

# Blood Pressure Regulation 2

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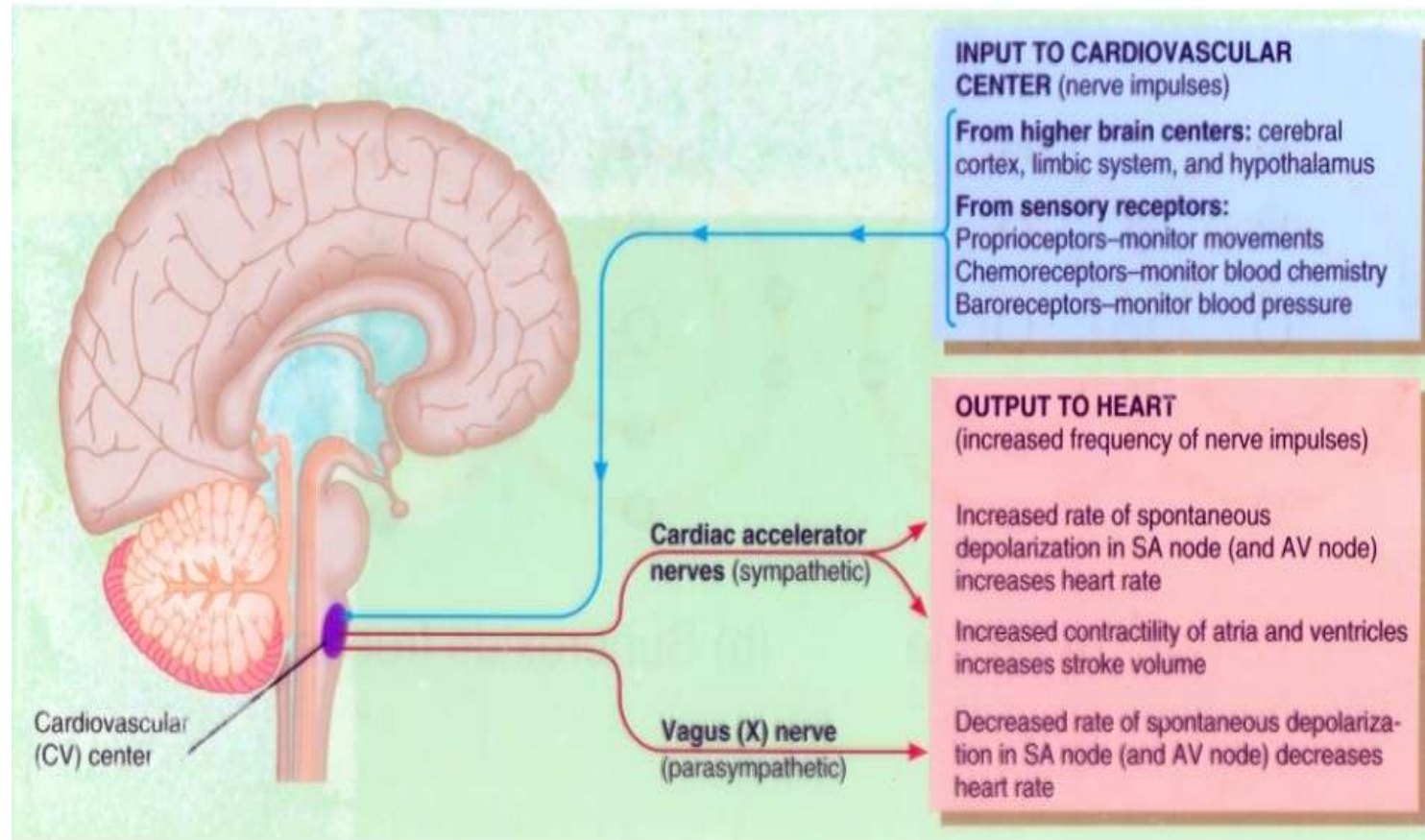
# Objectives

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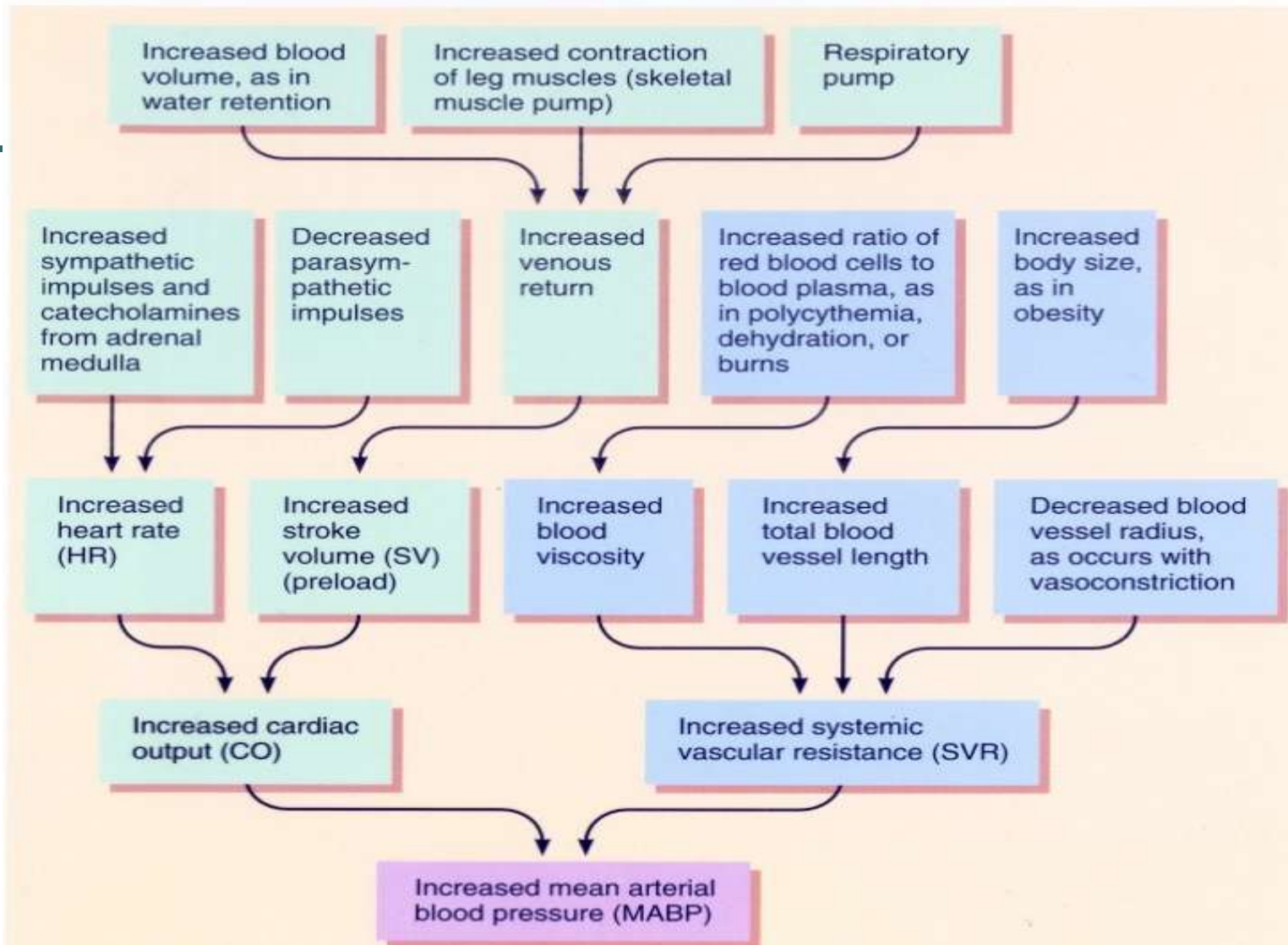
- Outline the intermediate term and long term regulators of ABP.
- Describe the role of Epinephrine, Antidiuretic hormone (ADH), Renin-Angiotensin-Aldosterone and Atrial Natriuretic Peptide (ANP) in BP regulation
- Point out the role of Kidney-body fluid system in long term regulation of BP
- Follow up the responses of the circulatory shock



