Physiology

Cardiovascular system

Sheet Slide

Number: 10

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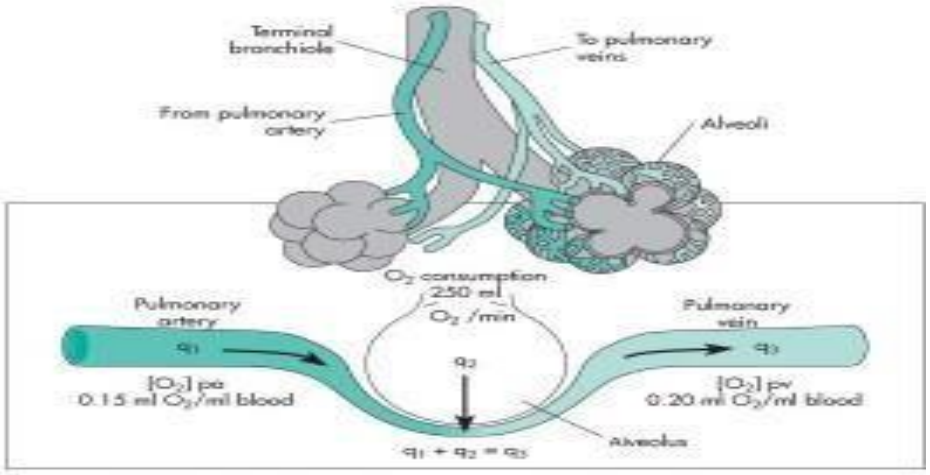
Note: This sheet was done with the help of sheets 14 and 15 from the last year

How to measure cardiac output

1. The direct method: not practical
2. Indirect methods:
   a. **Electromagnetic flowmeter**: We have two poles of a magnate (north and south). When a charged flow runs between the two poles, a current is thus formed and can be followed up by a galvanometer. Because blood is full of electrolytes, it is a charged flow, and the current that would be formed between the poles of the magnate is proportional to the flow. If we measure this flow per min, we can calculate the cardiac output. This method can be used around any artery, and many times. It is usually used in cardiac surgery.

   b. **Oxygen Fick Method**:



Follow the figure above:

- The oxygen content of the pulmonary vein equals the oxygen content of the pulmonary artery ***plus*** the oxygen that has been up-taken by the lung.
- The amount of oxygen that enters the lung per min. through the pulmonary artery (Q1) = cardiac output * oxygen concentration in the venous blood

Remember: The pulmonary artery carries venous blood.
{To know the total amount of oxygen we multiply the total blood by the concentration.}

- Q2= the amount of oxygen taken by the lungs from air through alveoli.
- Q3= the amount of oxygen that enters the pulmonary veins: This equals the cardiac output * arterial oxygen concentration.

*why do we say cardiac output?? This blood is returning to the heart and thus must equal the output.
*But why arterial? Pulmonary veins carry arterial blood.

- The Fick principle states that: Q3=Q1+Q2.
  Mathematics:
  - ✓ Q3= CO * O2 conc. In arterial blood.
  - ✓ Q1= CO * O2 conc. in venous blood.
  - ✓ Q2= oxygen uptake by the lungs from the air.
  - ✓ Q3=Q1+Q2
  - ✓ CO * O2 con in arterial = CO * con o2 venous + oxygen uptake.
    So, Oxygen uptake = CO $\{C_{AO2}-C_{VO2}\}$ → CO=Oxygen uptake/ $\{C_{AO2}-C_{VO2}\}$

Cardiac output = (oxygen consumption in 1min)/ (arterial concentration of oxygen – venous concentration of O2).
{When calculating, pay attention to the units}

*How to find the oxygen consumption?

- We measure it using a spirometer. You let the patient breathe and you measure concentration of inspired O2 after one or two minutes.

*How to find the arterial and venous concentrations of oxygen?

- All arteries have the same concentration of oxygen because the exchange happens only at the level of the capillaries. That's why you can take a sample from any artery to measure the concentration of oxygen in arterial blood.

Swan-Ganz catheter: A catheter with many ends to do several measurements at the same time. It has a thermometer, an area for injection, and pressure transducer.

- We can't take venous sample from any vein because the concentration of oxygen differs according to the metabolic needs. Thus we put this catheter in the pulmonary artery or the right ventricle (using the cubital vein). This is called **central venous blood**; as if we mix all blood and take the sample.

