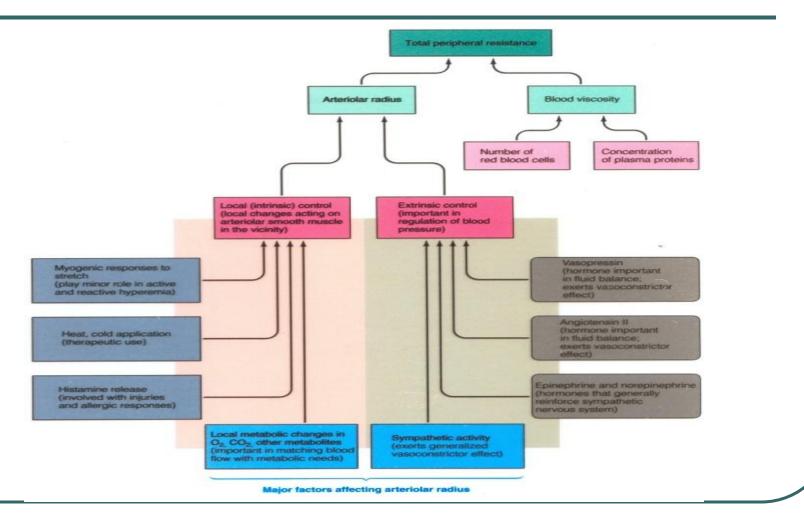
Blood Pressure Regulation 2

Faisal I. Mohammed, MD, PhD

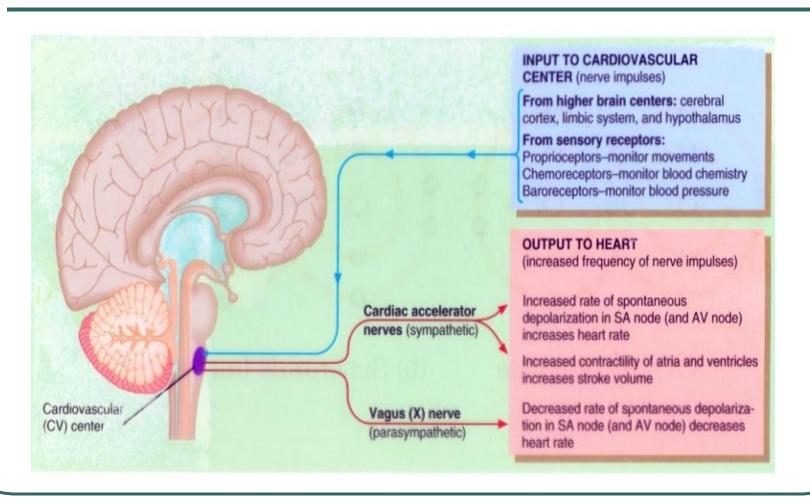
Objectives

- 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

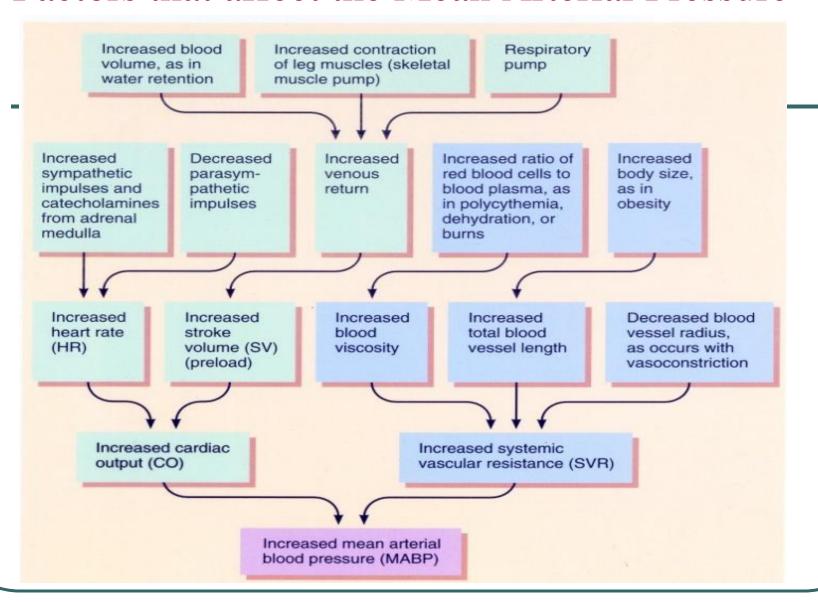
Factors affecting Total Peripheral Resistance



