Cardiac Output :-

- Stroke volume :- The amount of blood the ventricle pumps during one beat.
- End Systolic volume :- The volume of blood remaining in the ventricles at the end of systole
- End diastolic volume :- The amt of blood in the ventricles at the end of diastole just before contraction

$$SV = EDV - ESV$$

- Ejection fraction :- The fraction of EDV which is ejected per ventricular contraction [systole]

$$EF = \frac{SV}{EDV}$$

When ventricles are more efficient → more SV → EF is more
∴ EF tells us about the efficiency of ventricles.

- Cardiac output :- Amt of blood ejected by the ventricles during unit time.

minute cardiac output ⇒ 1 min

$$CO = SV \times HR$$

eg $CO = 70 \times 75$

$CO/min = 5250\ ml/min.$

If HR ↑ ⇒ CO ↑

but if HR is ↑ too much - diastolic filling time is reduced - too much ↓ in stroke volume
∴ the ventricle is not filling properly.

- FICK'S PRINCIPLE :- Used to determine the CO. - easiest way.

$$CO = \frac{O_2 \text{ consumption}}{\text{Arterio Venous conc difference}}$$

$$CO = \frac{O_2 \text{ consumption /min}}{PV_{conc} - PV_{conc} \text{ ml/L}}$$

$$= \frac{250 \text{ ml/min}}{190 - 140 \text{ ml/L}}$$

$$CO = \frac{250}{50} = 5 \text{ l/min}$$

- Cardiac work :-

work = displacement × Force

cardiac/stroke work = SV × Aortic pressure

min cardiac work = (SV × HR) × Aortic pressure

minute cardiac work = CO × Aortic pressure

Cardiac output => volume work => component of work of Internal work — work for generating C.O.

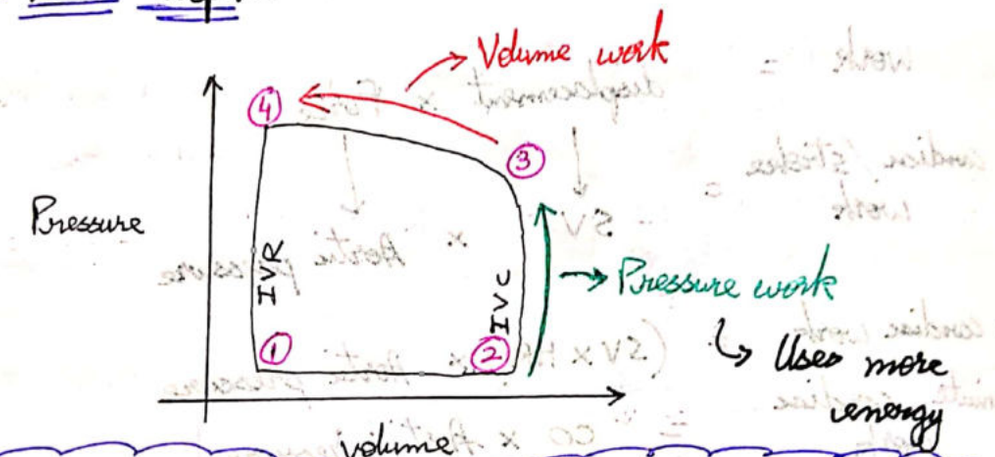
Aortic pressure => Pressure work / Internal work.

O_2 consumption ∝ minute cardiac work = CO × Aortic pressure

- The vol work of Rt & lft hrt is equal ∴ the cardiac output of both ventricles is equal.
But their Pressure works are different where the press

work done by lft hrt > than rt hrt.

