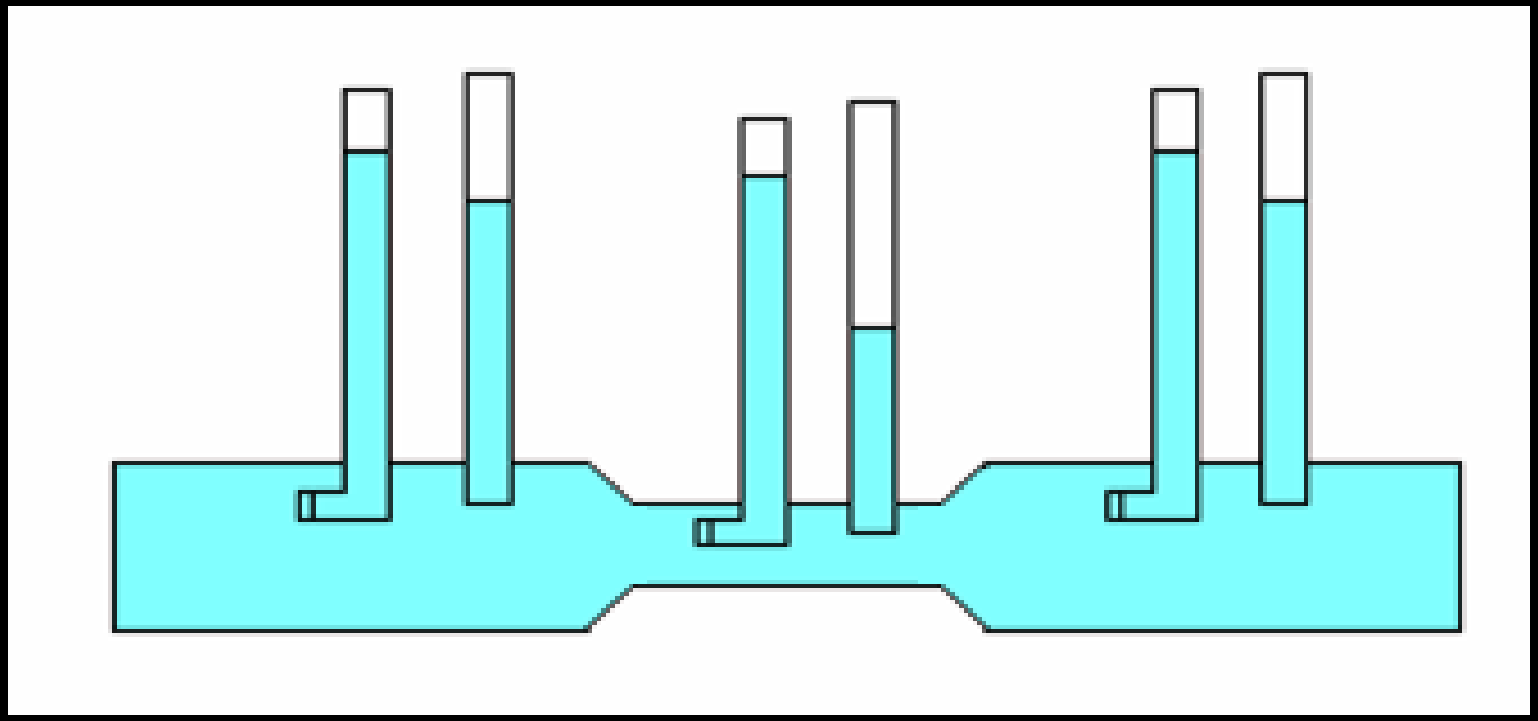
CARDIO-VASCULAR SYSTEM

- Dr. Chetna Ramanuj



REGULATION OF BLOOD PRESSURE

1. RAPID BLOOD PRESSURE CONTROL MECHANISM
   (Nervous regulating mechanism)

2. INTERMEDIATE BLOOD PRESSURE CONTROL MECHANISM

3. LONG-TERM BLOOD PRESSURE CONTROL MECHANISM

INTERMEDIATE BLOOD PRESSURE CONTROL MECHANISM

Salient features:

- These mechanisms come into play after several minutes of acute pressure changes and reach full function within a few hours
- These mechanisms play their role from few days to few weeks
- They try to control the alterations in blood pressure by altering the blood volume

INTERMEDIATE BLOOD PRESSURE CONTROL MECHANISM

- RENIN-ANGIOTENSIN VASOCONSTRICTOR MECHANISM
- STRESS RELAXATION AND REVERSE STRESS RELAXATION MECHANISM
- CAPILLARY FLUID SHIFT MECHANISM
- ABDOMINAL COMPRESSION REFLEX

RENIN-ANGIOTENSIN VASOCONSTRICTOR MECHANISM

- Renin is a small protein enzyme, released by the kidneys when the arterial pressure falls too low.
- Renin is synthesized and stored in an inactive form called “Prorenin” in the “Juxtaglomerular” (JG) cells of the kidneys
- JG cells are modified smooth muscle cells located in the walls of the afferent arterioles immediately proximal to the glomeruli

RENIN-ANGIOTENSIN VASOCONSTRICTOR MECHANISM

When the arterial pressure falls

Prorenin molecule in JG cells split and release renin

⇓

Renin acts enzymatically on other plasma protein, a globulin called

"Renin substrate" or "angiotensinogen"

⇓

It releases 10 Amino acid Peptide Angiotensin-I

A-I has a mild vasoconstrictor property ⇓ ⇓

Renin persist in blood for 30 min. to 1 hour to cause formation of Angiotensin-I during this entire time

the procedure mainly occurs in lungs by "converting" enzyme ⇓ ⇓ within few seconds of formation of A-I

Angiotensin-II (8 Amino acid peptide)