3- the indicator dilution principle

4- thermodilution method curve

Venous Return

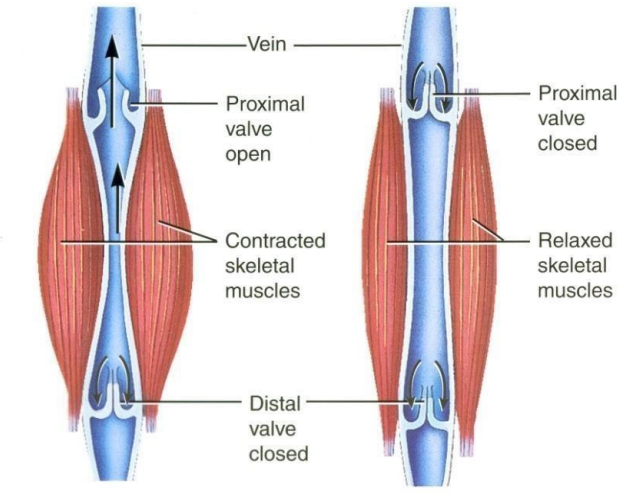
It is the amount of the blood that returns to either atrium per minute. It equals the cardiac output in normal conditions according to Frank-Starling (What comes to the heart has to leave. 1ml difference between ventricles per beat leads to the accumulation of 4liters in the heart after 1 hour).

Venous return is a flow and thus based on Ohm's law. So,
VR = CO = Δ P/R = (Venous pressure –Rt. Atrial pressure)/ resistance to venous return

How is the blood flow maintained unidirectional toward the heart against gravity?

1. The veins are embedded within skeletal muscles that when contract squeeze the veins and push the blood upward toward the heart.

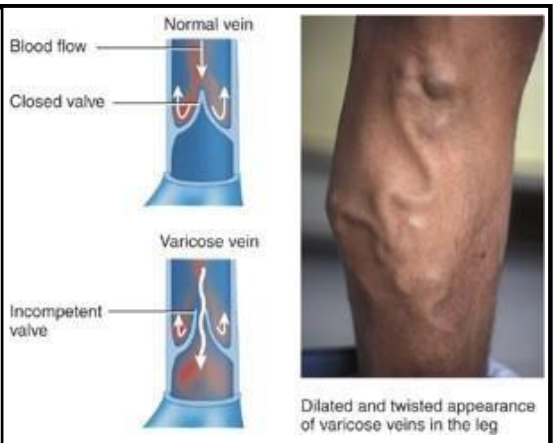
2. Veins have valves. When these valves are normal, they prevent blood from going down due to gravity. They close and keep little amount of blood. Because of this the blood is discontinuous and the pressure in the veins is maintained low (we need this low pressure for our normal pressure gradient).



(a) Contracted skeletal muscles (b) Relaxed skeletal muscles

Clinical correlate: varicose veins

When someone stands for a long time, blood will be collected around the valves. This causes the valve to slightly open and the blood column will become continuous. By time, the valve will become incompetent and this increases the pressure in the affected veins leading to varicose veins. These veins are congested, dilated, tortuous and blue.

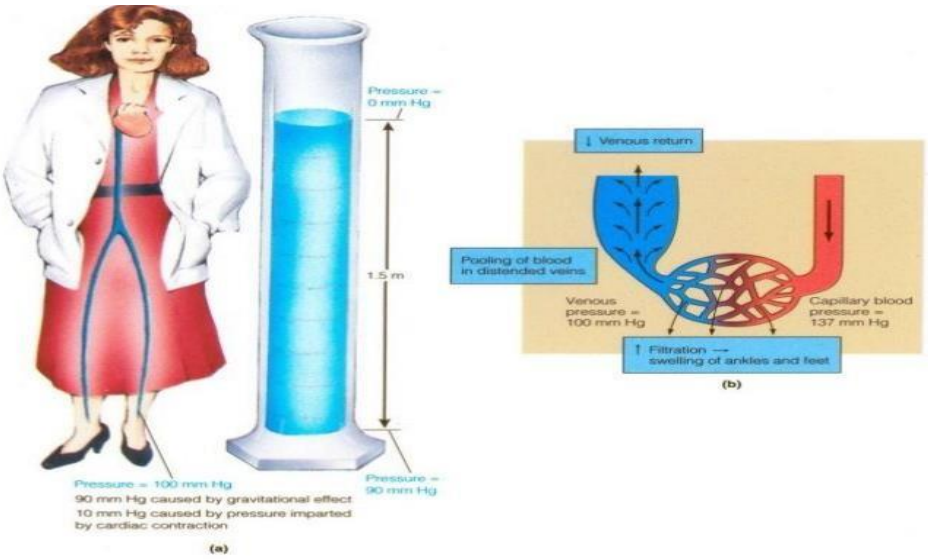


Let's assume that the right atrium is our zero point and from there to the toe we have a continuous blood vessel that has a length of 136 cm, what is the pressure at the lowest point?

