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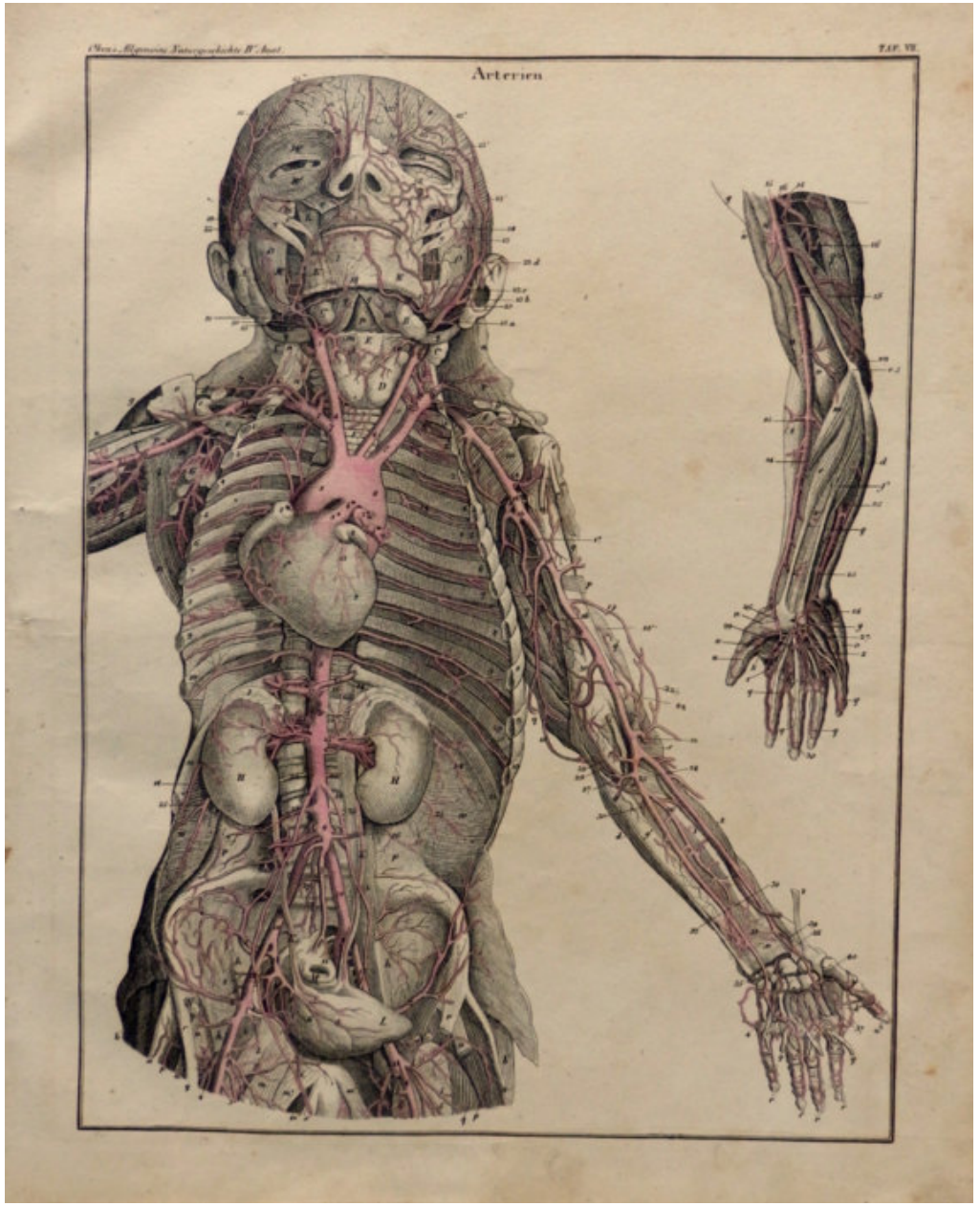


