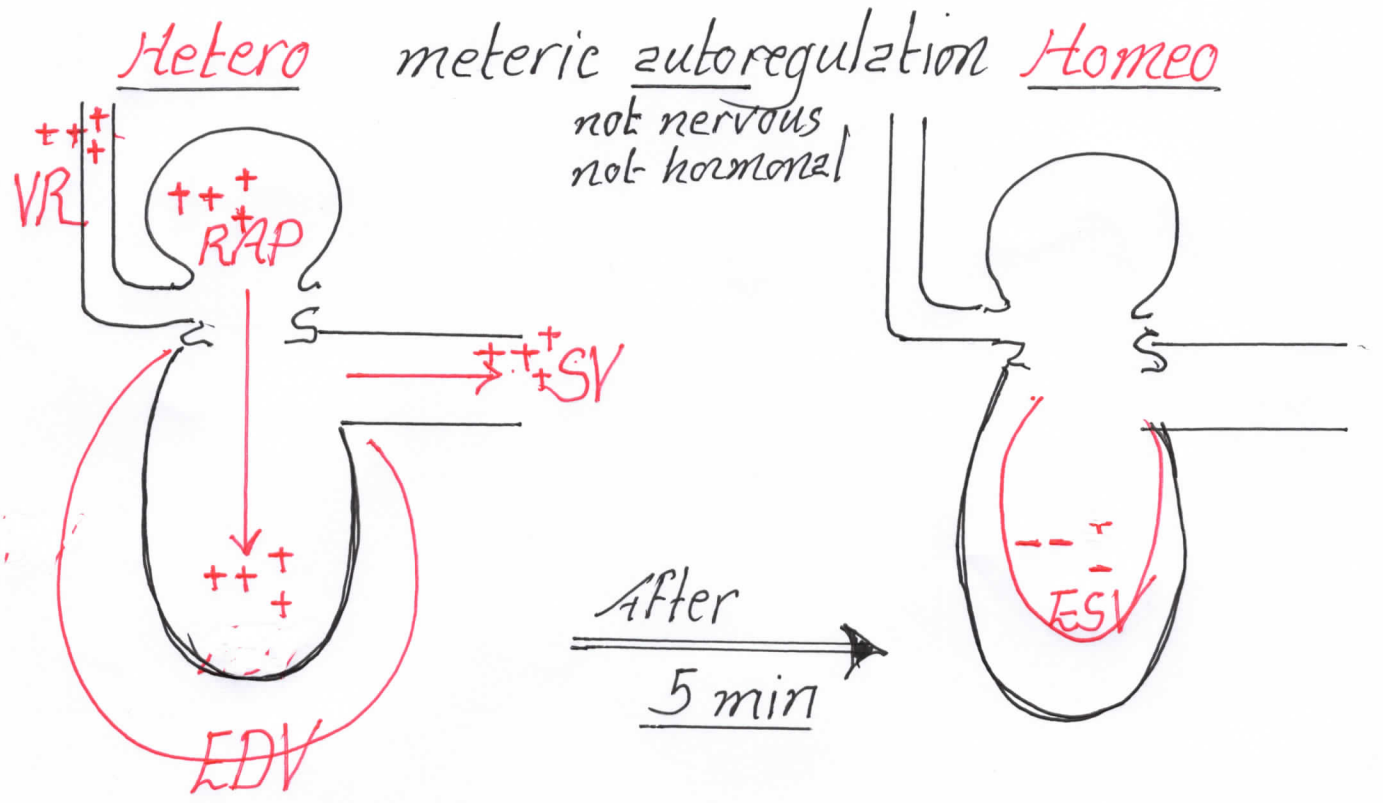
Relationship between EDV and SV

SV ∝ EDV within limits



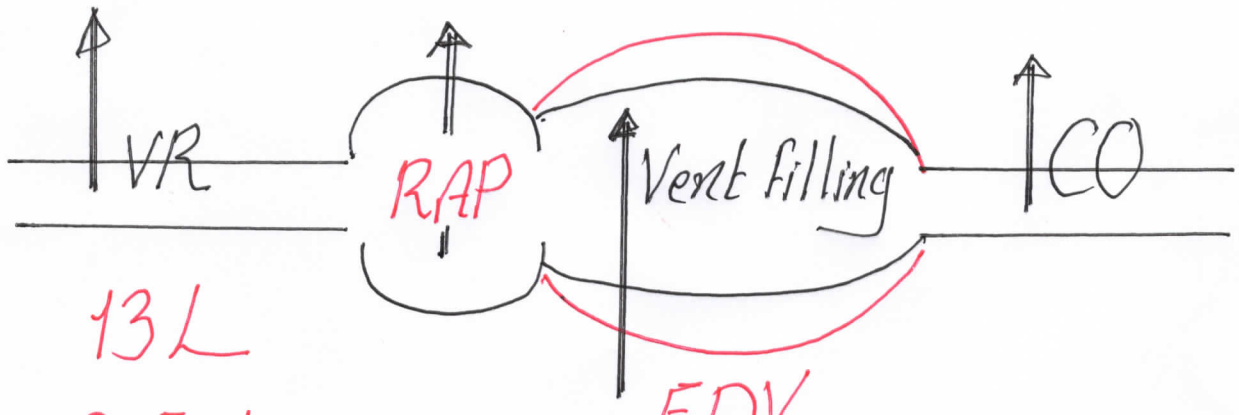
Effect of EDV on pressure-vol. loop

dashed line loop is control loop before ++ EDV



↑SV = ↑EDV − ESV
Hetero

↑SV = EDV − ESV↓
Homeo



13 L

2.5 times

EDV

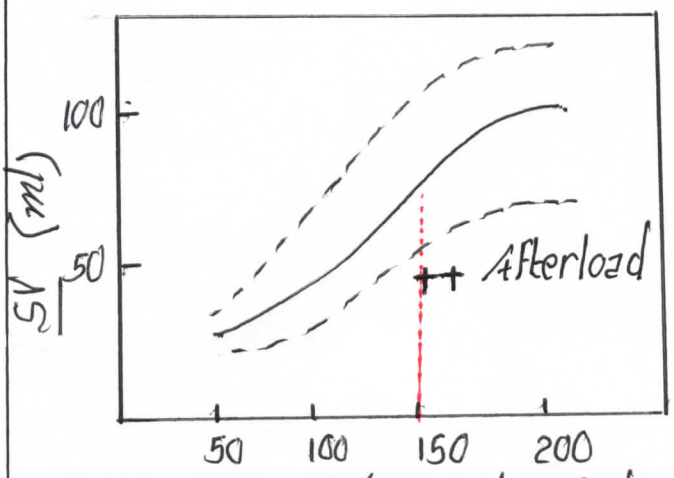
Hetero metric
