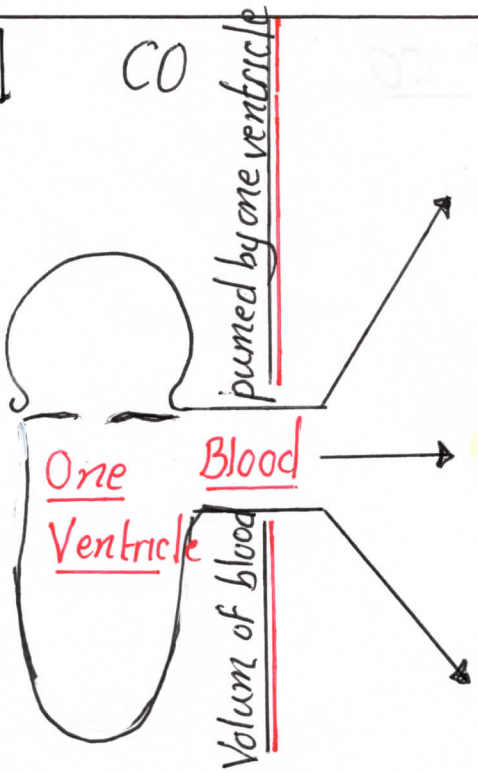
Cardiac output CO

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



- **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 x 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 ↓ x HR ↓ | = SV ↑ x HR ↑ | = SV ↓ x HR ↑

Exercise physiol. condition

Increased = SV ↑ x HR ↑

2 Stroke volume

3

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

Volume

Measured by

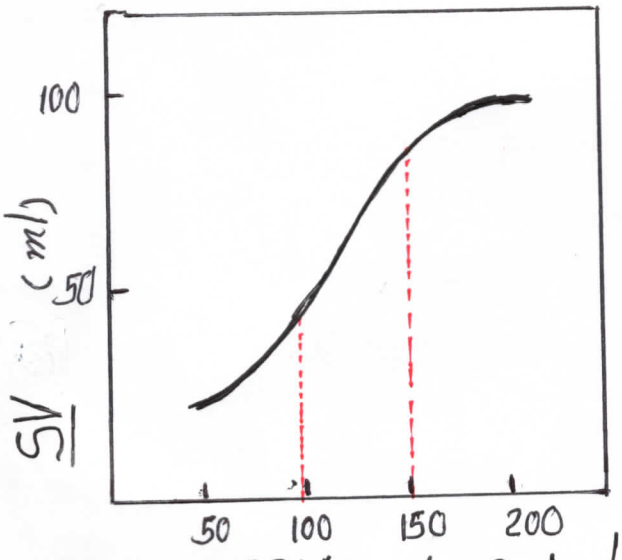
Sarcomere length / Myocyte

EDV (intact heart)

Depends on VR (RAP)

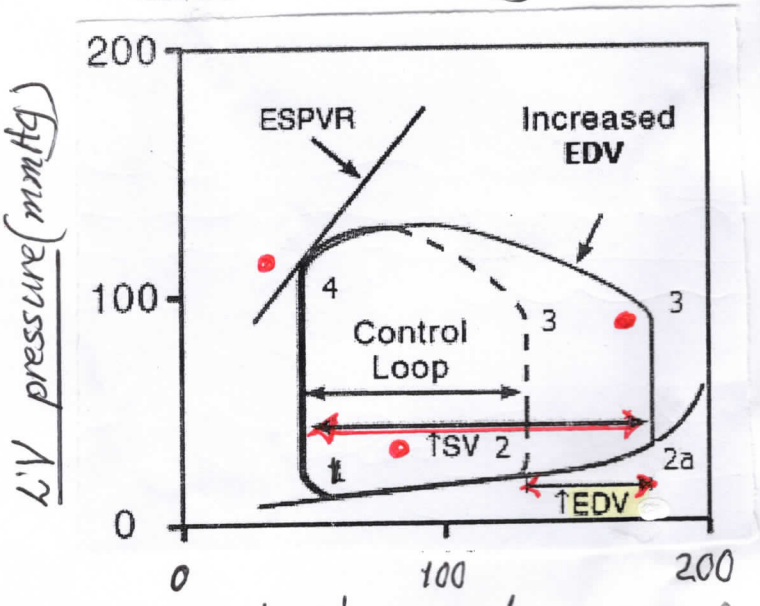
i.e Starling law

Heterometric autoregulatory mech.



