PHY
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CARDIAC OUTPUT

&

Venous Return

2nd edition

Created By :

Suhibe Al. Masry

Muhammad Al WATBAN

Approved by Dr. Tariq

Notes

Abdullah Al Faris

Eman Al Rasheadi

Ismail Raslan

Salman Al Sfaiyan

Figures:

Abdullah Al æqeal

Muhammad Al WATBAN

Ãmna Bljoun

Ãmna Bljoun

Ahmed Al Duraihem

To contact with the team :

ThePhyllteam@yahoo.de

First Version

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Cardiac output

Is the volume of blood ejected from each ventricles per minute

(either from the left or the right)

Average cardiac output is 5 L in resting condition

Stroke volume : the volume of blood ejected per beat

Cardiac output = Heart rate × Stroke volume

Any factor affecting (↑ or ↓) the heart rate or the stroke volume will affect the Cardiac output

$$\textbf{Cardiac Index} = \frac{Cardiac\ Output}{Body\ surface\ area}\ \textbf{/sq. meter}$$

high cardiac index means better Cardiac output.

Cardiac output = venous return ◄ in normal condition

Normal pressure in Right Atrium is : 0 - 2 mm Hg

If Right Atrium Pressure increases, the Cardiac output will increase, until the pressure reaches **4** mm Hg (the maximum)

But the Cardiac output can reaches 25-30 L in exercise conditions

Hypereffective : more cardiac output , can reach 25-35 L in severe exercise

Affected by physiological conditions

Hypoeffective : less cardiac output , reach less than 5 L

Affected by physiological conditions

Physiological variations :

Increasing cardiac output:

- Exercise (up to 700%)
- Eating (30%)
- increase metabolism
- High environmental temperature
- Pregnancy
- Anxiety (50-100%)
- Sympathemimitics, epinephrine

Decreasing cardiac output :

- Sitting or standing from lying position(20-30)
- Due to pulling effect of gravity

pathological variation:

Increasing Cardiac Output:

- Fever : will increase rate of metabolism
- Hyperthyroidism : will increase rate of thyroxin hormone that controls the rate of metabolism
- Anemia → low Oxygen → increase anaerobic metabolism →vasodilatation

Decreasing Cardiac Output:

- Hypothermia
- Hypothyroidism

} Cause vasoconstriction

- Myocardial diseases e.g. infarction, failure…
- Rapid arrhythmias

End of Diastolic Volume (EDV) :

It's the volume of blood remains in the ventricles at the end of the diastolic condition