RENIN-ANGIOTENSIN VASOCONSTRICTOR MECHANISM

- A-I is extremely powerful “vasoconstrictor”
- It persists in the blood only for 1 to 2 minutes
- Rapidly inactivated by “angiotensinase” enzyme

RENIN-ANGIOTENSIN VASOCONSTRICTOR MECHANISM

A-II has 2 peripheral effects that can elevate arterial pressure

1. Vasoconstriction occurs rapidly and intensely in arterioles

⇓

Increased HR

⇓

Increased BP

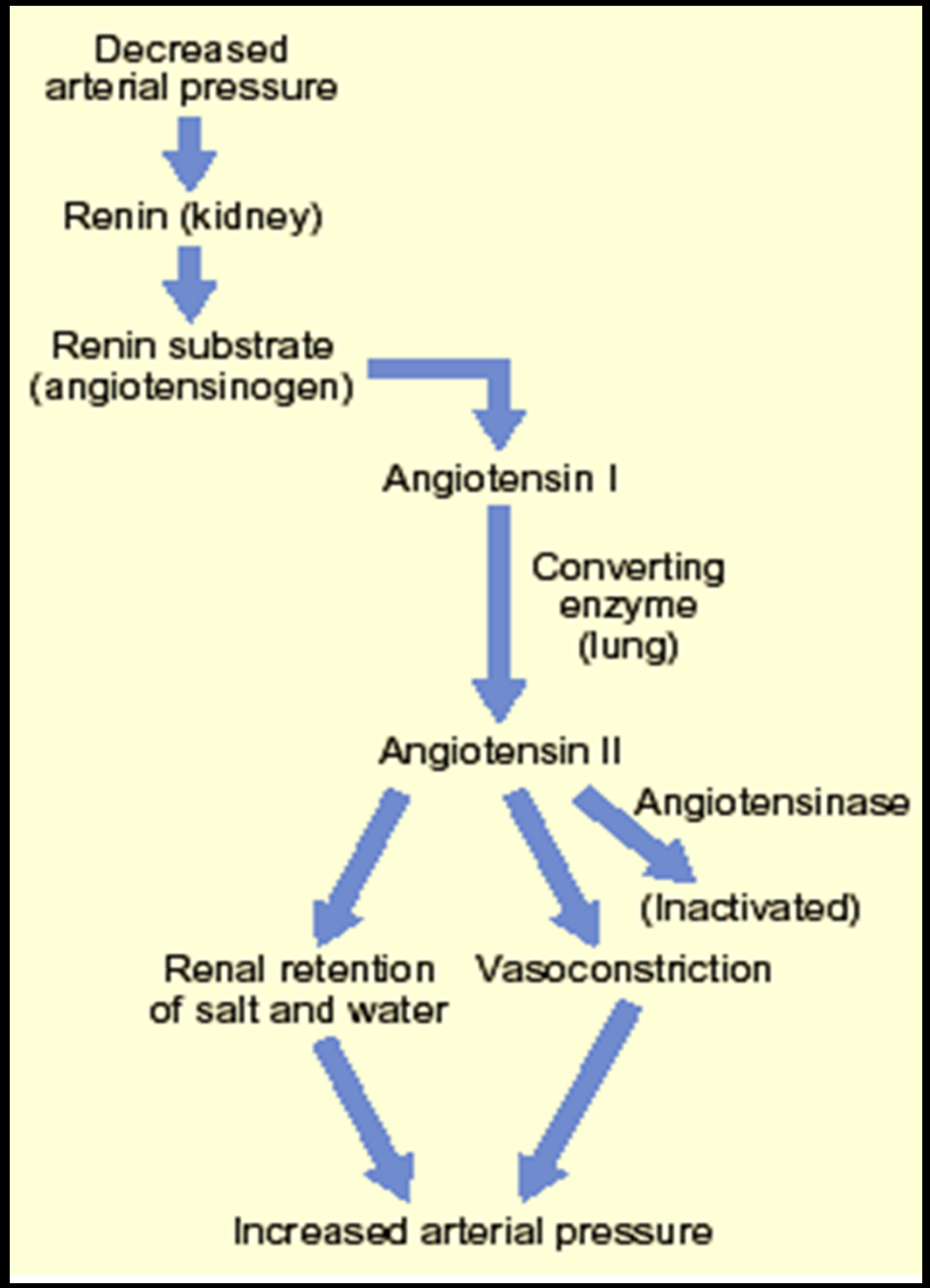
2. Act on kidneys to decrease the excretion of salt and water