- Hence O_2 consumption is more by lft hrt.
  ie. pressure work req more O_2 [more energy]
- ∴ When someone has systemic hypertension Press work of lft is ↑
- In aortic stenosis CO ↓ ie vol work ↓ but pressure work is too much ↑ ∴ total work is still ↑.
- In strenuous exercise => CO ↑ too much
  Aortic pressure may be normal
  ∴ Total work is not much ↑ as pressure work is not much change
- Pressure Volume loop :-



∴ Max work is done by the hrt from ② to ③
↳ Pressure work

- Cardiac Work & its relationship with Laplace's law :-

Acc to Laplace law ⇒ $P = \frac{2 \times \text{Tension in wall} \times \text{Thickness}}{\text{Radius}}$

$$P = \frac{2Th}{R}$$

∴ If T is ↑ P is ↑ but if R is ↑ P is ↓
thickness is ↑ P is ↑

Whenever in any disease the hrt becomes dilated ie its radius is ↑ it becomes less efficient bcoz it produces ~~less tension~~ => less pressure => less stroke volume.

To prod some pressure & stroke vol => tension shud be ↑
↳ its O_2 demand ↑ => less efficient.

eg:- congestive cardiac failure => CO is less.

↓

① Treatment by giving Venodilators => Veins dilate & the venous return ↓ ∵ most of the blood is collected in the peripheral veins => less blood is coming to the hrt system => Radius of Ventricles is ↓

Hence Venodilators are indirectly controlling the radius.

↳ ∵ At the same tension more pressure is generated => more stroke volume => more cardiac output

② diuretics => Tot blood vol ↓ => Tot Venous Vol ↓ => Venous return ↓

As a result radius is ↓.

Note:- Congestive cardiac failure => $\downarrow P = \frac{\downarrow T}{\uparrow R}$

↳ due to failure of myocardium T is ↓ => P is ↓

when P is ↓ less blood is ejected so R is ↑ due to collection of blood.

∴ Radus shud be ↓ — Venodilators, Diuretics

③ +ve Isometropic drugs => T ↑ eg:- digitalis.

⇓

P ↑ => more blood ejected

⇓

failure managed ← cardiac output ↑.

- Pressure generated by ventricles is to overcome the after load ie total peripheral Resistance.

④ Arteriodilators => Pressure in arterioles ↓ => ∴ Ventricles has to generate less pressure

—II O_2 demand ∝ minute cw = CO × Aortic pressure

In a patient with Ischemic heart dzs => supply of O_2 ↓ narrow coronary arteries

$$P = \frac{2 \times T \times \text{Thickness}}{R}$$

Treatment of Ischemia
- ⇓ O_2 demand => ↓ Cardiac work ie we have to reduce pressure work.
- or
- ⇑ O_2 supply

To ↓ pressure work => ↓ Aortic press by Arteriodilators

Venodilators => Radius ↓ => With less tension it can prod high pressure
↳ Nitrates

Patients with Angina
↓
Venodilators + Arteriodilators
Nitrates → Venodilation, Arteriodilation

Nitrates

Venodilation
Radius ↓
∵ with less O_2 [less T]
good press prod

Arteriodilation
With less P => good C.O.

- Pre load :- load on the cardiac muscle before contraction
  ↳ Vol present in ventricles at the end of diastole - EDV
  Vol on which the lft ventricle will work.

Preload ∝ EDV
Preload ∝ EDP [end diastolic pressure]

Lft atrial pressure ∝ EDV ∝ EDP

∴ Preload ∝ Lft Atrial pressure

Pulmonary Vein pressure ∝ Preload

- Pulmonary wedge pressure :- determined by Swan Ganz catheter (↓ Has a pressure transducer)
  It is also an index of Pre load.
  Indirect way to measure end diastolic pressure

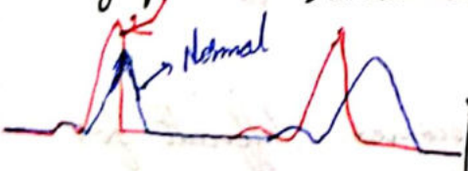
- Indices of Contractility :-

① Vel of dev of contraction = $\frac{dP}{dt}$ — change in P in lft ventricle per unit time
↓ Rate of Press dev during isovolumic contraction

② Ejection fraction = $\frac{SV}{EDV}$

If contractility is strong EF will be more.

