Q11 Compare and contrast renal and hepatic blood flow and its regulation (March 2012)

	RENAL	**HEPATIC**
Blood flow	1100ml/min, 20% CO	1500ml/min, 25% of CO
Oxygen consumption per 100g per min	6ml O2/min/100g of tissue	2ml O2/min/100g of tissue
Total oxygen consumption by weight in average adult	18ml/minute	50ml/minute
Main anatomical aspects	- one renal artery supplies each kidney, one renal vein drains each kidney into the IVC - 95% of flow goes to the renal cortex, 5% to medulla - two capillary beds: glomerular and peritubular	- 1/3 of blood supply is oxygenated blood from hepatic artery, 2/3 from portal vein - Branches of hepatic artery and hepatic vein travel together with branches of the bile duct in 'hepatic triads' with the arteriole and venule eventually joining to form a sinusoid, the specialized capillary system designed to optimize exchange with hepatocytes - venous blood drains via hepatic veins to IVC
Main function	Glomerular filtration	Metabolic activity
Demonstrates autoregulation?	Yes – to maintain strict MAP 75-170mHg	Yes but less than renal
Regulatory mechanisms	INTRINSIC - Autoregulation – the kidney maintains a constant blood flow over a range of perfusion pressures by two mechanisms: the myogenic stretch response of the afferent arteriole in response to increases in perfusion pressure will alter the amount of blood reaching the medulla, and metabolic autoregulation via locally derived metabolites - 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 hormones – endothelin, adenosine, nitric oxide, dopamine, bradykinin and Ach EXTRINSIC - 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	INTRINSIC → - Hepatic artery demonstrates some autoregulation, portal vein does not - There is a semi reciprocal relationship – hepatic artery resistance varies due to portal flow; this helps to maintain flow with portal vein fluctuations but not with decreases in hepatic artery flow EXTRINSIC → - Neural control via adrenergic system → hepatic artery has alpha and beta receptors, portal vein only alpha receptors - Hormonal control →glucagon, VIP, secretin all dilate vasculature and increase flow (mainly in the hepatic artery); ATII, vasopressin both constrict portal vein and hepatic artery flow - Feeding → increases GIT blood flow and indirectly increases hepatic flow - Exercise → reduces GIT blood flow and thus hepatic flow - PPV → Decreases blood flow secondary to decreased cardiac output