- Length from the right atrium = 136cm
- Pressure = blood column/ density of mercury= 136/13.6= 10 cmHg= 100 mmHg

Here lies the problem of standing still for a long time.

Abdominal pressures tend to increase venous pressures in the legs.



Central venous pressure (CVP)

- Pressure in the right atrium is called central venous.
- ❖ In the right atrium CVP is usually 0mmhg, but it might reach 20mmhg “maybe 30 sometimes!”
- Increases in right atrial pressure causes blood to back up into the venous system (venous return decreases) thereby increasing venous pressures.
- ❖ To monitor the right atrial pressure we use a Catheter “central venous line".

- CVP should be monitored in heart failure patients because increased CVP may lead to sudden death, especially when giving IVF fast and haphazardly! (Could be letha!). So, we give him IV fluid slowly and monitor the CVP. When it increases above normal we stop.
- Right atrial pressure (RAP) is determined by the balance of the heart pumping blood out of the right atrium and flow of blood from the large veins into the right atrium. When pumping is decreased or the venous return increased, right arterial pressure will increase.
- **Factors that <u>increase</u> RAP**:
  - **Increased blood volume**: It increases everywhere including in the right atrium.
  - **Increased venous tone**: more venoconstriction → push more blood to atria → increased pressure in right atrium and right ventricle too
  - **Dilation of arterioles**: Dilation of arterioles → less resistance → more blood flow→ increased pressure! "**arterioles are the main resistant vessels"
  - **Decreased cardiac function**: When contractility increases → ESV decreases→ RAP decreases because RAP is a reflection of how much blood is found in the ventricle. On the other hand, when it decreases RAP increases. That's why it should be monitored in patients with heart failure.

Remember: If there is an increase in venous return there is an increase in EDV, increased stroke volume and cardiac output "within physiological limit"s (Frank-Starling law)

<u>Factors affecting venous return</u>

1. **skeletal muscle contraction**: When muscles contract→ increased venous pressure → increased gradient between the veins and right atrium →increased venous return →increased cardiac output.
2. **Veno-constriction** & Arterio-constrction " by sympathetic stimulation!": <u>Veno</u>-constriction→ increased venous pressure → increased gradient →increased venous return →increased cardiac output.

   Note: main volume of blood is collected in veins, so arterio-constriction has less effect on the venous return (negligible), but vasoconstriction as a whole increases venous return due to veno-constriction although arteries are constricted too!

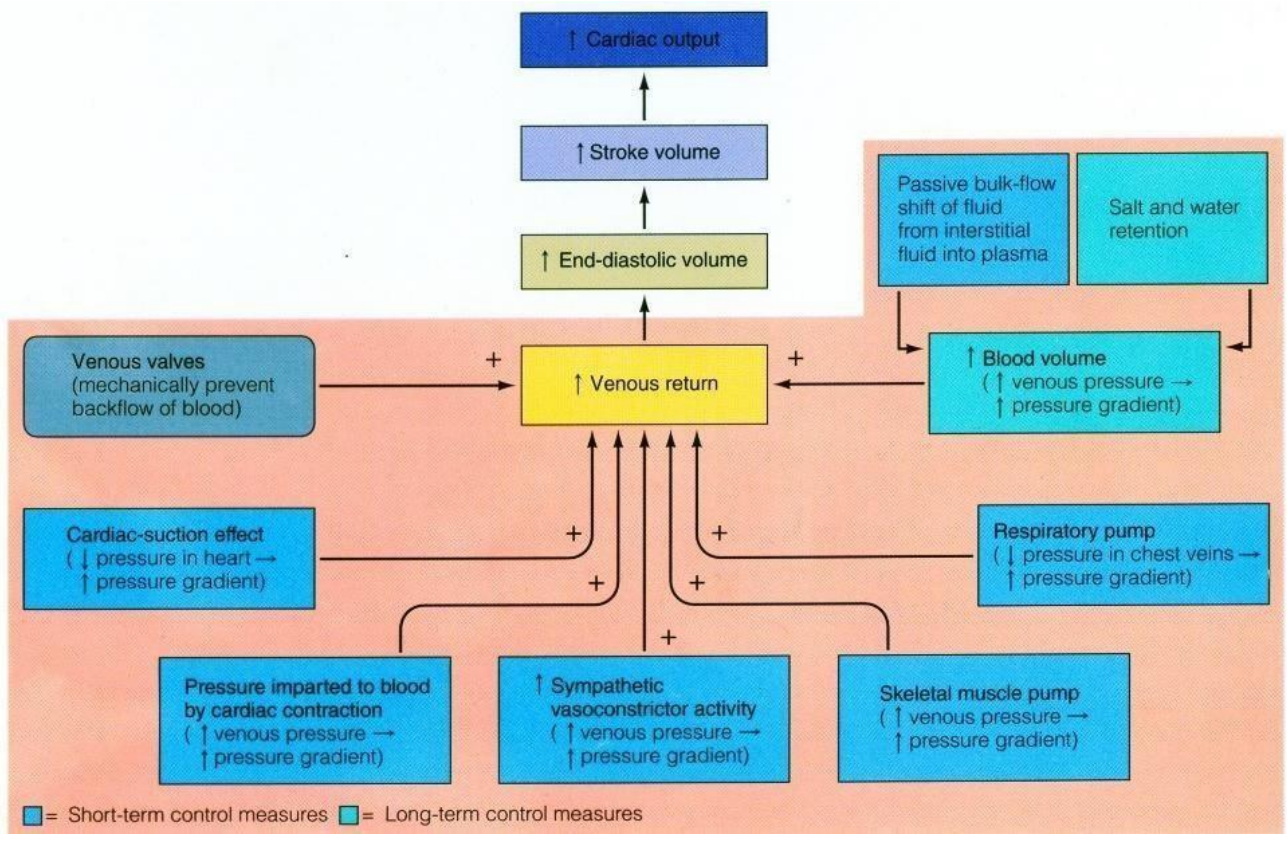
3. **Blood volume**: increased blood volume means more pressure →increased venous pressure →more gradient → more venous return → more cardiac output
4. **Respiration**:

   inspiration → volume in the chest increases→ pressure inside alveoli decreases to less than pressure outside "atmosphere" (according to boyle's law P1*V1=P2*V2) → less pleural pressure→ less right atrial pressure → more return.

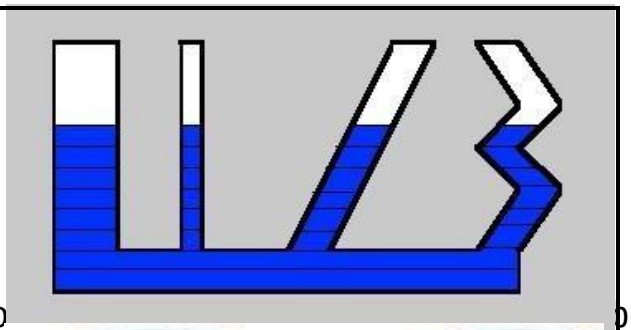
   Normally pressure in pleura is -4, during inspiration it becomes -6 instead of -4!, pressure in right atrium becomes -2 instead of 0 "gradient increased" →more venous return !

   Expiration: IPP returns to -4 , CO back to normal "CO and venous return are related to each other"

5. **Cardiac function**: if it increased → ↓ESV→ RAP ↓ → increased gradient →increased venous return →increased cardiac output. This is called cardiac suction.
6. Venous Return is decreased if **valves** are incompetent, "blood flows back" .



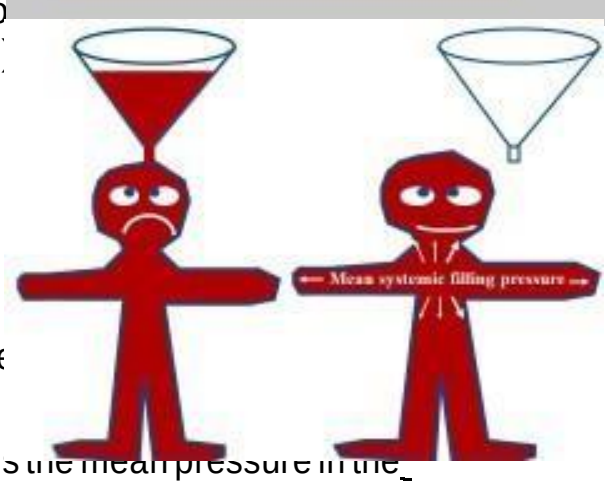
The Venous Return Curve



❖ The concept of Communicating vessels
(الاواني المستطرقة)

(if we stop the heart and equilibrate the blo be parallel in all these vessels (just like the figure)

- if we add more blood the level will rise in all vessels!
- If we lowered the pressure in one of them, water will flow to this vessel (from high to low pressure), until it is equal again.
- So the idea here is "same level means same pre **systemic pressure**.
- It is called mean <u>systemic</u> pressure because it is the mean pressure in the <u>systemic circulation</u>.
- At this point the arterial pressure will be equal to the mean systemic pressure.



