

Physiology - CVS

Done By

Heba Al Tahat

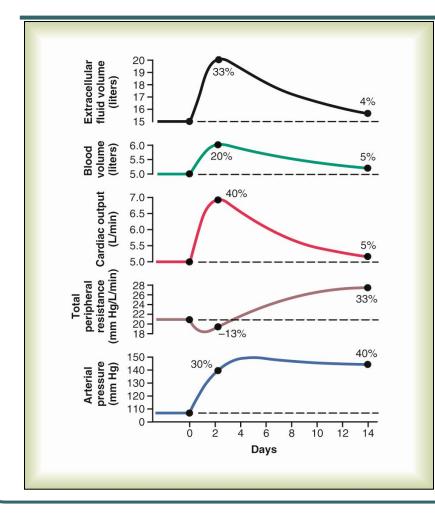
Corrected By

Dana Tarawneh

Blood Pressure Regulation 2

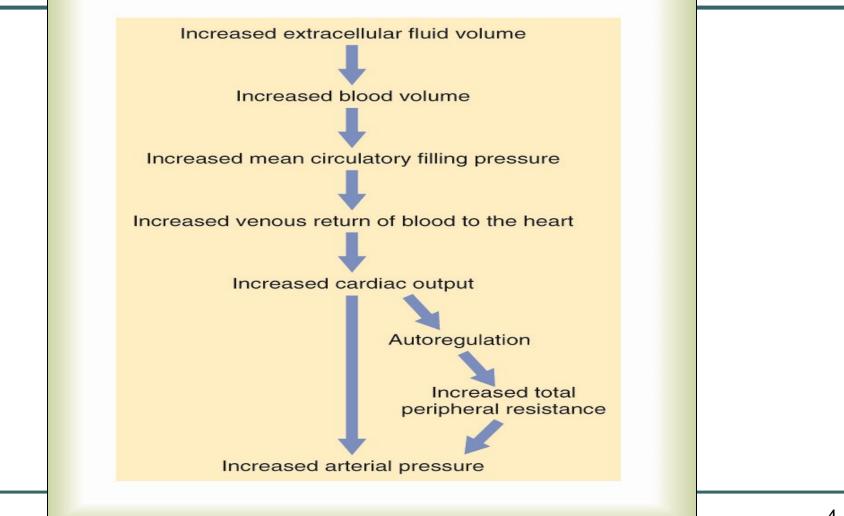
Faisal I. Mohammed, MD, PhD

Volume Loading Hypertension



- If few had blood transfusion, the volume loading hypertension would increase, this increases the ECF volume which increases the blood volume, and this increase causes an increase in the CO, and so does the pressure.
- The resistance in the beginning might decrease (receptive relaxation), because of the initial vasodilation due to the increase in volume, and then it returns back to normal and the resistance begins to increase.

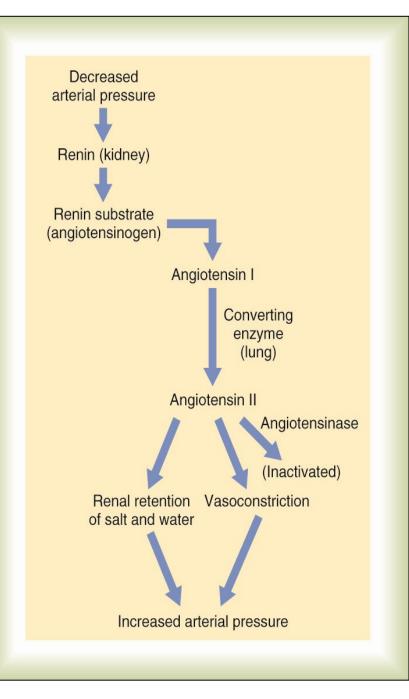
Effect of ECFV on Arterial Pressure



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Renin-Angiotensin System

- Renin is synthesized and stored in modified smooth muscle cells in afferent arterioles of the kidney.
- Renin is released in response to a fall in pressure.
- Renin acts on a substance called angiotensinogen to form a peptide called angiotensin I.
- AI is converted to AII by a converting enzyme located in the endothelial cells in the pulmonary circulation.