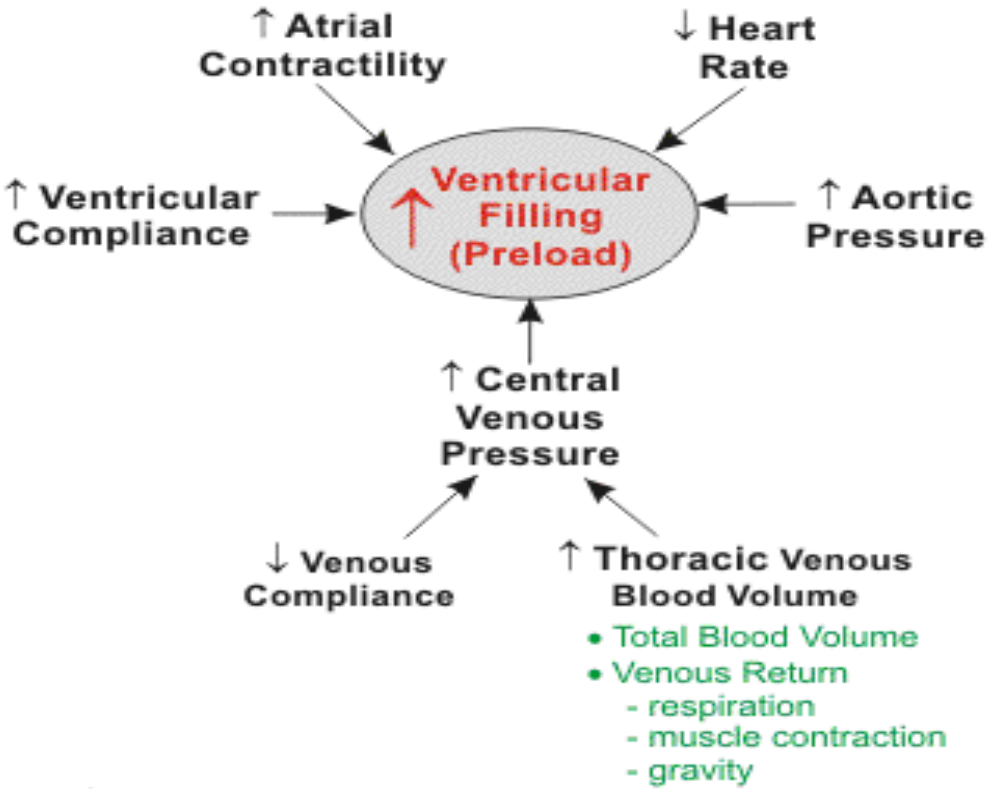
EDV : is also called Pre load

↑ EDV = ↑ Force of contraction

↑ Venous return → ↑ EDV → ↑ ejected blood → ↑ Force of contraction →

→ ↑ stroke volume → ↑ Cardiac output

Factors increases preload



↑Increase of atrial contractility

↑Increase of the ventricular compliance

↑In crease the central venous pressure (by ↓ the venous compliance)

↑Increase the thoracic venous blood volume

↑Increase the aortic pressure

↓ Decrease of heart rate

Frank- Starling mechanism

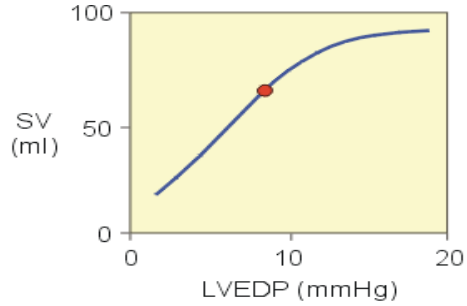


Figure 1. Frank-Starling mechanism. Increasing venous return to the left ventricle increases left ventricular end-diastolic pressure (LVEDP) and volume, thereby increasing ventricular preload. This results in an increase in stroke volume (SV). The "normal" operating point is at a LVEDP of ~8 mmHg and a SV of ~70 ml/beat.

Ability of the heart to change its force of contraction and therefore stroke volume in response to changes in venous return

- when the pressure or the EDV increase will lead to increase in force of contraction and will increase stroke volume "within limit"

Venous Return ↑ = EDV ↑ = Blood Volume ↑ = Force of contraction ↑

Venous Return ↓ = Cardiac Output ↓

Venous Return ↑ = Cardiac Output ↑

↑ : increase ↓ : decrease

Frank – starling's Law controls the normal performance of the heart

The Greater venous return = the greater Cardiac Output

ESV = EDV – Stroke volume

120 - 70 = 50 ml

End Systolic Volume (ESV) : is the volume remains in the ventricle after systolic contraction

After Load : the pressure against which the ventricle contract in order to eject blood

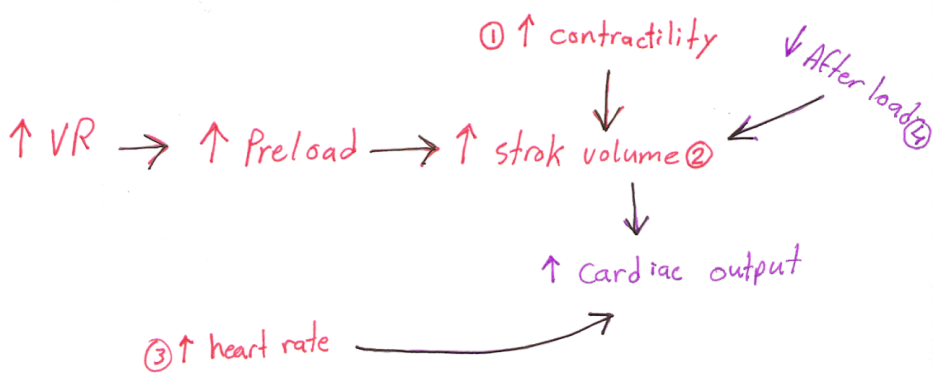
↑ After Load = ↓ Cardiac Output

↑ Preload = ↑ Cardiac Output

Factors increase After Load :

