CARDIAC OUTPUT

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END DIASTOLIC VOLUME (EDV)

Amount of blood present in ventricles just before contraction is called end diastolic volume. Its value is about **120ml.**

END SYSTOLIC VOLUME (ESV)

The volume left in ventricle at the end of systole (after ejection of blood). The normal value of end systolic volume is about **50ml**.

STROKE VOLUME

Amount of blood which left ventricle ejects during one beat is called stroke volume. The normal value of stroke volume is about **70ml.**

Stroke Volume = EDV – ESV

If left ventricle is more efficient, it will pump out more stroke volume. If left ventricle is less efficient, it will pump out less stroke volume.

EJECTION FRACTION

The fraction of end diastolic volume which is ejected per ventricular contraction is called ejection fraction. Ejection fraction is related with efficiency of ventricle.

Ejection Fraction = $\frac{Stroke\ Volume}{End\ Diastolic\ Volume}$

= $\frac{70}{120}$ = 0.6

Putting above values indicate 60% of blood is ejected per ventricular contraction.

Normal ejection fraction ranges from **50 – 75%**

CARDIAC OUTPUT

Amount of blood ejected by blood in one minute is called cardiac output.

Cardiac Output = Stroke Volume x Heart Rate

Suppose stroke volume is 70ml and heart rate is 75 beats/min, so

Cardiac Output = 70 x 75

= 5250 ml/min

Normally when heart rate goes up, cardiac output increases. But when heart rate becomes abnormally high, heart rate is so fast that it reduce ventricular filling time and left ventricle does not fill properly due to which stroke volume falls and in this case cardiac output does not increase.

FICK'S PRINCIPLE FOR MEASURING CARDIAC OUTPUT

$$Cardiac\ Output = \frac{Oxygen\ Consumption}{Arterial\ Oxygen\ Content - Venous\ oxygen\ conntent}$$

Arterial oxygen content is determined in systemic artery while cardiac venous content is determined in pulmonary artery.

If oxygen consumption = 250ml oxygen/min

Arterial oxygen content = 190 ml/Liter

Venous oxygen content = 140 ml/L

Then putting values in Fick's formula we get

Cardiac Output = $\frac{250}{190-140} = \frac{250}{50}$ = 5 Liter/min

Cardiac output of left ventricle and right ventricle is equal. During one minute, the cardiac output of right ventricle should be equal to blood flow of pulmonary system (as right ventricle pumps blood to lungs).

CARDIAC WORK

Cardiac work = Aortic pressure x Stroke Volume

Minute cardiac work is cardiac work per minute.

Minute cardiac work = Aortic pressure x Cardiac output

Cardiac output represent volume work while aortic pressure represent pressure work.

If cardiac output increase, heart has to work more. Similarly if cardiac output decrease, heart has to work less.

The work which is done by ventricle is not only dependent on cardiac output but also depends on the pressure against which cardiac output is maintained and this is called pressure work.

Pressure must be generated (in the aorta or pulmonary artery) to get certain volume out of ventricle. So if volume which is being ejected is increasing or decreasing, work will change accordingly. Similarly if pressure against which heart is pumping keeps changing, then work done by heart also changes.

Oxygen consumption is dependent on work of heart. Oxygen consumption (oxygen demand of heart) is directly proportional to work of heart. If heart is working more, it will consume more oxygen and vice versa.

In patients with ischemic heart disease, there is imbalance between oxygen supply and demand.

Volume work of right and left heart is equal. Pressure work of right and left heart is different. As left ventricle has to pump against higher pressure so left heart needs more oxygen as compared to right side of heart.

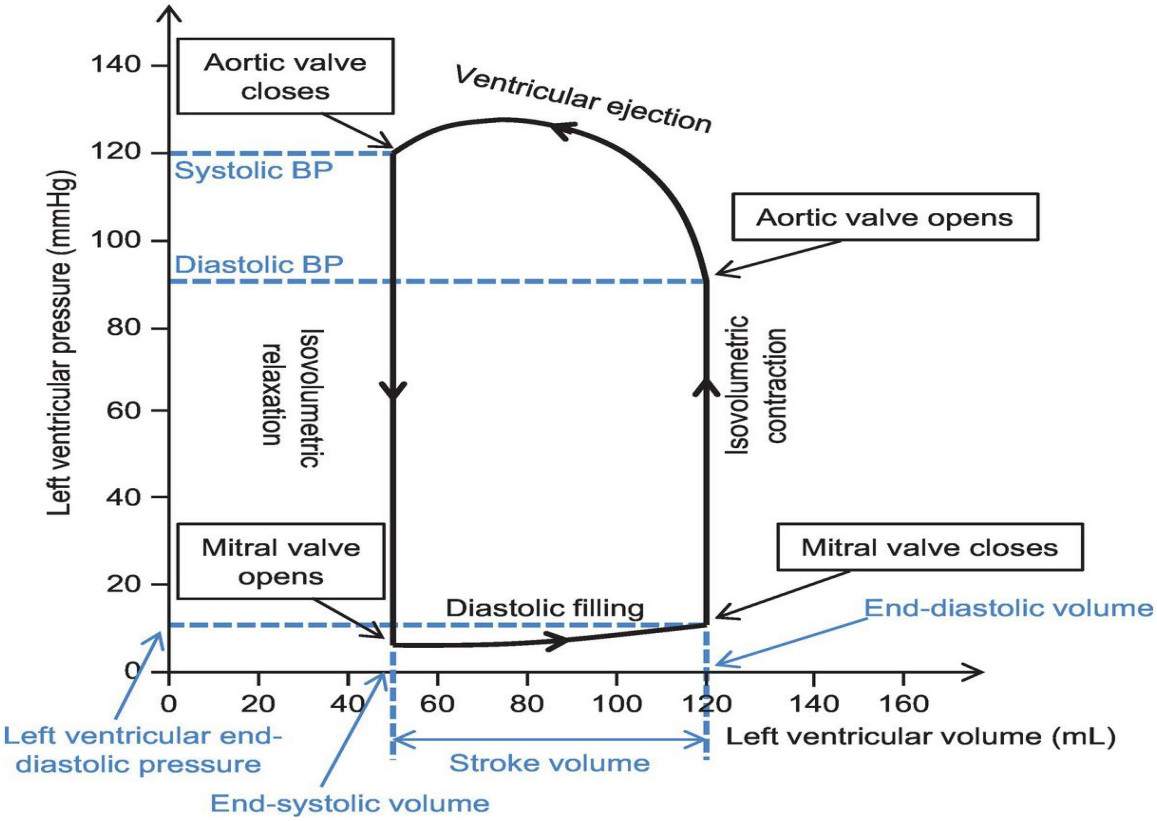
When someone has systemic hypertension, then pressure work on left heart is increased. When someone has pulmonary hypertension, pressure work on right heart is increased.