Nervous Control of the Heart

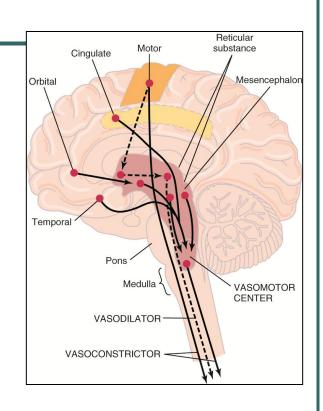


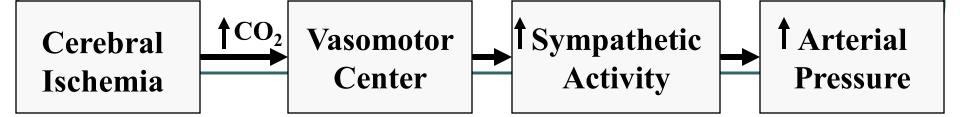
Factors that affect the Mean Arterial Pressure



CNS Ischemic Response

- CNS Ischemic response is activated in response to cerebral ischemia.
- Reduced cerebral blood flow causes CO2 buildup which stimulates vasomotor center thereby increasing arterial pressure.
- CNS Ischemic response is one of the most powerful activators of the sympathetic vasoconstrictor system.



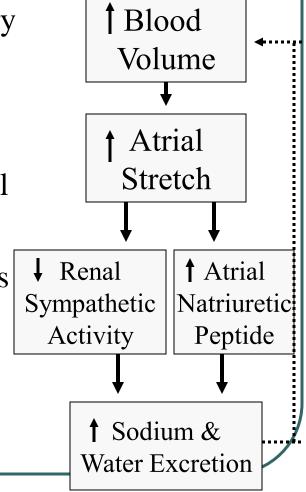


CNS Ischemic Response

- 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.

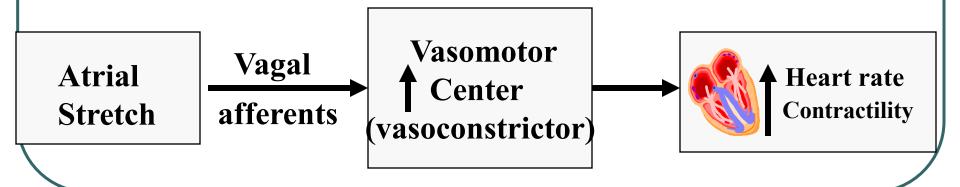
Atrial and Pulmonary Artery Reflexes

- 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 Na⁺ and water by:
 - Decreasing rate of antidiuretic hormone
 - Increasing glomerular filtration rate
 - Decreasing Na⁺ reabsorption



Bainbridge Reflex

- 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.



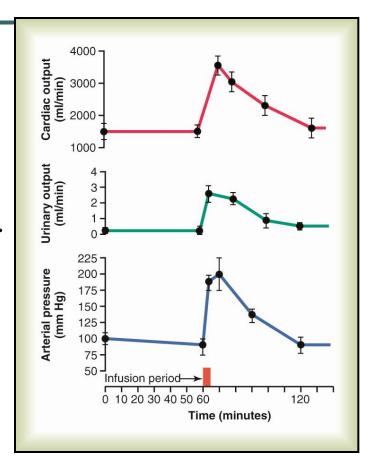
Blood Pressure Regulation

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

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

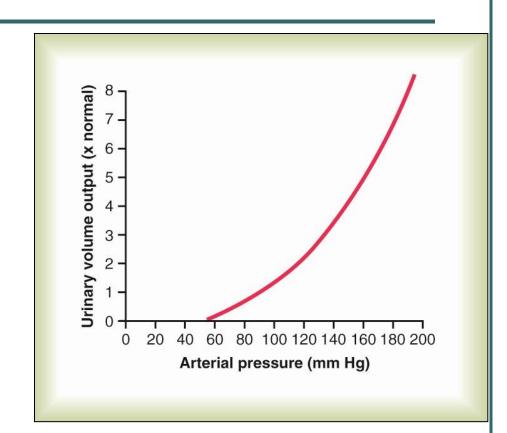
Renal Body Fluid System for Long Term Arterial Pressure Control

- 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.