ABO Blood Groups 2012-2

Question 5 LOA: 2	5.1 Describe the ABO blood types and their inheritance.	Inheritance – Mendelian **co-dominance of A and B antigens**. Complex oligosaccharides differing in terminal sugar. A and B phenotypes may be homozygous (AA, BB) or heterozygous (AO, BO) genotypes. **O – no antigens (universal donor), anti-A and anti-B antibodies** **A – anti-B** **B – anti-A** **AB - both antigens, no antibodies (universal recipient)** Most individuals have H antigen (terminal fucose coded by H gene) A – N-acetylgalactosamine added on the H antigen B – terminal glactose added. Similar antigens common in intestinal bacteria and possibly foods so rapidly develop antibodies to those not represent in own cells.	Understand co-dominance + Bold
	5.2 Why is Group O blood is used as a 'universal donor'? Additional if quick: i)How does the Rh system differ.	**O – no antigens (universal donor), anti-A and anti-B antibodies** Rh (first described Rhesus monkeys) C, D, E and others but only D clinically important (most antigenic). Rh only present on red cells so need exposure to Rh ve blood to develop antibodies. Occurs during transfusion and mixing at child birth or other bleeding (50% sensitized by transfusion) Antibodies take time to develop so first baby OK, second Rh +ve preg carries risk of HDNB (17% of 2nd Rh+ve preg if not treated). Rh antibodies IgG cross placenta. ABO IgM and don't.	Bold - absence of antibodies in O minimizing transfusion reactions.

Arteriolar Tone 1 2003-2

TOPIC: Neural factors affecting arteriolar tone _____ **NUMBER:** _____ 2a

OPENING QUESTION	Discuss the central neural control affecting arteriolar tone	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	1. Presence of a vasomotor centre situated in the CNS medulla with both vasoconstrictor and vasodilatory areas	1	3 of 5 to pass
	2. Medullary vasomotor centre is influenced by peripheral baroreceptors, peripheral chemoreceptors and higher neural centres	2	
	3. Noradrenergic vasoconstrictor fibres descend from medullary vasomotor centre via spinal cord to the smooth muscle in the walls of arterioles	3	
	4. Peripheral baroreceptors in carotid sinus and aortic arch respond largely to changing blood pressure and act to inhibit vasoconstrictor centre	4 what are the inputs?	
	5. Peripheral chemoreceptors in carotid bodies and aortic bodies respond to hypoxia and act to excite the vasoconstrictor centre	5	
SECOND QUESTION (if needed)	Describe the Volume (atrial stretch) reflex		
POINTS REQUIRED	1. Atrial stretch results in reflex afferent arteriolar renal dilatation		
THIRD QUESTION (if needed)			
POINTS REQUIRED			

Arteriolar Tone 2 2003-2

TOPIC: Local factors affecting arteriolar tone _____ **NUMBER:** _____ 2c

OPENING QUESTION	Discuss the local factors that affect arteriolar tone	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	Arteriolar tone changes to regulate local blood flow across a range of blood pressures. Two theories by which this occurs: myogenic or metabolic.	1	2 theories to pass
	Myogenic theory – distension of vessel with increasing pressure stretches the vascular smooth muscle leading to contraction of the muscle	2	
	Metabolic theory – vasodilator metabolites accumulate in tissues when blood flow falls leading to relaxation of vascular smooth muscle.	3	
	Vasodilators include local hypoxia and acidosis, CO2 build up, heat, potassium, lactate, histamine, adenosine	4	
	Serotonin causes localised vasoconstriction after vessel injury.	5	
	Prostacyclin (vasodilatation) and thromboxane (vasoconstriction) after local vessel injury		
	Endothelium Derived Relaxing Factor (nitric oxide) Many vasodilators act by activating EDRF		
	Endothelin - vasoconstrictor		

Arteriolar Tone 3 2003-2

TOPIC: Hormonal factors affecting arteriolar tone_____ **NUMBER:** _____ 2b

OPENING QUESTION	Discuss the hormones that influence arteriolar tone	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	1. Adrenaline is released from the adrenal medulla in response to sympathetic stimulation. It acts via alpha-1 receptors to constrict arterioles in most areas. It also acts via Beta 2 receptors to vasodilate muscle and liver blood vessels.	1	
	2. Noradrenaline although largely a neurotransmitter, is released from the adrenal medulla in response to sympathetic stimulation. It acts via alpha 1 receptors to constrict arterioles.	2	
	3. Angiotensin II is a generalised arteriolar constrictor. It is formed from angiotensin I in the lung.	3	1,2 and 2 others to pass
	4. Vasopressin is a potent arteriolar constrictor. It is released from the posterior pituitary.	4	
	5. Bradykinin is a tissue hormone that causes arteriolar dilatation.	5	
	6. Histamine is produced by basophils and mast cells and causes arteriolar dilation.		
	7. Serotonin ??		
	8. Adrenomedullin ??		