auto regulation

2 **Afterload** Aortic p. against which L.V. contracts

At any Preload, ++ Afterload

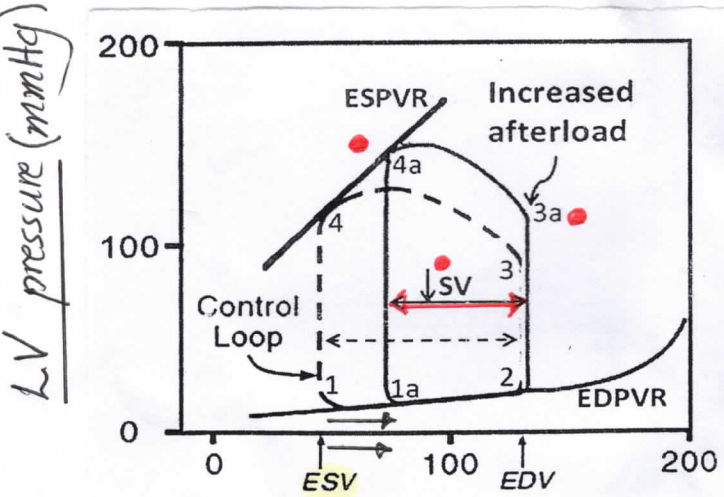
shifts relationship between preload & SV downwards & vice versa

Explanation -- degree & velocity of muscle shortening → -- SV



EDV (ml) « Preload »

Effect of changing afterload on stroke volume



LV volume (ml)

