Renin				
Description	Renin is a hormone secreted by the juxtaglomerular epithelial cells of the kidney is response to a fall in afferent blood pressure or decrease in sodium concentration. Renin converts angiotensinogen to angiotensin I, which is subsequently converted to angiotensin II by angiotensin converting enzyme (ACE). Angiotensin II is a potent vasoconstrictor and also stimulates secretion of aldosterone and anti-diuretic hormone. Aldosterone increases sodium reabsorption in exchange for potassium in the distal renal tubules. Thus, the reninangiotensin-aldosterone axis plays a major role in sodium and potassium homeostasis.			
Indication	Diagnosis of primary hyperaldosteronism Diagnosis of hyporeninaemic hypoaldosteronism Monitoring of mineralocorticoid replacement therapy.			
Additional Info	Renin is increased by spironolactone (cease therapy for 6 weeks before testing) and suppressed by B-blockers (cease therapy for 1 week before testing)			
Concurrent Tests	Aldosterone Aldosterone-renin ratio			
Dietary Requirements	Hypokalaemia and hyponatraemia should be corrected before testing.			
Interpretation	Normal value ranges differ among different laboratories using different assays. The assay in use at RLBUHT is for direct renin.    Direct renin (mlU/L)¹     Supine   4.2 – 60     Upright   5.3 – 99    Primary hyperaldosteronism is characterized by suppressed renin that cannot be stimulated by either sodium restriction or treatment with a diuretic, and a raised aldosterone that cannot be suppressed by saline infusion or administration of a mineralocorticoid. A paradoxical decrease in aldosterone on standing has also been reported. Causes include hyperplasia, benign adenoma and carcinoma. The aldosterone- renin ratio is useful in the investigation of primary hyperaldosteronism.			

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	ARR	Aldosterone (pmol/L)	Interpretation	
	<30	(	Primary	
			hyperaldosteronism	
			unlikely	
	≥30	<250	Primary	
			hyperaldosteronism	
			unlikely	
			(aldosterone	
		0.50	typically >400)	
	≥30	250 – 399	Primary	
			hyperaldosteronism not excluded	
			(suggest confirmatory testing	
	≥30	≥400	Consistent with	
			hyperaldosteronism	
			(suggest	
			confirmatory	
			testing)	
	Secondary hyperaldosteronism is characterised by elevated aldosterone and normal or raised renin. Causes include cardiac failure, diuretics, laxative 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 aldosterone.			
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Collection Conditions	EDTA plasma sample. If from a referral laboratory, please separate and send plasma frozen.			
Frequency of testing	As required			

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