Autoregulation 2016-1-B

Stem: Moving onto Physiology. Despite blood loss, he maintains adequate perfusion.			
Question 3 General principles of autoregulation (local, circulating, myogenic, neurological). LOA: 1	*a. What is autoregulation of tissue blood flow? Prompt: "What are the main features of autoregulation?"*	Capacity of tissues to regulate their own blood flow, which **remains relatively constant** despite moderate **changes in perfusion pressure.** This is achieved by **altering vascular resistance.**	Bold concepts to pass
	b. What are the proposed mechanisms involved in autoregulation?	**Myogenic:** Intrinsic contractile response of smooth muscle to stretch. As pressure rises, vascular smooth muscles surrounding the vessels contract to maintain wall tension (La Place Law, T = P x r). **Metabolic:** Production of vasodilator metabolites by active tissues → vessel vasodilation → ↑flow **Endothelial products** : vasoconstrictors (endothelin, thromboxane A2) and vasodilators (nitric oxide, prostacyclin). **Circulating neurohumoral substances**: vasoconstrictors (adrenaline, noradrenaline, vasopressin, angiotensin II) and vasodilators (kinins, VIP, ANP). **Neural :** Sympathetic (α-adrenergic receptors- vasoconstriction, β-adrenergic receptors – vasodilation) & parasympathetic (muscarinic receptors – vasodilation).	3 bold to pass with explanation
	*c. What are some **local** factors that lead to vasodilation?*	Hypoxia, hypercarbia, increased local temperature, hyperkalaemia, adenosine, acidosis, lactate, prostaglandins, histamine.	4 to pass

Autoregulation 2013-1

Question 1: LOCAL FLOW REGULATION LOA: 2	a. Describe the autoregulation of tissue blood flow. Prompt: what are the main features of autoregulation	Capacity of tissues to regulate their own blood flow Tissue blood **flow remains relatively constant** despite moderate **changes in perfusion pressure** through alterations in **vascular resistance.**	Three main features to pass
	b. How would this apply to autoregulation of cerebral blood flow?	**Constant flow over arterial pressure range 65-140 mmHg.** Sympathetic stimulation prolongs the plateau.	Bold including approximate range
	c. What are the proposed mechanisms involved in autoregulation? Prompt: What are some important metabolic changes that cause vasodilatation	**Myogenic**: **intrinsic contractile response of smooth muscle to stretch.** **Metabolic**: **production of vasodilator metabolites by active tissue**. Accumulation assoc. with decreased flow leads to vasodilation. Examples dec pO2, acidosis, high K, lactate, pCO2(brain and skin), local temp, adenosine (heart)	Both mechanisms & 2/5 metabolites

Autoregulation 2012-1

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 LOA: 1	1.1 What local factors can cause vasoconstriction or vasodilatation?	Vasodilatation: ↑ CO2, ↑ lactate, ↑adenosine, ↑ local temp; ↓ O2 or ↓pH Vasoconstriction: ↓local temp, autoregulation.	At least 4 to pass, and at least one in each group
	1.2 What is autoregulation in relation to blood flow?	Autoregulation: blood flow remains constant by compensating pressure changes with peripheral resistance. 1) **Myogenic**: as blood pressure rises, muscle fibres in the blood vessels contract. The muscles correspond to the wall tension which is maintained at fairly constant level. Wall tension is determined by the radius of the blood vessels (pressure x radius). So rise in pressure, leads to a reduction in the radius of the blood vessel. 2) **Metabolic**: active metabolites cause local vasodilatation.	Need bold & some details to pass.

Autoregulation 2010-1

1. (a)Describe how tissues regulate their own blood flow. Prompt- What are the proposed mechanisms of this process?	Most vascular beds have intrinsic capacity to compensate for moderate changes in perfusion pressure by changing vascular resistance and therefore maintaining constant bld flow. 1. Myogenic theory of autoregulation: Intrinsic contractile response of smooth muscle to stretch As pressure rises: blood vessels are distended → vascular smooth muscle surrounding vessels contract Law of Laplace: maintenance of given wall tension, as pressure rises, requires a decrease in radius 2. Metabolic theory of autoregulation Vasodilator substances tend to accumulate in active tissues when blood flow decreases → dilatation When blood flow increases → washed away Hypox, inc Co2, Inc H+, Inc lactate, inc K+, inc temp, histamine, adenosine	**1. Myogenic theory of autoregulation** **2. Metabolic theory of autoregulation** To pass must demonstrate understanding of both

