The Cardio-VASCULAR System

- ○ Anatomy
- ○ Histology
- ○ Pathology
- ○ Pharmacology
- ● Physiology
- ○ Microbiology

Lec #: **12**

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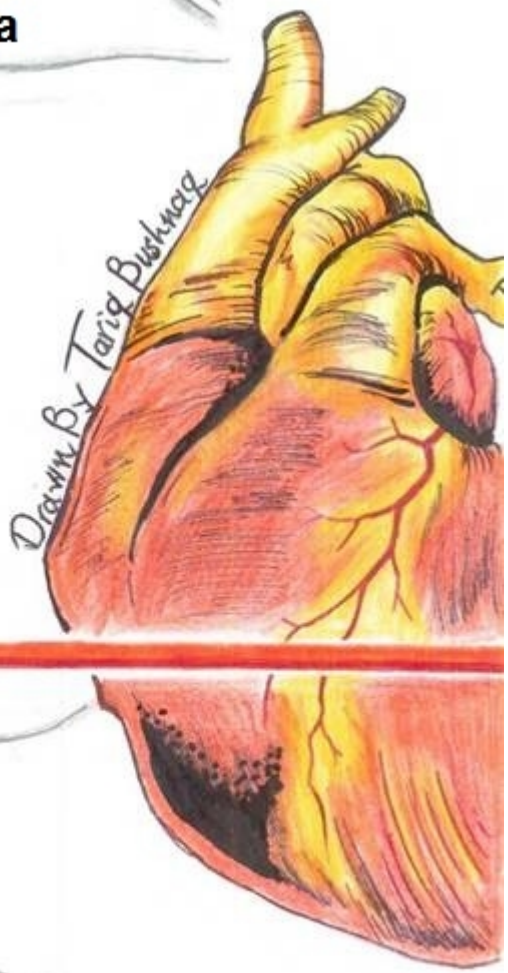
Done By: **Aseil & Wafaa**

- ○ Handout
- ● Sheet
- ○ Slide



CARDIAC OUTPUT & VENOUS RETURN

Note: when you see slides it means the doctor just read them so READ them

Factors that regulate cardiac output are those that regulate the stroke volume and those regulating the heart rate.

-Cardiac output curves

- The right atrial pressure and left atrial pressure are not that much different from one another, maybe one is 0 and the other is +2, unlike the difference between the right and left ventricles, especially during diastole.
- The pressure gradient around aortic valve during systole is very low, while it is very high during diastole (ventricular 0, aortic is 80)

We will talk about cardiac output curves, as we discussed yesterday, we put on the x-axis the right atrial pressure instead of the end diastolic volume since it's easier to measure.

As we said EDV is proportional to the atrial pressure, whenever EDV increases >> atrial pressure has to increase, why?

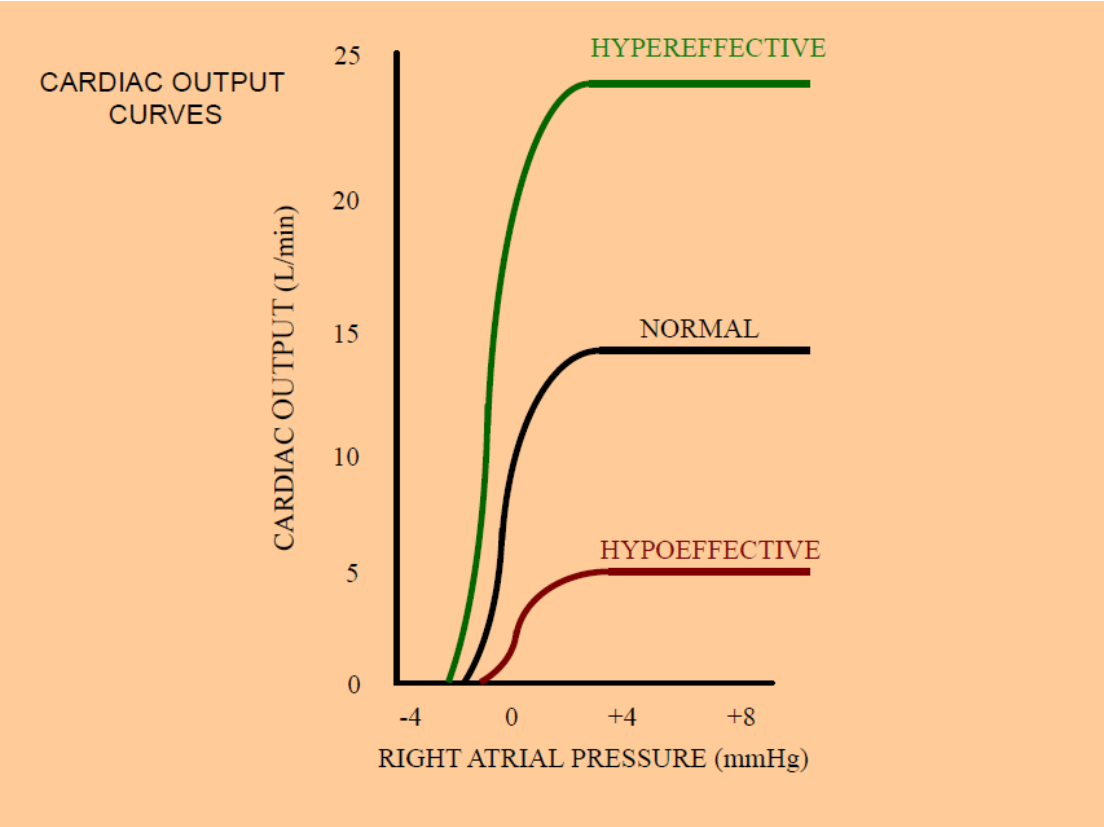
Blood has to flow from the atria to the ventricle, which means as the EDV increases, the end diastolic pressure increases as well.