Patients with high blood pressure are given arteriodilators. These are given so left ventricle pumps against less pressure.

In aortic stenosis, left heart has to work more as it has to pump against high pressure. In aortic stenosis, cardiac output drops but pressure generated in ventricle increases and total work increases.

During strenuous exercise, cardiac output increase too much but pressure work remains normal. Even though cardiac output increase, oxygen consumption increase too much. So work done by heart depends more on pressure work than volume work (cardiac output).

PRESSURE VOLUME LOOP



LAPLACE'S LAW

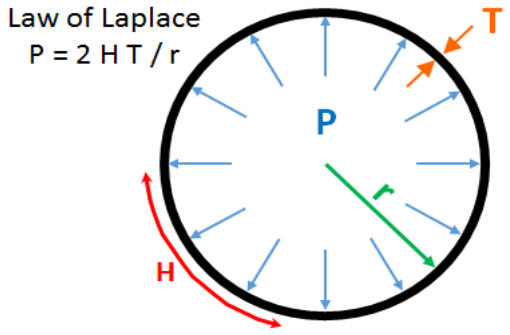
$$P = \frac{2\ X\ Tension\ X\ thickness}{Radius}$$

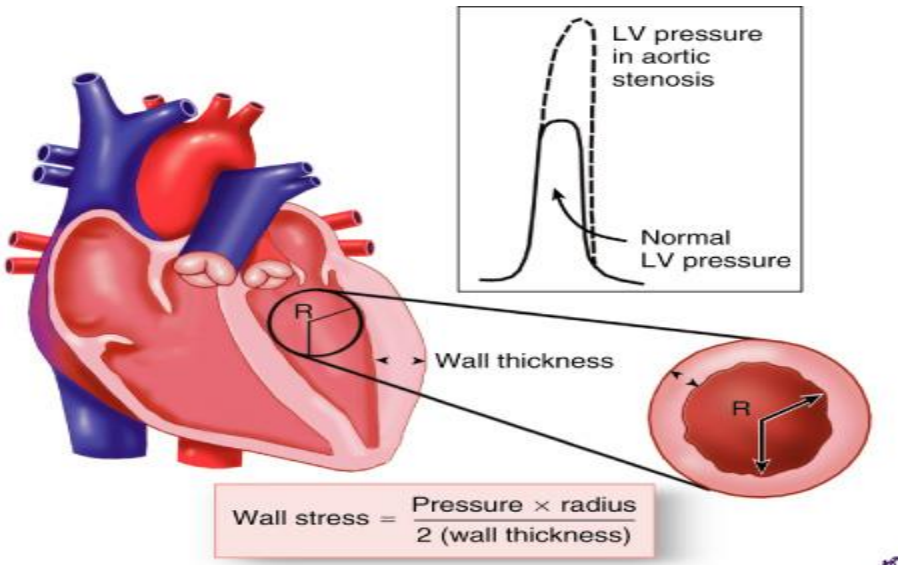
$$P = \frac{2Th}{r}$$

T: wall tension

h: wall thickness

r: radius of blood vessel





PRACTICAL APPLICATIONS OF LAPLACE LAW

1. **IN CONGESTIVE HEART FAILURE**

   Whenever heart becomes dilated (radius increase) e.g. as seen in congestive heart failure, heart becomes less efficient. Ventricular contractions become poor and blood cannot be ejected well so stroke volume decrease.

   Vasodilators are given to patients with congestive heart failure. Due to these vasodilators, blood will pool in the veins and venous return to heart decrease, which in turn reduce ventricular filling and radius of heart decrease. Due to this reduced radius, heart can produce more pressure and thereby increase ejection fraction. This pressure generated by ventricles is needed to overcome afterload.

   Diuretics are also given to patients with congestive heart failure. Due to diuretics, total blood volume is reduced thereby reducing venous return and leading to smaller radius of heart.

