

Long-Term BP Control mechanisms

1) Renal-body fluid feedback mechanism/ Direct mechanism

- corrects BP by causing appropriate changes in blood volume through diuresis and natriuresis

As arterial pressure increases from 60 mm Hg to 200 mm Hg
→ progressive increase in water excretion (pressure diuresis) and Na excretion (pressure natriuresis) in urine

when BP rises too high



Pressure natriuresis and diuresis



ECF volume and blood volume ↓



BP returns to normal

when BP falls too low



Reduce the rate of Na & water excretion



increase salt & water intake over a period of days



increases the blood volume



BP returns to normal level

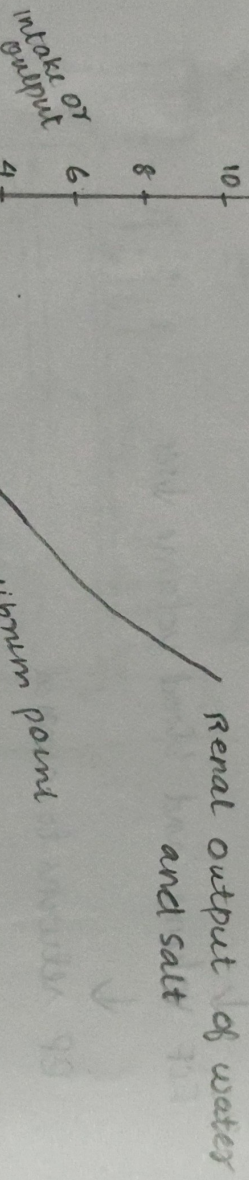
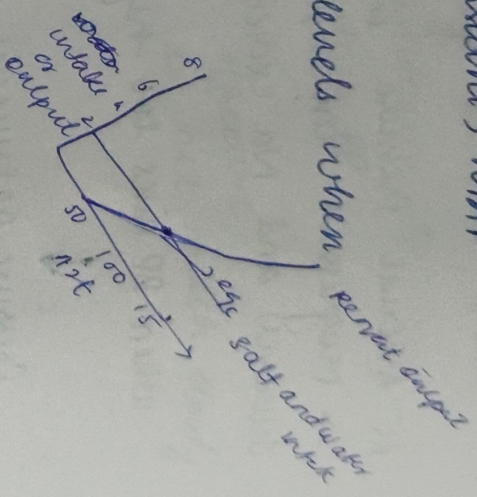
• 2 factors that determine the long-term control of BP by this mechanism are

- Renal output curve
- level of salt & water intake

as long as these 2 factors remain constant, MAP will also remain at a normal level

Arterial pressure deviates from normal levels when one of these factors is altered

Renal output curve



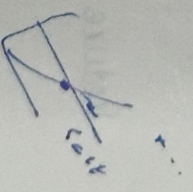
2) Renin-Angiotensin-Aldosterone system (RAAS)

Renin

A protein enzyme released by kidneys when arterial pressure falls too low

Synthesised and stored in an inactive form (Prorenin) in juxtaglomerular cells (JG)

when BP falls → intrinsic reactions in kidneys cause conversion of prorenin to renin



• Renin acts on renin substrate (Angiotensinogen) to release angiotensin-1 (narrow vasoconstrictor)

Angiotensin-1 is converted to angiotensin-II by ACE (Angiotensin converting enzyme) present in endothelium of lung vessels