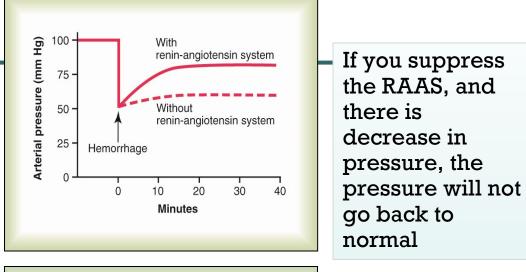


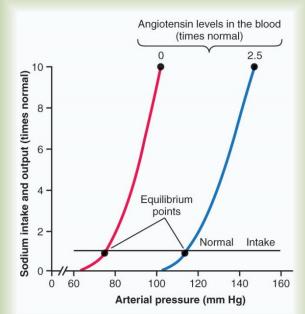
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- If the blood pressure decreased, then this will decrease the afferent arteriolar pressure, this will cause them to secrete renin. (look at the figure in the previous slide for the rest).
- Angiotensin I is converted mainly in the lungs due to the presence of ACE.
- Angiotensin II is a very potent vasoconstrictor and it has a positive inotropic effect on the heart, it increases the SV.
- It also goes to the adrenal cortex and stimulate the secretion of aldosterone which will increase sodium reabsorption, and this increases water reabsorption, increases the fluid, increases the pressure back to normal.
- If there is increase in blood pressure renin release is decreased.

Actions of the Renin Angiotensin System

- Causes vasoconstriction
- Causes Na⁺ retention by direct and indirect acts on the kidney
- Causes shift in renal function curve to right
- We can treat this shift by using angiotensin II inhibitors, because it is a potent vasoconstrictor and we must inhibit it. Their suffix is -pril
- While the suffix for ARBs is -sartan

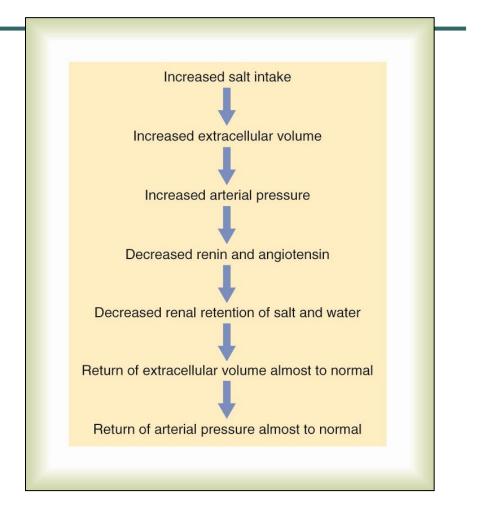




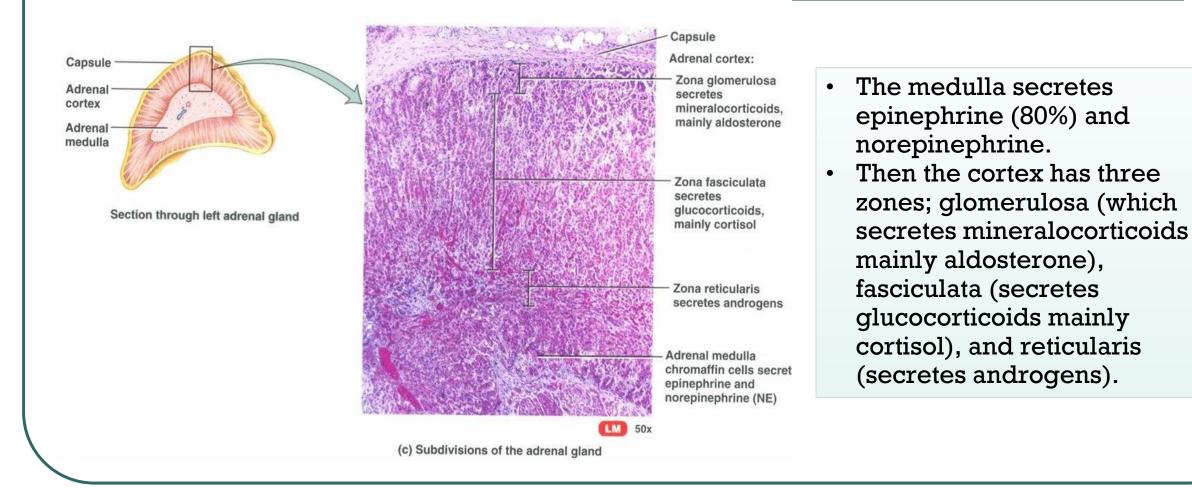
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Renin Angiotensin System: Effect of Na⁺ Intake