When right atrial pressure is 0, the cardiac output is 5L/min, increasing EDV, means increased right atrial pressure which increases the cardiac output, according to frank-starlings law, within a physiological limit if you reach the optimum length of the muscle, you reach the maximum increase in stroke volume (the maximum increase in cardiac output), if you exceed this limit, the cardiac output would decrease.

So according to the law, within intrinsic regulation, maximum output could reach 15L/min (this is the normal)

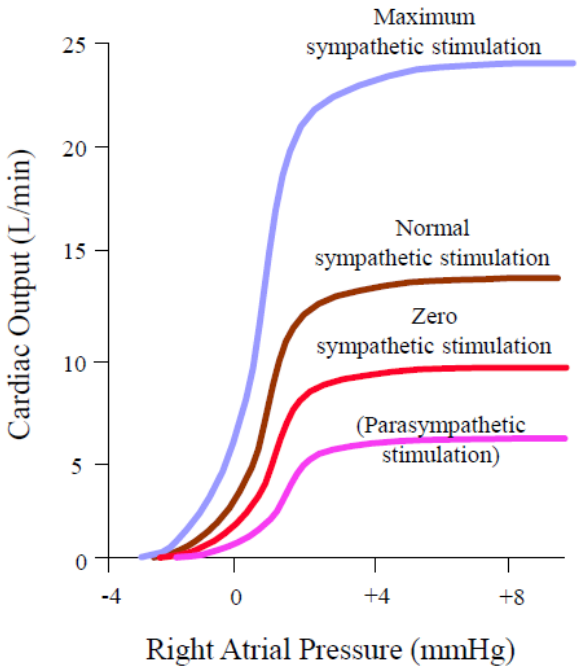
If you stimulate the heart (sympathetic stimulation), you will increase the contractility, meaning? When you increase contractility, with FIXED EDV, there is an increase in stroke volume and hence cardiac output, so at any value here on the right atrial pressure, the cardiac output has to increase >> positive inotropic effect, the curve here shown in green is for a HYPEREFFECTIVE heart.

While the red curve shows a HYPOEFFECTIVE heart, there is a decrease in maximum output for any given increase in value of right atrial pressure.



What are the causes?

Effect of Sympathetic and Parasympathetic Stimulation on Cardiac Output



The brown line represents the normal sympathetic stimulation of the heart, there is something called the basal tone for the sympathetic stimulation, meaning there is a basal rate of cardiac output which in this case is about 15L/min. this might increase or decrease, according to further stimulation or inhibition. Maximum sympathetic stimulation is seen in the blue curve, here there is "too much" cardiac output, this is a hypereffective heart (repeated info), if you inhibit the sympathetic stimulation, this might decrease the maximum cardiac output to about 10L/min.

Furthermore, the change in these curves affects the cardiac reserve, the cardiac reserve is between the normal value (5) and (15) so the normal cardiac reserve is? 10! (25-15) :P

But when we have a hypereffective heart! How much is the cardiac reserve?