# Nervous Control of the Heart

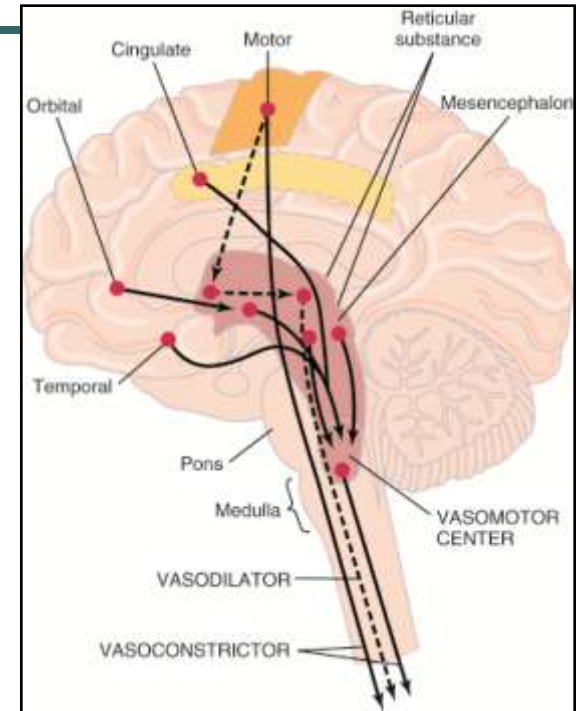


# Factors that affect the Mean Arterial Pressure



# CNS Ischemic Response

- CNS Ischemic response is activated in response to cerebral ischemia.
- Reduced cerebral blood flow causes CO<sub>2</sub> buildup which stimulates vasomotor center thereby increasing arterial pressure.
- CNS Ischemic response is one of the most powerful activators of the sympathetic vasoconstrictor system.



Cerebral Ischemia

↑CO<sub>2</sub>

Vasomotor Center

↑Sympathetic Activity

↑Arterial Pressure

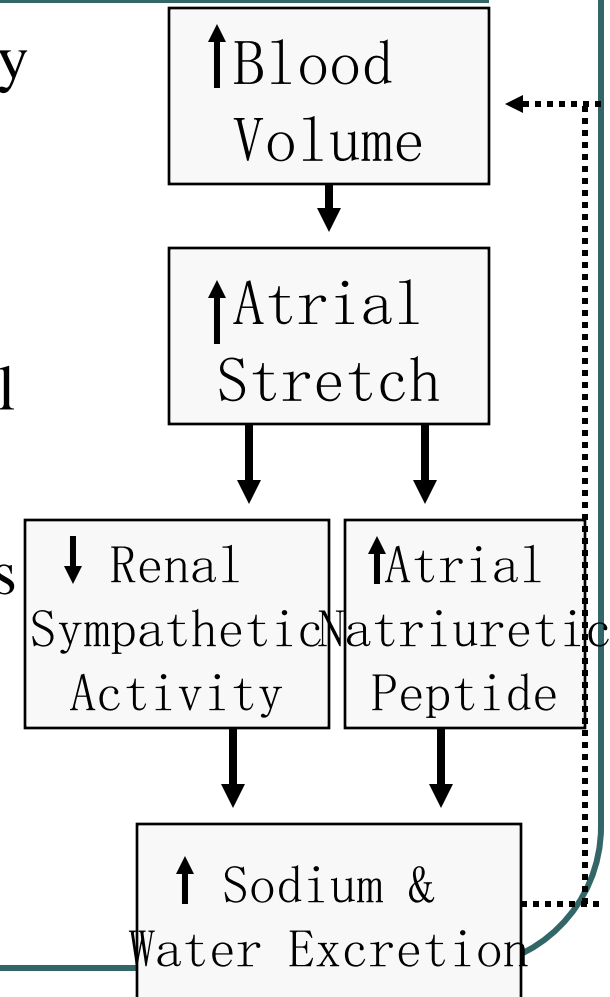
# CNS Ischemic Response

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- CNS Ischemic response is not activated until pressure falls below 60mmHg; greatest activation occurs at pressures of 15-20mmHg.
- Cushing reaction is a special type of CNS ischemic response.
- Prolonged CNS ischemia has a depressant effect on the vasomotor center.

# Atrial and Pulmonary Artery Reflexes

- Low pressure receptors in atria and pulmonary arteries minimize arterial pressure changes in response to changes in blood volume.
- Increases in blood volume activates low pressure receptors which in turn lower arterial pressure.
- Activation of low pressure receptors enhances  $\text{Na}^+$  and water by:
  - Decreasing rate of antidiuretic hormone
  - Increasing glomerular filtration rate
  - Decreasing  $\text{Na}^+$  reabsorption

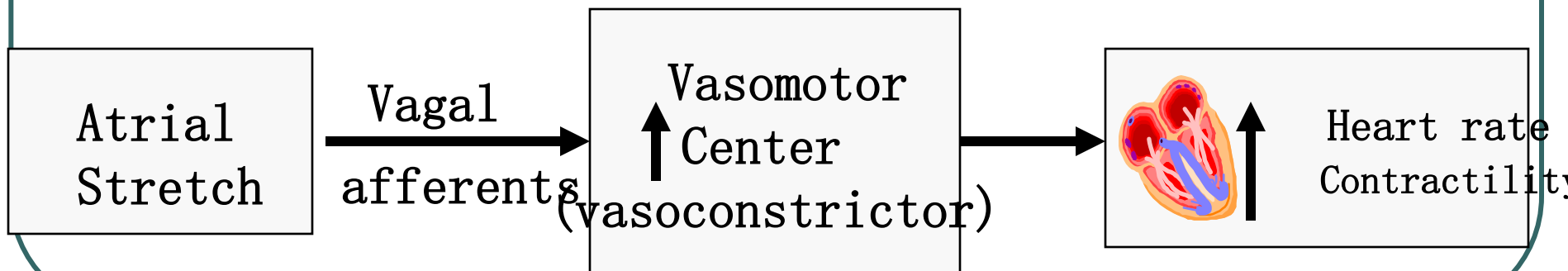




# Bainbridge Reflex

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- Prevents damming of blood in veins atria and pulmonary circulation.
- Increase in atrial pressure increases heart rate.
- Stretch of atria sends signals to VMC via vagal afferents to increase heart rate and contractility.