Relationship between EDV and SV

SV ∝ EDV within limits

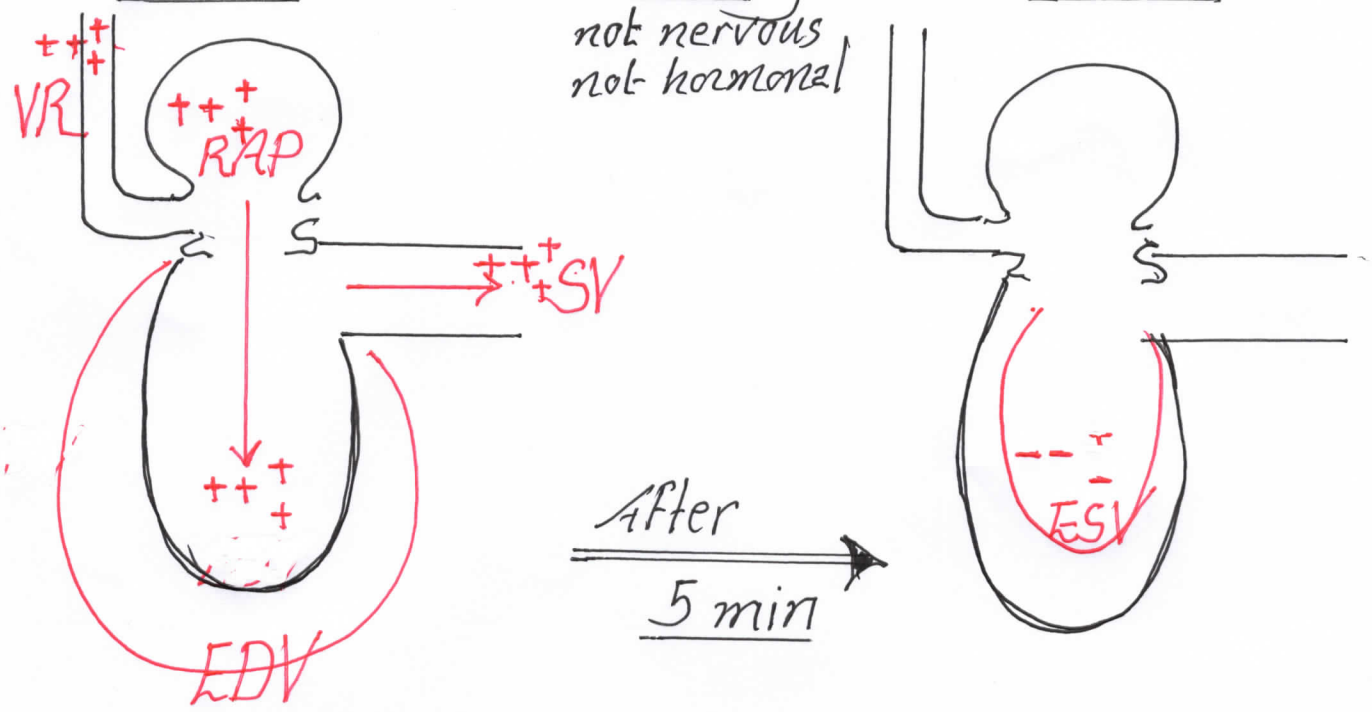


Effect of EDV on pressure-vol. loop

dashed line loop is control loop before ++ EDV

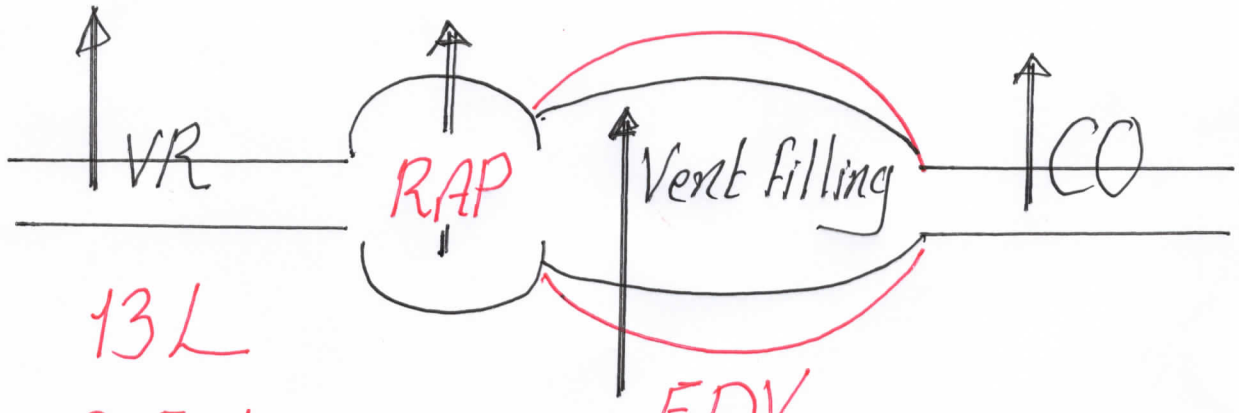
Hetero meteric autoregulation Homeo

not nervous
not hormonal



↑SV = ↑EDV − ESV
Hetero

↑SV = EDV − ESV↓
Homeo



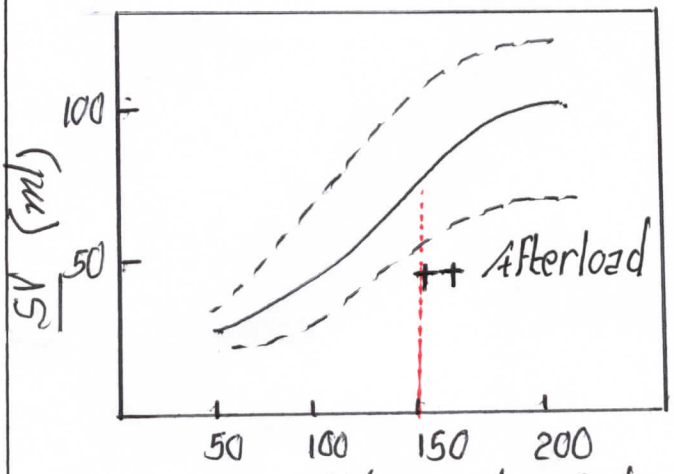