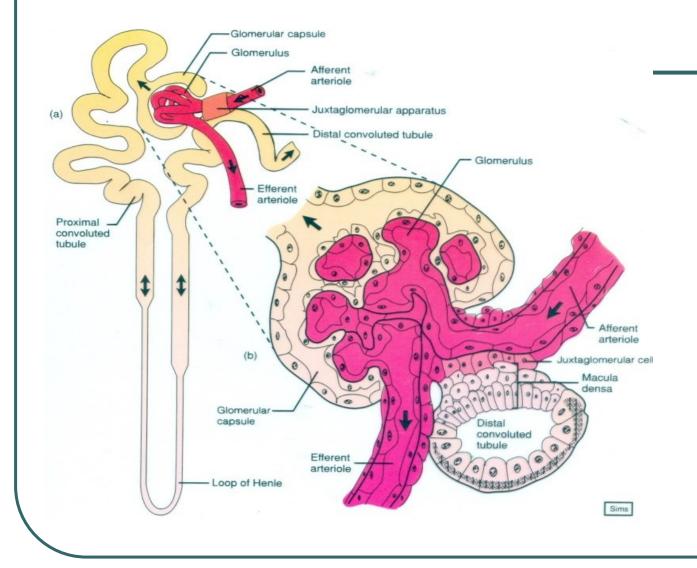
- RAS is important in maintaining a normal AP during changes in Na⁺ intake.
- As Na⁺ intake is increased renin levels fall to near 0.
- As Na⁺ intake is decreased renin levels increase significantly.
- RAS causes the Na⁺ loading renal function curve to be steep.



Adrenal Gland as the source of Aldosterone (cortex) and Epinephrine (medulla)



Juxtaglomerular Apparatus



- There are afferent and efferent arterioles, there are no veins in between , and the blood pressure in the arterioles is very high compared to the systemic circulation.
- There is no high difference between the afferent and efferent arterioles (from 60 to 40)

Factors Which Decrease Renal Excretory Function and Increase Blood Pressure

They cause vasoconstriction

•Angiotensin II

Aldosterone

Aldosterone does not cause vasoconstriction, it increases sodium reabsorption

•Sympathetic nervous activity

Endothelin

It is a local vasoconstrictor, not a systemic one. It is secreted from the endothelial cells.