   Positive ionotropic drugs such as digitalis increase tension on heart and thereby pressure generated by heart also increase.

2. **IN ISCHEMIC HEART DISEASE**

   Oxygen demand ∝ Minute cardiac work

   Minute cardiac work = Cardiac Output x Aortic pressure

   = Volume work x Pressure work

   In ischemic heart disease, coronary arteries are narrow due to which oxygen supply is reduced. To combat this oxygen supply deficit, we're interested in decreasing oxygen demand. Oxygen demand can be reduced by reducing cardiac work.

   We reduce cardiac work by reducing pressure work, as we don't want to reduce cardiac output.

Pressure work i.e. pressure in aorta can be reduced by giving arteriolodilators. Due to arteriolodilators, pressure in aorta drops as total peripheral resistance decrease. Venodilators are also given so with less radius, pressure of ventricle can increase.

PRELOAD

Preload is the load (pressure) on ventricle before it starts contraction, so it is the load on ventricle when ventricle is relaxed.

Preload is primarily the blood volume present in left ventricle when ventricle is relaxed. This volume is also called end diastolic volume.

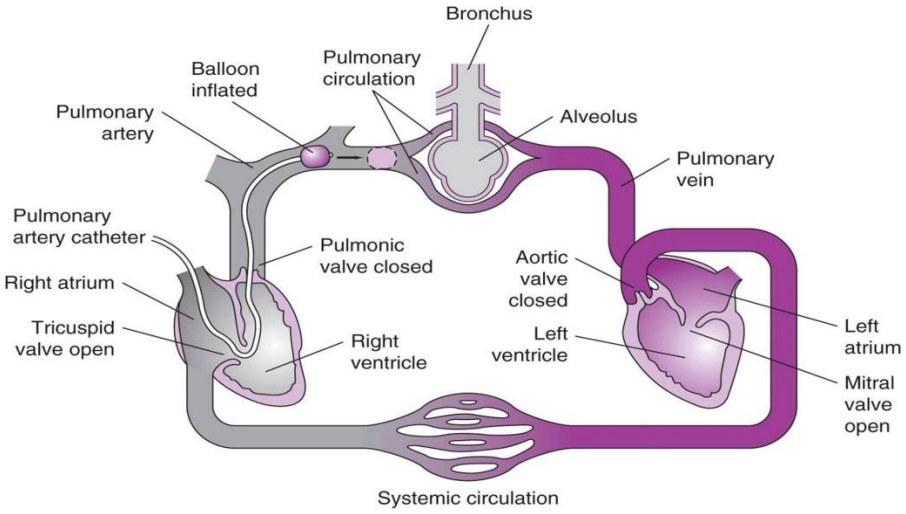
Preload ∝ End diastolic volume

When end diastolic volume increase:

- Preload increase
- Left atrial pressure increase
- Pulmonary vein pressure increase
- Pulmonary wedge pressure increase

Pulmonary Wedge Pressure

To determine pulmonary wedge pressure, we insert a catheter called Swan Ganz catheter from cubital vein and enter through superior vena cava and eventually into pulmonary artery. With the tip of the catheter, one vessel is blocked. At the tip of the catheter, there is pressure transducer which measures the pressure in pulmonary artery. In this way end diastolic pressure is measured as pressure in pulmonary artery will be the same as end diastolic pressure.



CONTRACTILITY INDICES

- Velocity of development of contraction i.e. how fast left ventricle contraction develops is given by

  $$\mathbf{v} = \frac{\Delta P}{\Delta t}$$

  where ΔP is change in pressure in left ventricle

  This formula determines the rate of pressure development during isovolumetric contraction

- Ejection Fraction is given by

  $$\text{Ejection Fraction} = \frac{stroke\ volume}{end\ diastolic\ volume}$$

  If contractility is more, bigger fraction of End diastolic volume will be ejected into aorta

  If contractility is less, smaller fraction of end diastolic volume will be ejected into aorta

- **Sympathetic stimulation**

  Sympathetic stimulation increase the velocity of contraction.

  Effects of sympathetic stimulation on heart:

  1. Increased velocity of contraction
  2. Increased peak left ventricular pressure
  3. Increased velocity of relaxation
  4. Decreased systolic interval due to increased velocity of contraction and relaxation

Systolic interval is mainly affected by contractility. Whenever contractility increase, systolic interval will increase.

Diastolic interval is the time interval between two contractions. Diastolic interval is mainly affected by heart rate. Increase in heart rate decrease diastolic interval.

AFTERLOAD

Afterload is the pressure against which left ventricle has to contract.

Afterload depends on total peripheral resistance. Arteriolodilators decrease afterload.