⇓

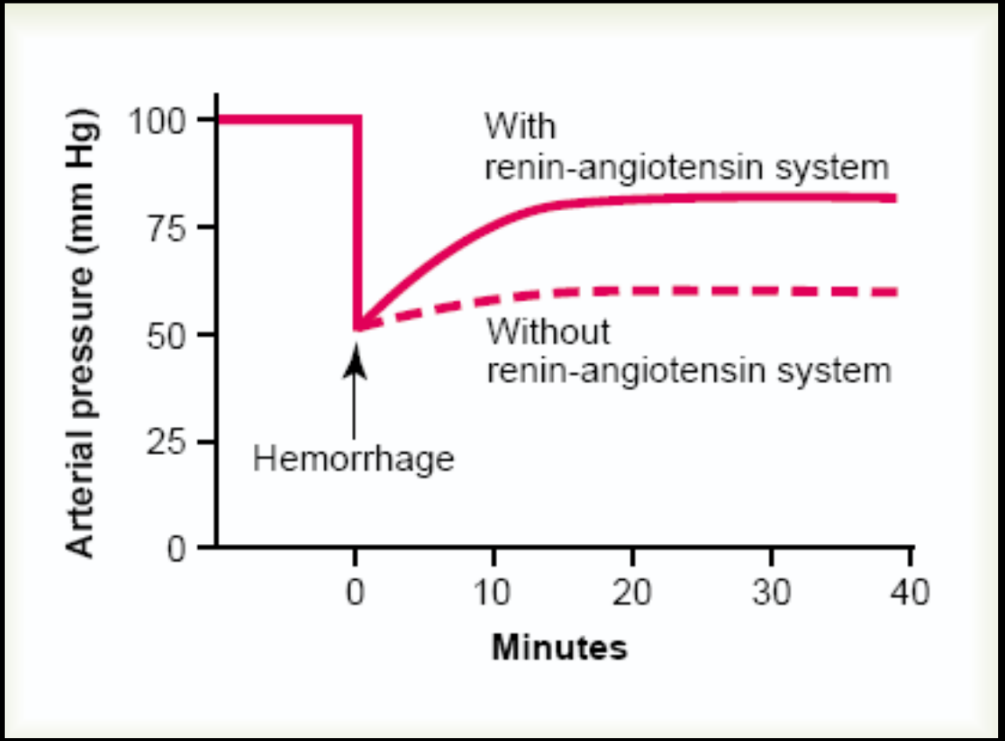
Increase ECF volume

⇓

Increase arterial pressure over a period of hours to days


Decreased arterial pressure
Renin (kidney)
Renin substrate (angiotensinogen)
Angiotensin I
Converting enzyme (lung)
Angiotensin II
Angiotensinase
(Inactivated)
Renal retention of salt and water
Vasoconstriction
Increased arterial pressure

Rapidity and intensity of the vasoconstrictor pressure response to Renin-Angiotensin system



- After hemorrhage, acute fall in BP to 50 mmHg.
- When R.A. system is functional : BP rises back to 83 mmHg
- If R.A. system not functioning : BP rises to 60 mmHg

❑ It can be a life saving service in “circulatory shock”

❑ It requires 20 minutes to become fully active

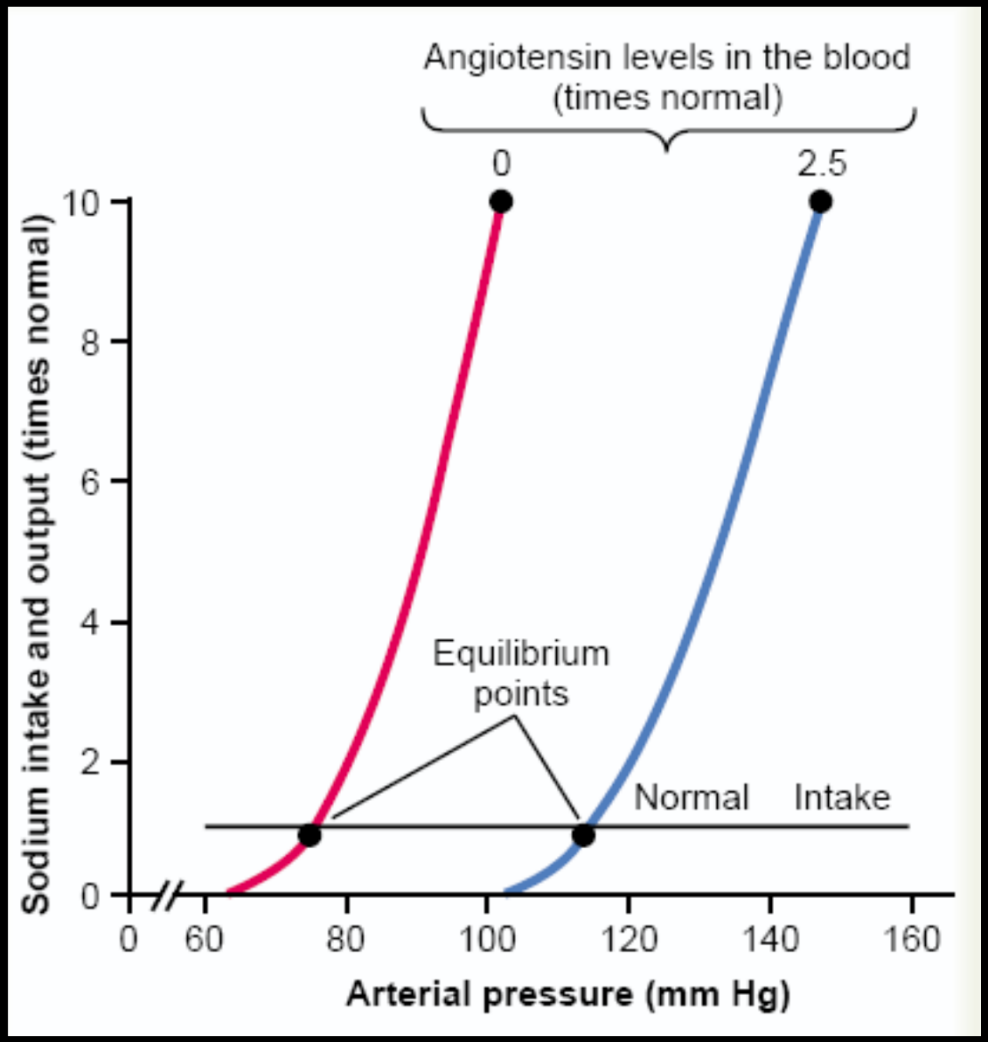
RENIN-ANGIOTENSIN VASOCONSTRICTOR MECHANISM

- Effect of angiotensin to cause renal retention of salt and water is also an important mean for long term control of arterial pressure