Autoregulation & Endothelin 2004-2

TOPIC: Endothelium and regulation of blood flow _____ **NUMBER:** _____

OPENING QUESTION	Describe how blood flow is regulated at the level of the endothelium.	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	1 Vasodilators: prostacyclins, **NO**, kinins	1 Tell me about local vasodilators.	
	2 Vasoconstrictors: **endothelin**, **thromboxane**, serotonin	2 Tell me about local vasoconstrictors.	
	3	3	
	4	4	
	5	5	
	6	6	
SECOND QUESTION (if needed)	7 What other general effects do endothelins have on the cardiovascular system?	7	
POINTS REQUIRED	1 Positive inotrope and chronotrope	1 Tell me about the effects on the heart and blood pressure	
	2 Rise in ANP/renin/aldosterone	2 Tell me about the renal effects	
	3 Decreased GFR and renal blood flow	3	
	4	4	
	5	5	
	6	6	

Baroreceptors 2015-2-C

Question 5 Cardiovascular Regulatory Mechanisms **Subject:** Phys LOA: 1	1. What are baroreceptors?	**Stretch** receptors in the adventitia layer of vessels	Bold
	2. Where are they located?	Located at **aortic arch** and **carotid sinus**, walls of right and left atria (SVC and IVC entrances) and pulmonary circulation.	Bold and 1 other
	3. What is their mechanism of action in hypotension?	In response to hypotension, the arterial **baroreceptors are less stimulated** because they are less stretched. Reduced baroreceptor discharge travels via glossopharyngeal and vagus nerves to the medulla resulting in an **overall increase in sympathetic discharge** to increase heart rate and stimulate vasoconstriction and **reduce vagal drive**.	Bold to pass and understand inhibitory concept

Baroreceptors 2014-1-C

Stem: A 60 year old man with a history of atrial fibrillation on warfarin presents to ED following a motor bike accident. His blood pressure on arrival is 80/40

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Baroreceptors **Subject:** Phys LOA: 1	**What are baroreceptors and where are they located?**	**Stretch receptors** Carotid, aortic, cardiopulmonary. In the adventitia of vessels. The **carotid sinus** and **aortic arch** receptors monitor the arterial circulation. Receptors are in the wall of the **right** and **left atria,** at the entrance of SVC and IVC and in the pulmonary veins as well as in **the pulmonary circulation** (collectively the cardiopulmonary receptors).	Bold to pass Carotid and aortic plus one other to pass
	What is their mechanism of action?	Very sensitive to changes in pulse pressure. Exert an **inhibitory** input via the tractus solitarius in the medulla. **Stimulated by distension** of the structures in which they are located, therefore discharge at an increased rate when the pressure in these structures rises. Increased baroreceptor discharge ***inhibits*** **the tonic discharge of sympathetic nerves and *excites* the vagal innervation of the heart.** Result is vasodilatation, venodilation and a fall in BP, bradycardia and decreased cardiac output.	Need mention of inhibitory nature of pathway and nerves affected (vagus, sympathetics)
	What is their action in this setting of acute blood loss?	Decreased blood volume and decreased venous return results in reduced stimulation of arterial baroreceptors and **increased sympathetic output.** The result is **reflex tachycardia and vasoconstriction.**	Bold to pass

Baroreceptors 2014-1-C

Stem: A 90 yo lady arrives by ambulance with confusion and agitation. She is hypotensive. **We will start with Physiology.**			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Baroreceptors / regulation of blood pressure (pp 589-592) **Subject:** Phys LOA: 1	1. What are baroreceptors and where are they located?	1. **Stretch receptors** in the walls (adventitia) of the heart & blood vessels, impt in control of BP (esp short term). Arterial- carotid sinus/Ao arch. Low pressure- Atria at entrance of IVC and SVC, Pulm veins and pulm circulation.	**Bold plus 2 locations.**
	2. What stimulates these receptors?	2. **Distension** of the structures above. More sensitive to pulsatile than constant pressure. Maximal firing at 150mmHg (@ Carotid sinus)	**Bold**
	3. What are their effects?	3. Inhibit tonic sympathetic drive& inc vagal drive=>**vasodilation, venodilation, hypotension, bradycardia (tachycardia in low pressure baroreceptors), ↓CO**. Allows rapid adjustments in BP in response to abrupt changes in posture, blood volume, cardiac output, or peripheral resistance	**3/5 end effects**