Stroke volume $\propto$ Preload

Stroke volume $\propto$ Contractility

Stroke volume $\propto \frac{1}{Afterload}$

FACTORS AFFECTING CARDIAC OUTPUT

1. **Contractility of myocardium** (direct relation)
   - Positive ionotropic agents increase contractility

- Negative ionotropic agents decrease contractility

2. **Preload** (direct relation)
   Preload further depends on:
   a) Blood volume: Increased blood volume increase preload
   b) Venomotor tone: increased venomotor tone increased blood volume
   c) Diastolic duration i.e. filling time of ventricle is directly proportional to preload. Diastolic duration depends mainly on heart rate

3. **Afterload** (inverse relation)
   - Arterioloconstrictors increase afterload
   - Arteriolodilators decrease afterload

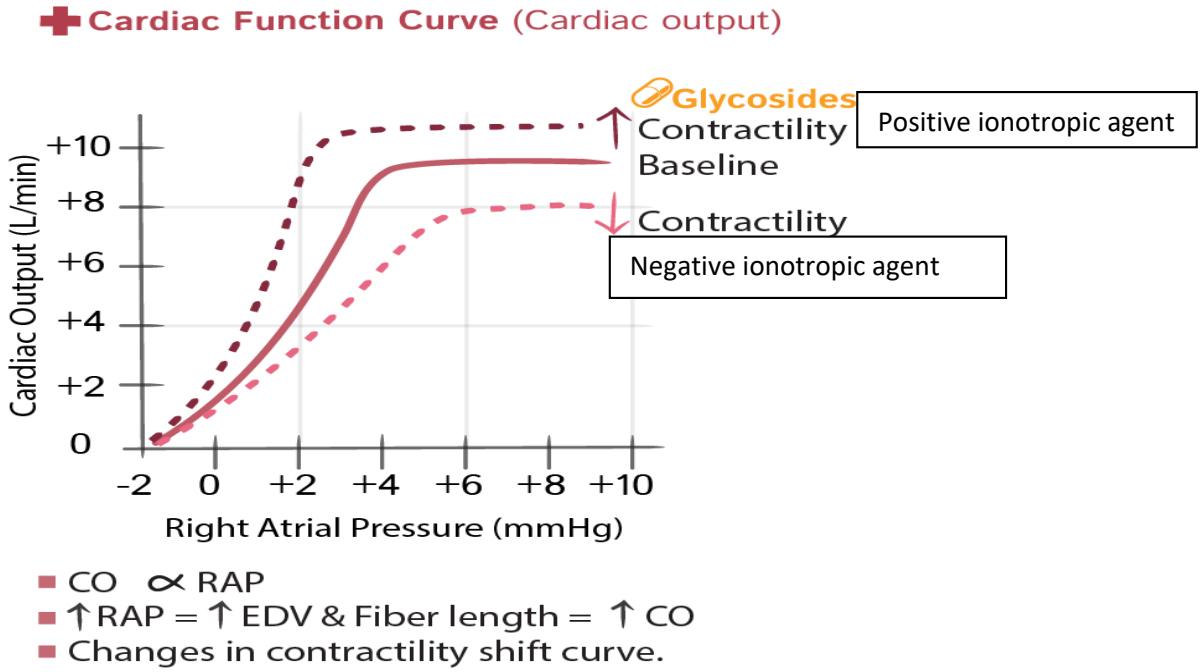
CARDIAC OUTPUT AND VENOUS RETURN

- Cardiac output depends on end diastolic volume
- EDV depends on ventricular filling
- Ventricular filling depends on right atrial pressure
- Right Atrial pressure depends on venous return

So as right atrial pressure increase, cardiac output also increase.

Whatever is the output of right ventricle, same will be output of left ventricle.

CARDIAC FUNCTION CURVE



Under low right atrial pressure, venous return increases.

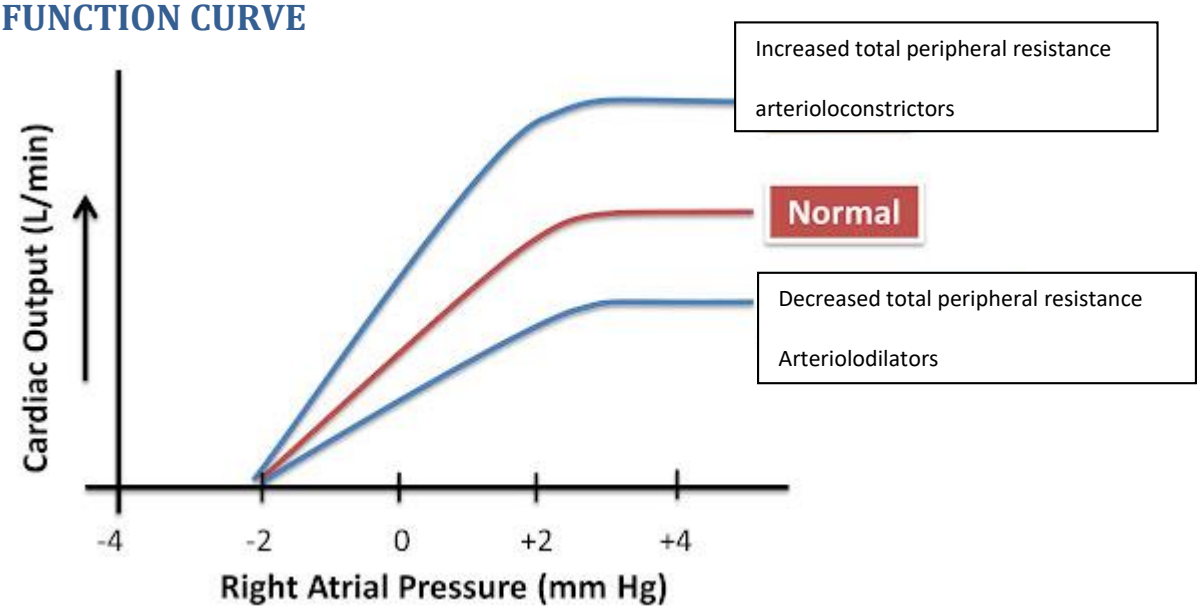
Under high right atrial pressure, venous return decrease

As right atrial pressure becomes +4mmHg, cardiac output cannot further increase as ventricles cannot be filled above pressure higher than +4mmHg.