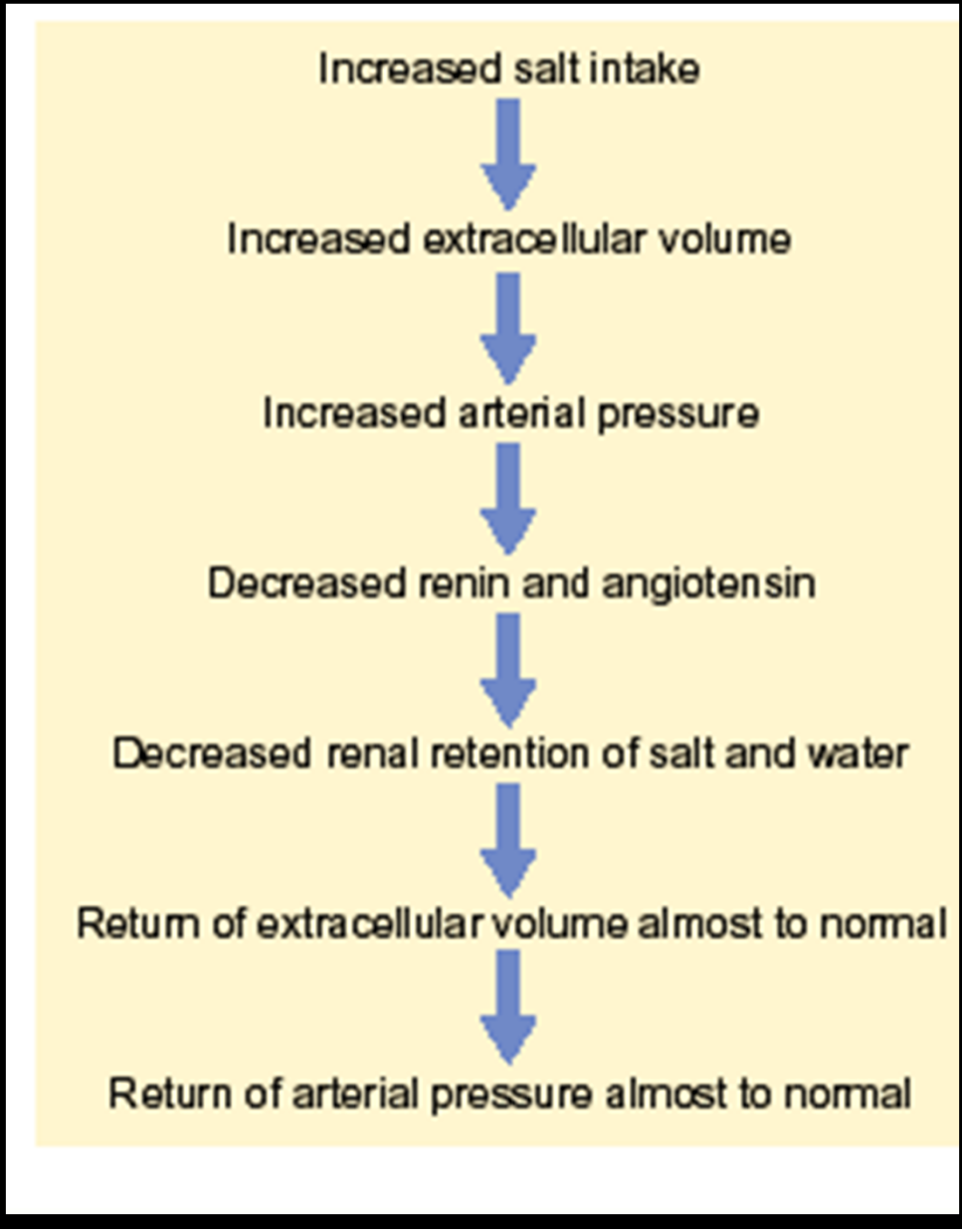
Angiotensin cause the kidneys to retain both salt and water in two ways

1. Act directly on the kidneys to cause retention
2. Causes adrenal glands to secret aldosterone which cause increased salt and water reabsorption by kidney tubules