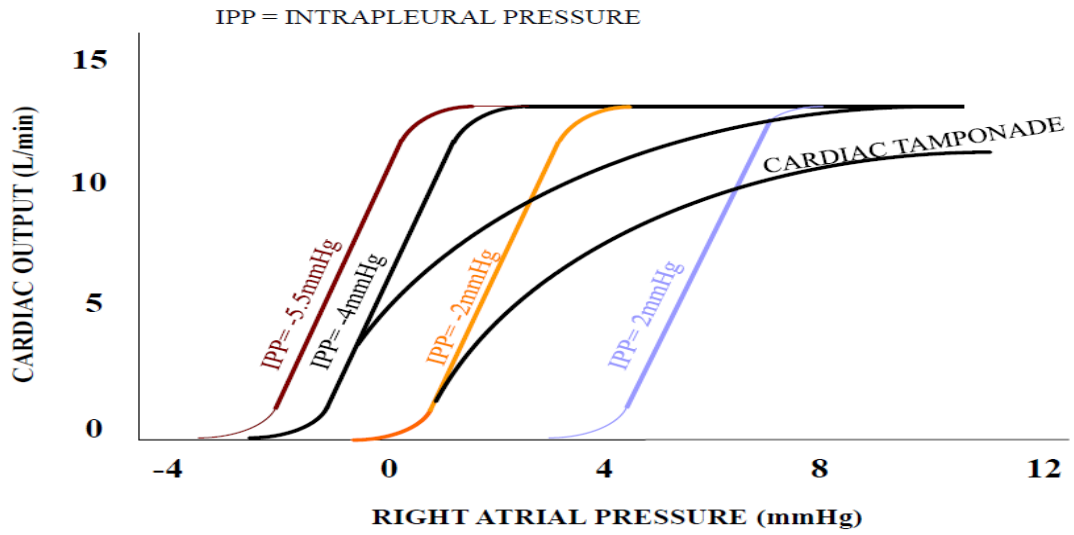
25 - 5 = 20 (with maximum sympathetic stimulation)

Cardiac reserve decreases when you inhibit the sympathetic stimulation, (curve without stimulation is 10) - (normal curve 5) = 5L/min so here the cardiac reserve is less

Parasympathetic stimulation MIGHT cause the pink curve (not very accurate in this picture).

Moving on to intrapleural pressure

Intrapleural pressure is the pressure around the heart, it is normally negative (normal IPP is -4mmHg according to Guyton) - unless there is pleural effusion- so with around -4mmHg intrapleural pressure, RAP is 0



If we decreased the IPP from -7mmHg, what would happen to RAP? -3

So the RAP is effected by IPP, it increases or deceases by the same difference in the IPP!

Another example, if IPP is increased to 2mmHg, how much is the RAP?

+6mmHG!

Why is this?

The amount of blood returning back to heart is proportional to pressure gradient between veins (sup and inf vena cava) and right atrium, this gradient acts as a driving force for the blood to enter the right atrium, let's assume pressure in veins is +5, and RAP is 0, then we have a gradient of 5! So as IPP decreases to -7 for example, RAP becomes -3 , now the gradient between the veins and RAP is increased right? It is 8 now! So the venous return would INCREASE, >> EDV would increase, shifting whole curve to the left.

Are we going to affect the maximum obtained CO? No, it stays the same according to frank-starling.

If we increase IPP, the whole curve is shifted to the right.

The curve for cadiac tamponade is a bit different, as here the fluid is exerting pressure on the heart itself! Which would slow down the filling of the heart, in order to fill the heart with a certain amount of blood, we need to overcome the new pressure exerted. (by the fluid in the pericardial space), so can we obtain the maximum CO? yes but at a much higher RAP!

Doctor read this slide

The Cardiac Output Curve

- Plateau of CO curve determined by heart strength (contractility + ↑HR)
- ↑ Sympathetics ⇒ ↑ plateau
- ↓ Parasympathetics (HR↑) ⇒ (? plateau)
- ↑ Plateau
- Heart hypertrophy ⇒↑'s plateau
- Myocardial infarction ⇒ (? plateau)
- ↓ Plateau

During sympathetic stimulation, plateau rises, however during parasympathetic stimulation, it is supposed to fall but we won't necessarily see that as it might not affect the plateau.

MI lowers the plateau as there is a decrease in the muscle mass >> less force exerted >> lower maximum cardiac output (plateau)

Then continued reading off this slide

The Cardiac Output Curve (cont'd)

- Valvular disease ⇒ ↓ plateau (stenosis or regurgitation)
- Myocarditis ⇒ ↓ plateau
- Cardiac tamponade ⇒ (? plateau)
- ↓ Plateau
- Metabolic damage ⇒ ↓ plateau

Valvular disease, lowers plateau as it is harder to push blood through stenosed valve, and during regurgitation blood goes back to the ventricles decreasing the output.

