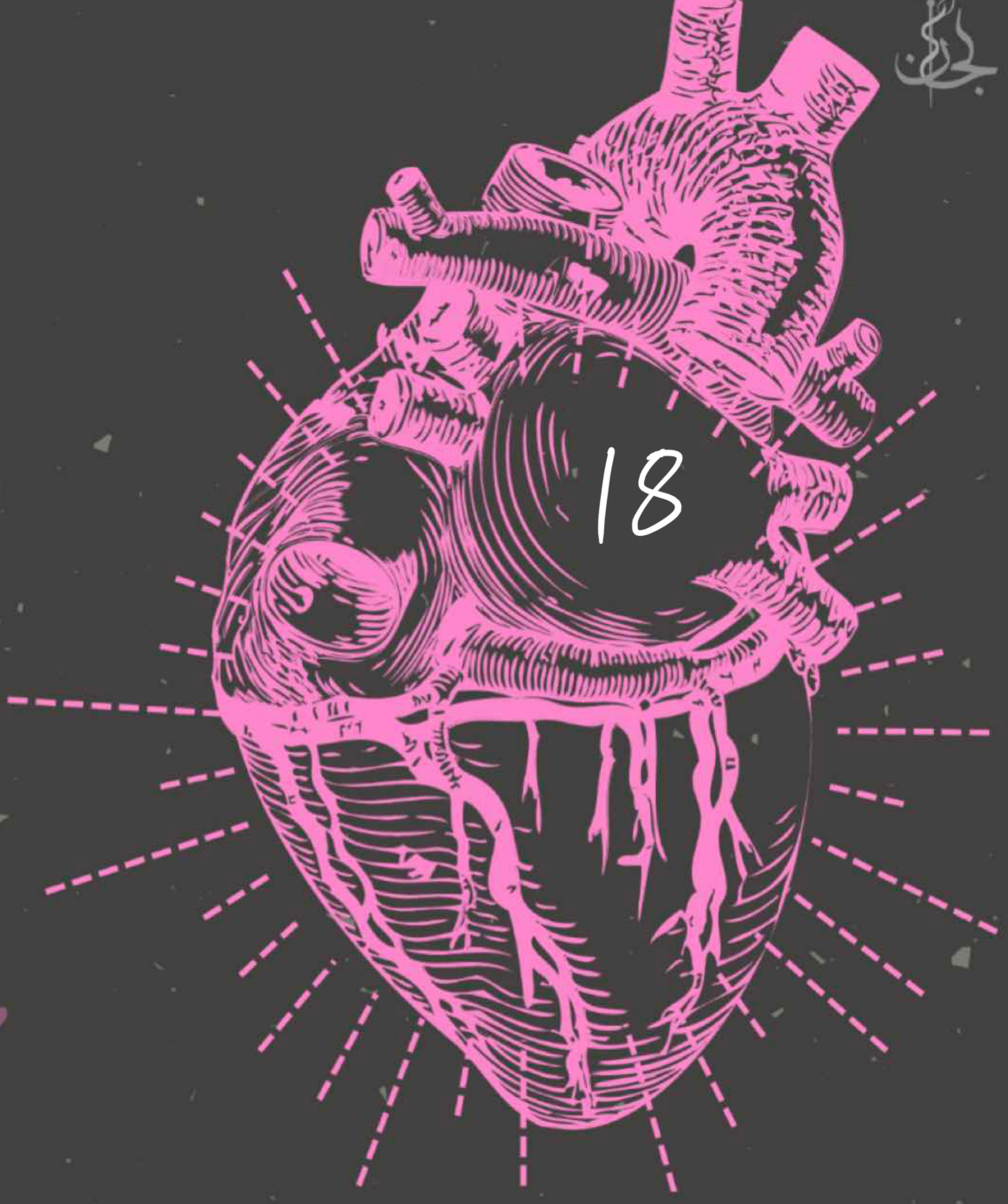
CVS



PHYSIOLOGY

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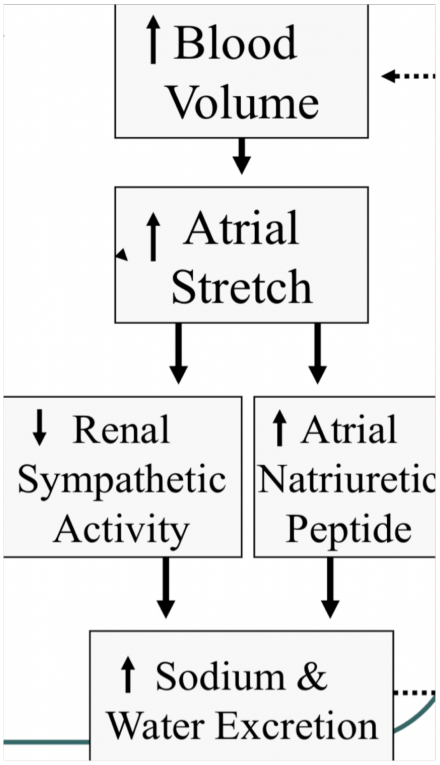
In this sheet we are going to continue our talk about the regulation of blood pressure

CNS ischemic response:

- If the atrial pressure reaches low value or under 60 mmHg, CNS will suffer from ischemia.
- If the cerebral blood flow reduced it will activate the vasomotor center, which will extensively increase the activity of the sympathetic nervous system which will cause vasoconstriction in order to increase atrial pressure.
- Prolonged CNS ischemia has a depressant effect on vasomotor center (auto-shutdown)

Atrial and pulmonary artery reflexes



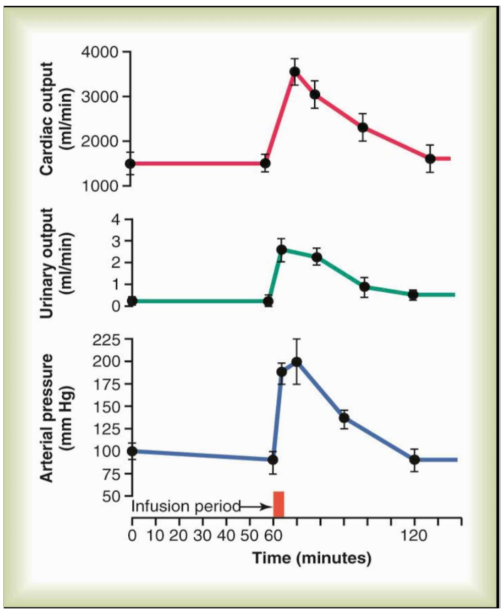
- If the baroreceptors, chemoreceptors, epinephrine, and norepinephrine try to maintain the blood pressure and we call it compensatory shock, but if it is not maintained or treated the patient will go under in-compensatory shock leading to organ failure and then death.
- Atrial-hypothalamic reflex: if there is increase in the volume of the right atrium it will inhibit ADH secretion, and if there is decrease in right atrium pressure or volume it will stimulate ADH secretion to rise the pressure.
- Atrio-renal reflex: if there is increase in pressure it will stimulate vasodilation of afferent arteriole.
- A blood pressure-lowering system that involves the hormone ANP, this hormone is produced mainly by the right atrium, although both atria can secrete it. The main action of ANP is to inhibit Na+ reabsorption and consequently promotes its excretion, water will osmotically follow Na+.
- **↑ECFV and ↑ MAP → the additional volume stretches the heart muscles → release of ANP → ↑ Na+ and water excretion → more urine is formed → ↓ ECFV and MAP ▪ ANP causes vasodilation in the afferent arterioles → ↑GFR → more urine is formed → Na+ and water are lost → ↓ECFV and MAP**
- Activation of low-pressure receptors enhances Na+ and water excretion by:
  a) Decreasing rate of antidiuretic hormone (atrial-hypothalamic reflex).
  b) Increasing glomerular filtration rate (atrio-renal reflex).
  c) Decreasing Na+ reabsorption.

Bainbridge reflex:

- When right atrial pressure increases, the resultant volume stretches the SA node and thus increases its discharge → HR↑
- Also, the stretch of atria sends afferent signals (through the vagus) to the brain to activate the vasomotor (vasoconstrictor + cardio-acceleratory) to further increase the HR and contractility (to a lesser degree) of the heart.
- This reflex Prevents damming of blood in veins atria and pulmonary circulation.