Under the effect of **positive ionotropic agent** e.g. Digitalis, cardiac output increases for a given preload as contractility is increased.

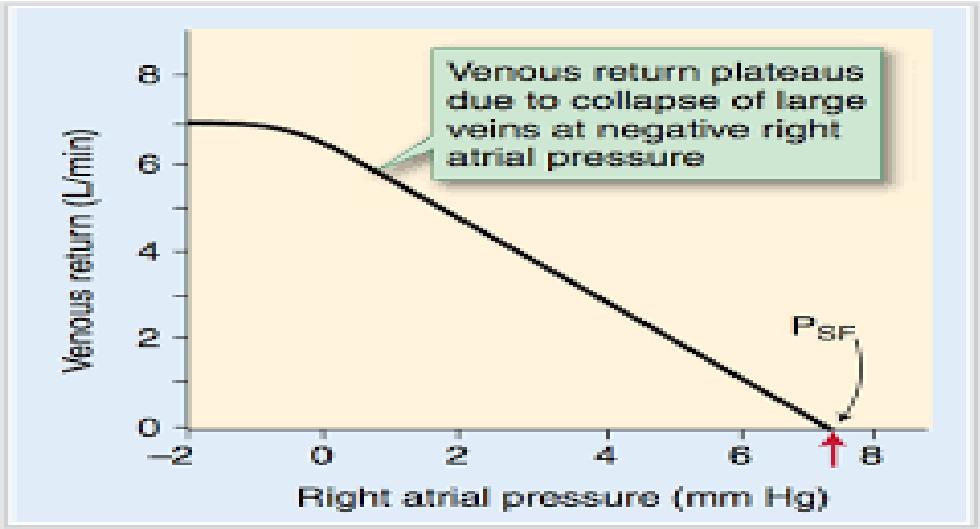
The effect of **negative ionotropic agent** e.g. calcium channel blockers is to suppress myocardial contractility. So for each right atrial pressure, cardiac output will be less than normal.

EFFECTS OF CHANGE OF TOTAL PERIPHERAL RESISTANCE ON CARDIAC FUNCTION CURVE



- **ARTERIOLODILATORS**
  - Resistance for outflow of heart decrease i.e. TPR decrease
  - Aortic pressure decrease i.e. the pressure against which ventricle has to contract decrease
  - Afterload decrease
  - Cardiac output increase
- **ARTERIOLOCONSTRICTOR**
  - Increased TPR
  - Cardiac output decrease

VASCULAR FUNCTION CURVE

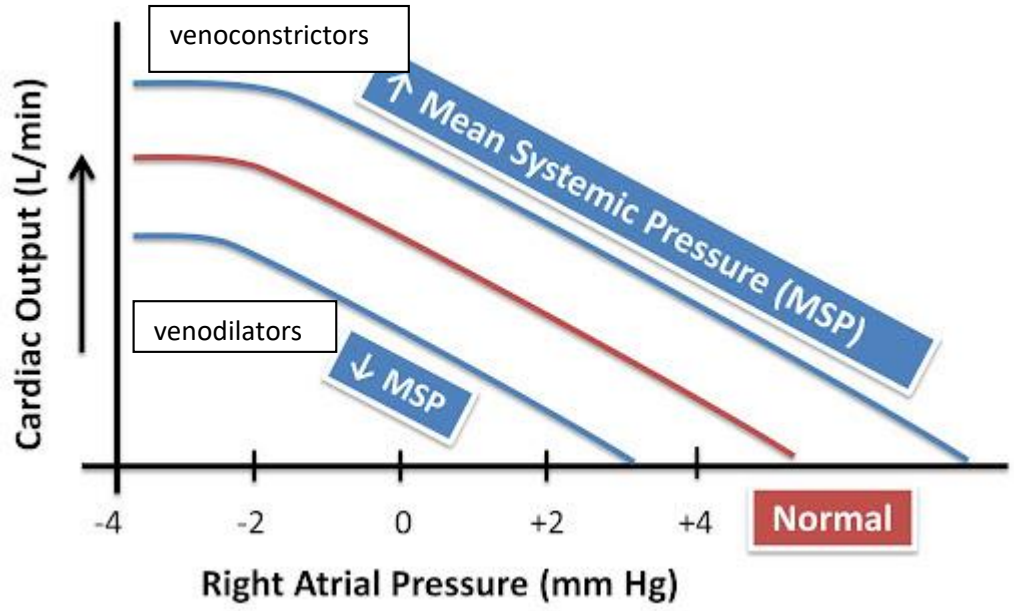


As Right atrial pressure progressively decrease, venous return increase. Or with increasing right atrial pressure, venous return decrease.

When pressure is less than zero venous return is so fast that major veins start collapsing so no further return in venous return can be seen.

At mean systemic pressure (normal value = 7 – 8 mmHg), venous return is zero.