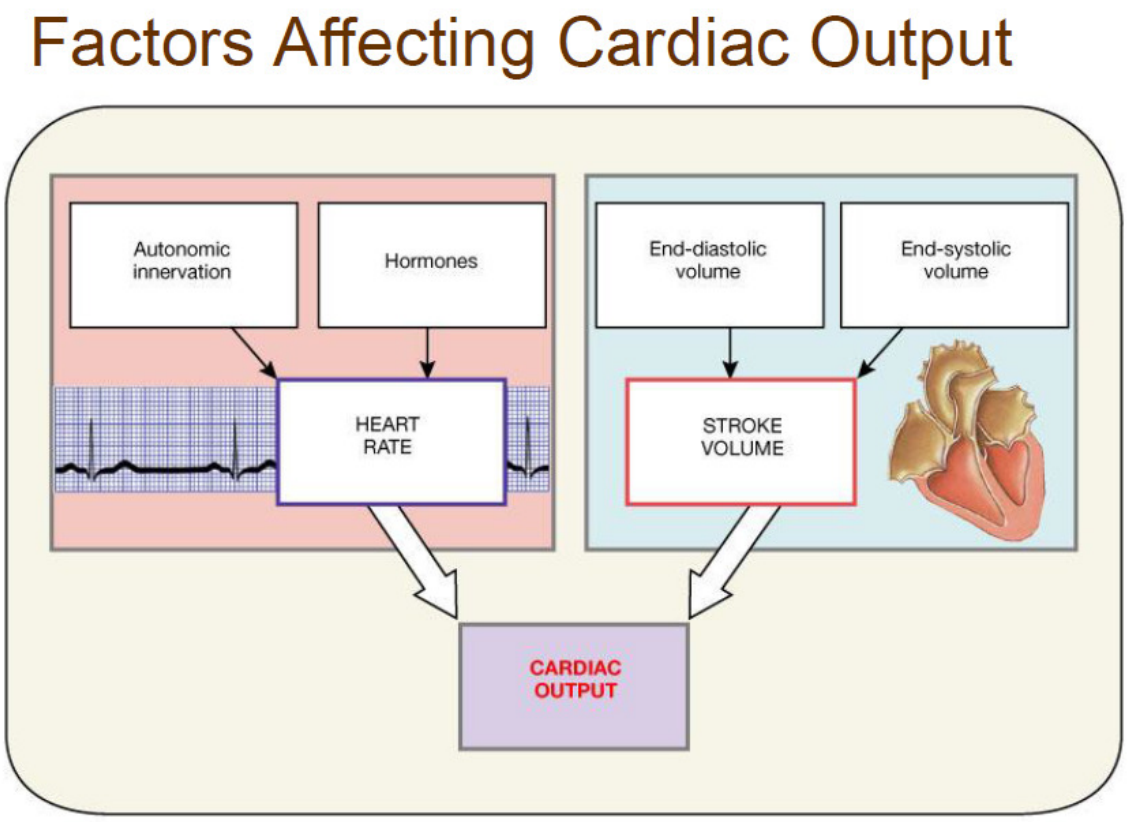
Cardiac tamponade may not change the plateau, but at very high pressure of pericardial space the maximum CO would decrease.

Factors affecting cardiac output

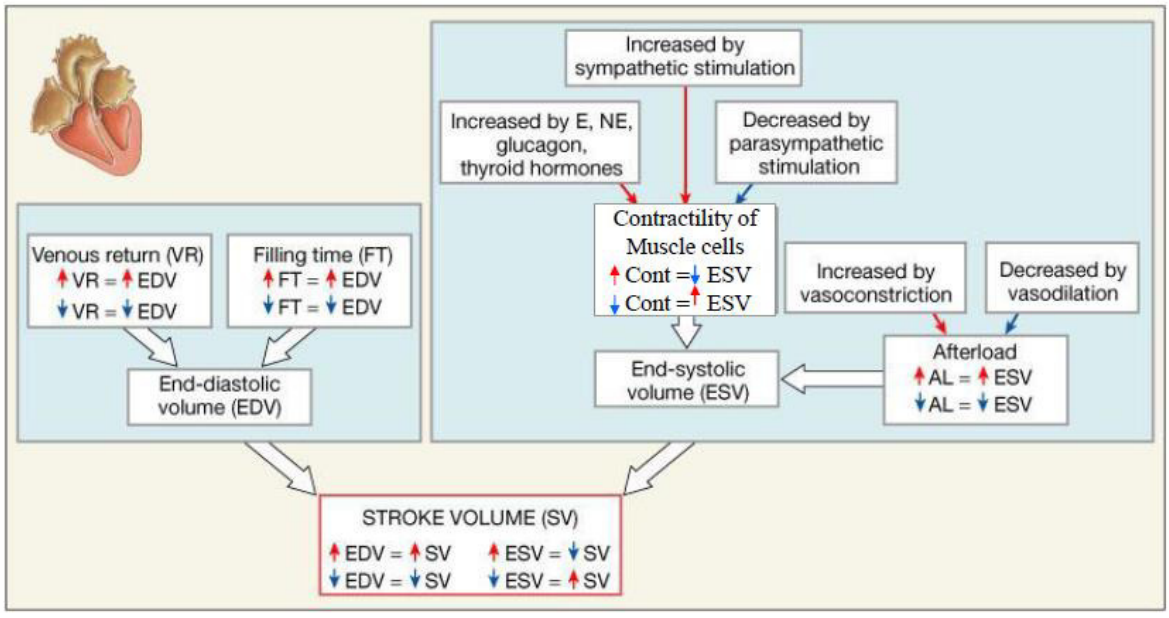
CO= heart rate x stroke volume

what affects each?