Baroreceptors 2008-2

OPENING QUESTION	**Where are Baroreceptors found in the body?**	**COMMENTS**
POINTS REQUIRED	1. **Stretch receptors in adventitia of vessel walls, major ones found in carotid sinus and aortic arch to monitor arterial side of circulation.**	
	2. Also "cardiopulmonary receptors" in right and left atria, and pulmonary circulation to monitor venous circulation	Both carotid sinus and aortic arch to pass
PROMPTS	Which blood vessels contain baroreceptors?	
SECOND QUESTION (if needed)	**What is the effect of vessel wall distension on a baroreceptor?**	
POINTS REQUIRED	**Stretch of vessel wall leads to increased baroreceptor discharge,** transmitted by afferents in glossopharyngeal and vagus nerves to **medulla. (vasomotor centre)** This results in release of inhibitory GABA which reduces sympathetic outflow, and excitatory effects on vagal motor neurones. Net effect is: **1. Inhibition of tonic discharge of vasoconstrictor nerves** **2. Excitation of cardiac vagal innervation** **Results in vasodilation, with decrease in BP, HR and CO.**	Bolded
PROMPTS	How does the baroreceptor respond to an increase in BP?	

Baroreceptors 2006-2

SUBJECT: PHYSIOLOGY

TOPIC: Arterial baroreceptors. Response to change in pressure **NUMBER: 2**
2006.2.2c

OPENING QUESTION	Please describe the arterial baroreceptors?	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	1 Located in the adventitia of the aorta at the apex of the aortic arch.	1 Where are they located	2/3 = pass
	2 Also in the adventitia of the internal carotid artery at the carotid sinus, immediately after the bifurcation from the common carotid.	2	
	3 The baroreceptors are stretch receptors, discharging at an increased rate in response to stretch of the arterial walls.	3	
SECOND QUESTION (if needed)	What is the role of the arterial baroreceptors in regulation of systemic blood pressure?		
POINTS REQUIRED	1 There is a basal activity in the afferent nerves (glossopharyngeal and vagus) from the baroreceptors.	1	
	2 Increased blood pressure stretches the baroreceptors.	2	Required for pass
	3 Stretch causes increased firing in the afferent nerves.	3	Required for pass
	4 The afferent nerves are inhibitory to the vasomotor centre.	4	Required for pass
	5 Inhibition of the vasomotor centre reduces central sympathetic outflow to the cardiovascular system.		
	6 Decreased blood pressure has an opposite effect.		
THIRD QUESTION (if needed)	What is the effect of chronic hypertension on the activity of the arterial baroreceptors?		
POINTS REQUIRED	1 They 'reset' to maintain normal basal activity at the elevated blood pressure.- reversible	1	

Baroreceptors 2004-2

TOPIC: Baroreceptors and arterial blood pressure _____ **NUMBER:** _____

OPENING QUESTION	What changes in arterial blood pressure do baroreceptors respond to?	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	1 Carotid sinus (rise or **fall**)	1 Where?	
	2 Aortic arch (rise)	2	
	3	3	
	4	4	
	5	5	
	6	6	
	7	7	
	8		
SECOND QUESTION (if needed)	What happens when the baroreceptors detect a fall in arterial pressure?		
POINTS REQUIRED	1 Dec firing rate of Hering's nerve	1 Sequence?	
	2 CN IX transmits to vasomotor centre	2	
	3 Dec parasympathetic outflow to heart	3	
	4 Inc sympathetic outflow to heart	4	
	5 Inc sympathetic outflow to vessels	5	
	6 Inc heart rate, contractility	6	
	7 Arteriolar and venous constriction		
THIRD QUESTION (if needed)	What is the Set Point?		
POINTS REQUIRED	1 Neutral MAP for vasomotor centre Around 100 mm Hg	1	
	2	2	
	3	3	
	4	4	

Blood Flow 2011-2

Question 1 LOA: 1	Describe the factors that control blood flow to the myocardium	**a. Local factors control radius of blood vessels (overall flow and regional flow)** Hypoxia – vasodilatation (? mediated via adenosine) Hypoxia is the main factor controlling flow (NB , extraction ratio of about 70%) NO2 – vasodilatation Local metabolites e.g. -K+, adenosine,CO2, lactate, PG **b. Neurogenic factors controlling radius of blood vessels (overall flow and regional flow)** Parasympathetic nerves Sympathetic nerves α – vasoconstriction, β vasodilatation Circulating catecholamine's **c. Pressure differences** Flow is dependant gradient between arteries and veins OR external compression from muscles. During systole ventricular muscle pressure limits flow especially to subendocardium of the left ventricle d.Viscosity of the blood	Need bold and 1 example of each to pass