Sympathetic stimulation :-



① ↑ dP/dt => +ve clinotropic effect
② More peak left Vent press => +ve inotropic
③ ↑ vel of relaxation
④ Systolic interval is less - Hrt becomes more dynamic

Note :- ↓ Systolic interval => ↑ contractility
↓ diastolic interval => ↑ HR

- **Afterload** :- load on muscle during contraction
  depends on Tot Peripheral R
  Press against which lft ventricle has to perform.

$\therefore$

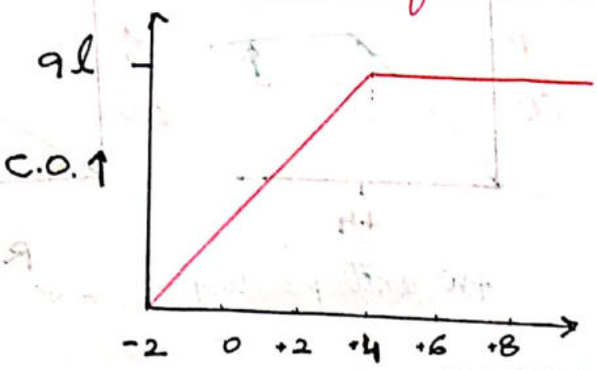
$SV \propto$ Preload

$SV \propto$ contractility

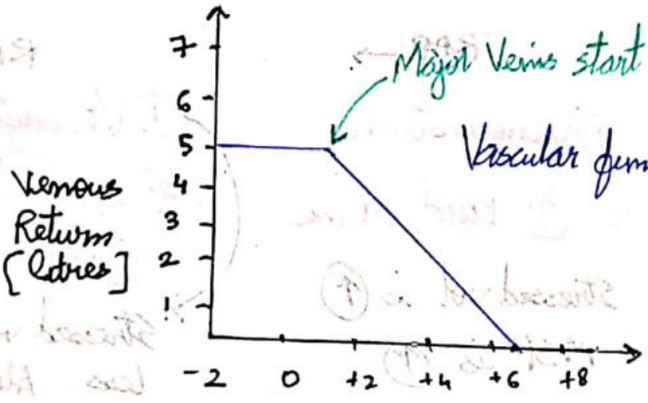
$SV \propto \frac{1}{\text{afterload}}$

- **Cardiac output & Venous Return** :-
  - Acc to frank starling law - As EDV / EDP is ↑ => ↑ contra
    ↓
    CO ↑ CO
  - C.O. is dependant on EDV which further depends on ventricular filling ∴ Vent filling depends on Atrial pressure which itself depends on venous return
  - If Atrial pressure ↑ within physio limits => Cardiac output ↑
  - As the CO of both ventricles is same any change in the rt atrial pressure will have an effect on filling of ventricles.
  - Whenever Rt atrial press ↑ it becomes difficult for venous return to enter the rt atrium ie venous return ↓

cardiac function curve



Around +4 pressure => max ventricular filling
After +4 => no ↑ in C.O. => cannot overfill the ventricles



At 6.5 mm Hg RAP => No venous return.

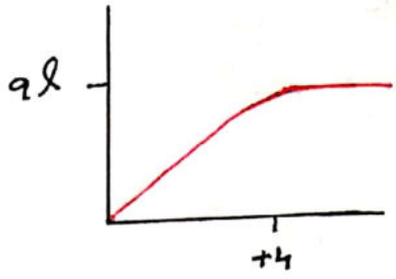
∴ Mean systemic pressure = 6.5 mm Hg => Normal
↳ driving force for maintaining venous return

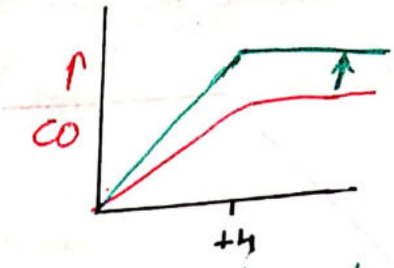
When RAP is too low => venous return faster => Major veins start collapsing.

