Decreased arterial pressure

↓

Detected in juxtaglomerular apparatus

↓

Release of rennin

↓

Coverts angiotensinogen to angiotensin I

↓

ACE converts to angiotensin II

↓ ↓

Acts directly on kidneys

Salt and water retention

Secondary to renal vasoconstriction, slower blood flow in peritubular capillaries, leading to increased resorption

Also weak direct effect on tubular cells

Acts on adrenal glands

Secretion of aldosterone

Increased Na absorption and K excretion and small amount H

CONN'S SYNDROME

Primary hyperaldosteronism

Usual findings – hypertension, hypokalaemia, hypomagnesaemia, metabolic alkalosis, NO OEDEMA

Thought to be implicated in 1-3% of hypertensives and may persist after surgical treatment if long standing.

Symptoms – none, weakness, muscle cramps, parasthesiae, polyuria, polydipsia

Causes – aldosterone secreting adenoma, idiopathic adrenal hyperplasia, unilateral hyperplasia

Investigations – rennin:aldosterone ratio (off antihypertensives), CT/MRI but often may not localise tumour

Treatment – surgical removal, spironolactone, amiloride

CAUSES OF HYPOKALAEMIA

Can lead to muscle weakness and direct tubular cell injury
Chronic low k is a cause of interstitial nephritis

With hypertension (usually have k excretion of >30mmol/day)

High plasma rennin	Low plasma rennin
Renovascular disease	Primary hyperaldosteronism
Accelerated phase ^bp	Carbenoxalone
Cushing's syndrome	Liquorice excess
Rennin-secreting tumour	11-ß-hydroxy steroid dehydrogenase deficiency
	Liddle's syndrome
	Glucocorticoid suppressible hyperaldosteronism

Without hypertension (usually high plasma activity)

Diuretic usage (urinary excretion may be high or low)
GI loss
Salt wasting CRF
Bartter's syndrome
Gitelman syndrome
Secondary hyperaldosteronism

The enzyme is also inhibited (or, rather, saturated) by very high plasma concentrations of cortisol, such as occur in patients with the ectopic ACTH syndrome (see Chapter 12.7.1). These patients develop the clinical and biochemical features of Conn's syndrome before (or without) becoming floridly cushingoid. Indeed, the lowest levels of plasma K+ (less than 2.5 mmol/litre) in the presence of plasma Na+ over 145 mmol/litre should suggest the diagnosis of ectopic ACTH rather than primary hyperaldosteronism.