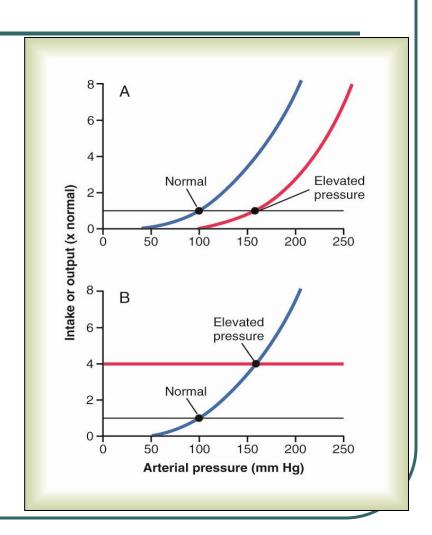
Pressure Natriuresis and Diuresis

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



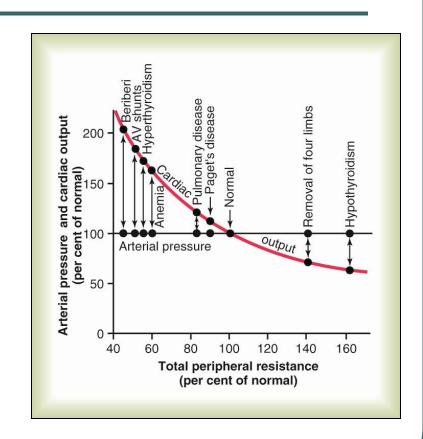
Graphical Analysis of Renal Body Fluid Mechanism

- The major determinants of longterm 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.



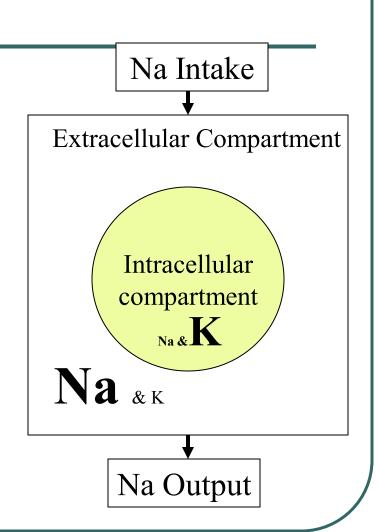
Failure of Total Peripheral Resistance to Elevate Long-term Arterial Pressure

- 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.

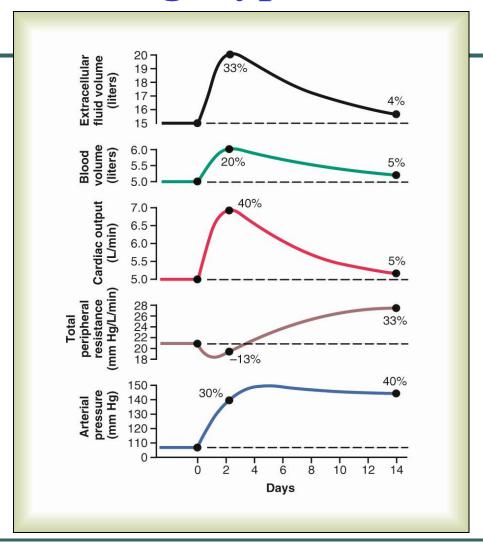


Sodium is a Major Determinant of ECFV

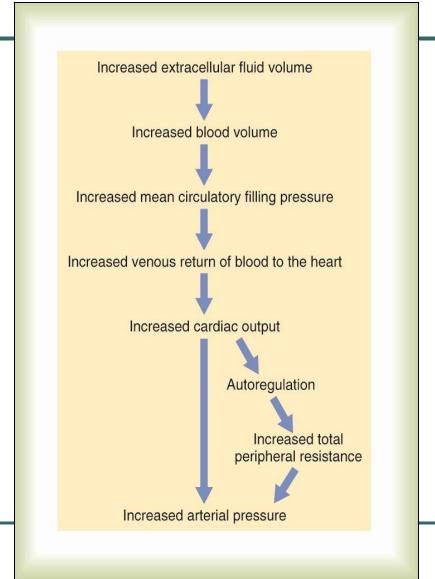
- As Na⁺ intake is increased; Na⁺ stimulates drinking, increased Na⁺ concentration stimulates thirst and ADH secretion.
- Changes in Na⁺ intake leads to changes in extracellular fluid volume (ECFV).
- ECFV is determined by the balance of Na⁺ intake and output.