# Blood Pressure Regulation

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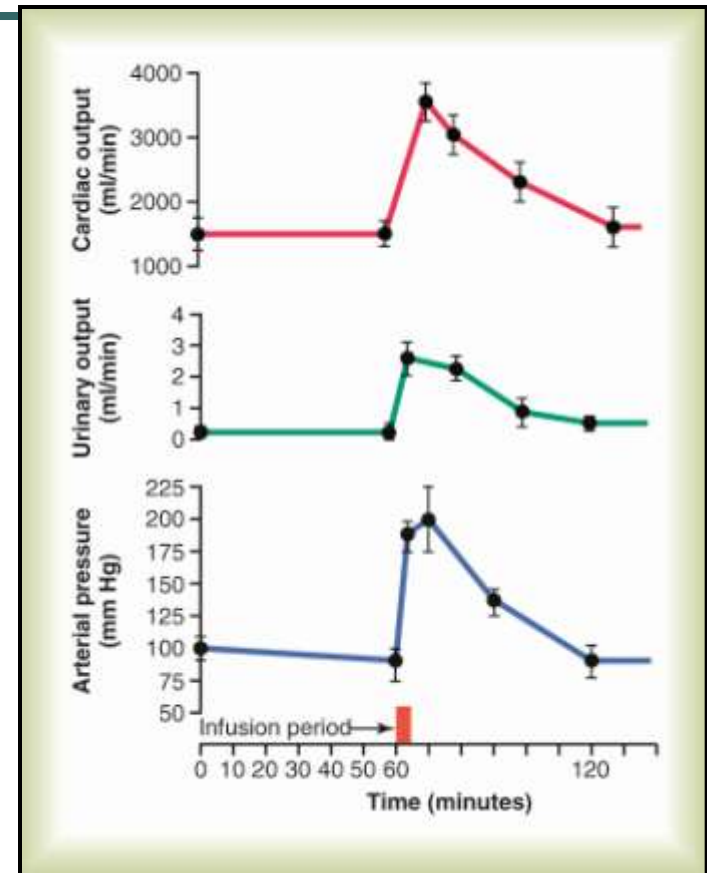
- Mean Arterial Pressure (MAP) = 1/3 systolic pressure + 2/3 diastolic pressure

$$CO = \frac{MAP}{TPR}$$

$$MAP = CO * TPR$$

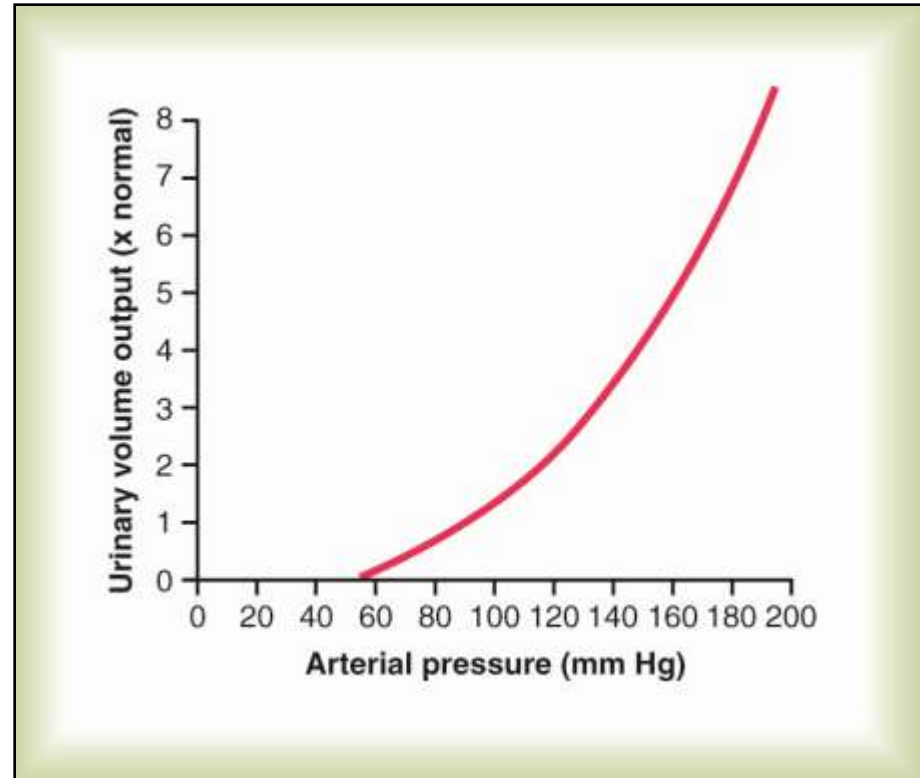
# Renal Body Fluid System for Long Term Arterial Pressure Control

- Plays a dominant role in long term pressure control.
- As extracellular fluid volume increases arterial pressure increases.
- The increase in arterial pressure causes the kidneys to lose Na and water which returns extracellular fluid volume to normal.



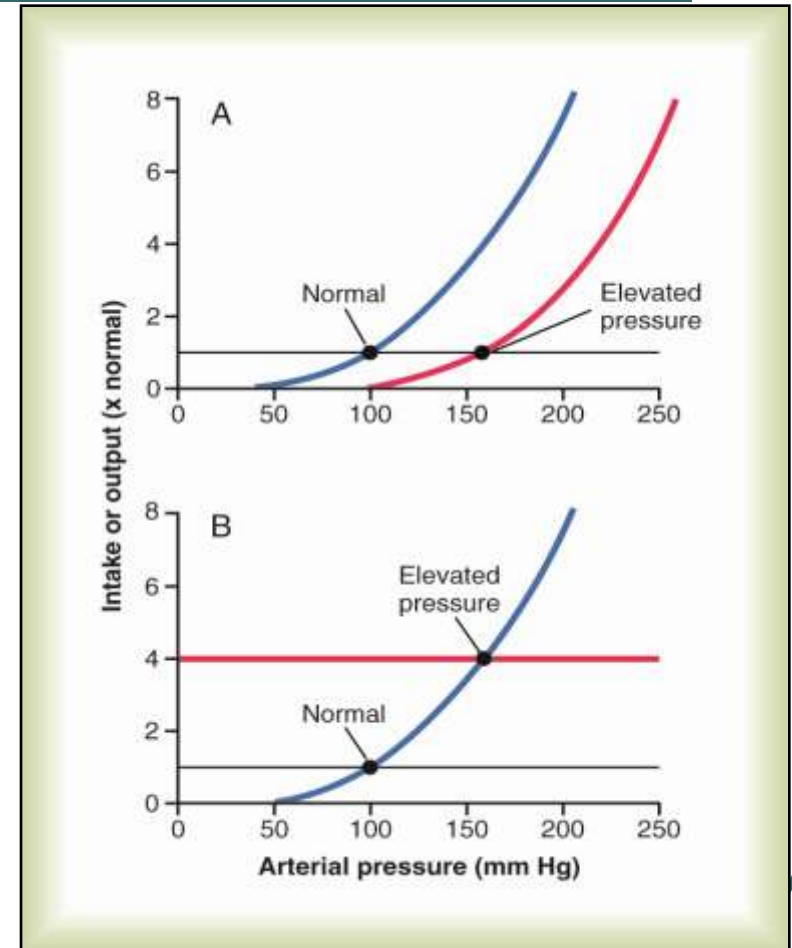
# Pressure Natriuresis and Diuresis

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



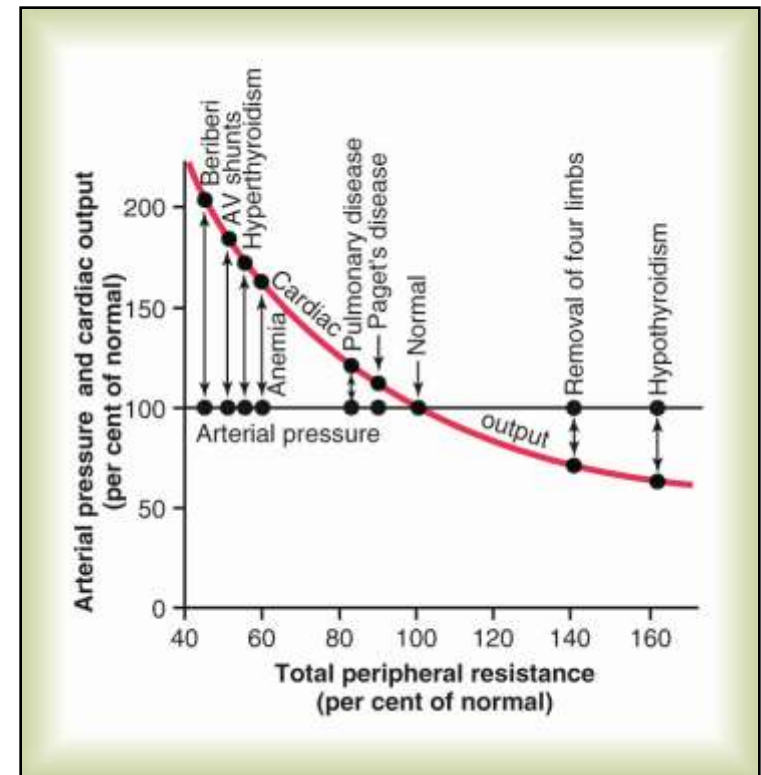
# Graphical Analysis of Renal Body Fluid Mechanism