VASCULAR FUNCTION CURVE UNDER IONOTROPIC EFFECTS



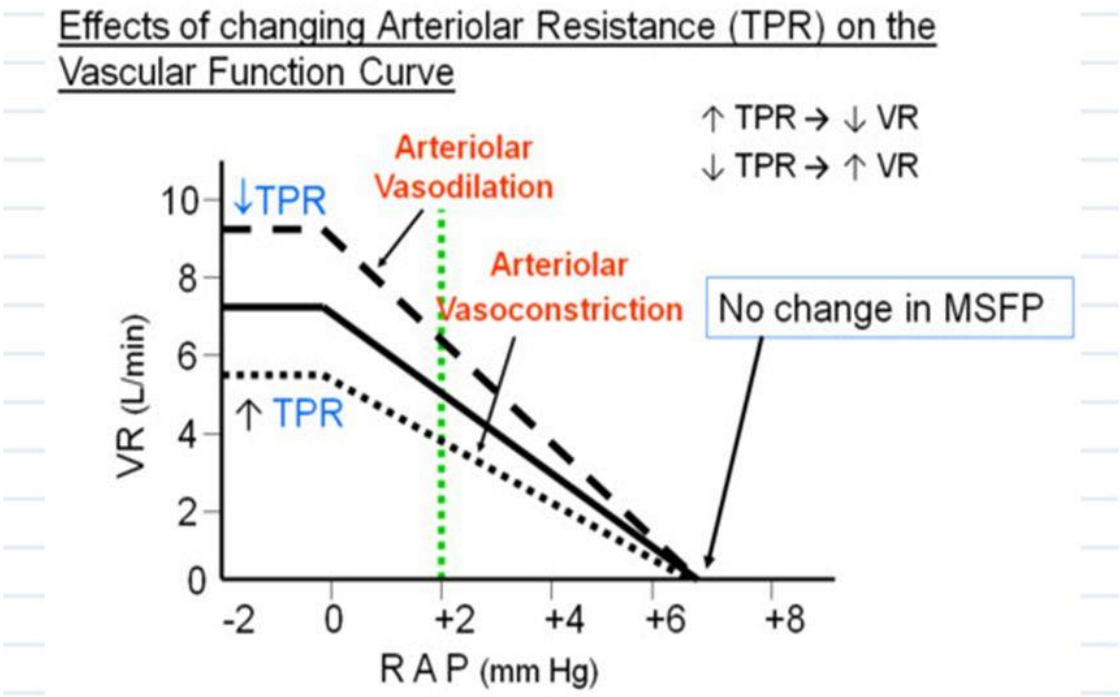
- **Venodilators** decrease venous return as blood is pooled in veins.
  Reduced blood volume e.g. due to hemorrhage will decrease venous return.

Stressed volume maintains the mean systemic pressure. Due to venodilators, mean systemic pressure is reduced as blood moves more towards unstressed volume.

- **Venoconstriction** increase venous return as stressed volume increase.
- Under effects of venoconstrictors or blood transfusions,
    - Blood volume is increased
    - Venous return increase
    - Cardiac output increase
    - Stressed volume increase
    - Mean systemic pressure increase

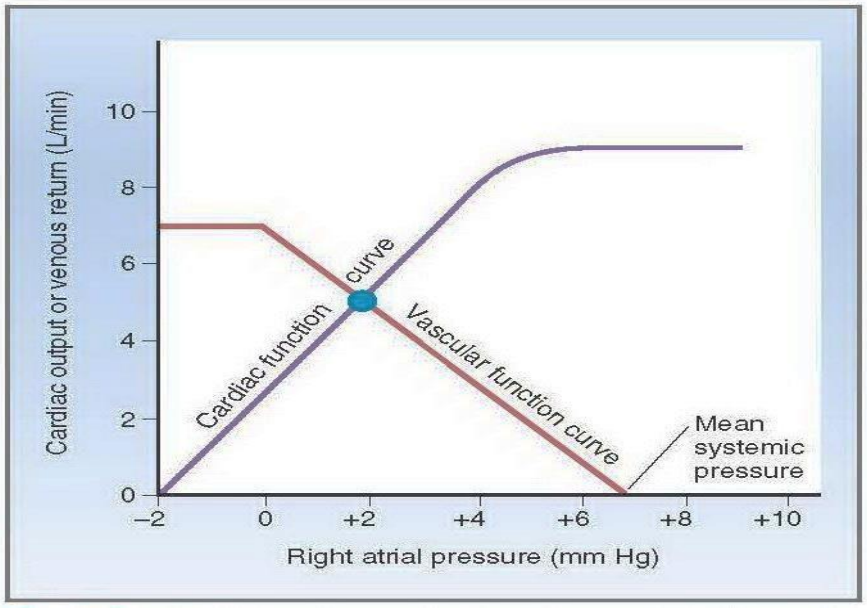
      So for every given right atrial pressure on graph, venous return will be increased.