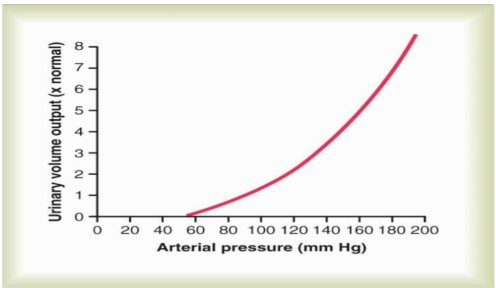
Renal body fluid system for long term arterial pressure control:

- A volume of blood is infused intravenously and thus the ECFV is increased.
- The extra volume will raise the MSFP. Consequently, the venous return and CO are increased.
- The resultant increase in CO will raise the MAP.
- The middle curve is the effect of this increased arterial pressure on urine output, which increases to multiple folds. Along with this tremendous loss of fluid in the urine, both the cardiac output and the arterial pressure returned to normal.
- Kidneys have extreme capability to eliminate excess fluid volume from the body in response to high arterial pressure, to return the arterial pressure back to normal.



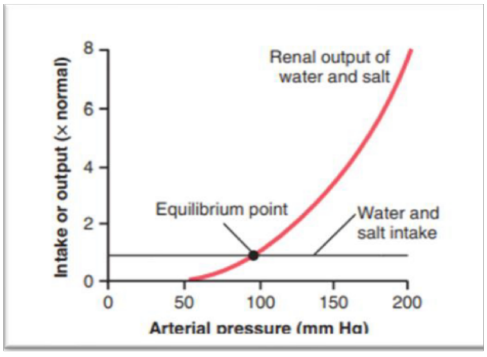
The adjacent figure represents the renal output curve for water and salt in response to rising arterial pressure.

- The effect of pressure to increase water excretion → diuresis.
- The effect of pressure to increase Na excretion → natriuresis.



To maintain a constant BP, sodium and water intake must equal their output (urinary volume).

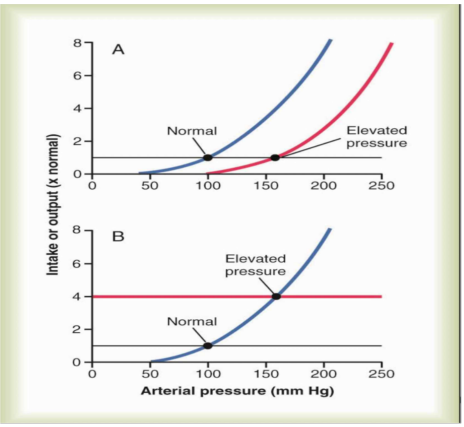
- The horizontal line represents the net water and salt intake, and the red curve is the same renal output curve.
- The only place on the graph at which output equals intake is where the two curves intersect, called the **equilibrium point.**



The two primary determinants of the long-term arterial pressure level are:

1.The degree of pressure shift of the renal output curve for the same water and salt intake.

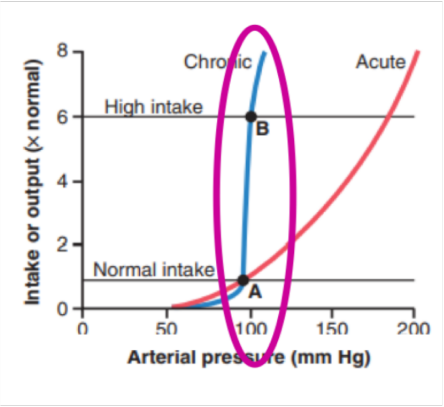
2.The level of the water and salt intake.



a) In this case, the normal curve is shifted to the right. The intake is not altered. But the kidneys are functioning abnormally (for example angiotensin II levels are elevated). Shifting the renal curve to a higher value will increase the arterial pressure (the equilibrium point is also shifted to the right).

b) Changing the level of salt and water intake also can change the arterial pressure (equilibrium point is also shifted to a higher value). Conversely, a decrease in the intake level would reduce the arterial pressure.

c) Luckily, with normal functioning kidneys, increased salt intake causes only small changes in arterial pressure. Over a long period of chronic intake of salt, the renal curve becomes much steeper to maintain normal BP.

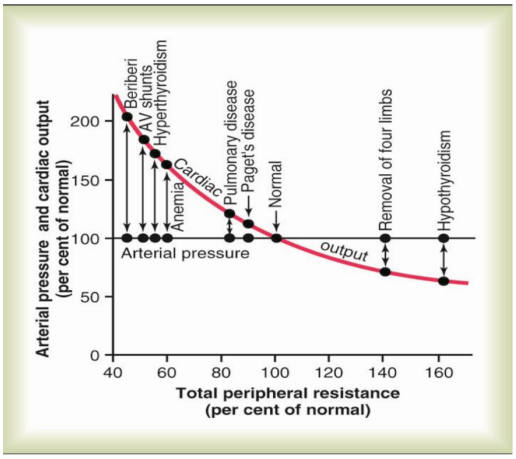


Renal body fluid feedback system has an infinite gain:

- One of the causes that baroreceptors do not play a role in long-term regulation of BP (other than being adaptable) is because their gain is small. In contrast, the renal system has almost infinite gain.
- Now, what does gain mean? It is a measure of effectiveness of a particular control system to resist changes in homeostasis. Gain = Correction / error
- The higher the gain the higher the effectiveness of a control system. For example, if blood pressure is elevated from 100 mmHg to 150 mmHg, the baroreceptor reflex can decrease the pressure to almost 120 mmHg. What is the gain in this situation? Gain = (120-150)/20 = - 1.5, the negative sign indicates a negative feedback mechanism. You can notice that the gain is so small because the short-term compensatory measures have limited ability to minimize a change in blood pressure.
- On the other hand, the renal system returns the blood pressure exactly to the original value (100 mmHg). Because the error is almost zero, the gain is infinite.

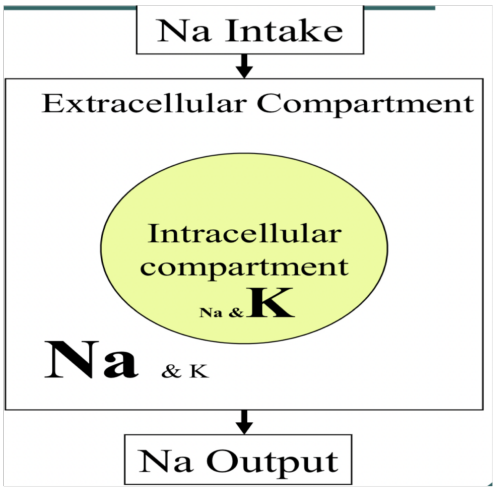
Failure of Total Peripheral Resistance to Elevate Long-term Arterial Pressure:

- Changing the TPR has no effect on arterial pressure on the long term, because when the kidneys are normally functioning, blood pressure will be adjusted by increasing or decreasing the cardiac output.
- If TPR is decreased (after the ingestion of vasodilators for example), the kidneys respond and retain more fluids to increase the CO and maintain a constant arterial pressure. That's why diuretics are prescribed with vasodilators (to counteract the compensatory measures →decrease CO and consequently decrease arterial pressure).



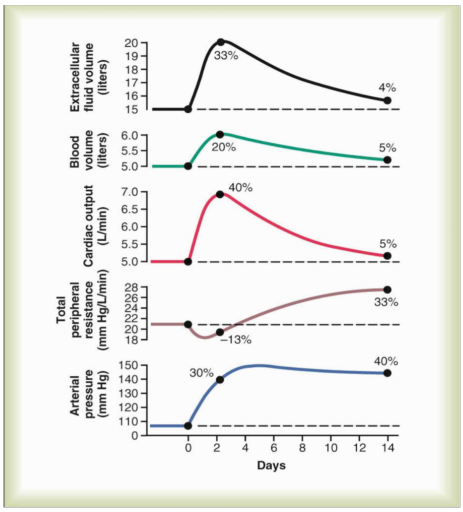
- Conversely, if TPR is increased, the kidneys respond and excrete more Na+ and water in the urine to decrease the ECFV and consequently the CO.
- One must alter the renal function curve in order to have long-term changes in arterial pressure. Changing renal vascular resistance does it lead to long-term changes in arterial pressure not the TPR.

Sodium is a Major Determinant of ECFV:



- Why sodium? Because it is the major cation in the ECF. Changing Na+ concentration will change the ECFV because water follows sodium osmotically.
- As Na+ intake is increased; Na+ stimulates drinking, increased Na+ concentration stimulates thirst and ADH secretion.
- Changes in Na+ intake leads to changes in extracellular fluid volume (ECFV).
- ECFV is determined by the balance of Na+ intake and output.