Doctor read this slide



What affects stroke volume?



When venous return increases, EDV increases, and vice versa, when EDV increases the stroke volume increases, as EDV decreases the SV would decreases.

When ESV increases SV decreases and vice-versa

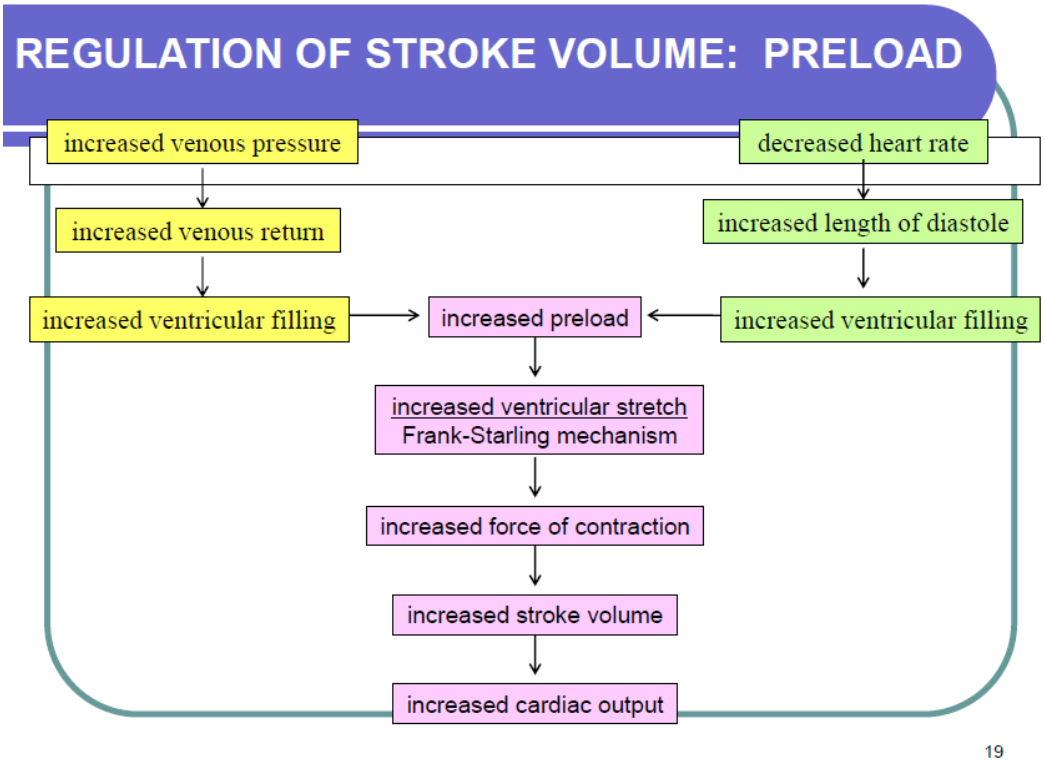
Increase in sympathetic stimulation > increased heart rate & contractility >> less ESV > higher SV

Vasoconstriction increases afterload > higher ESV > lower SV

Vasodilation decreases afterload > lower ESV > higher SV

Heart rate increases by

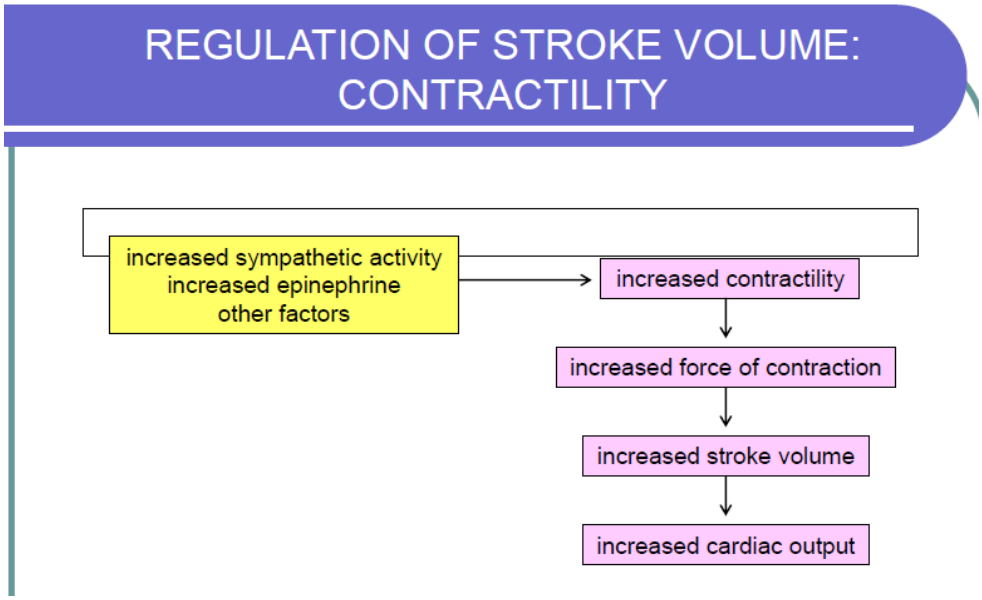
1. Increasing the venous return (Bainbridge reflux), whenever there's an increase in atrial pressure because of an increase in its volume, the heart rate increases.
2. Hormones (Epinephrine, norepinephrine, thyroxine)
3. Sympathetic stimulation.