- Effect of Inotropic drugs on cardiac output :-

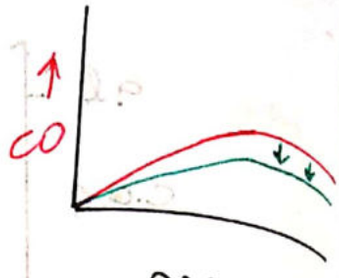
① +ve Inotropic drugs :- eg digitalis.
For a given preload cardiac output ↑

② -ve Inotropic agent :- Ca^{++} channel blockers eg Verapamil.



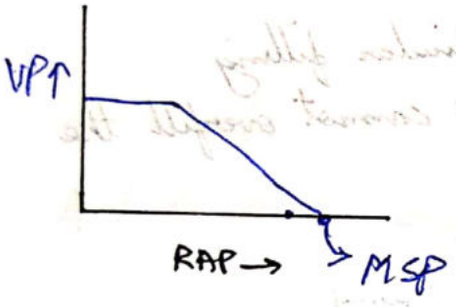


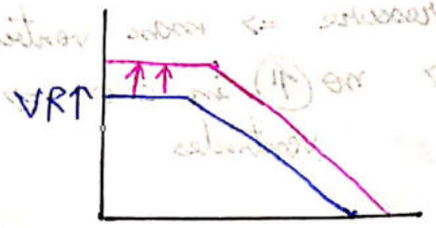
+ve inotropic drug

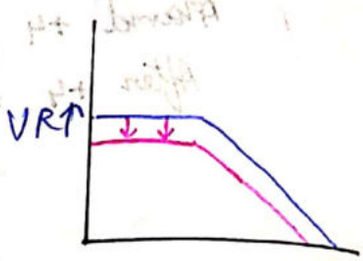


-ve inotropic drug

- Affect on Vascular funct curve :-



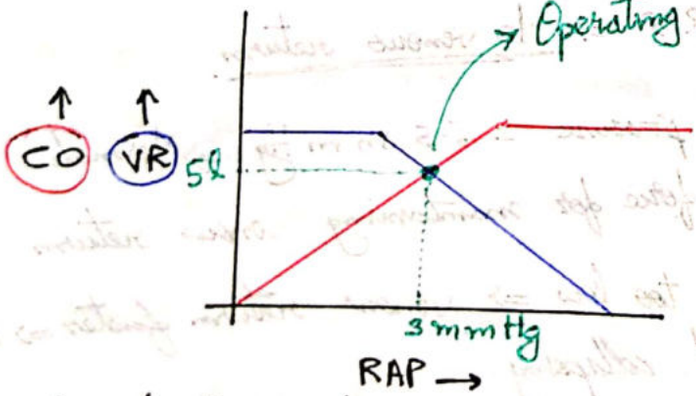




① Venoconstriction
② ↑ Blood volume
Blood transfusion ←
Stressed vol is ↑
MSP is ↑

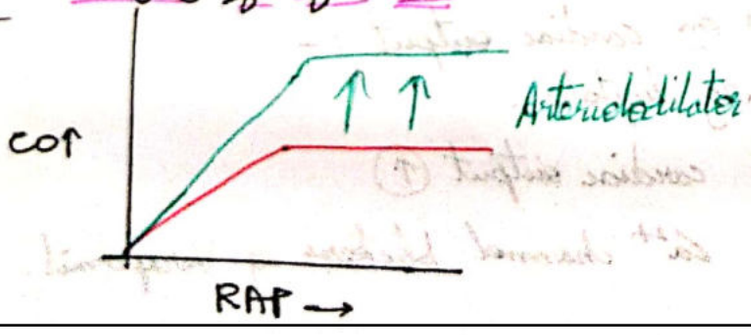
① Venodilater
② Hemorrhage
[① Blood volume]
Stressed vol becomes less. Also MSP is also reduced.