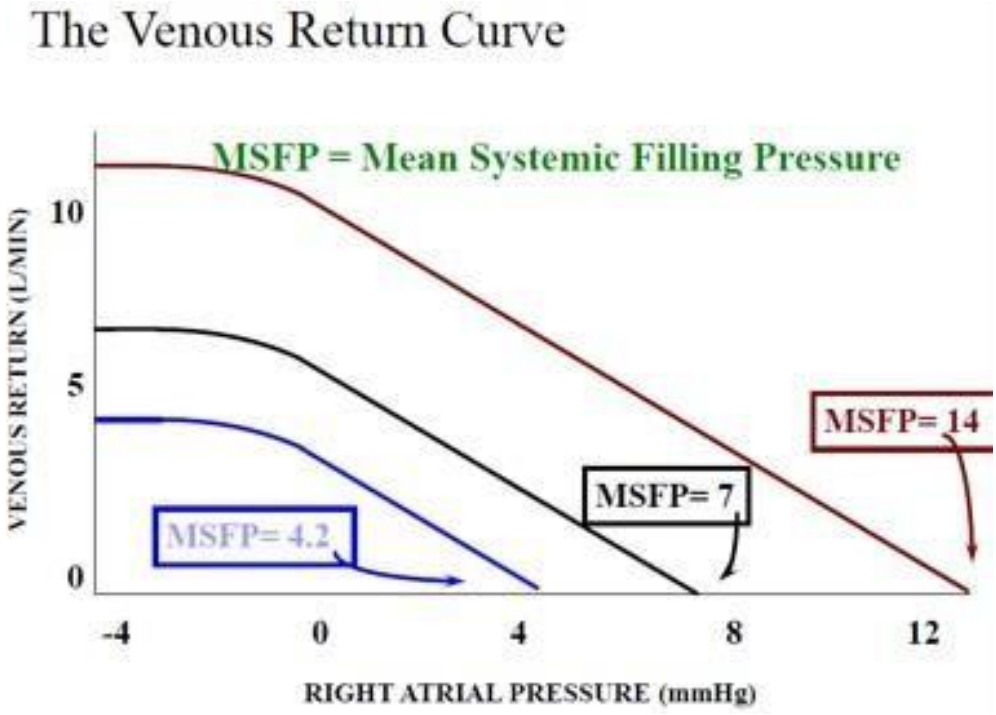
"The first person to measure the mean systemic pressure was Guyton, he first measured it in goats, then tried to do so in humans and found it was 7-8 mmhg"

- Mean systemic pressure = **mean systemic filling pressure** (MSFP) = **7- 8mmHg**

- If the pressure in the right atrium equals to MSFP, this means that there is <u>no venous return</u> "Gradient is 0" {there must be a gradient between the right atrium and the pressure in veins in order to create a blood flow "from high to low pressure"}

- When right atrial pressure decreases below 8mmHg (MSFP), venous return will increase.
  If pressure in right atrium is 6mmHg there's a little flow. When it's 5mmHg, flow will increase "more" and so on …{See figure below, the black curve in the middle}
  That's why it's called <u>filling</u> pressure; because the difference (gradient) between MSFP and atrial pressure is what causes its filling.
  When it reaches zero, the venous return will be 5L/min

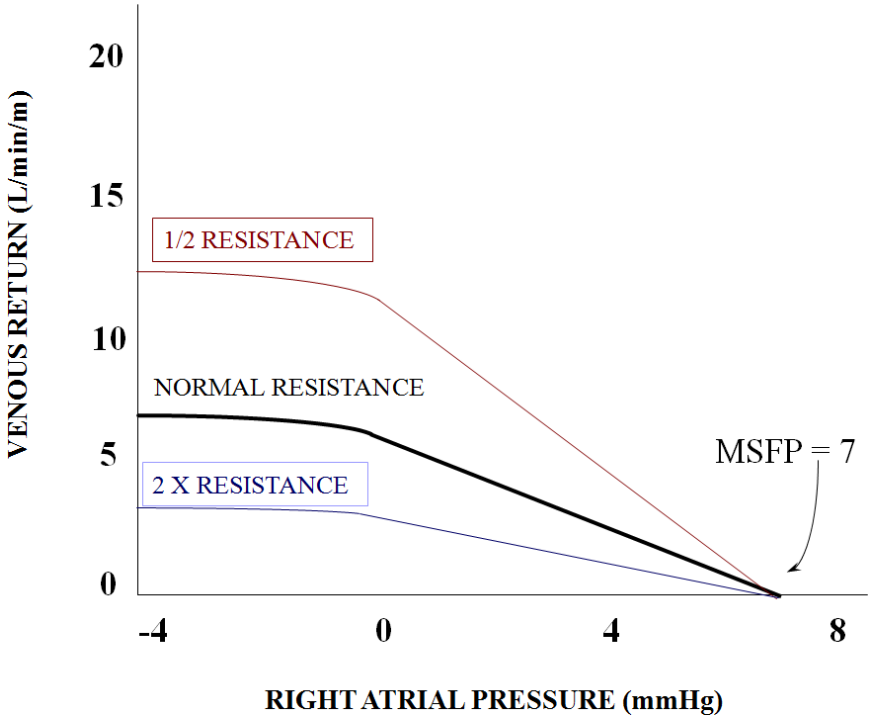
When pressure in the right atrium becomes negative, vessels will collapse. It will open when there is blood collecting in. Here, the venous return will no more increase (plateau in the figure below).