- The major determinants of long-term arterial pressure control.
  - Based on renal function curve
  - Salt and water intake line
- Equilibrium point is where intake and output curves intersect.
- Renal body fluid feedback system has an infinite gain.



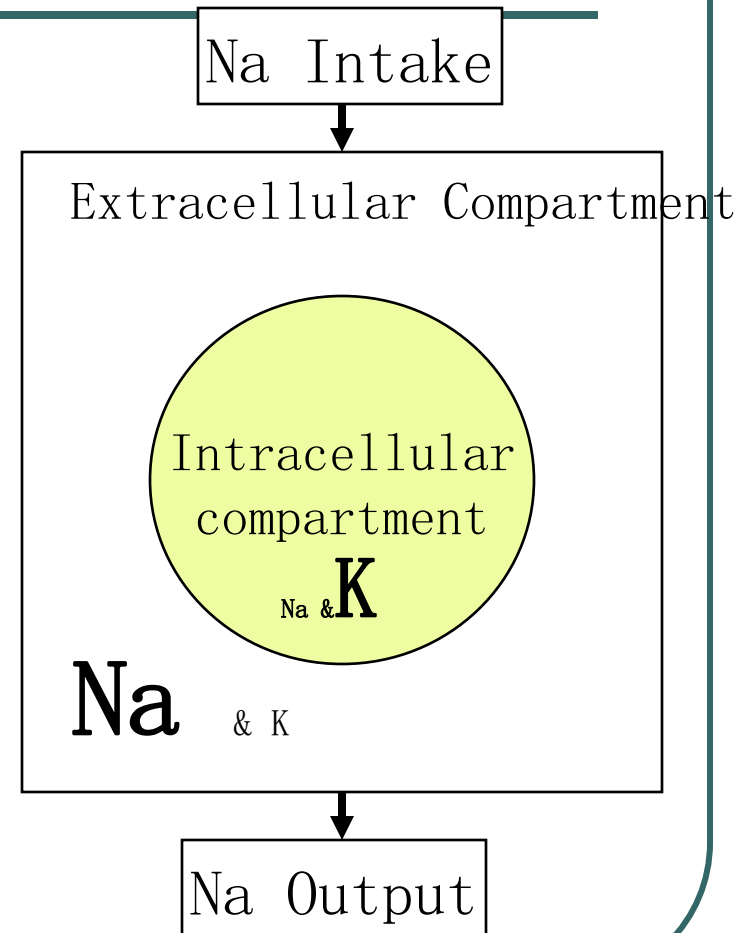
# Failure of Total Peripheral Resistance to Elevate Long-term Arterial Pressure

- Changes in TPR does not affect long-term arterial pressure level.
- One must alter the renal function curve in order to have long-term changes in arterial pressure.
- Changing renal vascular resistance does lead to long-term changes in arterial pressure .

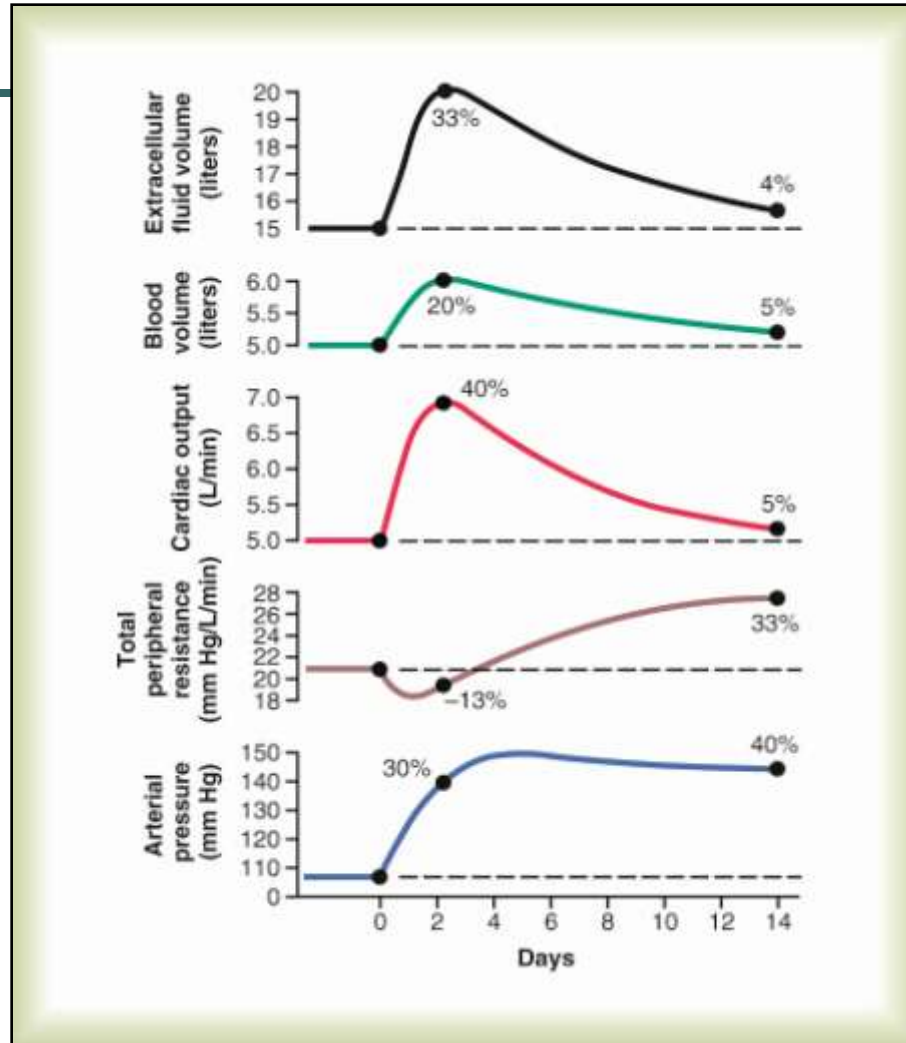


# Sodium is a Major Determinant of ECFV

- As  $\text{Na}^+$  intake is increased;  $\text{Na}^+$  stimulates drinking, increased  $\text{Na}^+$  concentration stimulates thirst and ADH secretion.
- Changes in  $\text{Na}^+$  intake leads to changes in extracellular fluid volume (ECFV).
- ECFV is determined by the balance of  $\text{Na}^+$  intake and output.