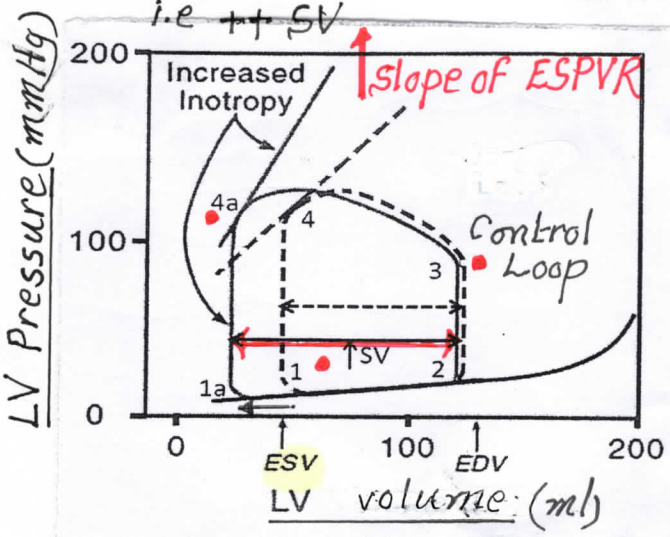
Effect of sudden ++ in afterload on pressure-volume loop

3 **Inotropic**

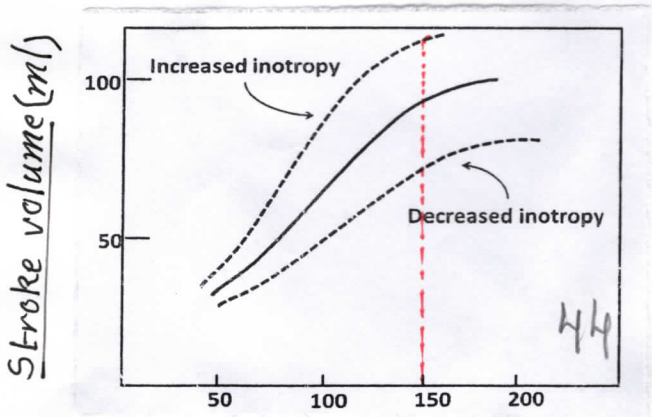
At any particular preload & afterload,

+ve inotropics ⟶ ++ SV & vice versa

- Ventricle contracts at same EDV & ejection starts at same afterload (3a) But aortic valve closes at lower ESV (4a) i.e ++ SV
- ESPVR shift upwards & Lt.



LV volume (ml)



End diastolic volume (ml)

- **Ejection fraction** $EF = \frac{SV}{EDV}$

  normally greater than 55% (or 0.55)

  Clinically is used as an index of contractility.

  +ve inotropic ⟶ ++ EF & vice versa

- **Cardiac Function curves**

  CO ∝ RAP

  Explanation Within limits,

  ++ RAP ⟶ ++ EDV (Starling law)

  ++ CO ⟵ ++ SV ⟵ Force of cont.

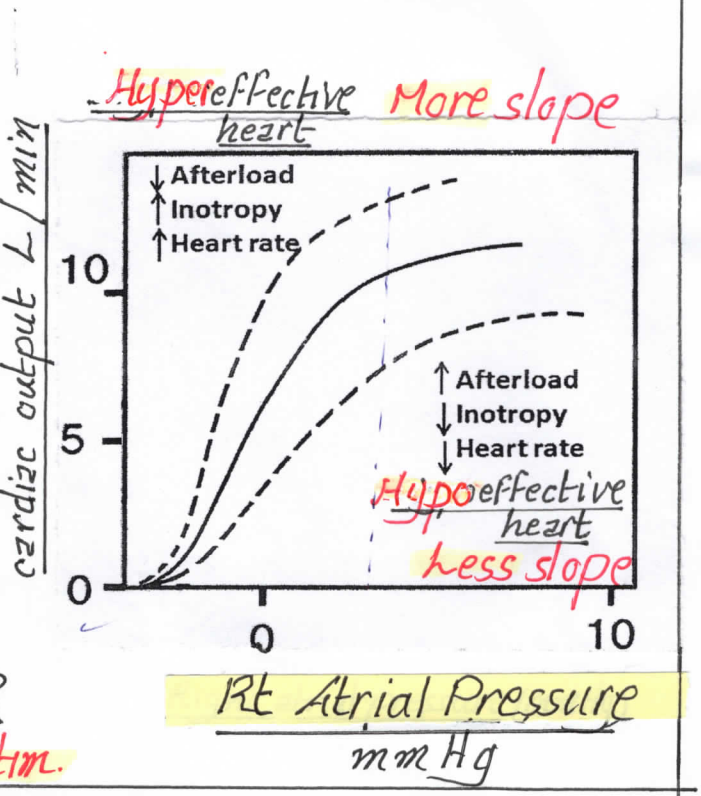
  Intrinsic (heterometric autoregul.)

  no external (nervous or hormonal) F.