What affects MSFP?

1. **IV fluid infusion**: increased fluid "blood volume" increases venous return→ shift the curve to right and upward =14 (the red curve). Increased venous return increases cardiac output thus increasing pressure.
   That's why when someone has hypotension we give him/her IV fluid.
2. **Hemorrhage and dehydration**: means low blood volume →shifting the curve to left and downward =4.2 (the blue curve)

Important note: increasing or decreasing TPR (total peripheral resistance) has no effect on MSFP because resistance is mainly in the arterioles, and arterioles contain little amount of blood, so it won't affect the MSFP. While veno-constrction or dilation affects the MSFP!



When you increase or decrease the resistance, MSFP says the same but venous return changes:

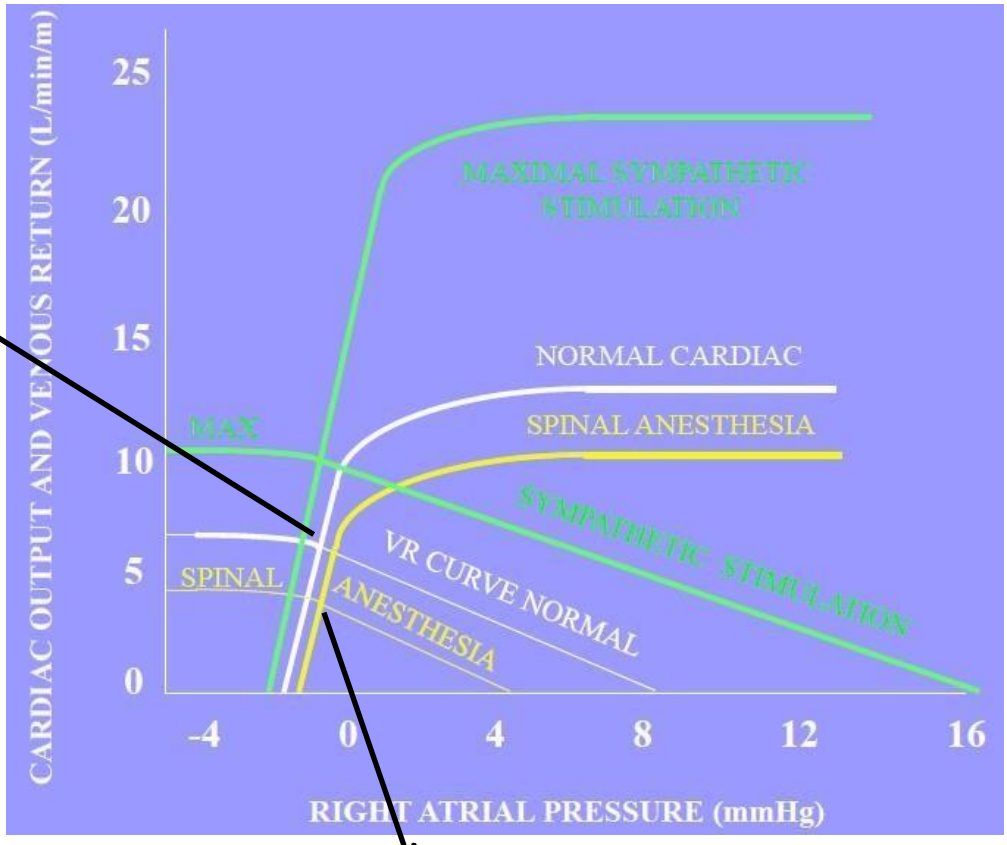
- When the resistance is half the normal, venous return will increase.

- When the resistance is increased, venous return will decrease.

*Other factors affecting MSFP will be explained more in the next lecture.

This figure combines the cardiac output and venous return curves.

This is the point of intersection between normal CO curve & normal VR curve (when cardiac output equals venous return). It represents **the working CO**.



- Spinal anesthesia (sympathetic inhibition) decreases both CO and VR. The heart will work at the new intersection point.
- Sympathetic stimulation will increase CO & VR. The heart will work at the intersection point between the new curves.