Volume Loading Hypertension:



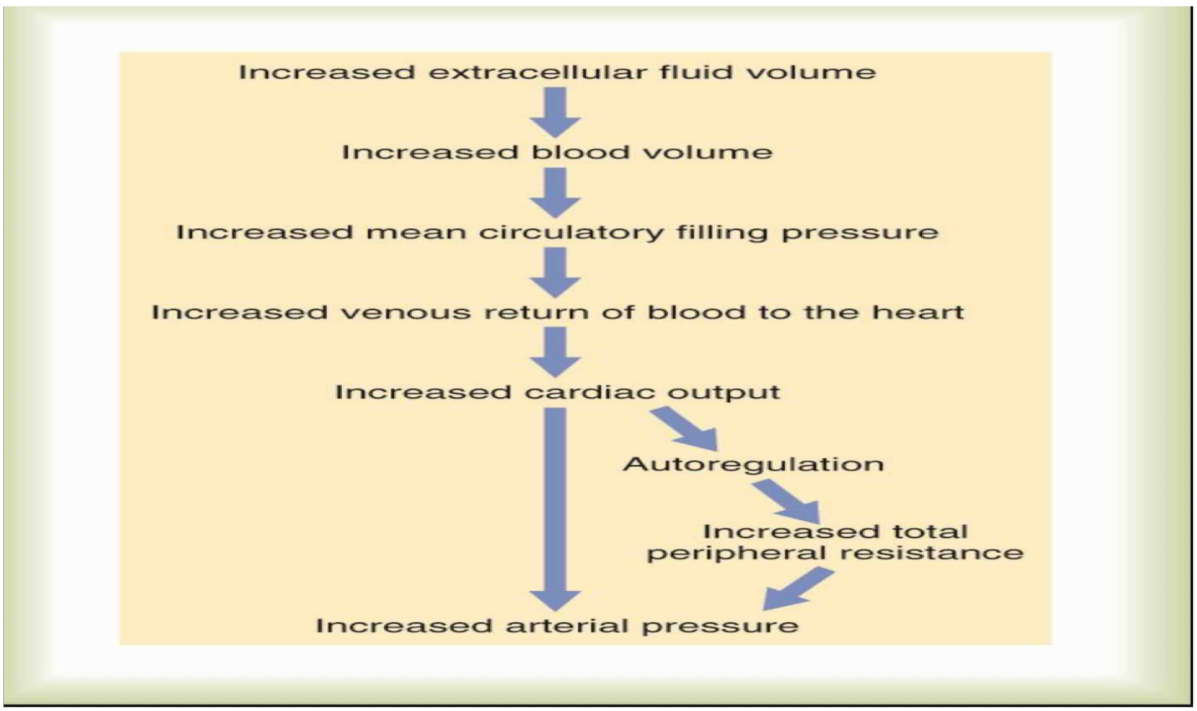
a) Increasing the ECFV.

b) Increased ECFV increases blood volume.

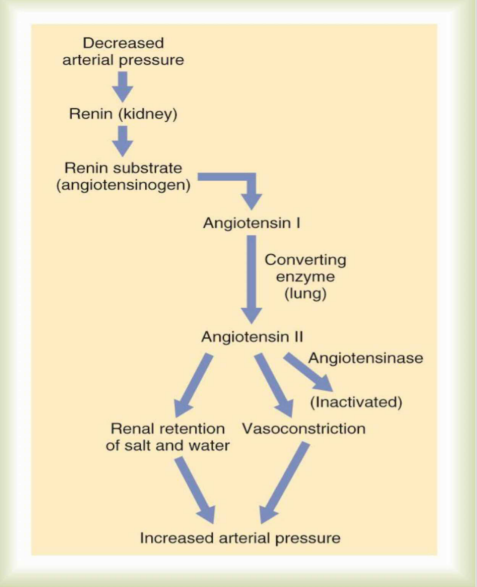
c) The increased volume increases venous return and thus CO.

d) Increasing the CO decreases the TPR a little bit through receptive relaxation of the vessels [this decrease was caused by the baroreceptor mechanism because the arterial pressure was suddenly increased, after a few days the receptors adapt and the TPR increases back to normal levels]

e) This will increase the arterial pressure, but if the kidneys are normal, the BP will be adjusted.