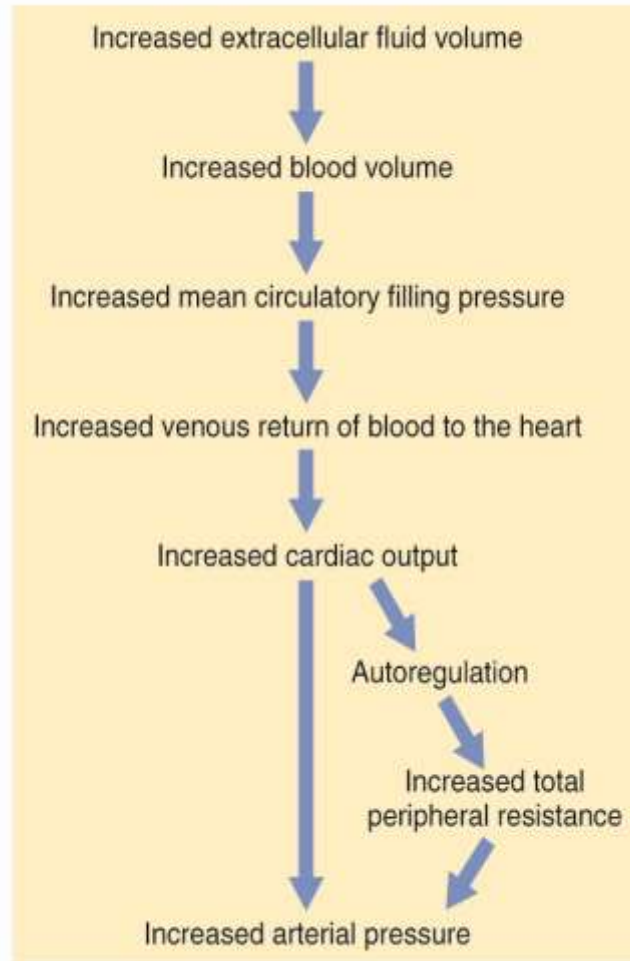


# Volume Loading Hypertension





# Effect of ECFV on Arterial Pressure



# Intermediate / Long term Regulation of BP

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## 1. Epinephrine – Adrenal medulla system

works as intermediate term needs ~ 10 min. to work causes vasoconstriction

## 2. ADH (vasopressin) system needs ~ 30 min to work causes vasoconstriction

# Long term Regulation of BP...cont

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## 3. Renin-Angiotensin-Aldosterone system ~ 1 hour to be effective

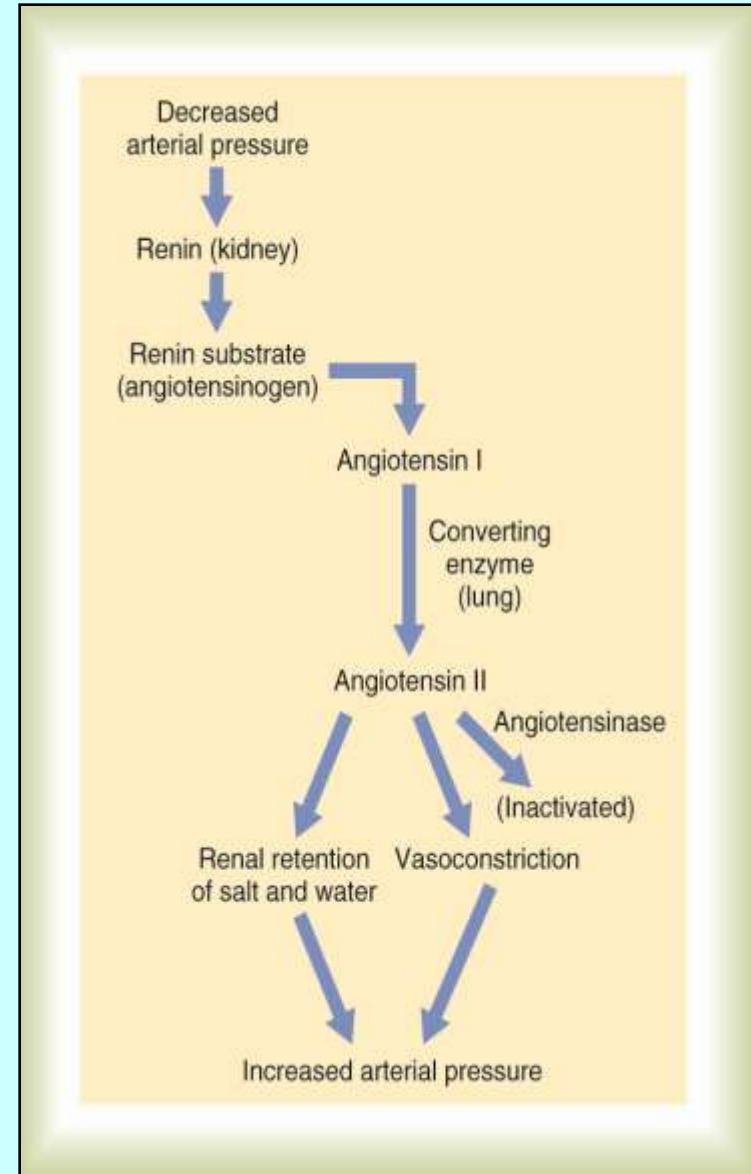
Angiotensinogen (14 a.a peptide) converted into Angiotensin I (10 a.a peptide) by Renin that come from afferent arteriolar cell, the angiotensin I is converted into angiotensin II (8 a.a peptide) by Angiotensin converting enzyme mainly in the lungs.

Angiotensin II (A II) is very potent vasoconstrictor. AII also stimulates aldosterone synthesis and secretion from the adrenal cortex (Zona glomerulosa), aldosterone increases  $\text{Na}^+$  reabsorption from the renal nephron and so water.

AII is also a positive inotropic agent

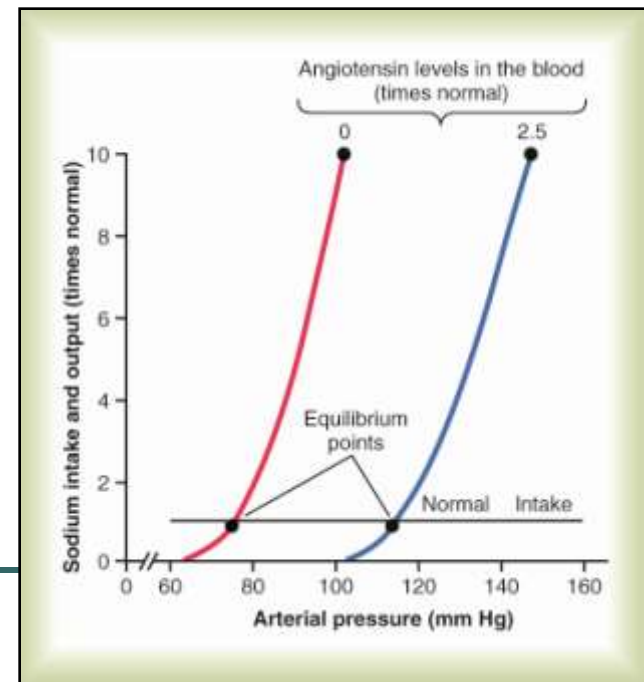
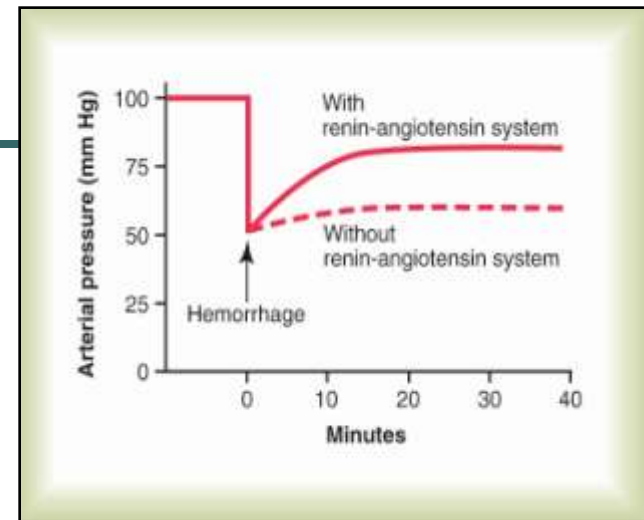
# Renin-Angiotensin System

- 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.