13 L
2.5 times

Hetero metric
auto regulation

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

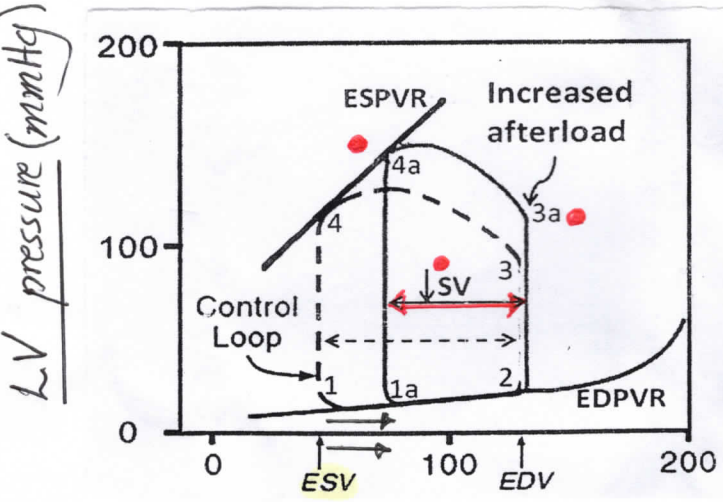
At any Preload, ++ Afterload
shifts relationship between preload & SV downwards & vice versa

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



EDV (ml) « Preload »

Effect of changing afterload on stroke volume



LV volume (ml)