EFFECT OF CHANGE OF TOTAL PERIPHERAL RESISTANCE ON VASCULAR FUNCTION CURVE



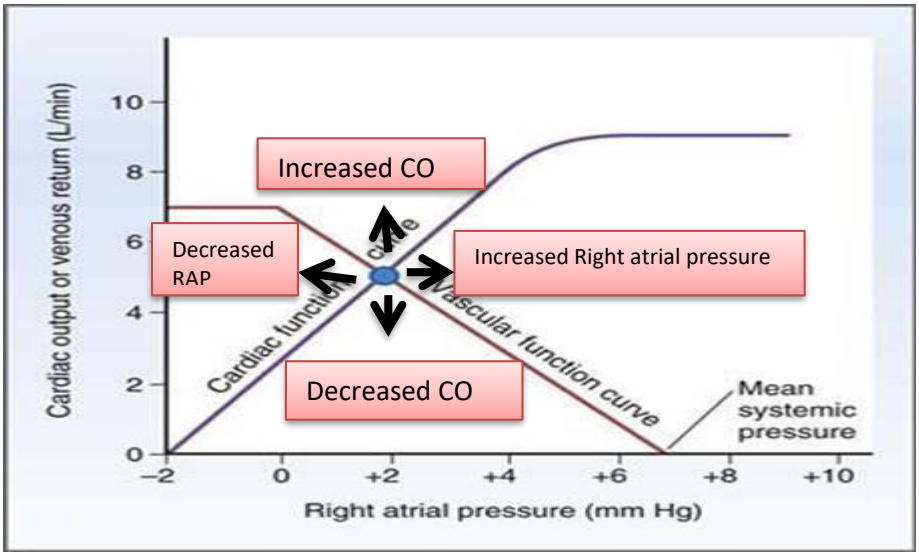
- **ARTERIOLODILATOR**
    - More blood moves to venous side
    - Increased venous return
    - Clockwise movement of curve on graph
- **ARTERIOLOCONSTRICTOR**
    - Less blood coming to venous side due to increased TPR
    - Counter clockwise movement of curve on graph

CARDIAC FUNCTION CURVE AND VASCULAR FUNCTION CURVE



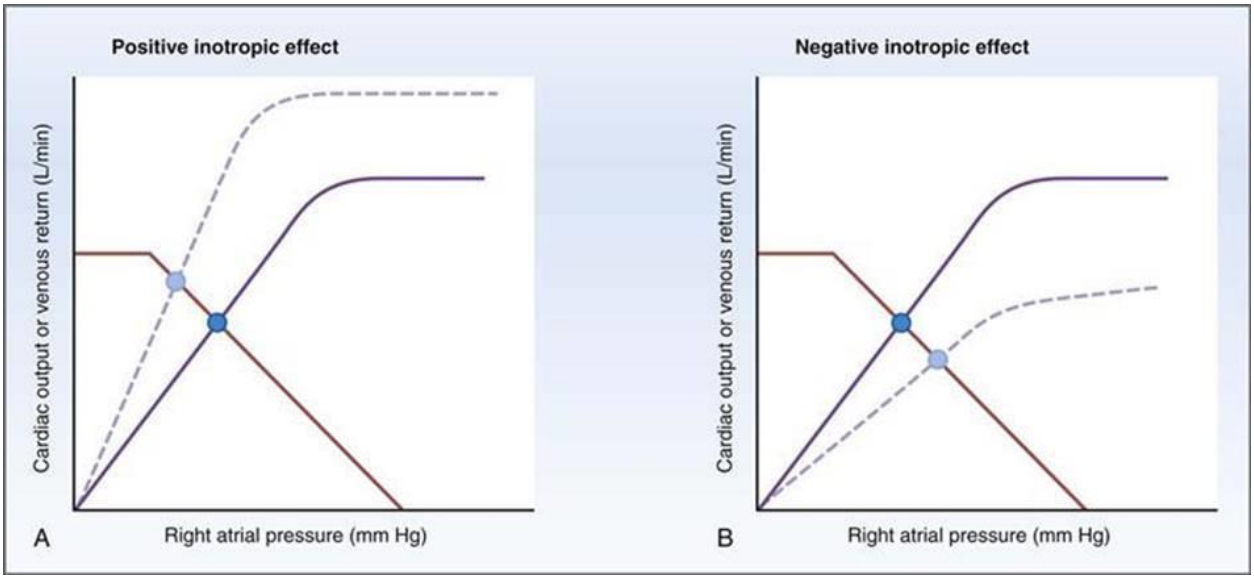
The intersection point of the cardiac function curve and vascular function curve is called **operating point** or equilibrium point.

SHIFT IN OPERATING POINT



- When operating point moves to the right, it indicates increased right atrial pressure
- When operating point moves to the left, it indicates decreased right atrial pressure
- When operating point moves upward, it indicates increased cardiac output
- When operating point shifts downward, it indicates decreased cardiac output

EFFECTS OF IONOTROPIC AGENTS ON OPERATING POINT

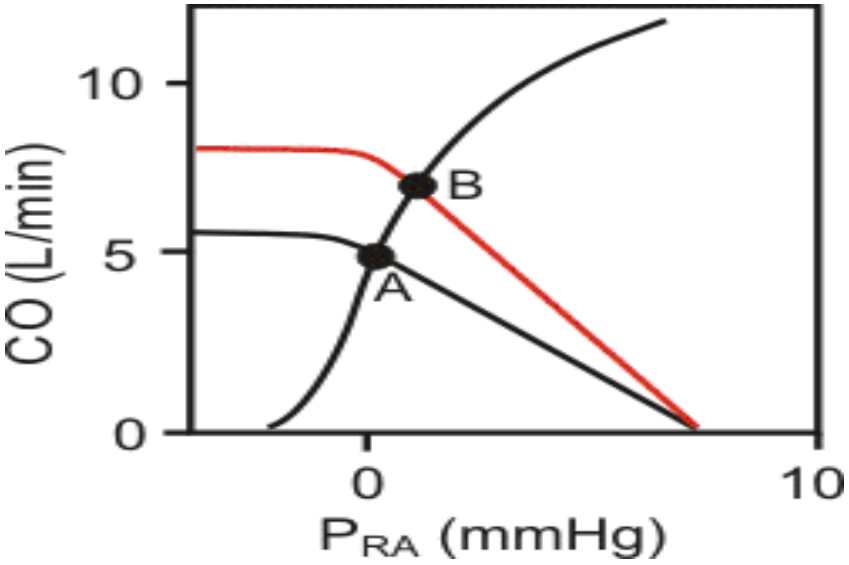


Due to positive ionotropic agent, operating point is increased so cardiac output is increased.