Blood Pressure 2012-2

TOPIC	QUESTIONS	KNOWLEDGE (essential in bold)	NOTES
Question 1 Control of Blood Pressure LOA: 1	1.1 How is blood pressure maintained in the setting of acute blood loss?	1. <u>seconds/minutes</u> - **baroreceptors** (increased discharge with stretch, afferent nerve fibres pass to vasomotor area of medulla which in turn inhibits tonic discharge of vasoconstrictor nerves leading to drop in BP) - **chemoreceptors** (stimulation leads to peripheral vasoconstriction and rise in BP) - CNS ischaemic receptors 2. <u>minutes/hours</u> - **renin-anglotensin system** - blood volume changes - fluid shift through capillaries 3. <u>Longer term</u> - renal compensation via aldosterone - blood volume changes - salt intake	**Bold to pass + must understand baroreceptors**
	1.2 What other factors influence the vasomotor centre?	**Direct stimulation** - CO_2 , hypoxia **Excitatory inputs** - from cortex via hypothalamus - from pain pathways and muscles - chemoreceptors (carotid & aortic) **Inhibitory inputs** - from cortex via hypothalamus - from lungs - from baroreceptors	**Must get 2 of 3 bold**
Question 2	Please draw and label a	▪ Tidal volume 500 mL	**The candidate must be able to label**

Blood Pressure 2010-1

1. a) Please draw a diagram of the changes in systolic and diastolic pressure as blood flows through the systemic circulation.

-suggest: pressure on y-axis, and label the various parts of the systemic circulation on the x-axis.

1 b) How does the total cross-sectional area of vessels change through the systemic circulation?

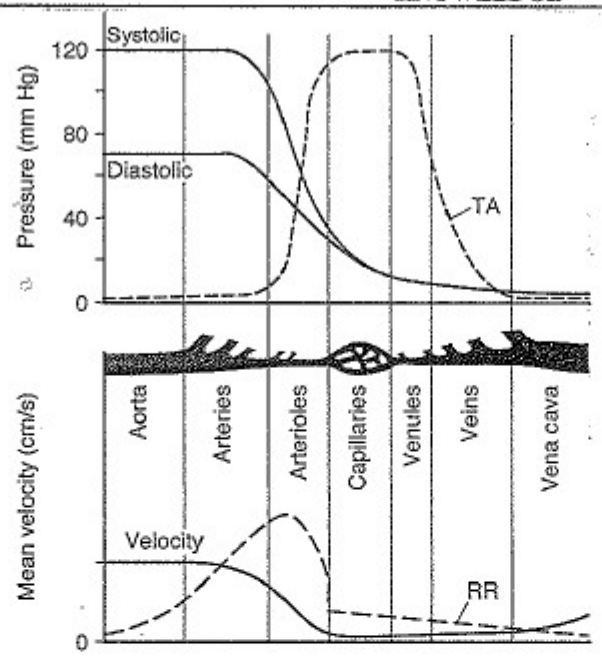


FIGURE 32–27 **Diagram of the changes in pressure and velocity as blood flows through the systemic circulation.** TA, total cross-sectional area of the vessels, which increases from 4.5 cm^2 in the aorta to 4500 cm^2 in the capillaries (Table 32–9). RR, relative resistance, which is highest in the arterioles.

w of Medical

TA = total cross-sectional area of vessels
RR = relative resistance (highest in arterioles)

- pressure falls very slightly in large and medium-sized arteries because resistance to flow is small
- pressure falls rapidly in small arteries and arterioles, which are main sites of peripheral resistance against which heart pumps
- magnitude of pressure drop along arterioles varies depending on whether constricted or dilated
  1. small pressure change in large and medium-sized arteries
  2. rapid fall in pressure in small arteries and arterioles
  3. **mean** pressure at end of arterioles is 30-38mmHg
  4. **Pulse pressure** 5mmHg at ends of arterioles

TA – maximal in capillaries and venules (about 10 x that in arterioles)

Need to pass:

1. Correct shape of pressure diagram
2. Rapid fall in pressure in arterioles
3. TA is maximal in capillaries, and venules

Blood Pressure 2007-1

TOPIC: Arterial pressure regulation _____ **NUMBER:** _____

OPENING QUESTION	What are the major factors affecting the regulation of arterial pressure?	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	1 Seconds/minutes: baroreceptors, chemoreceptors, CNS ischaemic receptors	What systems enable responses to changes in arterial pressure for example with acute haemorrhage? Describe the baroreceptor reflex.	Must describe baroreceptors and angiotensin to pass.
	2 Minutes/hours: stress (stretch) relaxation, renin-angiotensin vasoconstriction, blood volume change and fluid shift through capillaries		
	3 Longer term: renal compensation via aldosterone, blood volume changes, salt intake	What happens in the longer-term?	