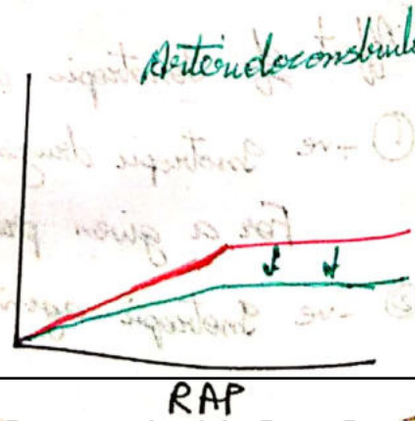
- Combined curve :-



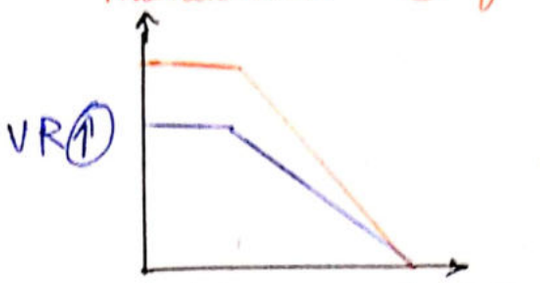
Operating pt / Equilibrium pt
↳ Heart is operating at this position.

- Changing of After load :-



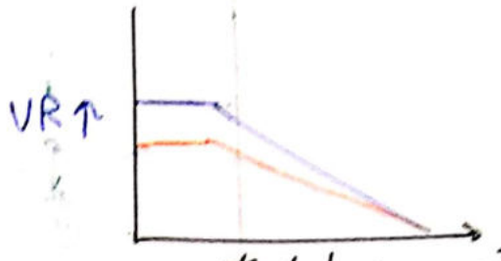


Arteriolodilation ⇒ ↓ Afterload



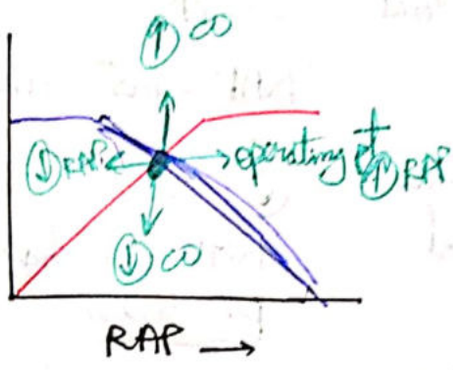
clockwise movt of the curve

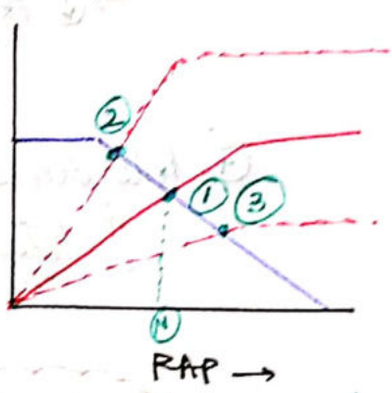
Arteriolaconstriction ⇒ ↑ Afterload



counterclockwise movt of the curve

Note :- In both the curves MSP is not changed.





(2) operating pt moved upward & ~~leftward~~

↓

CO ↑

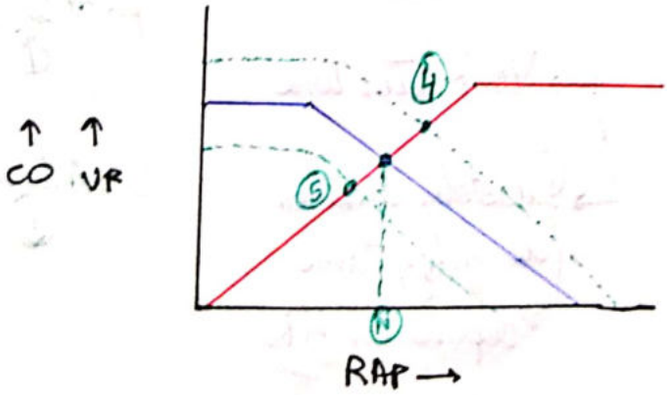
Atrial press ↓

(2) +ve inotropic agent

(3) -ve inotropic agent

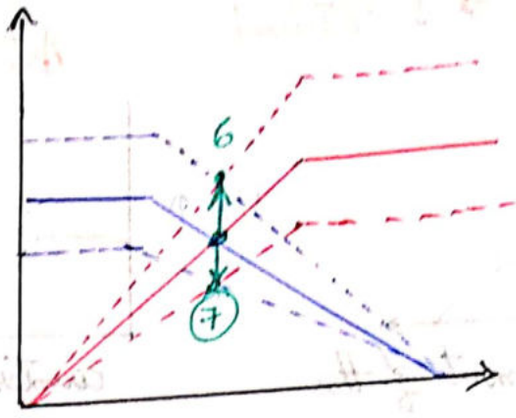
↳ operating pt down & right

CO ↓ Atrial press high.