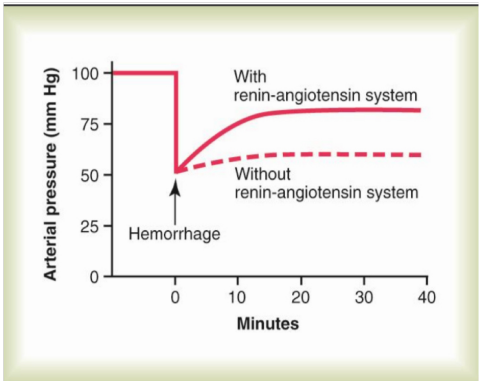


Renin-Angiotensin System

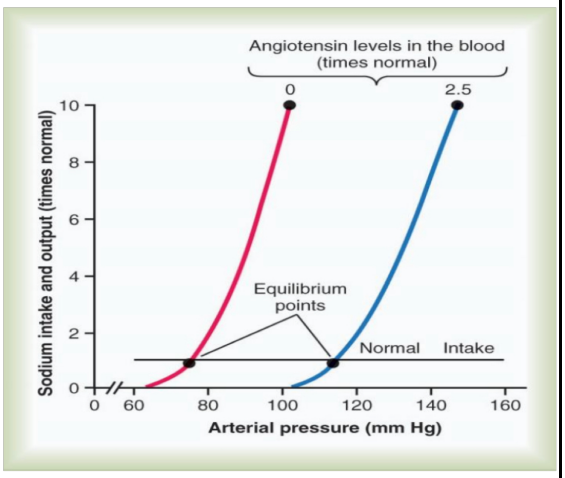


- Renin is synthesized and stored in modified smooth muscle cells in afferent arterioles of the kidney.
- Renin is released in response to a fall in pressure.
- Renin acts on a substance called angiotensinogen to form a peptide called angiotensin I.
- AI is converted to AII by a converting enzyme located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

Actions of the Renin Angiotensin System:



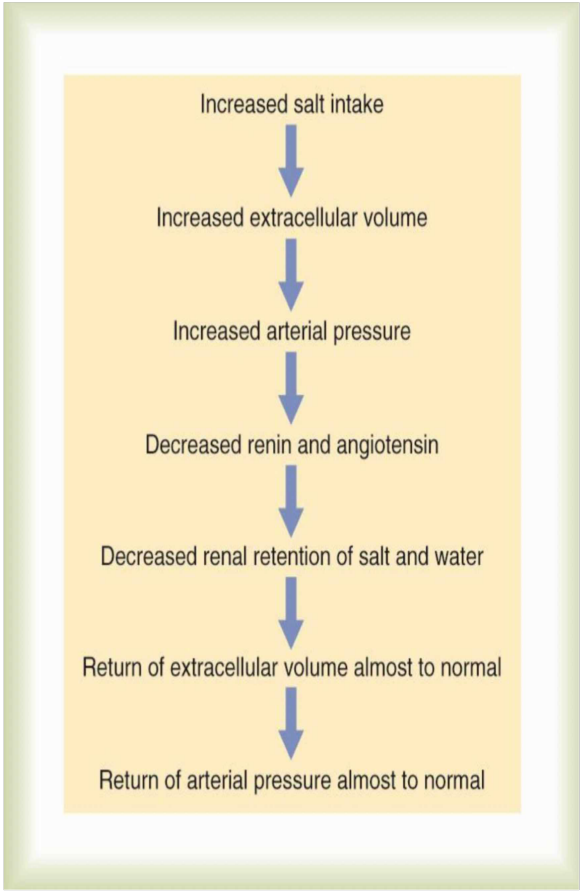
- the effect of hemorrhage on the arterial pressure under two separate conditions:
  1. with the renin-angiotensin system is functioning, the arterial pressure rose back to a higher value.
  2. without the system functioning, more time is spent to elevate BP, and the maximum value is far less than the normal value.
- The renin-angiotensin system is powerful in returning the arterial pressure back to almost normal values after severe hemorrhage. Therefore, sometimes it can be lifesaving, especially in circulatory shock.



- The figure shows different levels of Angiotensin II in the blood. The normal level is something in between the blue and red curves (the additional green curve).
- When the angiotensin system is blocked and no Angiotensin II is formed, the renal curve is shifted to the left (equilibrium point is shifted to the left → lower BP at the same salt intake level).
- When angiotensin II level is above the normal level (like in hypertension), the curve is shifted to the right (equilibrium point is also shifted to the right → higher BP at the same salt intake level).
- Angiotensin II Causes vasoconstriction and Na+ retention by direct and indirect acts (through aldosterone) on the kidney → Causes shift in renal function curve to right
- ACE inhibitors (captopril) and angiotensin receptor blockers are used to treat hypertension, because they shift the renal curve to the left (towards normal levels).

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.