  It reaches a plateau at 13 L/min,

  i.e 2.5 times normal CO

  i.e Heart pumps 2.5 times VR without nervous or hormonal stim.



- **Myocardial O_2 consumption** MVO_2

  8 ml/min/100 gm = 6 ml contraction & relaxation; 2 ml (basal) cellular activities & ionic pump

  Factors affecting MVO_2:

  A) Proportional ++ in MVO_2 with

  ++ HR , ++ Afterload (ABP) & +ve inotropic

  B) Smaller ++ in MVO_2 with

  ++ preload e.g valve regurgitation i.e less anginal pain

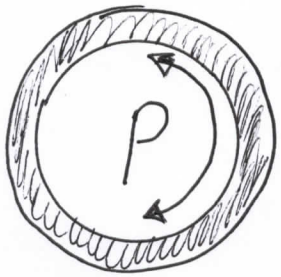
  Major part of MVO_2 is consumed to generate tension:

  - Laplace law T (tension) ∝ P (intravent. p) x r (radius of V)
  - Wall stress $S \propto \frac{T}{W} \quad (P \times r)$

	Length - Tension Preload EDV - SV curve	Vent. P - V loop	Explanation	Notes
Increased Preload	++ EDV ⟶ ++ SV on same curve	++ SV due to ++ EDV (↑ width of loop) Semilunar valves open & close at same P Slope of ESPVR same	More muscle shortening and more velocity of shortening	++ Preload (EDV a ++ VR b ++ atrial co -- Preload (ED a ++ HR (-- diastolic pe b -- Vent comp hypertrophy infarction.
Increased Afterload	Curve is shifted downwards i.e -- SV at same preload & vice versa	-- SV due to ++ ESV ↓ width of loop Semilunar valves open & close at Higher P Slope of ESPVR same	Less muscle shortening and less velocity of shortening	
+ve inotropy	Curve is shifted upwards i.e ++ SV at same preload & vice versa	++ SV due to -- ESV ↑ width of loop Semilunar valves open & close at same P Slope of ESPVR increases	More muscle shortening and More velocity of shortening	V max Slope of ES are indices myocardia Contractili

$$T \propto P \times r$$

Tension ∝ Pressure x Radius



$$\text{Wall stress «S»} \propto \frac{T \text{ Tension}}{W \text{ wall thickness}}$$

$$O_2 \text{ consumption} \propto S \text{ i.e} \propto \frac{P \times r}{W}$$

e.g

50% increase in afterload (aortic p) i.e hypertension

⟶ 50% increase in S

⟶ 50% increase in MVO_2

explanation $S \propto \frac{P \times r}{W}$

50% increase in preload (volume) e.g valve regurgitation

⟶ 14% increase in S

⟶ 14% increase in MVO_2

explanation $S \propto \frac{P \times r}{W}$ (not V)

Only 14% ++ in r ⟶ 50% ++ in V

as $V \propto r^3$

So, little ++ in O_2 consumption with valve regurgitation

Cardiac reserve CR

- **Def** Maximal % increase in CO in response to increase in body needs

- **Person** | **CR** | **CO ++ from 5L/min**

Well trained athlete	700%	35
Normal young adult	300 - 400%	15 - 20
Elderly people	200%	10
Heart failure	0%	0

- **CR mechanisms**

$CO = HR \times (EDV - ESV)$ Hypertrophy

① **HR reserve** Most important

Maximum 220 - age in years i.e at 20y 200/min

So, HR reserve = 200 - 75

Cause Symp stim. Catecholamines

Limits ++ HR → -- Diastolic period → -- Vent filling → -- CO

② **SV reserve** Maximum 200 ml at 20y

a ↑ EDV cause ↑ VR limit Starling law

b ↓ ESV cause Symp stim & catecholamines +ve inotropy

limit limited ESV

③ **Hypertrophy** with chronic strain on myocardium