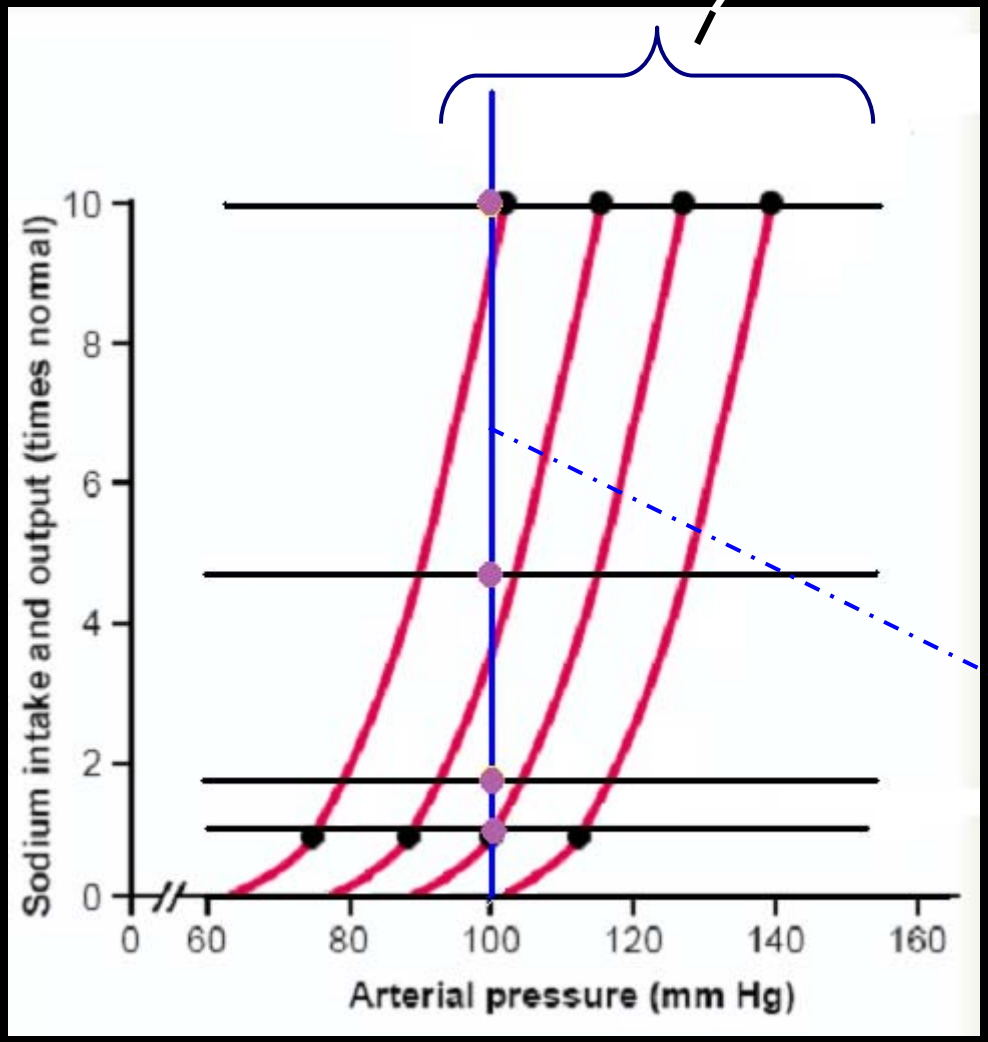
QUANTITATIVE ANALYSIS OF ARTERIAL PRESSURE CHANGES CAUSED BY ANGIOTENSIN



Role of angiotensin system in maintaining arterial pressure despite wide variations in salt intake



Effect of angiotensin on renal curves (times normal)



0

0.2

1.0

2.0

Angiotensin production at different sodium intake (times normal)

Sodium loading renal function curve

- 50 fold change in Na^+ intake (1/5 to 10 times) will lead to rise in BP only 4 to 6 mmHg when R.A. system is working {as seen by animal experiments}

- In human, 1/15 to 10 times normal intake of Na^+ {150 fold} will lead to rise in BP of 17 mmHg only when R.A. system is working

STRESS RELAXATION AND REVERSE STRESS RELAXATION MECHANISM

STRESS RELAXATION MECHANISM :

- It refers to vasodilatation occurring due to stress on the vascular smooth muscles.
- When pressure in the vessels become too high, the vessels become stretched and continue to stretch for minutes or hours.
- This causes relaxation of blood vessels simply by vascular tone adjustment.
- This leads to an increase in the capacity of the arterial system with a concomitant fall in blood pressure

STRESS RELAXATION AND REVERSE STRESS RELAXATION MECHANISM

REVERSE STRESS RELAXATION MECHANISM :

- Operates when the BP is low due to less stress on the vessel walls and tries to restore it back to normal.
- Example: when BP falls due to prolonged slow bleeding, there occurs tightening of blood vessel walls by vascular tone adjustment secondary to less stress on the vessel wall (reverse stress relaxation mechanism)
- This mechanism tries to restore the BP back to normal
- This mechanism can correct up to 15 % change in blood volume below normal.

CAPILLARY FLUID SHIFT MECHANISM

This mechanism helps in restoring both low and high BP back to normal

When BP is raised :

The mean capillary pressure is also high resulting in shift of fluid from circulation to the interstitial fluid compartments. This reduces the blood volume to restore the arterial pressure.

CAPILLARY FLUID SHIFT MECHANISM

When BP is lowered :

The mean capillary pressure is also low resulting in absorption of fluid from interstitial fluid compartments to the circulation. Thus the blood volume is increased which helps to restore the blood pressure back to normal

ABDOMINAL COMPRESSION REFLEX

When baroreceptor or chemoreceptor reflex is elicited or whenever any factor stimulate sympathetic vasoconstrictor system:

nerve signals are also transmitted to skeletal nerves to skeletal muscles

⇓

Increase basal tone of muscle

⇓

Compresses all venous reservoir of the abdomen

⇓

Helps to translocate blood out of abdominal vasculature towards heart

LONG-TERM BLOOD PRESSURE CONTROL MECHANISM

RENAL BODY FLUID SYSTEM FOR ARTERIAL PRESSURE CONTROL

When the body contains too much ECF

⇓

The arterial pressure rises

⇓

The rising pressure in turn has a direct effect to cause the kidneys to excrete the excess ECF

⇓

Thus returning the pressure back towards normal

Pressure diuresis:

An increase in arterial pressure of only a few mmHg can double the renal output of water.