Factors Which Decrease Renal Excretory Function and Increase Blood Pressure:

1. Angiotensin II

2. Aldosterone

3. Sympathetic nervous activity

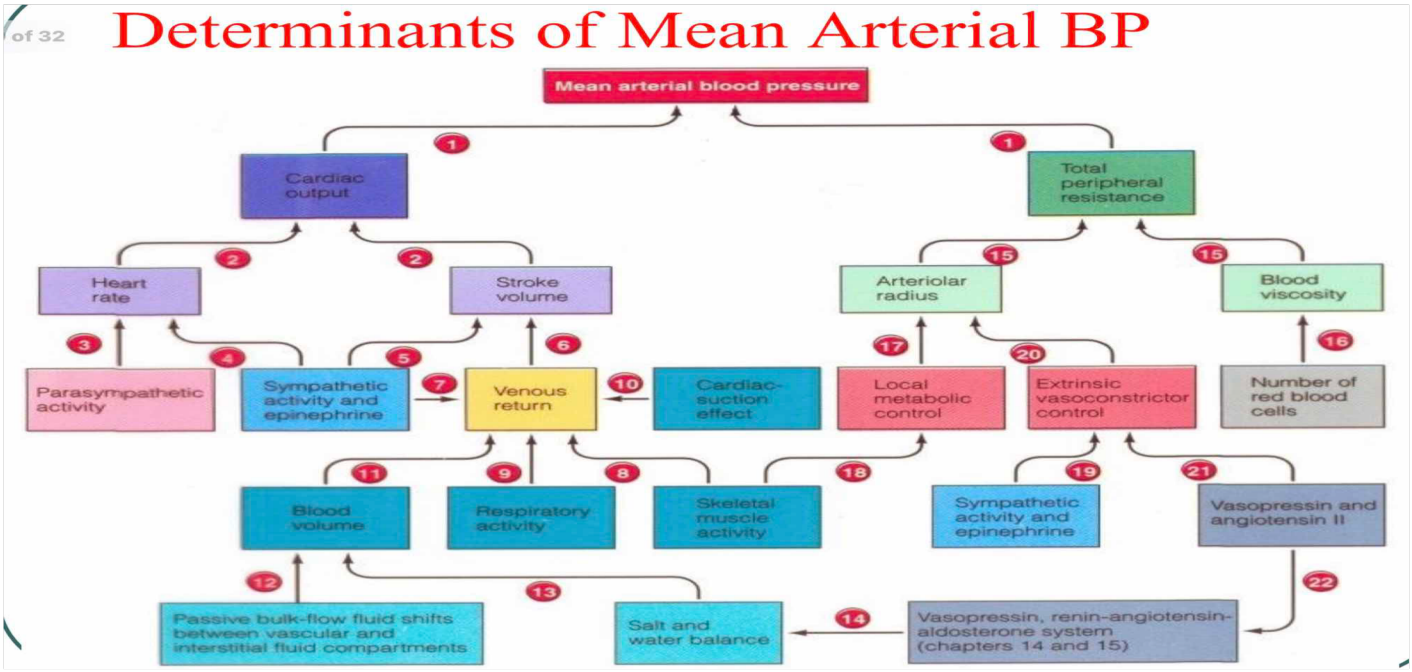
4. Endothelin (vasoconstrictor released by endothelial cells).

Factors Which Increase Renal Excretory Function and Reduce Blood Pressure:

1. Atrial natriuretic peptide

2. Nitric oxide (local vasodilator)

3. Dopamine (vasodilator)



Everything in the previous figure has been explained in detail.

Just go over it quickly to revise the main concepts of BP regulation.

Consequences and Compensations of Hemorrhage:

1. Following severe blood loss, the reduced volume leads to a decrease in venous return and a fall in CO and arterial blood pressure.

2. The baroreceptor reflex increases the sympathetic and decrease parasympathetic activity to the heart.

3. The heart rate is increased to compensate for the decreased SV.

4. Increased sympathetic activity to the veins produce generalized veno-constriction, increasing the venous return.

5. Sympathetic stimulation increases the contractility of the heart, to beat more forcefully and eject more blood → increased SV.