Other factors affecting venous return:

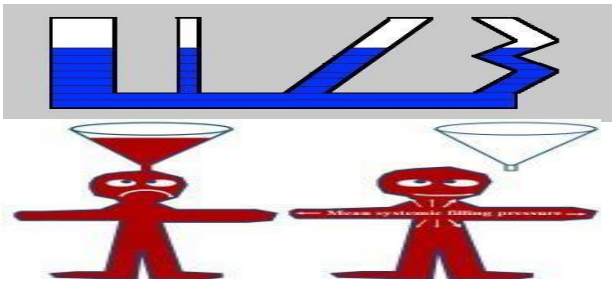
*Note: you will not be asked about these in the mid exam. They will be further explained in the next lecture and you will be asked about them in the final.

- **Arteriolar dilatation** → ↓RVR (resistance to venous return) → ↑venous return
  Because VR = (MSFP - RAP) /RVR
- **Thiamine deficiency** (Beriberi): the artery becomes flexible → arteriolar dilatation→ ↑venous return
- **AV fistula**: An arteriovenous fistula is an abnormal connection or passageway between an artery and a vein. It may be congenital or surgically created for hemodialysis treatments (in this case it's called AV shunt).

  Hemodialysis = means that blood is taken from an artery to be filtered and then returned back to a vein.

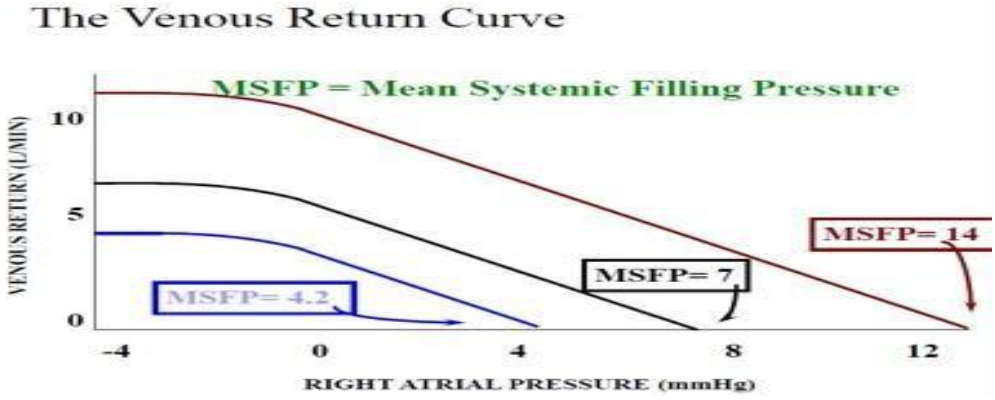
  AV shunt >> shunts blood from an artery to a vein bypassing the capillary network >> resistance decreases >> increase in VR. VR increases acutely, it then returns to normal.
- **Hyperthyroidism** → ↓RVR→ ↑venous return
- **Anemia**: it ↓RVR; why? Because the number of RBCs and this decreases viscosity.

RECAP: we started talking about the venous return curve. In this curve we relate the right atrial pressure with the vinous return (we put the right atrial pressure on the X axis and by that we can compare between this curve and the cardiac output curve which also has right atrial pressure values on the X axis). We said also if we stop the heart and equalize the pressure, the pressure will be the mean systemic pressure, because we are talking about the systemic circulation, now all over the circulation the pressure is the same, and it was measured by Guyton to be 7-8 mmHg.



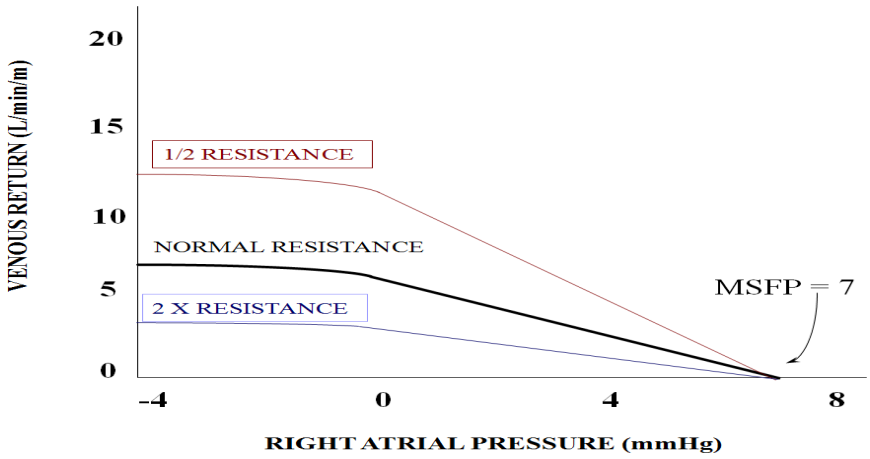
Now if the right atrial pressure equals this value, there will be no venous return (at point 0 of venous return we call it mean systemic filling pressure (MSFP)) . but if the right atrial pressure decreases then the blood will move to right atrium , until you reach 0 right atrial pressure the venous return at that point will be about 5 liters (look at curve A in the figure) , then when you become in negative pressure (which is actually vacuum) there will be collapse of large vessels , and they will open when the accumulated blood around the collapsed part of the vessels reaches a high enough pressure to open them to allow some blood to pass through them until they close again (they will open and close repeatedly) to the extent that we have almost plateau (in which the venous return will not increase significantly with decreasing right atrial pressure) .