Factors Which Increase Renal Excretory Function and Reduce Blood Pressure

•Atrial natriuretic peptide

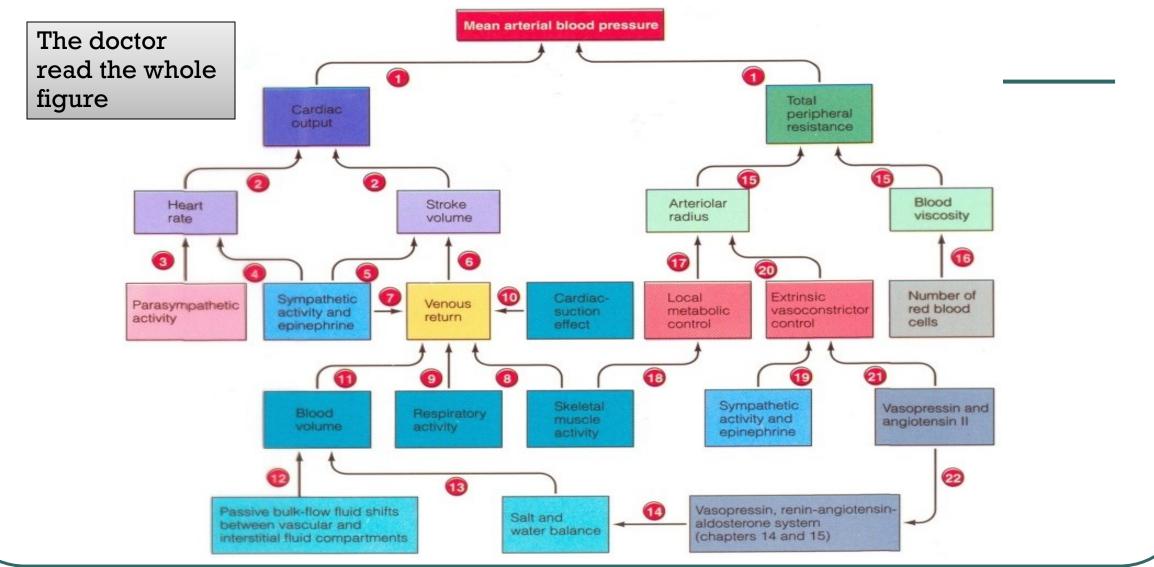
ANP increases GFR because it causes vasodilation of the afferent arterioles.