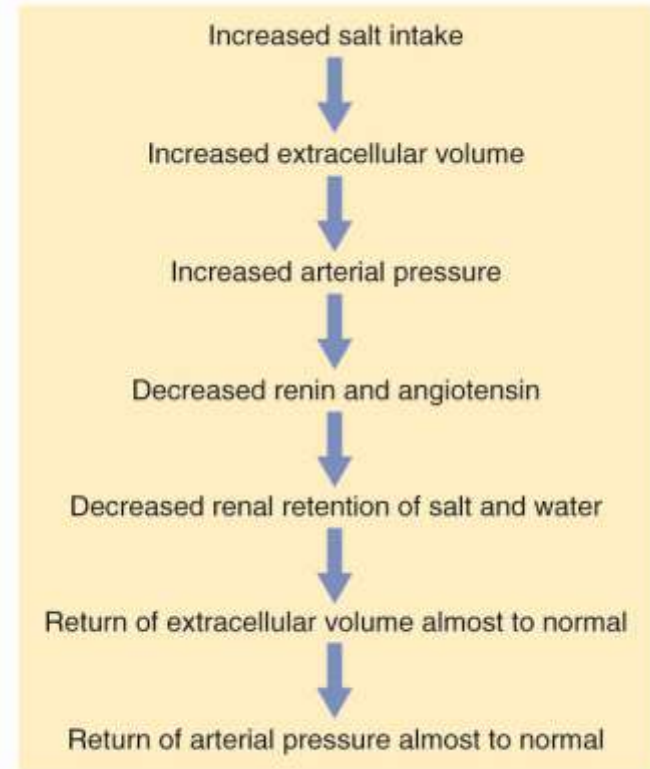
# Actions of the Renin Angiotensin System

- Causes vasoconstriction
- Causes  $\text{Na}^+$  retention by direct and indirect acts on the kidney
- Causes shift in renal function curve to right

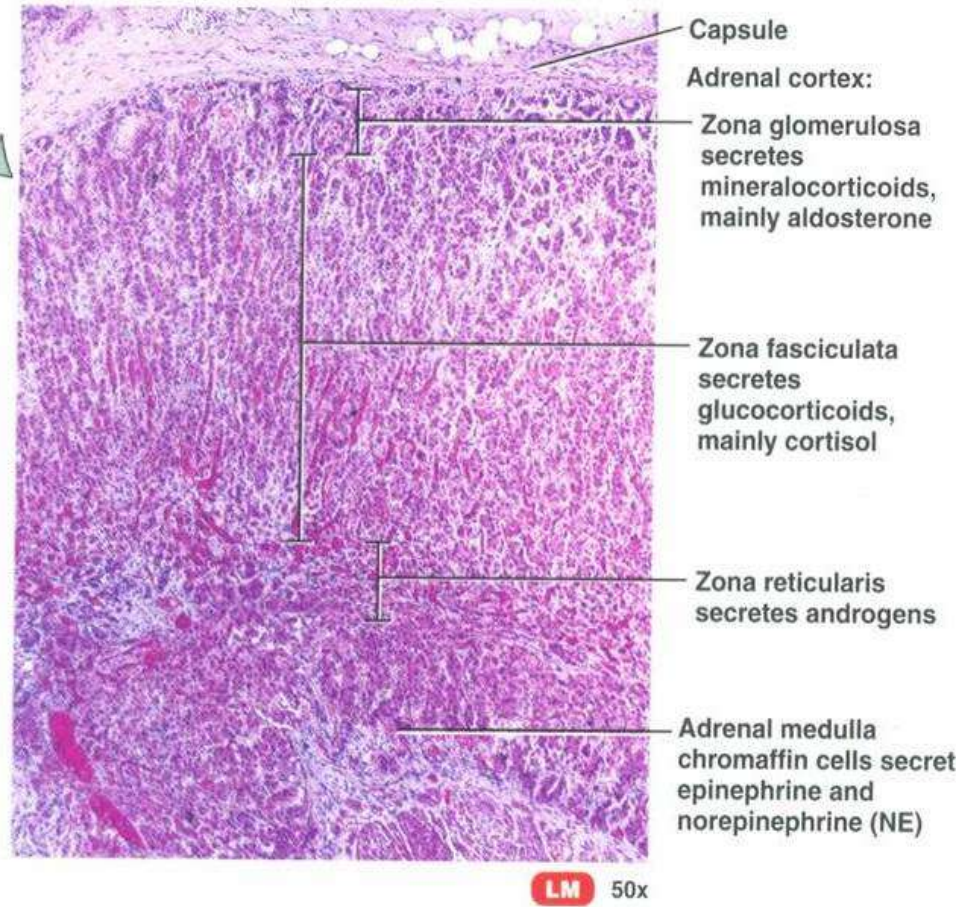
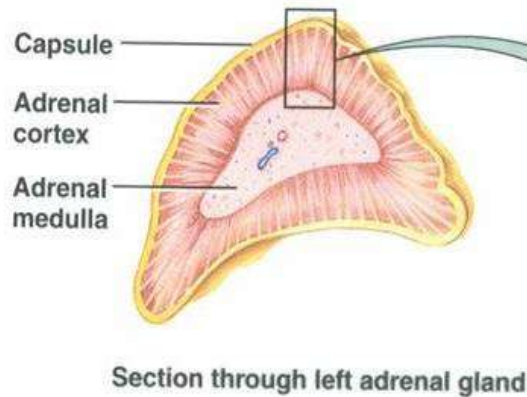