Note that in MSFP : we say systemic because it is in the systematic circulation and filling because it causes filling of the heart .



Now how can you increase this MSFP ?

By increasing blood volume (infusion, too much drinking …) , venoconstriction…, that all shift the curve right and upward (see curve B above). But if you have bleeding , dehydration … that will shift the curve downward and to the left (curve C).



How does resistance affect MSFP ?

We will talk in upcoming lectures about the total peripheral resistance that lies mainly in arterioles. Arterial system doesn't have much blood, so changing the diameter of the arterioles (changing the total peripheral resistance) will change how these vessels will press on their blood content which is in this case a relatively small amount of the whole blood content, so it will not have any significant effect on the MSFP, while changing the diameter of the veins will affect a large amount of blood which will result in significant changes in MSFP,

so applying this , if you increased the mean arterial resistance (not the resistance for venous return) the MSFP will be the same except the flow (venous return in this figure) will change (with decreased TPR the venous return will increase and vise versa) .

Factors that affect venous return:

- one condition is called beriberi (thiamine deficiency) having elastic arterial walls that aren't going to recoil so low resistance exists , then arterial dilation will occur and we will have more flow. (in slides we have decreased RVR , which means resistance to venous return not total peripheral resistance).

  NOTE that venous return = (MSFP- RAP)/ RVR … RAP: right atrial pressure.

- AV fistula / AV shunt = we connect arteries and veins, this is done in patients with renal failure, in this case you shortcut the circuit decreasing resistance ->increasing flow (this occurs acutely , then we will have balance in resistance).

- Hyperthyroidism → increased metabolism (T3/T4 causes O2 consumption which causes releasing of vasodilators that ↓ resistance ↑ flow (RVR = resistance to venous return not total peripheral resistance).

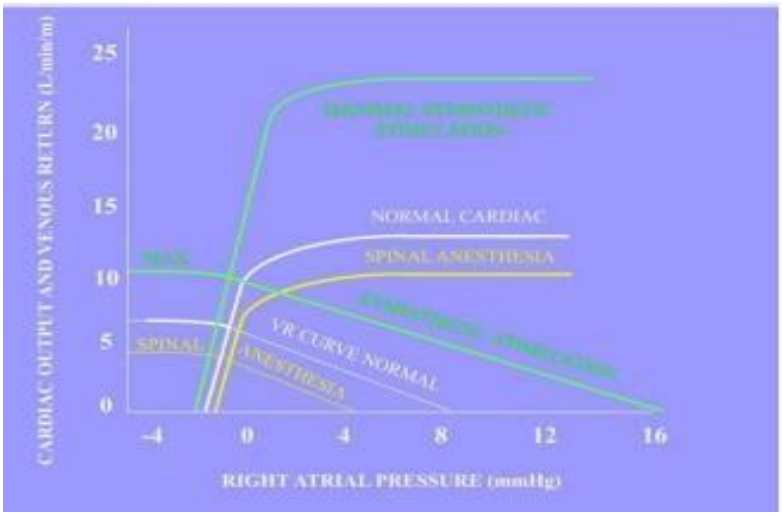
- Anemia : decreasing in RBCs that decreases viscosity then ↓ resistance then ↑ flow.

- Decreased blood volume leads to decreased MSFP which decreases the flow.

- Decreased venous compliance (as if we have venoconstriction) ↑ MSFP and ↑ flow.

- Decreased sympathetic causes venodilation → decreasing venous return.

- Obstruction (in femoral v. For example) of veins ↑ resistance (like in abdominal tumors or - normally- in pregnancy that sometimes causes varicose veins which ends after delivery).



Here we have 2 curves together **(cardiac output & venous return) curves** . The working cardiac output is the point of intersection between the 2 curves , where the cardiac output = venous return Anything that shifts the curves will change the point of intersection (for example, sympathetic inhibition that happens in spinal anesthesia).

End of preview