Increase of aortic pressure : Hypertension

Disease of aortic valve : Aortic Stenosis



①Contractility increases by :

- Sympathetic
- Positive Inotropic agents :
  - Sympathetic stimulation
  - Catecholamine
  - Thyroid hormones
  - Ca^{+} Increase in extracellular fluid

②Stroke volume increases by :

- Ca^{+} increase
- epinephrine , nor-epinephrine
- Increases of contractility

③Heart rate increases by :

- Catecholamine (epinephrine , nor-epinephrine)

After load decreases by : decrease of Atrial Blood Pressure during Diastole

Note !

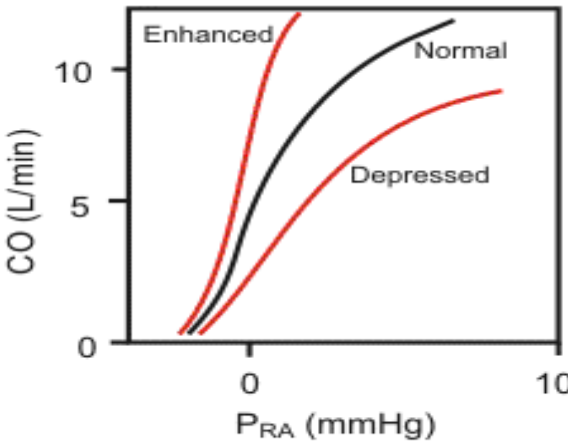
When :

↓ After Load = ↑ Cardiac Output , ↑ After load = ↓ Cardiac Output

↓ Preload = ↓ Cardiac Output , ↑ Preload = ↑ Cardiac Output

*Cardiac output pressure curve

The pressure is the same but Cardiac Output changes due to physiological and pathological factors .



Extrinsic Regulation of Cardiac output : (factors outside the heart)

Nervous :

- **Sympathetic** will increase Heart Rate and stroke volume because it affects the contractility
- **Parasympathetic** will decrease Heart Rate doesn't affects stroke volume because it doesn't supply the ventricles **BUT** if there was a significant change in the Heart Rate the Cardiac Output will decrease because **CO =SV x HR**

Chemical

- Potassium
- Calcium (ionized Ca)
- Thyroxin
- Catecholamine

Measurement of cardiac output

There are 2 methods to calculate Cardiac Output :

❶ Echocardiograph techniques **(not important to know)**:

- estimate stroke volume then we multiply it by heart rate cardiac output

❷ Fick's principle :

Arterial level – venous level × blood flow (Cardiac output)

$$\text{Blood flow (cardiac output)} = \frac{amount}{arterial - venous}\ L/M$$

- **Arterial level** : the concentration of Oxygen in pulmonary veins or any artery
- **Venous level** : the concentration of Oxygen in pulmonary artery ONLY
- **Amount** : is the Oxygen observed by the lungs per minute
  :It's measured by device that measures Oxygen in expiration and inspiration

Arterial level presents oxygenated blood

Venous level presents deoxygenated blood

e.g. if the oxygen consumption is 250 ml

the O_2 of the pulmonary artery = .2 ml

the O_2 of the pulmonary vein = .15 ml

how much is the cardiac output ?

$$\frac{250}{.2 - .15} = \frac{250}{.05} = 5000$$

Venous return

..Is the blood volume which return to the right atrium per minute

It should equal the same amount that leaves the left ventricle (Cardiac Output)

Venous return ↑ = cardiac output ↑ (in normal condition)

Factors controlling the venous return :

Skeletal muscle pump :

- whenever there is skeletal contraction it well increase the venous return

exercise causes skeletal contraction

Respiratory pump :

- during inspiration the pressure will be negative in the thoracic cavity and that well lead the convert the right atrium and **increase** the venous return

Blood volume :

- the greater the blood volume is , the **more** venous return well accrue

Pressure gradient :

- the pressure in right atrium is ZERO , and the pressure in the venules is 18 mm Hg , so if right atrium pressure increased the venous return will **decreased**

Venous pressure :

- it's the contraction of veins by **sympathetic stimulation** and it will increase the venous return