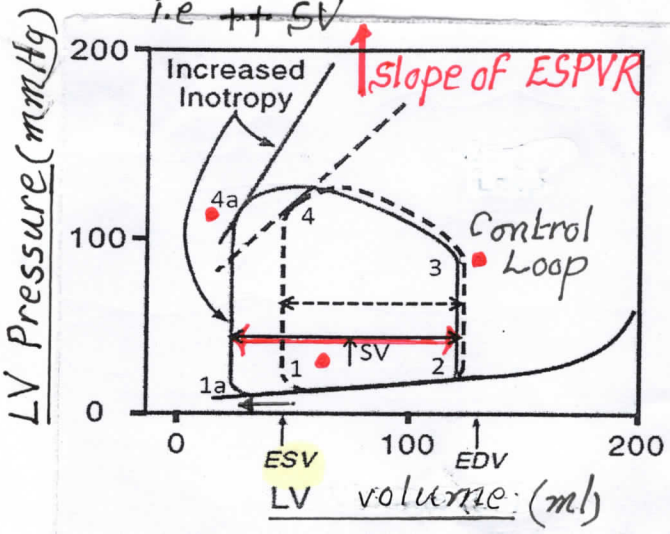
Effect of sudden ++ in afterload on pressure-volume loop

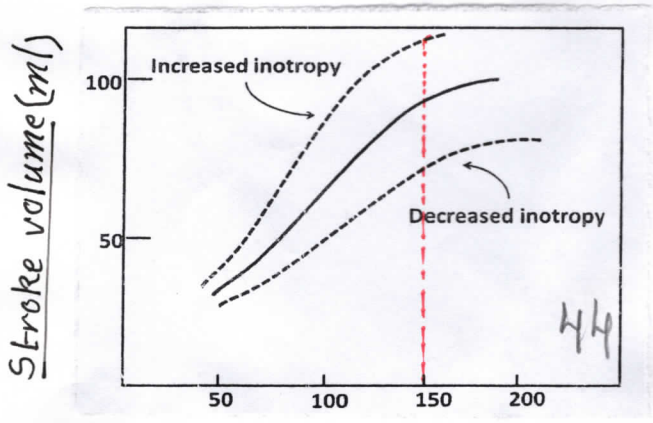
3 Inotropic

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.





- **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.)

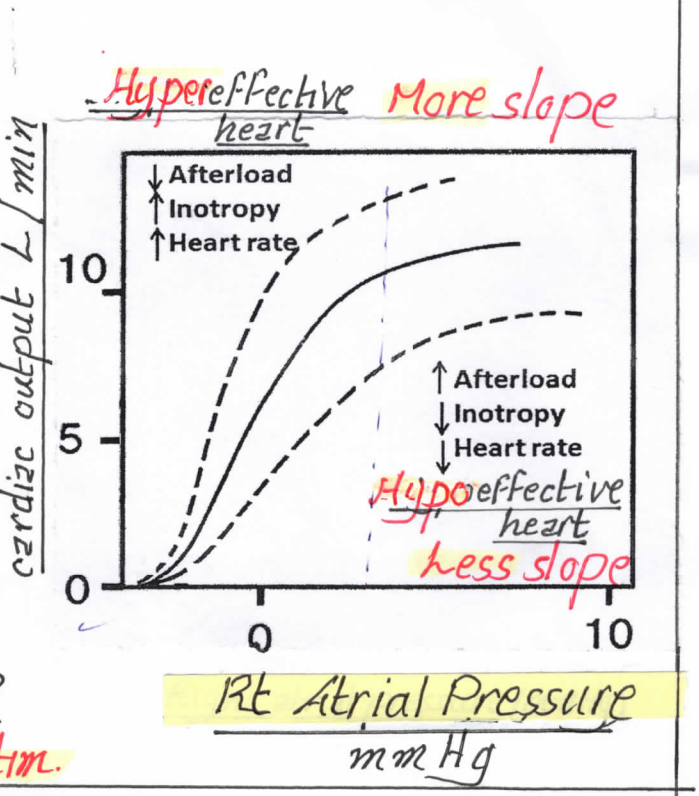
  no external (nervous or hormonal) F.

  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

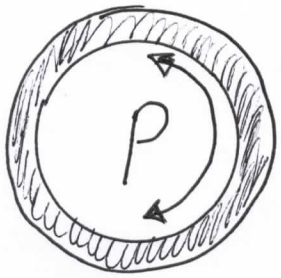
  Major part of MVO_2 is consumed to generate tension:

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

	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



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

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

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**

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