Cardiac Output 2012-1

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1: LOA: 1	1.1 Describe the factors affecting Cardiac Output	**CO=SVxHR** SV related to contractility, preload and afterload, HR controlled by intrinsic rate, autonomic, exogenous factors, heat, thyroid	Bold to pass + 2 mechanisms from each SV and HR
	1.2 What are the physiological responses to moderate blood loss?	↓venous return, stimulation of **baroreceptors**, inc catecholmine release, ↓ renal blood flow – activation **of renin angiotensin system** fluid shifts, hepatic synthesis of proteins, inc RBC production	Bold to pass

Cardiac Output During Exercise 2012-1

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1	1.1 Describe what happens to Cardiac Output during exercise. *Prompt: By what mechanisms?*	Increases (CO= SV x HR) ↑venous return and hence ↑end diastolic volume, ↑myocardial contractility, so ↑stroke volume. ↑ sympathetic drive and heart rate	**Increases + one mechanism stroke vol + one mechanism heart rate**
	1.2 What are the local mechanisms that maintain a high blood flow in exercising muscles?	↓in tissue PO_2, ↑tissue PCO_2, and accumulation of K^+ and other vasodilator metabolites, ↑ temperature in active muscle	**Need 3 to pass.**

Central Venous Pressure 2003-2

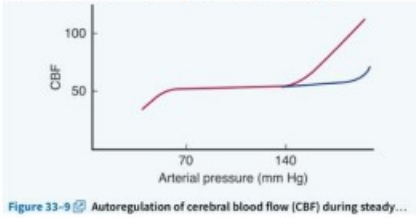
TOPIC: Factors Affecting CVP _____ **NUMBER:** _____ 4c

OPENING QUESTION	What is the normal Central Venous pressure at rest	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	1 = Pressure in Right Atrium = 0 (range –5 to +5)	1	Must pass
		2	
SECOND QUESTION	Describe the factors that determine Central Venous Pressure		
POINTS REQUIRED	1 Balance between venous return, and ability of heart to pump out of RA	1	Must pass
	2 Factors affecting venous return: Gravity, intraabdominal pressure (eg pregnancy), hypo/hypervolaemia, venodilation (drugs/fainting), sympathetic tone (venoconstriction), arteriodilation (sepsis, drugs, anaphylaxis), resistance to venous return (tamponade, tumour)	2	2 examples to pass
	3 Factors affecting ability of heart to pump blood: Myocardial contractility, Hypertrophy (Athlete) Cardiac Failure, Myocardial Infarction (RV), Arrhythmias, Atrial Fibrillation (Volume & filling time, and contractility), Resistance to RV = Pulm valve stenosis, PE, LVF, Hypoxia, tension PTx	3	2 examples to pass

Cerebral autoregulation 2015-1-D

Stem: Her GCS has fallen to 8. We will now move onto Physiology			
Question 3 CNS Autoregulation / Cushing response Subject: Phys LOA: 1	1. What factors affect cerebral blood flow?	1. MAP at brain level MVP at brain level ICP Viscosity of the blood Local constriction/dilatation of cerebral arterioles	Pass in bold **3/5**
	2. What is the mechanism of the Cushing response?	2. Increase in ICP results in Decr CBF – ischaemia of VMA – SNS output incr - Incr systemic BP – stimulation of baroreceptors – stimulation of vagal outflow – decr HR and RR	**Explains concept**
	3. What is the Monro-Kellie doctrine?	3. The volume of blood (75mL), CSF (75mL) and brain (1400g) in cranium must be relatively constant. Negative effects on these therefore if additional intracranial volume eg SDH / EDH occurs	**Explains concept**