Gravity :

- whenever you stand without moving , the venous return will **decrease**

< Related

More salt and water increase the venous return

When the fluid moves from interstitial space to capillaries it will increases the venous return

In exercise : the venous valves make the blood unidirectional and that will increase venous return

Prolong standing causes edema and increase the pressure in the foot

Pressure will decrease from small veins to large veins

The pressure in large vein nearly 0 to 1

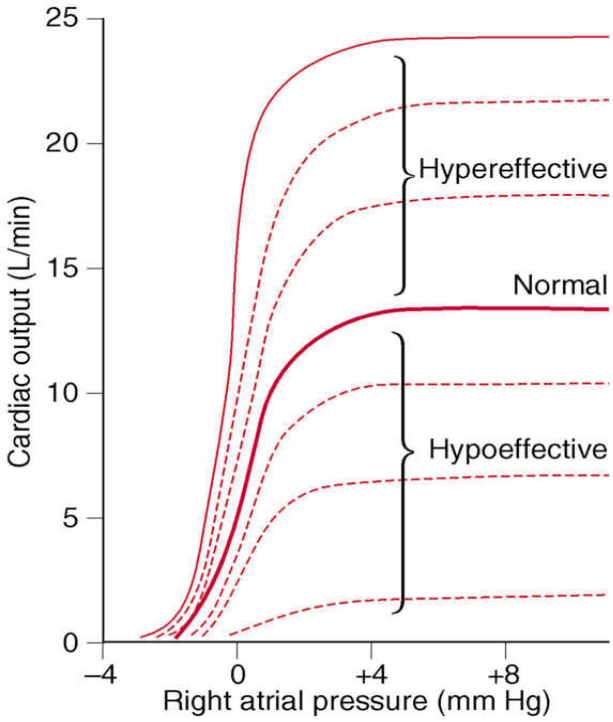
Increase pressure gradient = increase venous return

In sleeping position , the difference of pressure in heart and foot less than standing position

↑ pressure gradient = ↑ venous return

Venoconcentration =↑ venous return Venodialation =↓ venous return

Relationship between Cardiac output and Right Atrial Pressure (RAP)



This figure shows the relationship between Cardiac Output and right atrial pressure

- The normal RAP is : between (–1 → +2) mm HG

When the RAP increases the cardiac output will increase

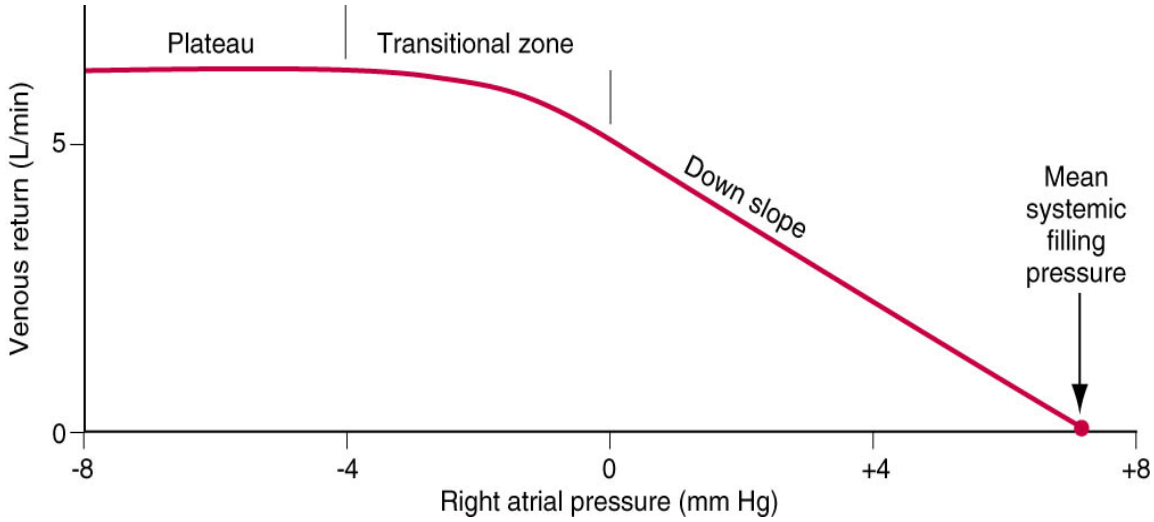
- If the pressure in RA increases abnormally , the Cardiac output will remain the same (no changes)

But if the RAP increases in normal range the cardiac output will increase

This means that the venous return also will increase

- The relationship between the cardiac output and right atrial pressure RAP follows Frank-Starling law in normal ways.