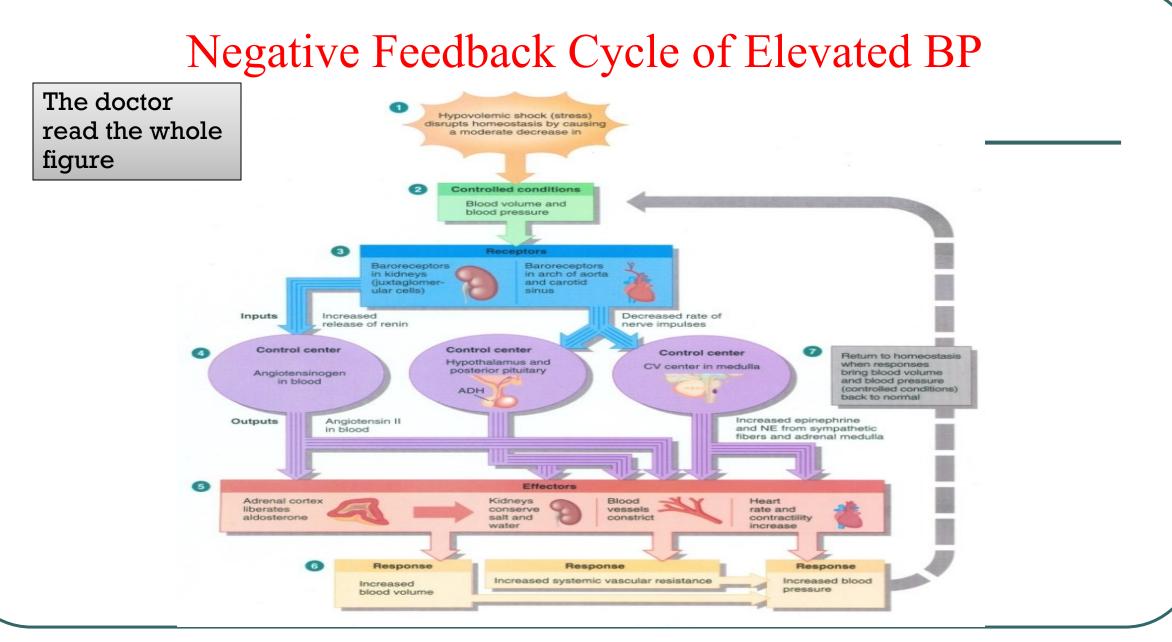
Nitric oxide

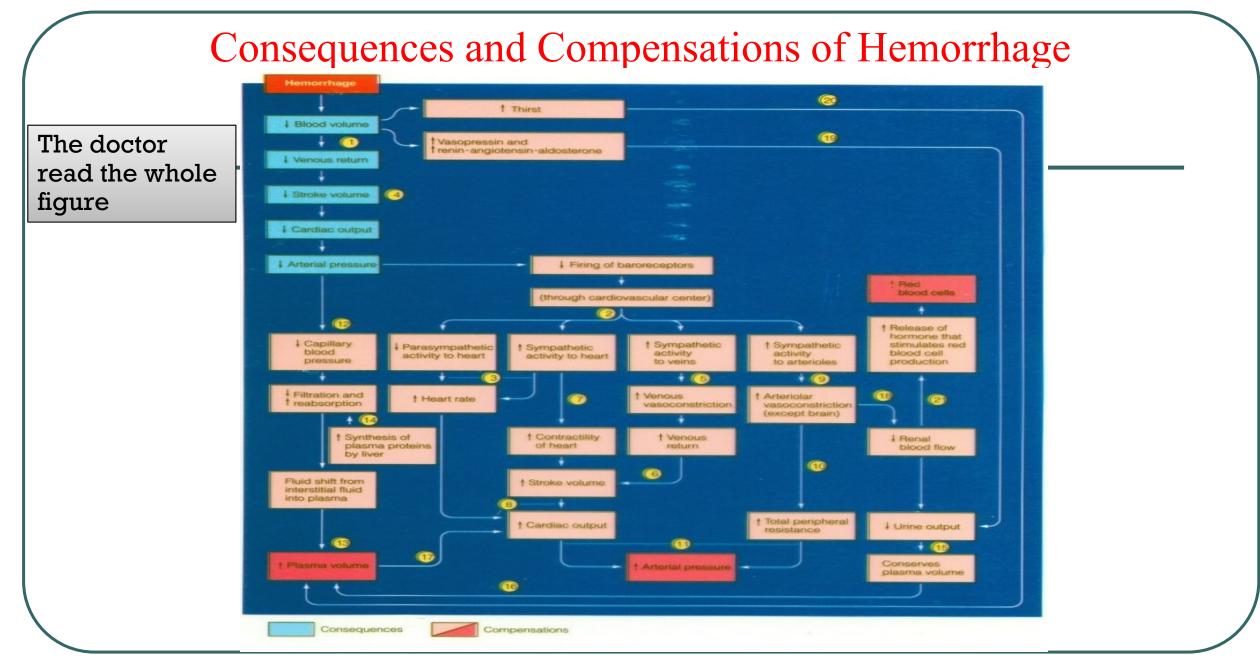
NO is a local vasodilator

• Dopamine Dopamine is a systemic vasodilator

Determinants of Mean Arterial BP







- There are different types of shock
- The type in the figure above is compensatory shock, hemorrhagic or circulatory shock. Dehydration, or hypovolemic shock.
- If someone has an MI, then they will undergo a cardiogenic shock. The heart stopped pumping and there is no pressure, this is cardiogenic shock.
- There is also neurogenic shock, due to excess feelings of sadness or happiness, there is extensive inhibition of the sympathetic system and vasodilation which decreases blood pressure greatly.
- There can also be allergic shock, due to sensitivity to a substance for example, this causes the increase of histamine. This increases the permeability of capillaries and then fluid oozes to the interstitial fluid and increases blood volume. This leads to redness and edema.
- Septic shock might also be caused from the release of endotoxin from invading bacteria. This endotoxin causes extensive vasodilation. This type of shock is a high CO shock, because the endotoxin will cause the uncoupling of oxygen and the tissue cannot use this oxygen and senses it is ischemic, so vasodilation occurs to "supply" this tissue, and CO increases.

Thank You