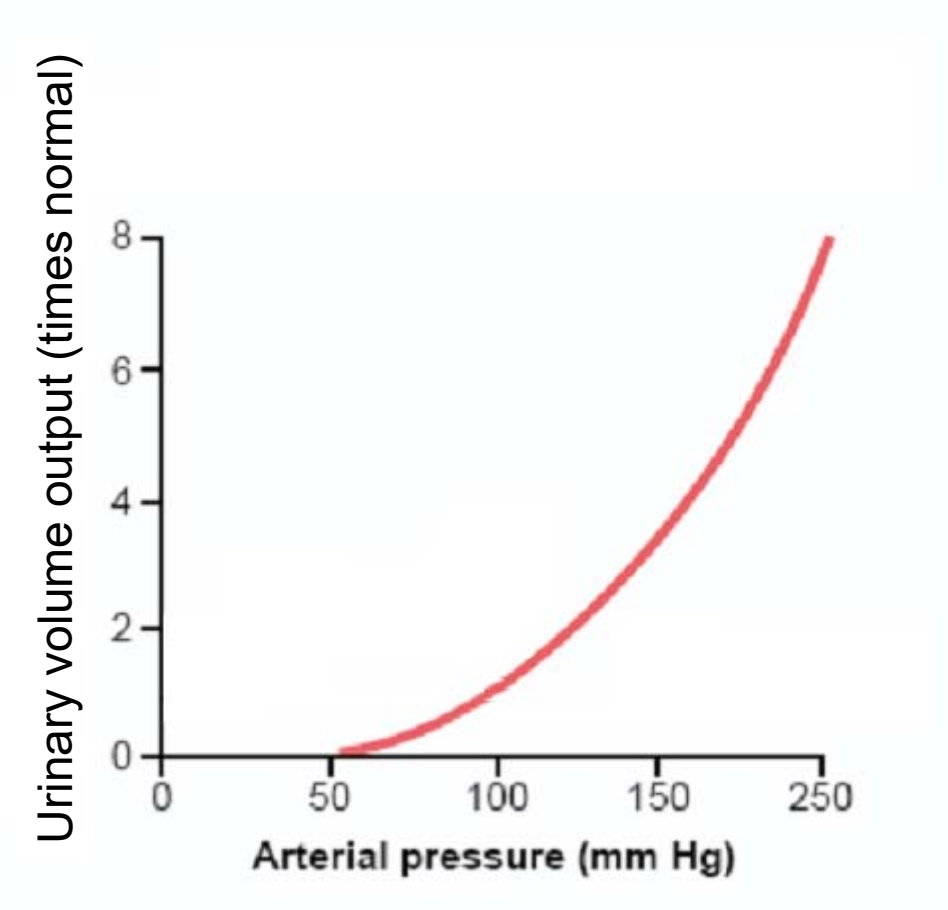
This is called pressure diuresis

Pressure natriuresis:

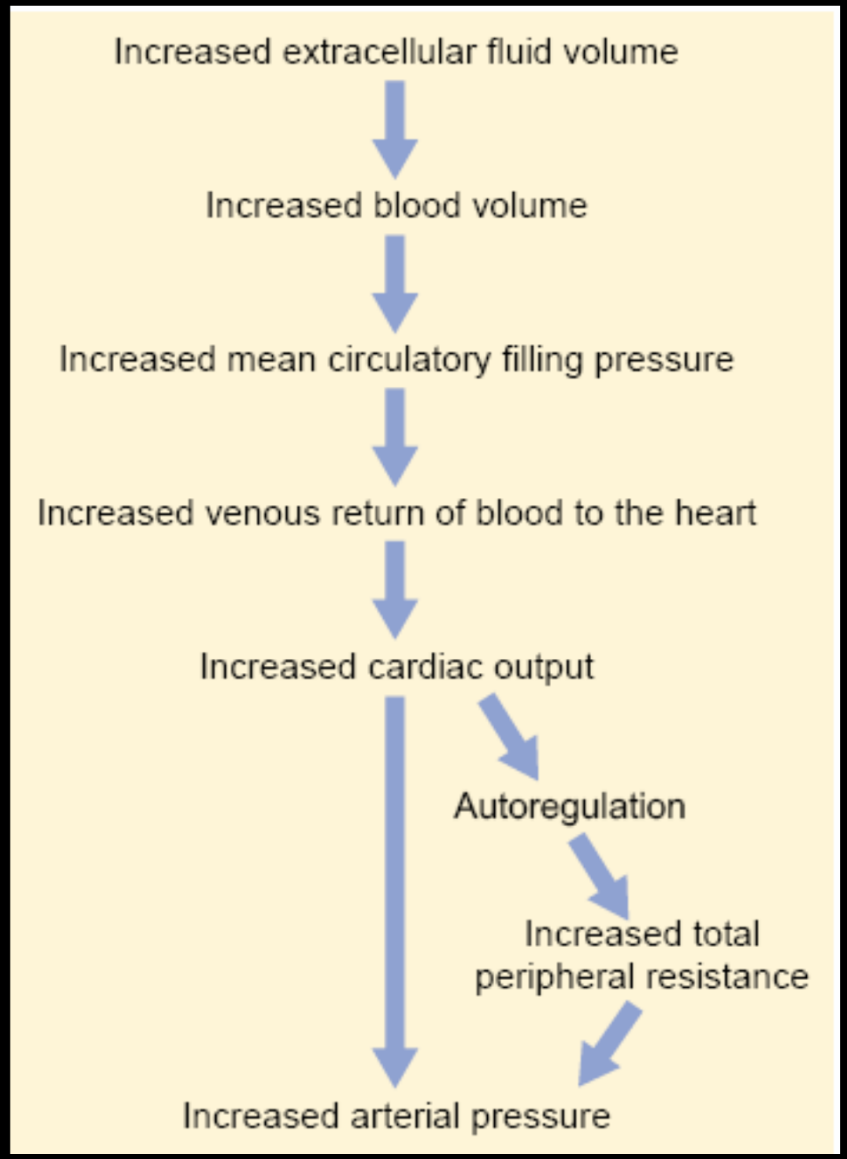
An increase in arterial pressure of only a few mmHg can double the renal output of salt.