Venous return effect on stroke volume

Increased venous return> increases EDV > increased preload > increased S.V

Increase in afterload, increases ESV which in turn decreases SV (opposite to preload)



Cardiac contractility:

Affected by autonomic nervous system and horomes, how do we measure it?

Very hard, it is usually computerized, we integrate dP/dt. Change in pressure over change in time, but not just ANY dP/dt we take the MAXIMUM pressure, do you remember the ventricular pressure-volume curve of the cardiac cycle from lecture 10?

يا زم خليني أتذكر شو أكلت مبارح أول

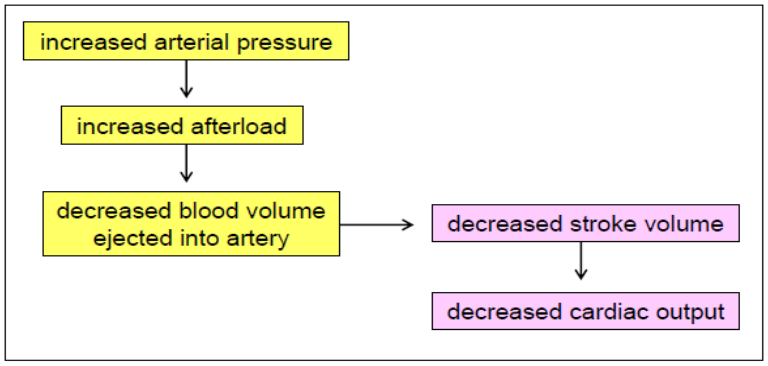
We use that curve and take the highest slope of dp/dt to calculate cardiac contractility.

Cardiac Contractility

- Best is to measure the C.O. curve, but this is nearly impossible in humans.
- dP/dt is not an accurate measure because this increases with increasing preload and afterload.
- $(dP/dt)/P_{ventricle}$ is better. $P_{ventricle}$ is instantaneous ventricular pressure.
- Excess K^+ decreases contractility.
- Excess Ca^{++} causes spastic contraction, and low Ca^{++} causes cardiac dilation.

What is the effect of afterload?

REGULATION OF STROKE VOLUME: AFTERLOAD



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End of first half

Venous return: (skip to slide 31)

What is venous return? Amount of blood returning back to heart per minute and it has to equal cardiac output according to frank starling, what goes in goes out (in normal conditions, or else heart failure will take place)

So venous return = CO which equals $\Delta P/R$ "the Resistance here is for the venous return, don't confuse it with TPR for CO!

If you look at the veins you will see that their valves direct the blood unidirectionally (when they close > blood tries to go back but it can't) which maintains the venous blood pressure low.

But if the valves were not present, blood would accumulate down "according to gravity" and the pressure would be very high, right?

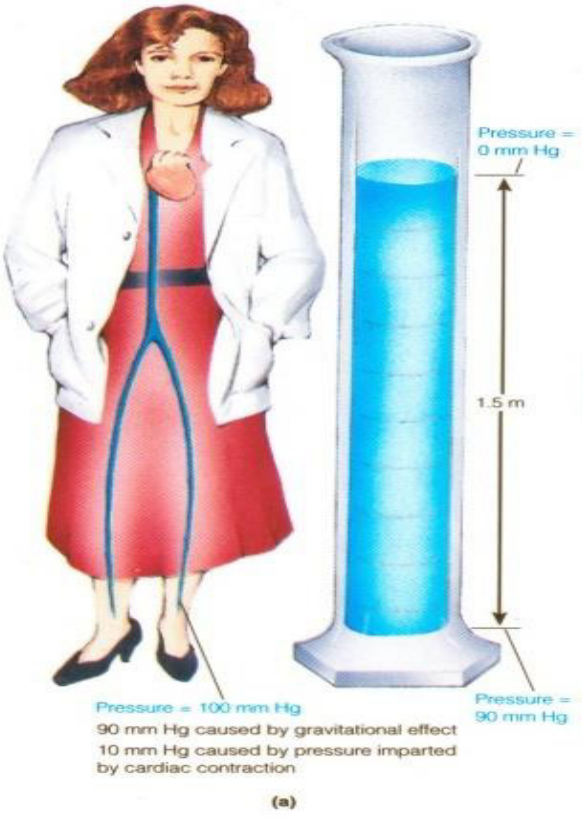
Remember !