# Renin Angiotensin System: Effect of Na<sup>+</sup> Intake

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

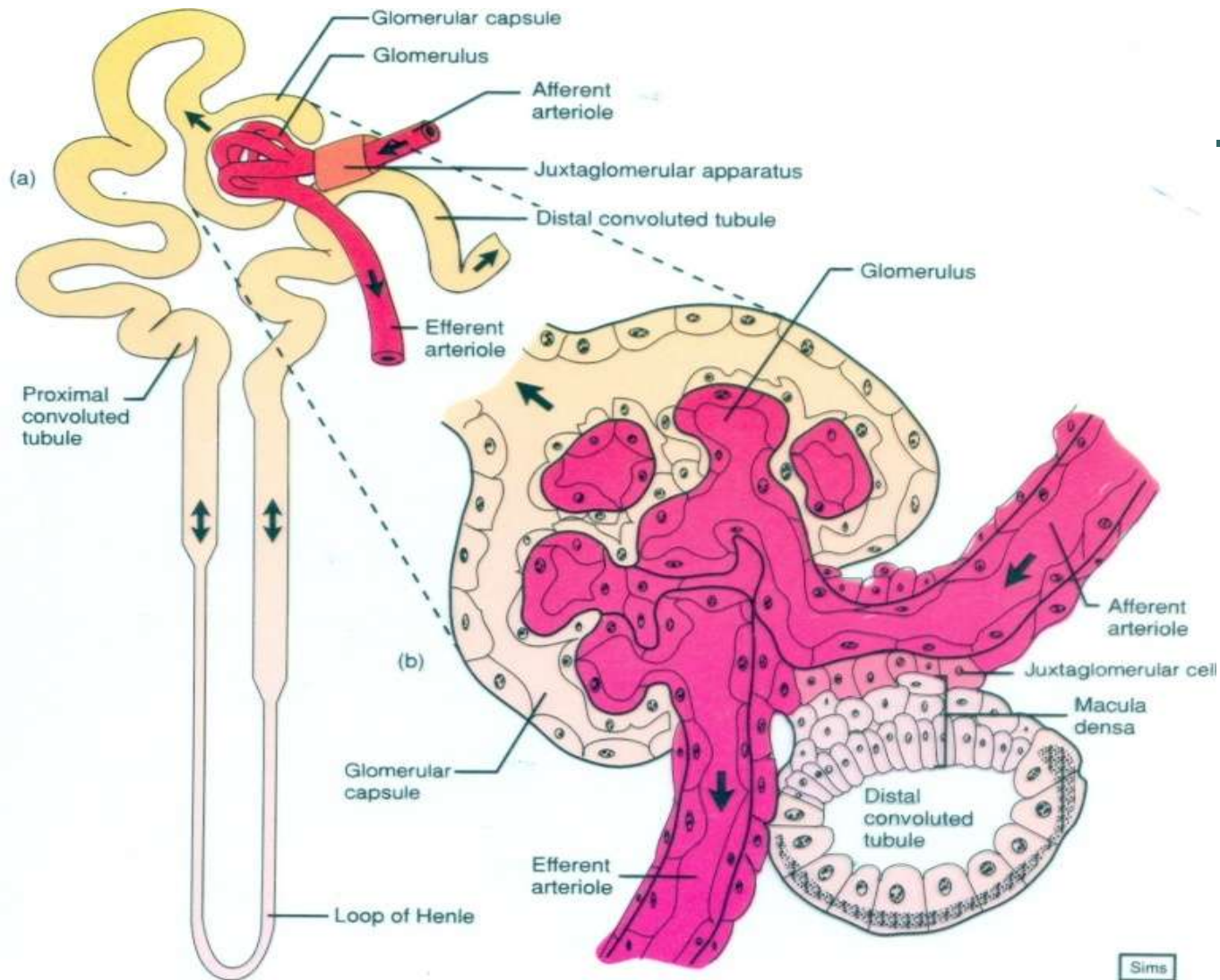


# Adrenal Gland as the source of Aldosterone (cortex) and Epinephrine (medulla)



(c) Subdivisions of the adrenal gland

# Juxtaglomerular Apparatus





## Long term Regulation of BP ...cont

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4. **Atrial Natriuretic peptide (ANP)**: An 28 a.a peptide released mainly from the Rt. Atrium in response to stretch. It causes increase in GFR so increase  $\text{Na}^+$  and water. Its concentration decreases when BP is low and its concentration increases if BP is high, mainly due volume overload

# Factors Which Decrease Renal Excretory Function and Increase Blood Pressure

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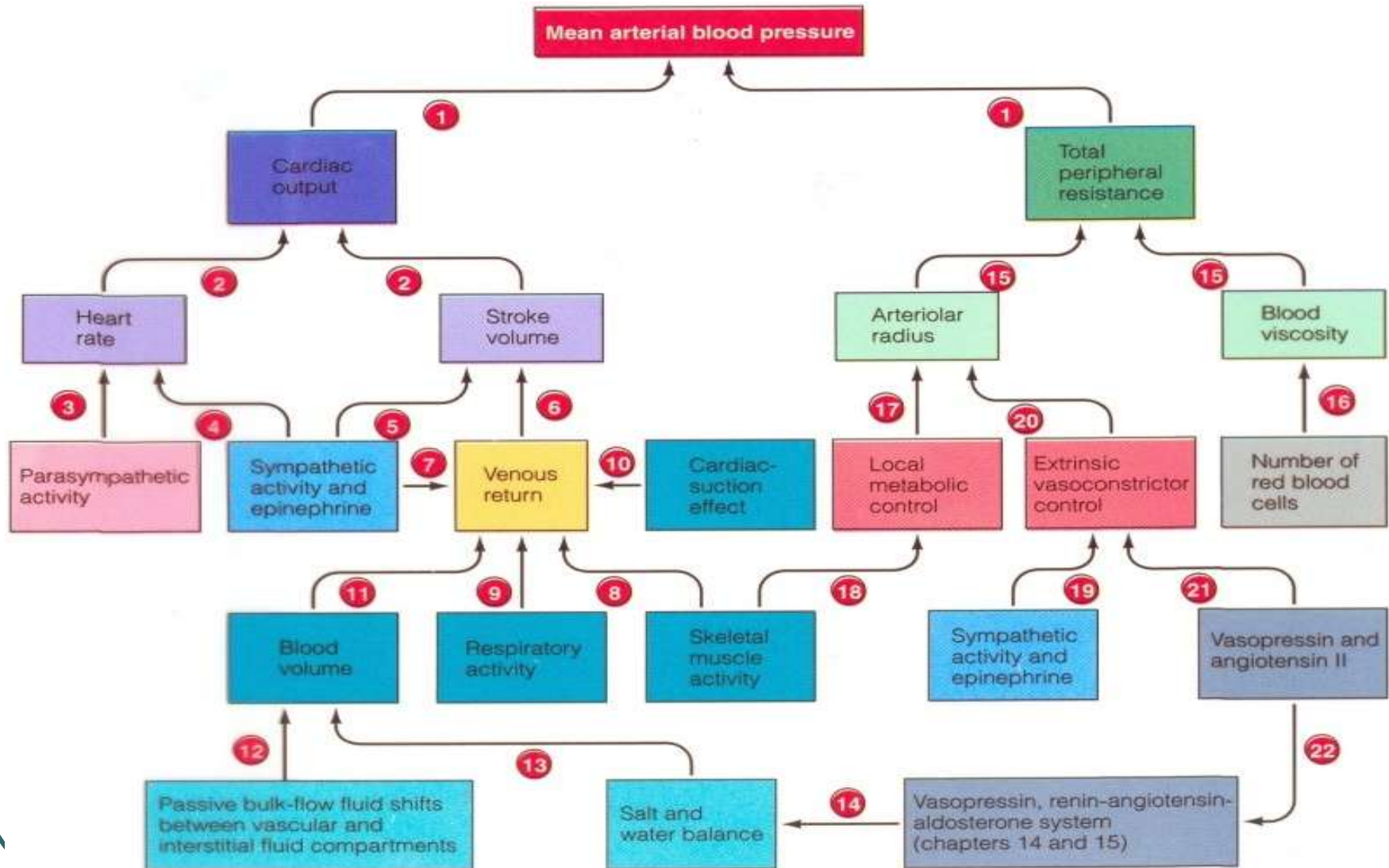
- Angiotensin II
- Aldosterone
- Sympathetic nervous activity
- Endothelin