6. The increased HR and SV collectively increase CO.

7. Sympathetically induced generalized arteriolar vasoconstriction leads to an increase in TPR.

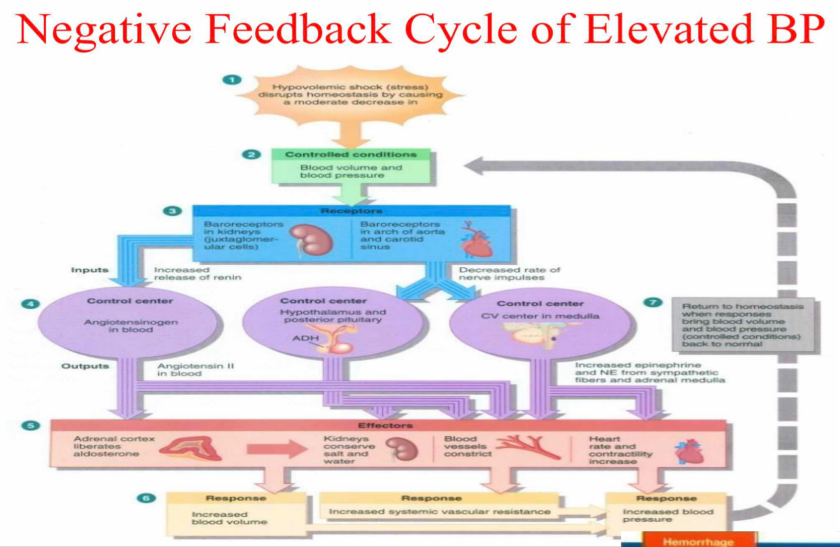
8. Together, the increase in CO and TPR increase the arterial pressure.

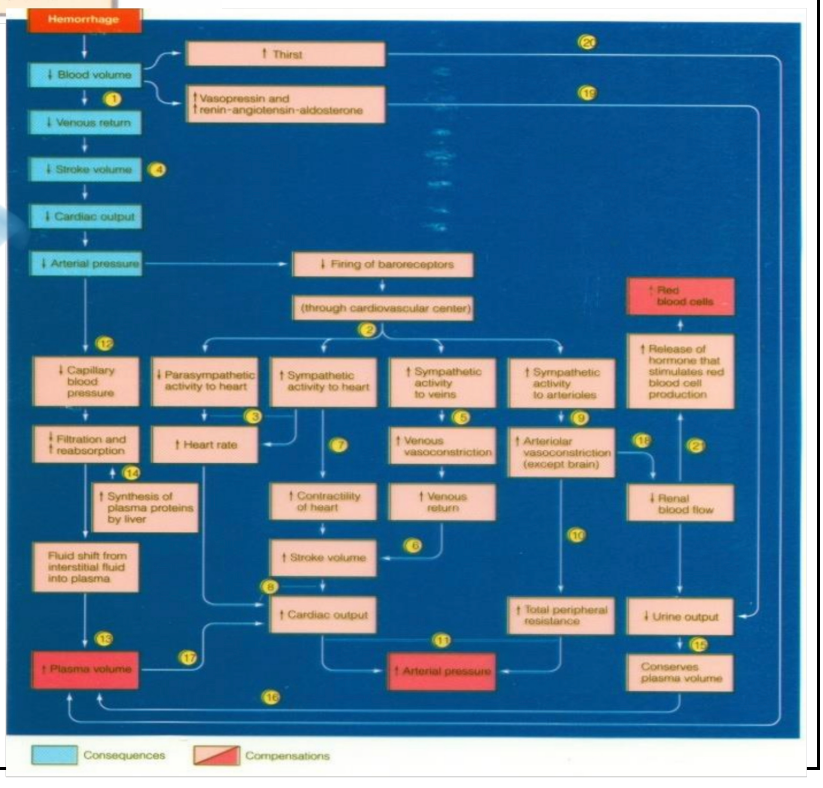
9. Urinary output is reduced, thereby conserving water that normally would have been lost from the body. This additional volume helps expand the reduced plasma volume.

▪ **Reduction in urinary output results from decreased renal blood flow (afferent arteriole vasoconstriction → ↓GFR).**

10.The reduced plasma volume triggers the secretion of ADH and activates the renin-angiotensin-aldosterone hormonal pathway, which further reduces urinary output and elevate BP.

11. Thirst is also stimulated; the resultant fluid intake helps restore plasma volume.





Shock types:

1. Neurogenic shock: it results in either passive vasoconstriction or vasodilation
2. Cardiogenic sock: due to previous heart disease e.g IHD
3. Allergic shock: release of histamine → increase permeability of the capillaries → excessive edema → shock
4. Septic shock: due to some released chemicals it will uncouple the oxygen with the blood causing hypoxia and then shock

Our journey with physiology is done

I hope you enjoyed studying the material

And I hope ya'll getting high scores

Good luck

لا تنسونا من دعواتكم