This is called pressure natriuresis

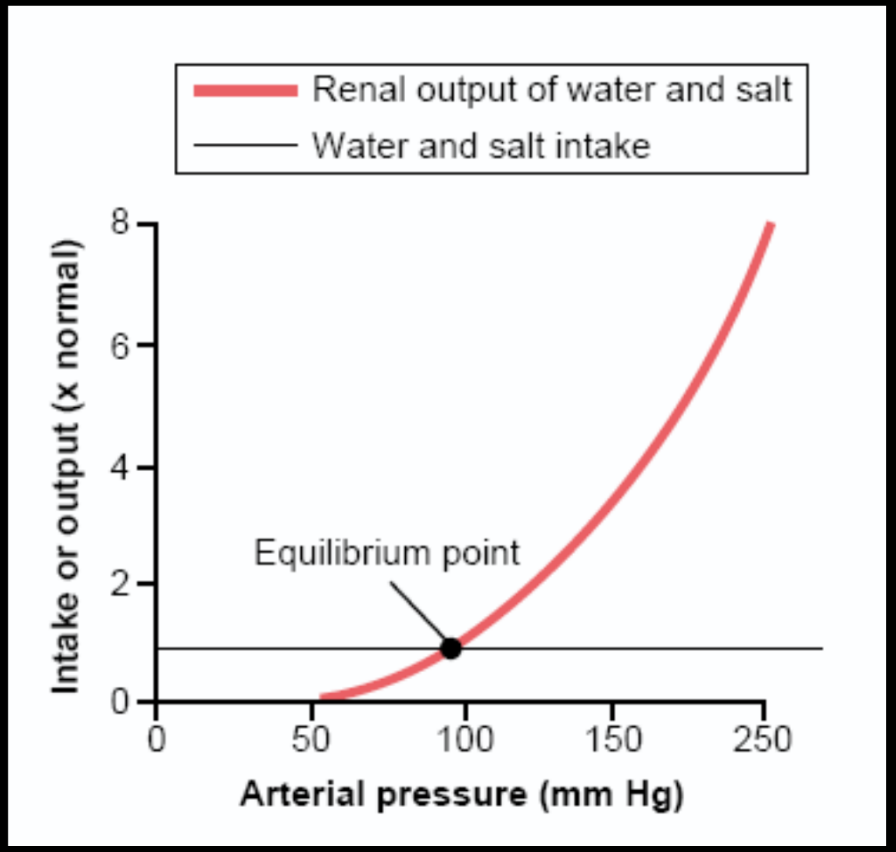
RENAL OUTPUT CURVE OR RENAL FUNCTION CURVE



- At 50 mmHg : Urine output 0
- At 100 mmHg : Urine output normal
- At 200 mmHg : Urine output 6-8 times normal


Increased extracellular fluid volume
Increased blood volume
Increased mean circulatory filling pressure
Increased venous return of blood to the heart
Increased cardiac output
Autoregulation
Increased total peripheral resistance
Increased arterial pressure

Infinite feedback gain principal:



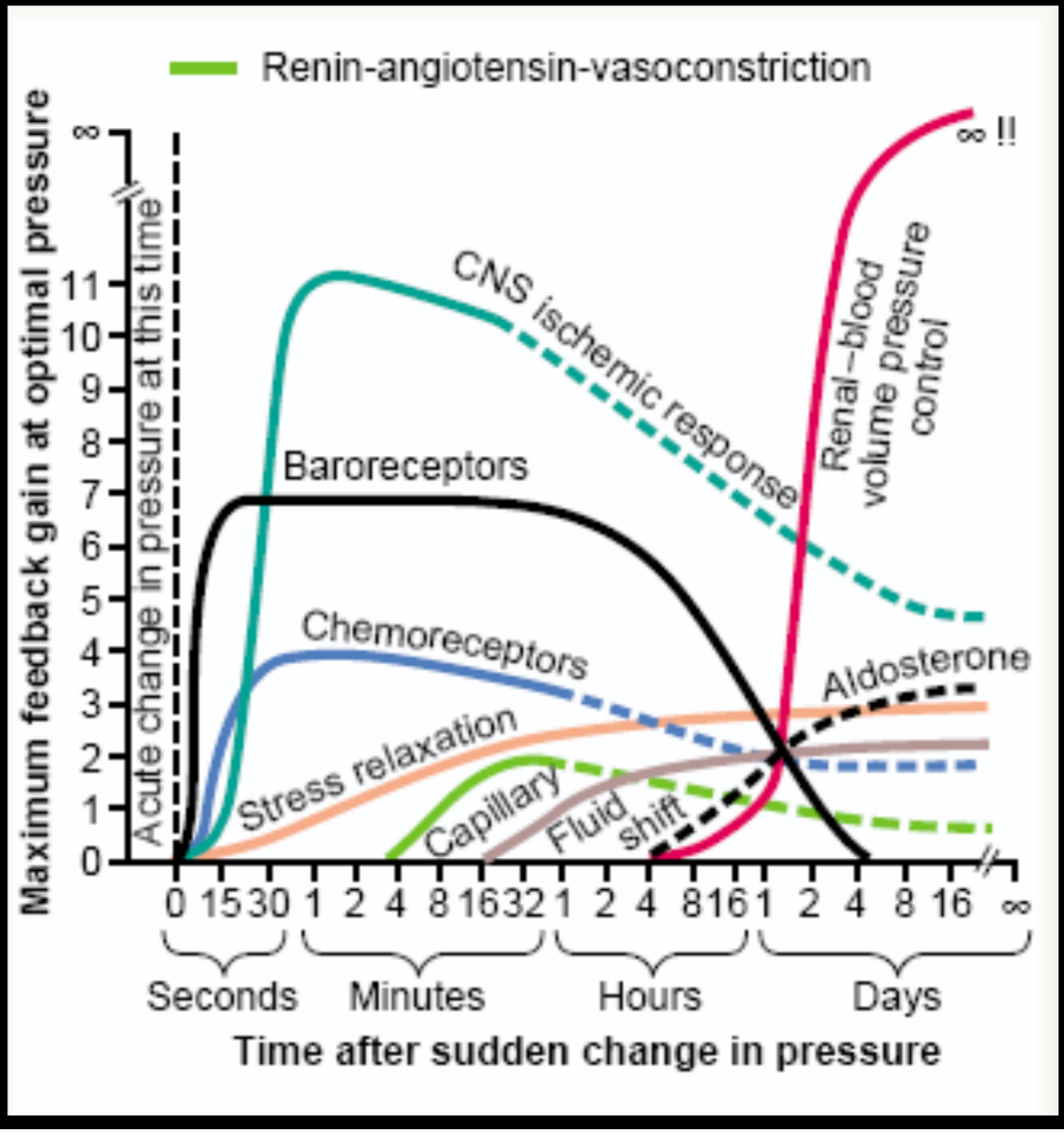
EQUILIBRIUM POINT : the point at which output equals the intake