(4) Blood transfusion [or venoconstriction] → ↑ CO, High Atrial press

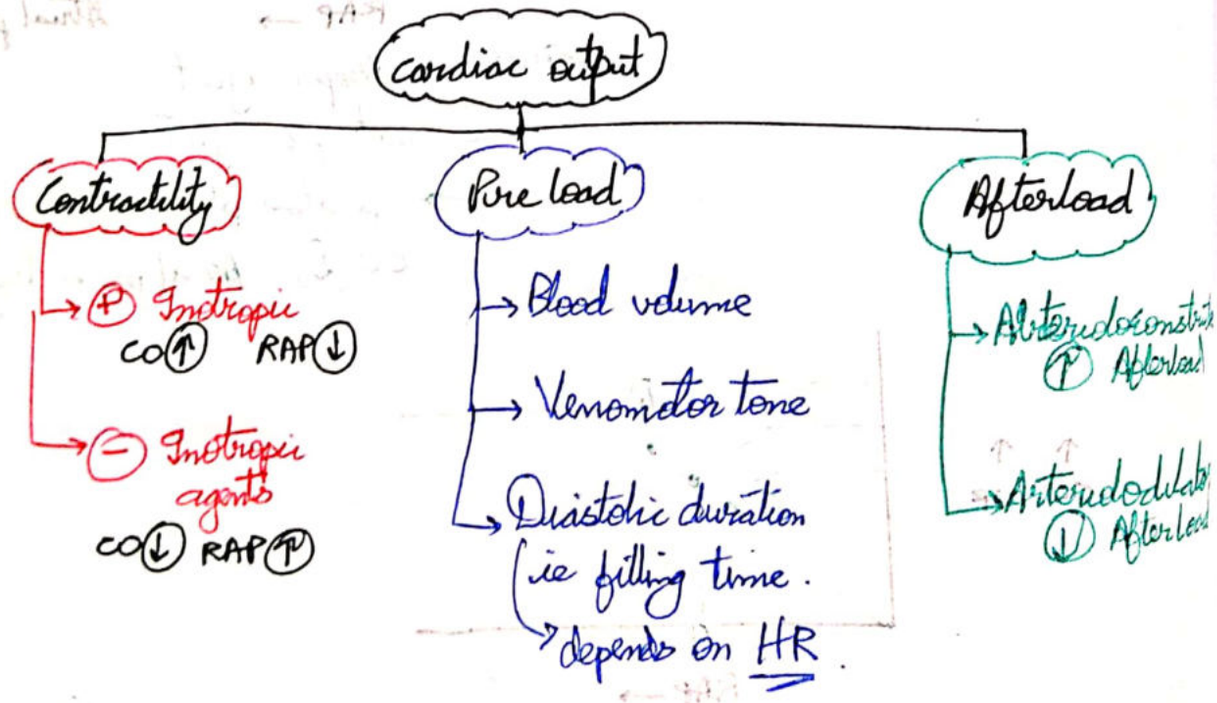
(5) Venodilator → ↓ CO, ↓ RAP



RAP

(6) Arteriodedilator ⇒ pt shifted upwards
↳ (↓) Afterload CO (↑)
RAP – not changed

(↑) Arteriodeconstrictor ⇒ (↓) CO
(↑) Afterload RAP – unchanged
pt down



CO ∝ Preload

(↓) Preload ⇒ CO (↓)
RAP (↓)

(↑) Preload ⇒ CO (↑)
RAP (↑)