Angiotensin-II is a powerful vasoconstrictor

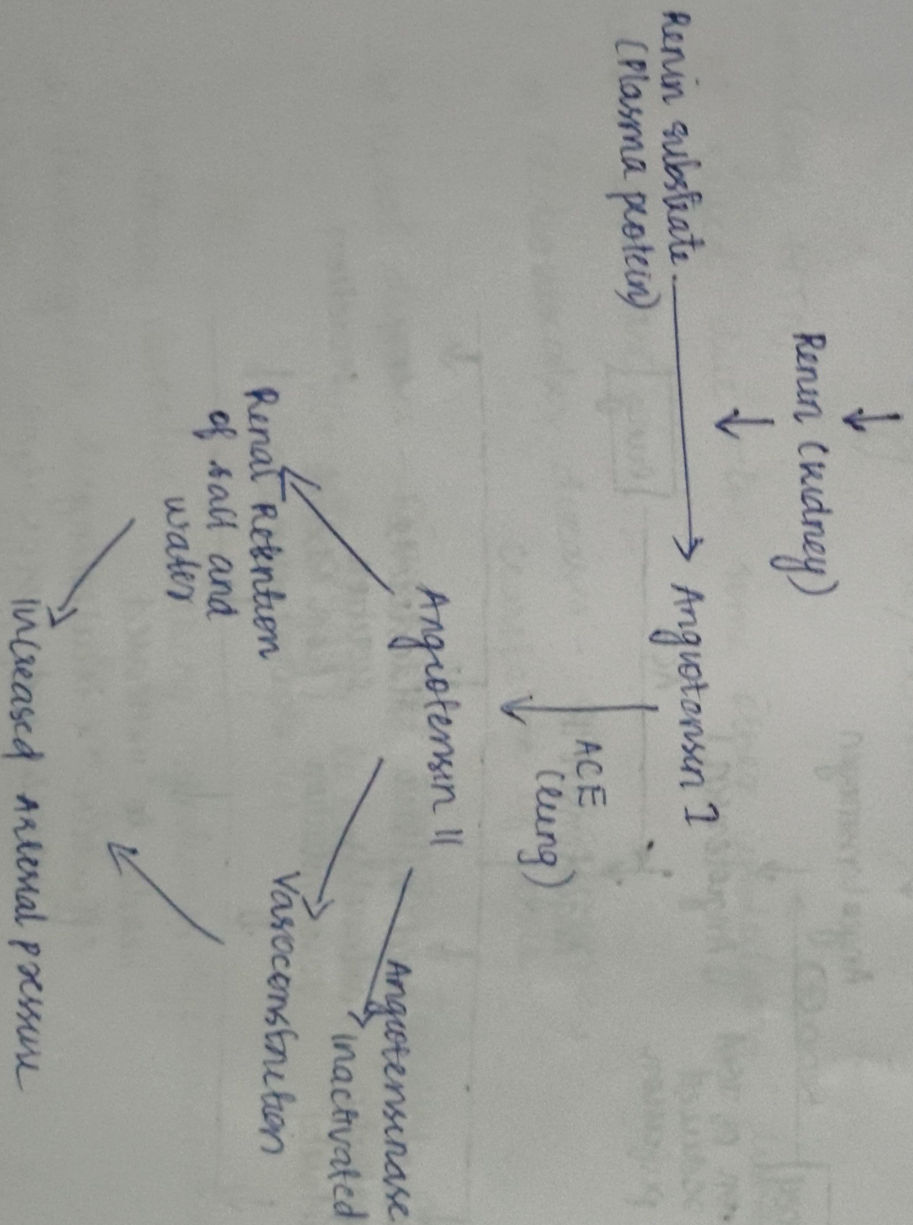
⇒ Angiotensin-II produces 2 effects:

a) Vasoconstriction occurs intensely in arterioles and much less in the veins → increase PR → increase BP

Mild constriction of veins → $\uparrow$ VR $\rightarrow$ $\uparrow$ CO

b) Decrease excretion of salt and water by kidneys → $\uparrow$ ECF volume $\rightarrow$ $\uparrow$ BP Actual pressure

Decreased arterial pressure



Angiotensin causes kidneys to retain both salt & water in 2 ways

• Acts directly on kidneys to cause salt-water retention

• causes adrenal glands to secrete aldosterone which retains salt & water reabsorption

↓
increases ECF volume

↓
increases the arterial pressure.

RAAS