Infinite feedback gain principal:

- 1ST Assume that arterial pressure rises to 150 mmHg, renal output of water and salt is about 3 times as great as intake
  Therefore, the body loses fluid → the blood volume decreases → the arterial pressure decreases
  This –ve balance of fluid will not cease until the pressure falls all the way back exactly to the equilibrium point

- If arterial pressure falls below the equilibrium point, the intake of water and salt would be greater than the output → body fluid volume increases → arterial pressure rises until it return to the equilibrium point

Various arterial pressure control mechanisms at different time level



HEART RATE

DEFINITION :

no. of heart beats per minute

Normal value :

60 – 100 / minute (average 72) in adults

< 60 is called : Bradycardia

> 100 is called : Tachycardia

Factors affecting the heart rate

- **AGE:**

After birth, as age increases, vagal tone increases and HR decreases, but in old age HR is slightly higher due to fall in vagal tone.

Foetal HR : 140 – 150

At Birth : 130 – 140

At 12 years : upto 100

Adults : 70 – 80

Old age : upto 100

Factors affecting the heart rate

- **GENDER**:

HR is slightly higher in females as compared to males due to

- Lower systemic BP
- More Resting sympathetic tone

Factors affecting the heart rate

- **BODY TEMPERATURE:**

HR is directly related to the body temp.

- HR rises with rise in body temp. For each 1° F rise in body temp., HR increases by about 10 beats / min.

  It also produces vasodilatation causing fall in BP.

- Fall in Body temp. decreases HR.

  It also produces vasoconstriction causing BP to rise.

❖ **Thus, HR is *inversely* related to the systemic BP (Marey's Law)**

Factors affecting the heart rate

- **DISEASES:**

INCREASE IN INTRACRANIAL TENSION:

Rise in ICT

⇓

decreased blood supply to medulla producing local hypoxia and hypercapnia

⇓

Stimulation of VMC

⇓

Restoration of blood supply to medulla by increase in BP (Baroreceptor mechanism)

⇓

Decreased HR

Increased ICT is associated with bradycardia (Cushing Reflex)

Factors affecting the heart rate

- **DISEASES:**

THYROTOXICOSIS:

It is associated with high resting HR

Factors affecting the heart rate

- **DRUGS:**

Drugs like epinephrine increases HR due to direct action on heart

Factors affecting the heart rate

- **EMOTIONS:**

Emotions like

- Excitement, fear, anger etc. are associated with *tachycardia*

- Sudden shock, grief etc. are associated with *bradycardia*

Factors affecting the heart rate

- **EXERCISE:**

HR increases in linear pattern with the severity of the exercise because :

- ❖ Increase in sympathetic activity
- ❖ Decrease in vagal tone
- ❖ Increase in body temperature
- ❖ Release of catecholamines and thyroxine
- ❖ Change in blood chemistry (hypoxia and hypercapnia)

CONTROL OF HEART RATE

Mainly by two mechanisms:

1. Through cardiac innervation
2. Through medullary cardiovascular centres

Regulation of heart rate

Mediated via vasomotor center (VMC)

- **Marey's reflex (via Baroreceptor reflex) :**

Increased B.P. → stimulation of baroreceptors → Impulses through IX and X cranial nerves → nucleus of tractus solitarius → stimulation of vasodilator area and inhibition of vasoconstrictor area → increase in vagal tone and Decrease sympathetic tone → reflex bradycardia

Regulation of heart rate

Mediated via vasomotor center (VMC)

- **Bain bridge reflex (via Cardiopulmonary reflex):**

Increased venous return → stimulation of stretch receptors In right atrium → afferent impulses through vagus → Inhibition of vasodilator area → decrease in vagal tone → Tachycardia

Regulation of heart rate

Mediated via vasomotor center (VMC)

- **Chemo receptor reflex :**

low po2, high pco2 and acidosis → stimulation of chemoreceptors → Impulses through IX and X cranial nerves → nucleus of tractus solitarius → stimulation of vasomotor center → tachycardia

Regulation of heart rate

Mediated via vasomotor center (VMC)

- **Cushing reflex (via CNS ischemic reflex) :**

Increase ICP → Increases B.P. due to CNS ischemic reflex → Increase B.P. in turn causes reflex bradycardia by Baroreceptor reflex

- **Role of higher centers :**

some parts of cerebral cortex & limbic system increases heart rate while some other parts decreases heart rate

Regulation of heart rate

Mediated via vasomotor center (VMC)

- **Role of peripheral afferents :**

example- painful stimuli produces tachycardia

- **Role of respiratory center :**

during inspiration impulse spill over from respiratory center to VMC and heart rate increases (it is called sinus arrhythmia)

Regulation of heart rate

Mediated without vasomotor center (VMC)

- **Role of thyroid hormones & temperature :**

T3, T4 & increase temperature directly stimulates SA node and cause tachycardia

HEART RATE

REGULATORY REFLEXES:

- Marey's Reflex : ↑ B.P. ↓ es HR
- Bain bridge Reflex : ↑ B volume ↑ es HR
- Chemo receptor Reflex : ↑ pco_2 ↑ es HR
- Cushing's Reflex : ↑ ICP ↓es HR

DISCLAIMER

- All figures are taken from Guyton and Hall Textbook of Medical Physiology, 12th Edition.