Volume Loading Hypertension



Effect of ECFV on Arterial Pressure



Intermediate / Long term Regulation of BP

- 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 vasocnstriction

Long term Regulation of BP...cont

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

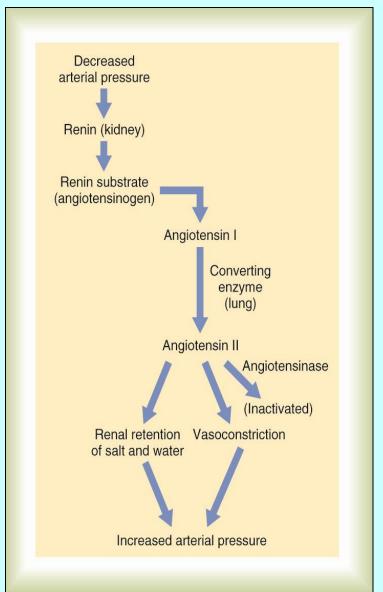
Angiotensinogen (14 a.a peptide) converted into Angiotensin I (10 a.a peptide) by Renin that come from afferent arteriolar cell, the angiotensin I is converted into angiotensin II (8 a.a peptide) by Angiotensin converting enzyme mainly in the lungs.

Angiotensin II (A II) is very potent vasoconstrictor. AII also stimulates aldosterone synthesis and secretion from the adrenal coretx (Zona glomerulosa), aldosterone increases Na⁺ reabsorption from the renal nephrone and so water.

AII is also a positive inotropic agent

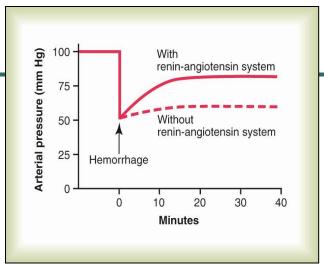
Renin-Angiotensin System

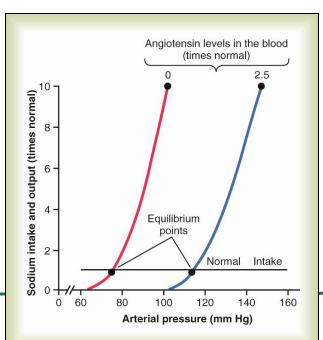
- Renin is synthesized and stored in modified smooth muscle cells in afferent arterioles of the kidney.
- Renin is released in response to a fall in pressure.
- Renin acts on a substance called angiotensinogen to form a peptide called angiotensin I.
- AI is converted to AII by a converting enzyme located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



Actions of the Renin Angiotensin System

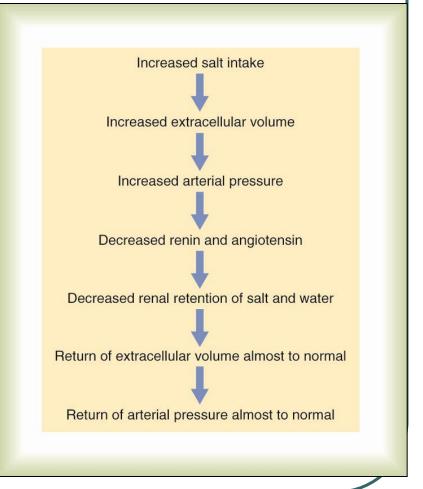
- Causes vasoconstriction
- Causes Na⁺ retention by direct and indirect acts on the kidney
- Causes shift in renal function curve to right



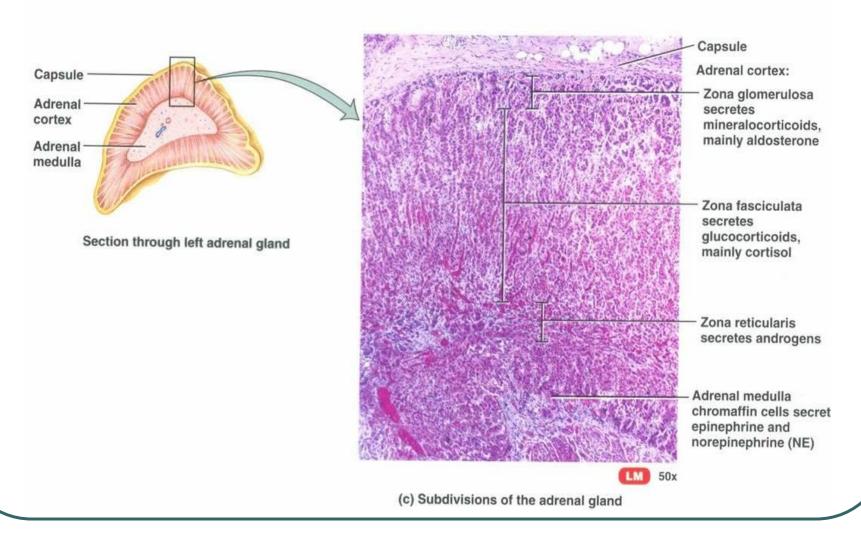


Renin Angiotensin System: Effect of Na⁺ Intake

- 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 Giomerular capsule Glomerulus Afferent arteriole Juxtaglomerular apparatus Distal convoluted tubule Glomerulus Proximal convoluted tubule Afferent arteriole (b) Juxtaglomerular cell Macula densa Glomerular capsule Distal convoluted tubule Efferent arteriole - Loop of Henle Sims

