

Q21 Outline the functional anatomy, and the physiological factors, that determine oxygen delivery to the renal medulla (Sept 2011)

FUNCTIONAL ANATOMY

- The kidneys are unusual in that they contain two capillary beds, the glomerular and peritubular beds
- The renal artery enters the kidney through the hilum and then branches progressively to form the interlobar arteries, arcuate arteries, and afferent arterioles, which lead into the glomerular capsule
- Most efferent arterioles leave the glomerulus and form the peritubular capillaries which supply the renal tubules
- The efferent arterioles situated just above the corticomedullary border descend down into the medulla, dividing many times to form bundles of parallel vessels called vasa recta. Only the centermost vasa recta descend all the way to the papilla. They carry postglomerular blood, which contains less serum and is more viscous. They then reform into ascending vasa recta which eventually rejoin to form veins by which deoxygenated blood leaves the kidney.
- Blood flow to the renal medulla is far less than cortical blood flow, ~ 100ml/min, however this is not low in an absolute sense.

RENAL BLOOD FLOW

Renal blood flow = 1100ml/min (20-25% of cardiac output)

Arterial oxygen content = $(1.34 \times \text{hemoglobin concentration} \times \text{SaO}_2) + (0.0031 \times \text{PaO}_2)$

Oxygen delivery DO_2 (ml/min) = cardiac output (Q) x arterial oxygen content (CaO_2)

Hence oxygen delivery to the kidney will vary depending on cardiac output (which is dependent on heart rate, preload, afterload and contractility), haemoglobin concentration, and respiratory factors that affect the PaO_2 and SpO_2 (such as FiO_2 and atmospheric pressure).

FACTORS AFFECTING RENAL BLOOD FLOW –

- INTRINSIC
 - Autoregulation – the kidney performs autoregulation (ie, maintains a constant blood flow over a range of perfusing pressures) by two mechanisms
 - Pressure autoregulation – myogenic stretch response of the afferent arteriole in response to increases in perfusion pressure will alter the amount of blood reaching the medulla
 - Metabolic autoregulation – actions of locally derived metabolites will also alter vascular tone
 - RAAS system – release of renin stimulated by sympathetic nerve activity and tuboglomerular feedback catalyses the production of angiotensin II, which causes constriction of the afferent and efferent arterioles (thus reducing medullary blood supply)
 - Other vasoactive mediators
 - Endothelin – causes constriction of the afferent arteriole and reduction in medullary blood flow
 - Adenosine – released in response to tuboglomerular feedback, causing constriction of the afferent arteriole
 - Prostaglandins – increase blood flow in the renal cortex, decrease it in the medulla
 - Nitric oxide – dilates the afferent arteriole and increases renal blood flow
 - Dopamine – reduces the intrarenal expression of ATII receptors thus reducing its effects
 - Bradykinin – increases medullary blood flow
 - Acetylcholine – increases medullary blood flow
- SYSTEMIC
 - Sympathetic stimulation – causes constriction of afferent arteriole and reduction in medullary flow
 - High protein meal – raises glomerular capillary pressure and increases renal blood flow
 - BGL – increases renal blood flow