- Positive ionotropic agent
  - Heart operates at less right atrial pressure
  - Heart contracts strongly
  - Ejection fraction increases
  - Atrial pressure drops
  - Venous return increase

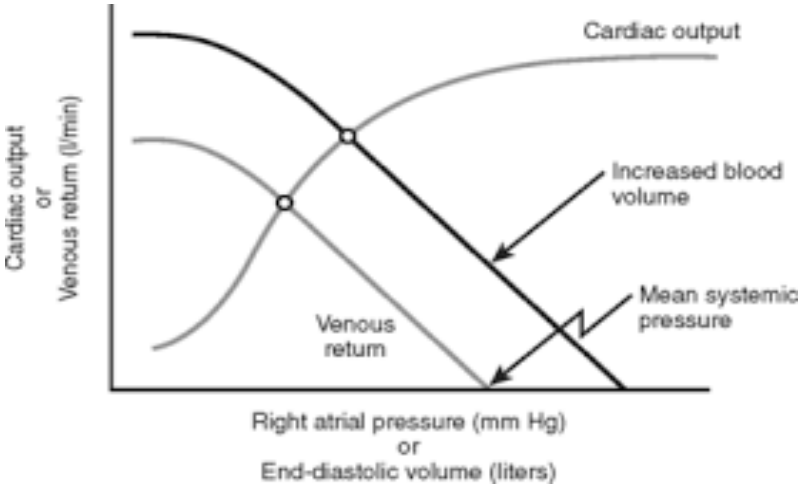
- Negative ionotropic agent
  - Cardiac output is decreased
  - Heart operates at high atrial pressure

EFECT OF VENODILATORS ON OPERATING POINT



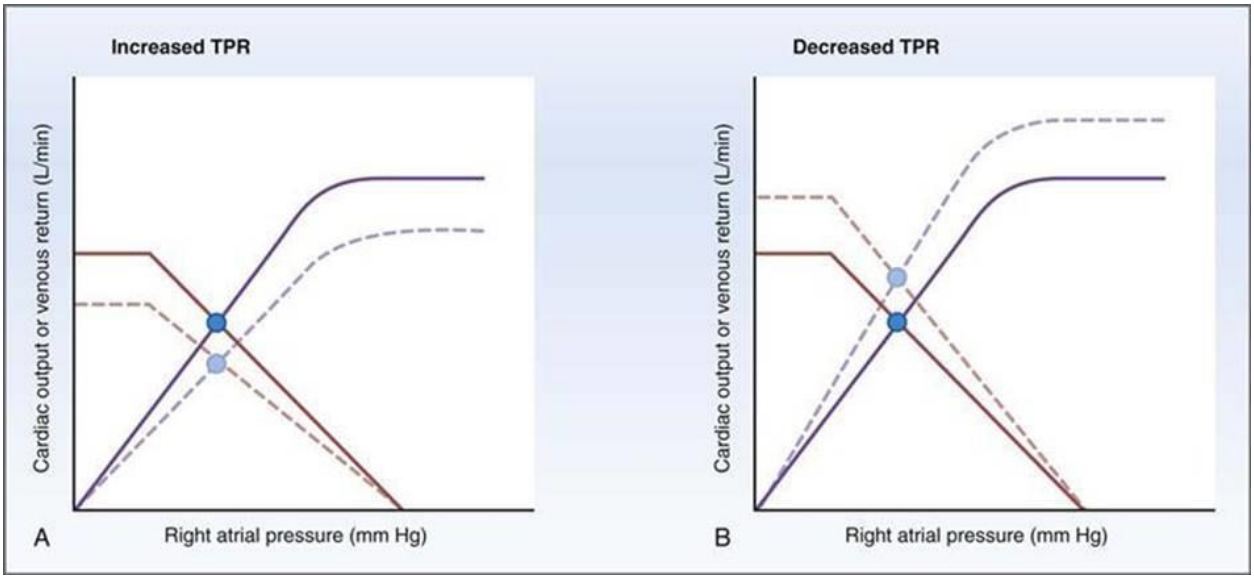
Under the effects of venodilators, cardiac output is decreased as right atrial pressure is decreased.

EFFECT OF VENOCONSTRICTORS OR INCREASED BLOOD VOLUME ON OPERATING POINT



Due to venoconstrictors, atrial pressure and cardiac output is increased

EFFECT OF TPR ON OPERATING POINT



- **ARTERIOLODILATOR**
  - Decreased TPR
  - Decreased afterload
  - Cardiac output increase
  - Right atrial pressure doesnot change

- **ARTERIOLOCONSTRICTOR**
  - Increased TPR
  - Increased afterload
  - Cardiac output decrease
  - Right atrial pressure does not change