Long term Regulation of BP ...cont

4. Atrail 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 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

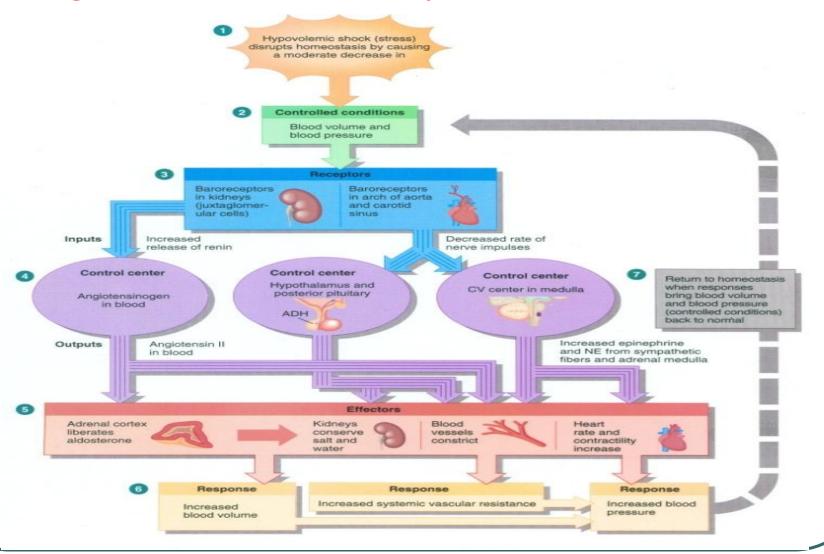
- Angiotensin II
- Aldosterone
- Sympathetic nervous activity
- Endothelin

Factors Which Increase Renal Excretory Function and Reduce Blood Pressure

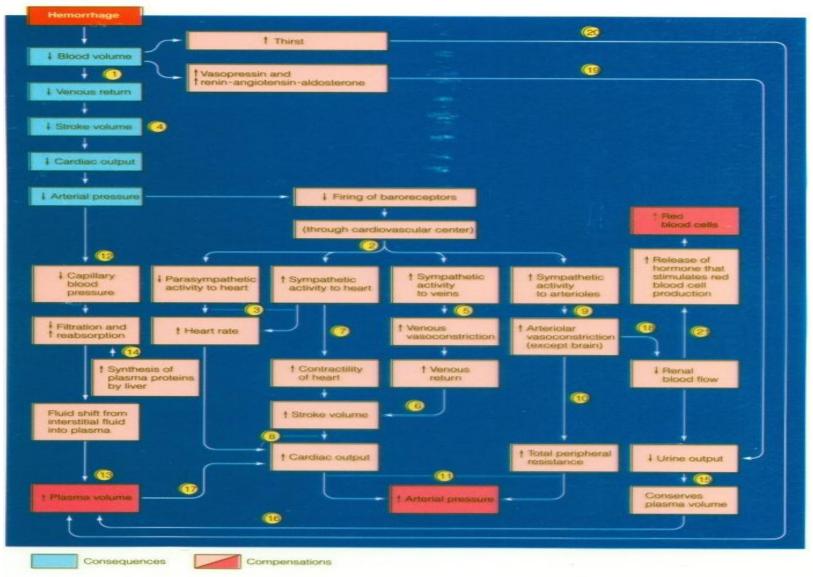
- Atrial natriuretic peptide
- Nitric oxide
- Dopamine

Determinants of Mean Arterial BP Mean arterial blood pressure 1 Total Cardiac peripheral output resistance TF) 15 Blood Arteriolar Heart Stroke viscosity radius volume rate 20 Number of Local Extrinsic Sympathetic Venous Parasympathetic red blood activity and suction metabolic vasoconstrictor return activity cells control effect control epinephrine **a** 8 9 Sympathetic Skeletal Vasopressin and Blood Respiratory activity and muscle angiotensin II volume activity epinephrine activity 22 13 Vasopressin, renin-angiotensin-Passive bulk-flow fluid shifts Salt and aldosterone system between vascular and water balance (chapters 14 and 15) interstitial fluid compartments

Negative Feedback Cycle of Elevated BP



Consequences and Compensations of Hemorrhage



Thank You