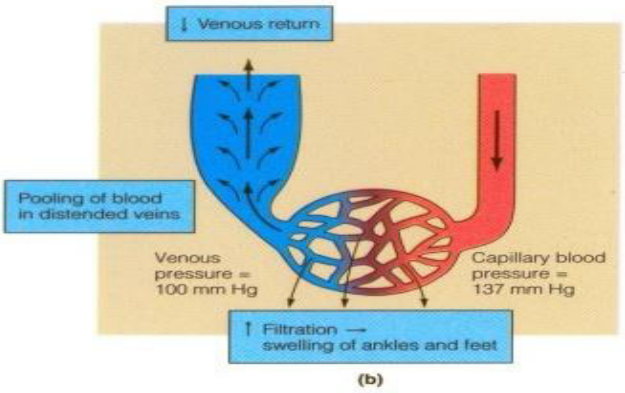
Pressure = water column (if you remember in physics, pressure = density * **g** * height of water column)

The contraction of the muscles open the valves, increase the venous pressure and directs the blood towards the heart.

So, one of the very important factors that increases venous return is " skeletal muscle contraction"

In the picture





A woman is standing for a long time, so the valves may open "valves incompetence" and blood will be collected<<<< now, what's the pressure down there at her leg, if the length between her heart and her leg = 136 cm?!

you know that " density of mercury = 13.6 " :p

so pressure down in her leg = column of water= 136 cm

in Hg = 10 cm " 136/ 13.6 = 10 cm of Hg" = 100 mm Hg *pretend you understood this part like we pretend we understand embryology*

so when does the pressure reach 136 mmHg? in case of incompetent valves" ex:

1- Varicose veins .... The veins here will be dilated , filled with blood , so will appear tortuous ; and because its deoxygenated blood , their color will be blue !

2- Pregnancy = increasing pressure on the veins

3- Tumors

4- Abdominal pressure increases venous pressure

So with increased venous pressure the blood is pushed back to the heart.
When RAP "right atrial pressure " increases > blood backs up into venous system, so here venous pressure should increase>> to increase venous return

RAP= central venous pressure "CVP" = Zero,may abnormally reach +20.

In "heart failure" almost = 20 mm Hg ,,, here "we have to control their CVP " what happens if we give them fluids ? ↑ CVP > ↓ venous return >causing death !

So **How to control CVP not exceed 20 ?**

Using CVP-Line "catheter" . through chest to> SVC> Rt atrium , and connect it with a Manometer (used to measure the pressure)

↑contractility"↓ ESV ">> >↓ RAP and vice versa !
Right atrial pressure is determined by the balance of the heart pumping blood out of the right atrium and flow of blood from the large veins to it.

Sometimes we can watch it by "Jugular pulsation" while the patient is sitting down at 45 degree position, here we can know CVP by watching C,A and V waves

**Remember :

A = atrial systole
C= ventricular contraction
V= ventricular relaxation

**in case of Tricuspid stenosis there'll be some abnormal waves
So if you see the wave راجع هيك >> tricuspid stenosis "regurgitation"
C-wave raised more !!

Factors that increase RAP

1- ↑blood volume (hypervolemia)
2- ↑ venous tone>>↑venous return
3- Dilatation of the arterioles >>↓ resistance "Co= ΔP/R >↓R=↑Co"
4- ↓ cardiac function "contractility">>↑ESV>>↑RAP

- ## Factors that increase venous return

**↑venous return >>↑ EDV , the amount of blood that's going to be collected in the ventricles is affected by the venous return which increases by:

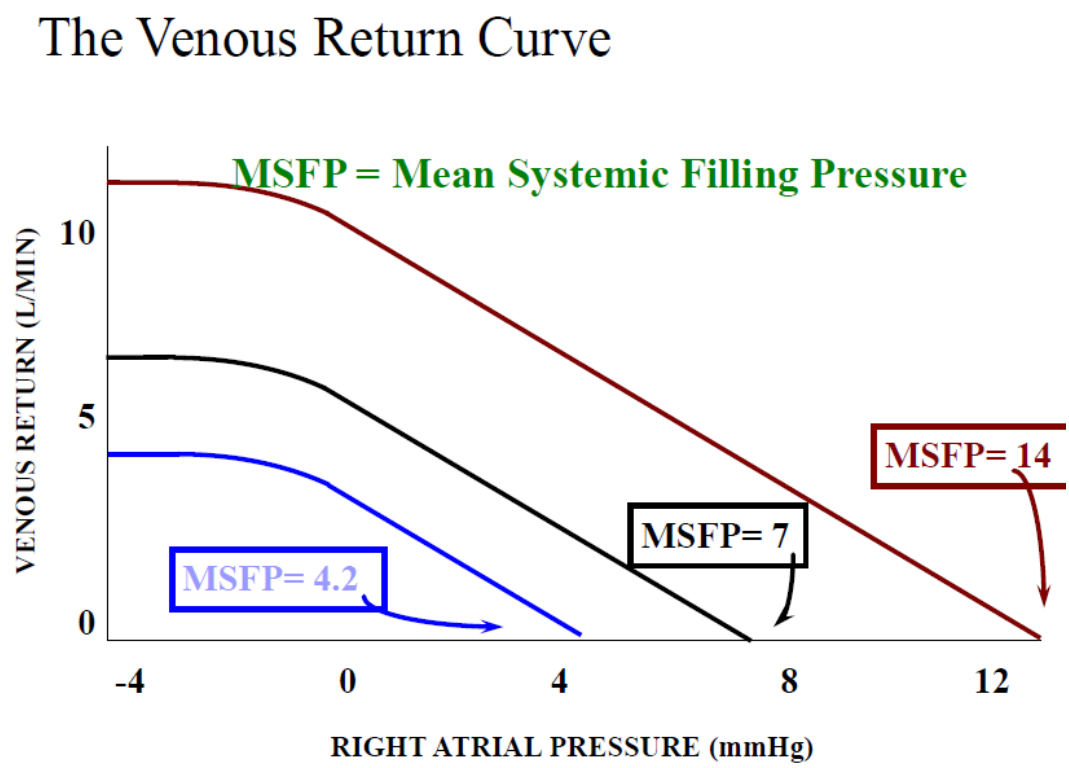
1- Skeletal muscles contraction or pump >>open the valves>>pushing blood towards the heart
2- Intact valves ,, so in varicose ↓venous return>↓EDV >↓Stroke volume >↓Co
3- Respiratory pump "↑inspiration" ↑venous return> ↑ EDV> ↑ SV > ↑ CO
4- Sympathetic stimulation >> venous constriction >↑resistance because of increased pressure >↑gradient >↑venous return
5- ↑contractility (cardiac suction) >↓ESV>↓RAP "↓ESV>↓RAP>↑gradient between the veins and atrium >↑venous return
6- ↑blood volume "by infusion in case of hypotension"> this will ↑ venous pressure >↑EDV>↑Co so what?! "remember ohm's law : ↑CO=↑mean arterial pressure"

- ## Venous return curve

Remember: first we discussed "CO curve" alone… now we'll discuss "venous return curve" finally we're going to combine them ☺

First: assume that we stopped the pumping action of the heart
Here : 1- there's blood in circulation . 2_-we equalized the pressure in the circulation "cause the column is equal so the pressure will be same "
Now ! how much is the pressure?!
Guyton " he's the first one who measures this pressure"
It was first measured in animals= 7 mm Hg , = 8 mm Hg in humans all over the body
So , RAP will be also 8
Now , if we ↓RAP "let's say to 7 " what'll happen ? >>> there will be venous return due to pressure gradient

But when it was 8, venous return was zero !! why? Since in all parts of the body it was also 8 so no pressure gradient to move blood!



X-axis >> RAP
Y-axis>>venous return
**note
*VR "venous return"↑↑ when we ↓RAP ,, until we reach zero blood= 5L
What do we call this pressure =8 ? "mean atrial pressure" it's the average pressure in the systemic circulation if we stopped the heart
What does it cause? >> filling of the heart
So we call it " mean systemic filling pressure (MSFP)"
Here we conclude that " when RAP = mean systemic filling pressure >>venous return= zero"
Whenever we ↓RAP>>↑VR "until we reach zero in pressure difference"

**Normally

The pressure around venous return especially big veins "ex. SVC" is Negative
What will happen to the veins??!
They'll collapse, and only open when the blood will accumulate there!
And here the venous return will stay almost the same "we call it plateau" plateau happened when veins collapsed inside chest by –ve pressure >>here veins will stay the same closed and then opened by accumulation of blood and so on!!
** by infusion > more blood volume >>↑pressure>↑mean systemic filling pressure
**how to decrease venous return??>>hemorrhage

Look at the curve on the previous curve:

*either all the curves are shifted to the left by bleeding (decreased blood volume) Or to the right by infusion (increased blood volume)

Effect of resistance:

**mainly due to arterioles

"neither venous constriction nor dilation will affect the resistance, mean systemic filling pressure is only affected by the arterioles."

And that was the last topic included in the exam

Second half done by Wafaa