- If the cardiac output is constant and venous return is high that will mean more blood pressure in tissues will cause edema and other diseases



This figure shows the relationship between venous return and right atrial pressure

Mean Systemic filling Pressure (MSP) : when the heart stops contracting , the pressure remains in the vessels is called " mean systemic filling pressure "

Normal mean systemic filling pressure in artery is is 7 mm HG

When the heart stops , the range of blood volume is 5 L :

- 4 in veins
- 1 in arteries

When the RAP become more negative , it will cause collapse of large veins and will stop the venous return

⬢The more positive RAP the less venous return

⬢Very low or very high RAP will affect the venous return

MSP depends on blood volume : when the body has more than 5 L of blood

If the veins has 4 L (total capacity) , and arteries has more than 1 L(1 L is max capacity) = increase in MSP

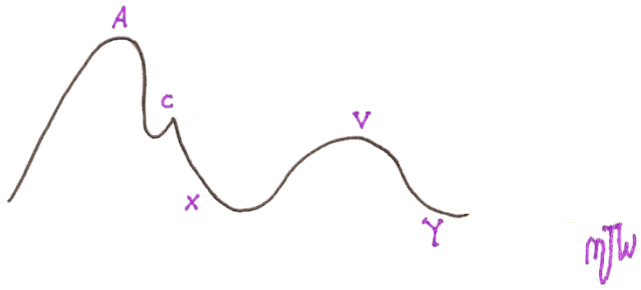
If the body has only 4 L of blood , all will be in the veins and MSP = ZERO

Jugular venous pressure

Is the indirectly observed pressure over the venous system

So if there is increased pressure in venous system it will be shown in the jugular vein

It helps in detecting heart and lung disease



- **3 positive waves (upward):**

A wave : when the right atrial contract, some blood will return to SVC creating **A** wave (because there is no valve between them)

C wave : right ventricular contraction during isovolumetric contraction . this will cause bulging of Tricuspid into the right atrium

V wave : atrial filling (when the atrial is filled with blood , some will remain in jugular vein and cause **V** wave)

- **2 Downward waves :**

X : the atrium relaxes and tricuspid valve move downward

Y : when the tricuspid valve opens , more blood will come from atrium to the ventricle (ventricle filling)

Raised Jugular venous pressure

If you can see the jugular vein when patient is at 45^{o} position then might be a high pressure caused by :

1. Increase right ventricle filling pressure , e.g. the right heart failure , fluid over load

2. Obstruction of blood flow from right atrium to right ventricle :

    - e..g tricuspid stenosis , when the valve is tight , some of the blood will return to the jugular vein
    - more blood transfusion caused raised in Jugular vein

3. Superior vena caval obstruction e.g. : retrosternal thyroid goiter

    - If the thyroid goiter remains the sternum, it will cause high pressure in the Jugular venous

4. positive intrathorasic pressure e.g. : pleural effusion , pnemothorax

    - The entrapleural pressure usually negative , when a person get stabbed , the pressure will be positive which may collapse the lungs then it will increase the jugular venous

    - The pressure should be negative . fluid cause positive pressure

Reduces venous return in right atrium and increases Jugular vein pressure

Why JVP increases only in right heart failure ?

Because when there is left heart failure this will cause edema in the lungs. But in right heart failure edema will be in lower limb or liver enlargement

Venous Return(VR) notes

Sympathetic : supplies the blood vessels = venocontraction

- Cause increase in heart rate and force of contraction
- ↑constriction = ↑in venous return
- Supplies all the heart

Parasympathetic : Doesn't supply blood vessels

- Cause decreasing in heart rate ONLY
- Act on the atrium ONLY
- Supplies SA nodes

Veins :

- Veins has valves , to prevent blood from flowing back
- Of there a damage in venous valve , that will decrease the venous return

That's all :)