# Factors Which Increase Renal Excretory Function and Reduce Blood Pressure

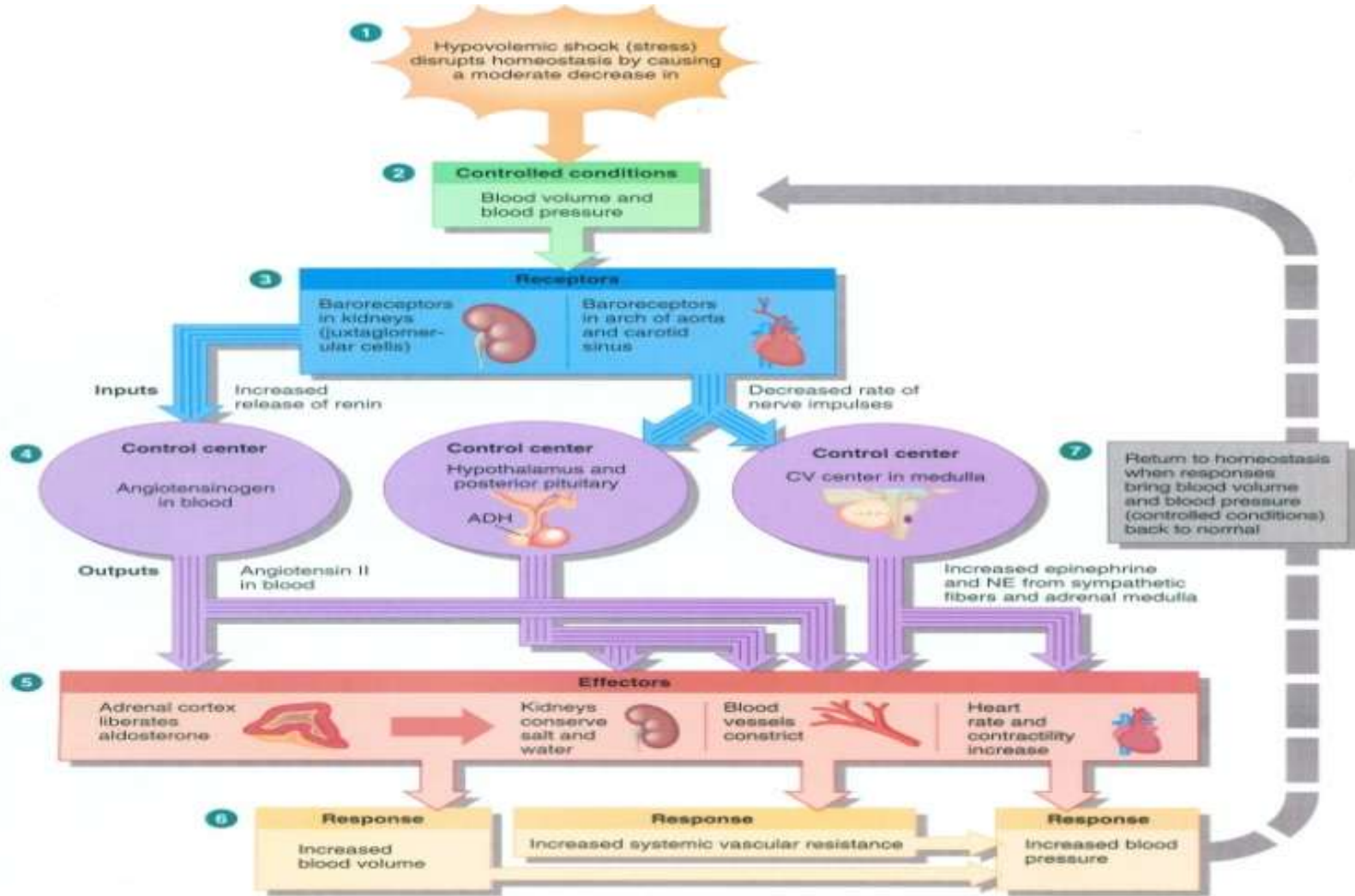
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- Atrial natriuretic peptide
- Nitric oxide
- Dopamine

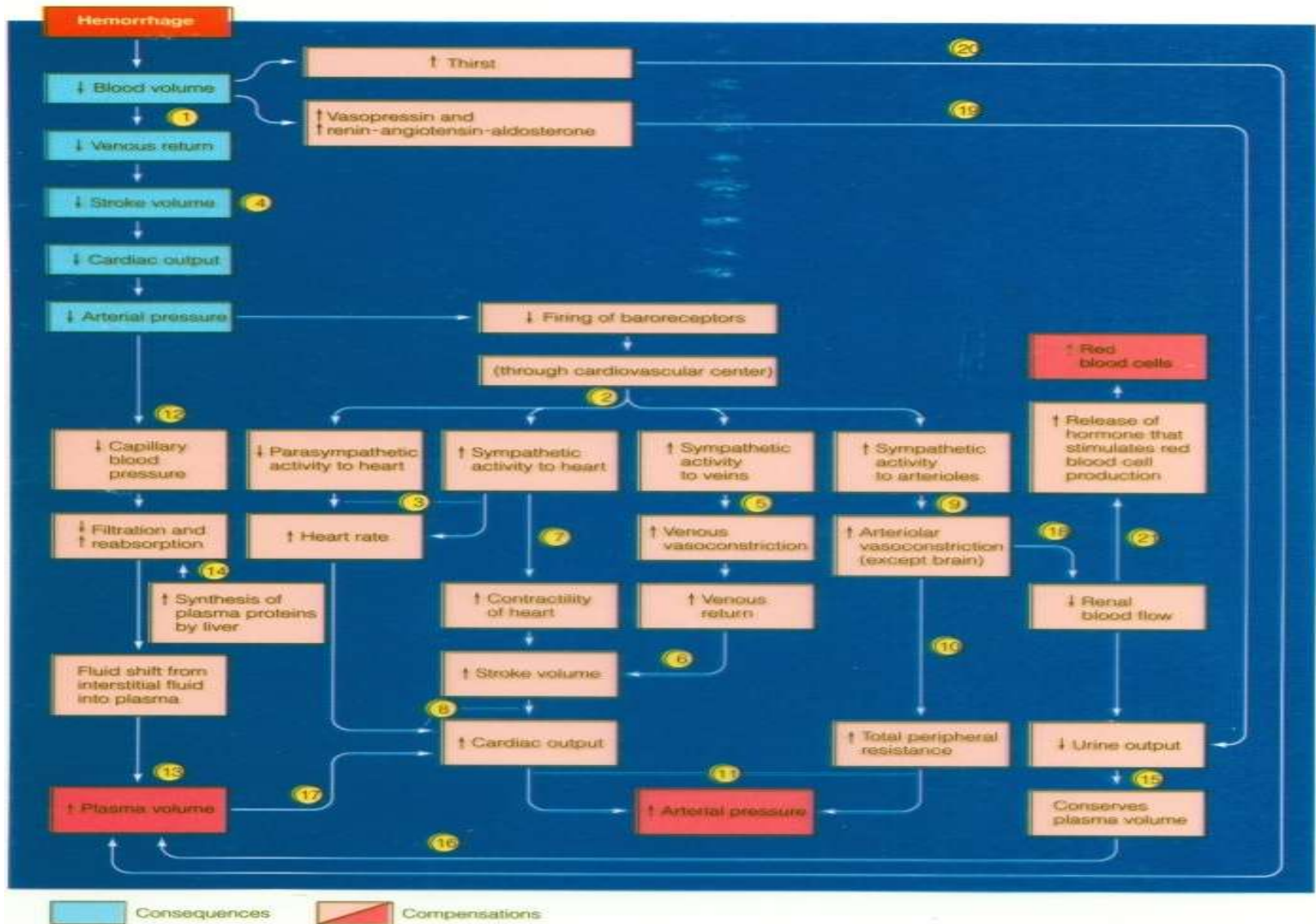
# Determinants of Mean Arterial BP



# Negative Feedback Cycle of Elevated BP



# Consequences and Compensations of Hemorrhage



# Thank You

