Aldosterone

Description	Aldosterone is secreted by the zona glomerulosa of the adrenal cortex and stimulates sodium transport across cell membranes in the distal renal tubules. Aldosterone plays a major role in the homeostasis of sodium and potassium and maintenance of arterial blood pressure in the sodium-depleted state. The renin-angiotensin system regulates aldosterone secretion. Low sodium concentrations or low blood volume causes release of renin from kidney cells, which mediates release of aldosterone.
Indication	Suspicion of primary hyperaldosteronism due to aldosterone-secreting adenomas (Conn's Syndrome). To differentiate between adenomas and hyperplasia as the cause of primary hyperaldosteronism. Diagnosis of hyporeninaemic hypoaldosteronism
Additional Info	Pharmacological interference may be observed in women taking drospirenone, a synthetic progestin with anti-mineralocorticoid activity and aldosterone-receptor antagonism. Angiotensin, oestrogens, laxatives, oral contraceptives, sodium restriction, and thiazide diuretics lead to elevated aldosterone concentration. Aminoglutethimide, ACE inhibitors such as Lisinopril and captopril, deoxycorticosterone, prolonged heparin therapy, and saline decrease aldosterone concentration.
Concurrent Tests	Renin Aldosterone-renin ratio Sampling of adrenal vein aldosterone and cortisol can be used to differentiate between adenomas and hyperplasia as cause of hyperaldosteronism
Dietary Requirements	Salt restriction may be required in some cases
Interpretation	Normal value ranges differ among different laboratories using different assays. Elevated concentrations of aldosterone are observed in primary aldosteronism due to aldosterone secreting adenomas (Conn's Syndrome). Primary aldosteronism is characterised by suppressed renin activity and cannot be stimulated by either sodium restriction or treatment with a diuretic, or by demonstrating lack of suppression of aldosterone following saline infusion or administration of a mineralocorticoid. The aldosterone- renin ratio is useful in the investigation of primary aldosteronism and in the interpretation of aldosterone results.

	<table><tr><th>ARR</th><th>Aldosterone (pmol/L)</th><th>Interpretation</th></tr><tr><td><30</td><td></td><td>Primary hyperaldosteronism unlikely</td></tr><tr><td>≥30</td><td><250</td><td>Primary hyperaldosteronism unlikely (aldosterone typically >400)</td></tr><tr><td>≥30</td><td>250 – 399</td><td>Primary hyperaldosteronism not excluded (suggest confirmatory testing</td></tr><tr><td>≥30</td><td>≥400</td><td>Consistent with hyperaldosteronism (suggest confirmatory testing)</td></tr></table> Aldosterone <140 pmol/L post- salt loading suggests primary hyperaldosteronism unlikely. Pseudo- primary aldosteronism is due to bilateral adrenal hyperplasia. Sampling of adrenal vein aldosterone and cortisol can be used to differentiate between adenomas and hyperplasia as the cause of hyperaldosteronism. In hyperplasia, both adrenals secrete high levels of aldosterone. Secondary hyperaldosteronism may be observed in laxative abuse, cardiac failure, diuretic abuse, and Bartter's Syndrome (a rare inherited defect in the thick ascending limb of the loop of Henle). Primary hypoaldosteronism is characterised by an undetectable aldosterone with markedly raised renin. Causes include Addison's disease and congenital adrenal hyperplasia. Secondary (hyporeninaemic) hypoaldosteronism is characterised by low/undetectable renin and aldosterone. Causes include diabetic nephropathy, ACE inhibitors, NSAIDs and ciclosporin. See also laboratory handbook entry for renin.
Collection Conditions	EDTA plasma sample
Frequency of testing	As required