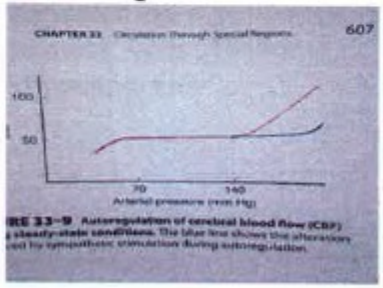
Cerebral Blood Flow 2017-2-C

Stem: A 75-year-old man presents following a collapse. His GCS is 6/15. We will start with Physiology.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Cerebral blood flow **Subject:** Physiology LOA: 1 *Ganong,25th edition. Chapter 33 Circulation through special regions. Cerebral blood flow and its regulation.*	(a) What factors affect cerebral blood flow?	1. **Intracranial pressure** 2. **Mean arterial pressure** 3. **Mean venous pressure** at brain level 4. Local factors: pH, pCO2 – constriction and dilatation of cerebral arterioles 5. Blood viscosity	**2 of 3 Bold**
	(b) What is meant by the term autoregulation of cerebral blood flow? (You may draw a diagram).	The process by which CBF is maintained at a constant level (approx. 750ml/min) despite variation of arterial pressure (MAP 65 – 140mmHg)	Understanding of concept.  Figure 33-9 Autoregulation of cerebral blood flow (CBF) during steady...
	(c) What is the Monro-Kellie doctrine? *(prompt: what is the relationship between volume of blood, volume of CSF, and brain tissue)*	**Volume** of blood, CSF and brain tissue **must be relatively constant.** When ICP rises, cerebral vessels are compressed resulting in reduced cerebral blood flow. Rise in venous pressure also causes decreased cerebral blood flow by decreasing effective perfusion pressure and compressing cerebral vessels.	Understanding of concept.

Cerebral Blood Flow 2015-2-D

Stem: Moving onto Physiology. He is becoming progressively hypertensive and bradycardic.			
Question 3 Cerebral Circulation **Subject:** Phys LOA: 1	What are the factors that determine cerebral blood flow?	**Intracranial pressure** **Mean arterial pressure** **Mean venous pressure** at brain level Blood viscosity Local constriction/dilation of arterioles	**Bold** and 1 other to pass
	Describe the autoregulation of cerebral blood flow (Prompt: what happens to cerebral blood flow when blood pressure changes?)	**Maintains CBF at constant rate** (~750ml/min) **across a range of perfusion pressures** (MAP 65-140mmHg)	**Bold** to pass
	The patient's bradycardia and hypertension is caused by the head injury. Describe the mechanism responsible.	Cushing reflex – **increased ICP compromises blood flow to medulla** → **sympathetic outflow** from vasomotor centre → increases BP in attempt to restore medullary flow → **stretch of baroreceptors** → vagal stimulation→ bradycardia	**Bold** to pass Vagal stimulation OK instead of stretched baroreceptors

Cerebral Blood Flow 2014-1-C

Stem: A 30 year old woman who is 35 weeks gestation presents with a severe headache and a BP of 160/100. We will begin with physiology.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Autoregulation of cerebral circulation **Subject:** Phys LOA: 1	1.1 What factors affect cerebral blood flow?	1.1 • **Intracranial pressure** • **Mean arterial pressure** • Mean venous pressure • Local factors: pH, pCO_2, cause constriction and dilatation of cerebral arterioles • Blood viscosity	1.1 Bold +1
	1.2 Describe autoregulation of cerebral blood flow. You can draw a diagram if you wish.	1.2 The process by which **CBF is maintained at a constant level despite variation in perfusion pressure.** Average CBF is 54 ml/100g/min between MAP 65- 140 mmHg	Able to draw a plateau region with a range for MAP of 50 – 150 mm Hg. 
	1.3 What is the Monroe-Kellie doctrine? (optional if run out of time)	1.3 Due to the fact that brain tissue and spinal fluid are essentially incompressible, **the volume of blood, spinal fluid and brain tissue must be relatively constant.** So when ICP rises, the cerebral vessels are compressed resulting in reduced cerebral blood flow (CBF)	Need to pass 2/3 part to pass.

Cerebral Blood Flow 2011-2

Question 1: LOA: 1	1)What factors determine cerebral blood flow? 2)What is the Monro-Kellie Doctrine?	1) **Intracranial Pressure** 2) Local Constriction and dilation of cerebral arterioles 3) **Mean Arterial pressure** 4) Viscosity of Blood 5) Mean Venous pressure at brain level Volume of blood (75ml), CSF (75ml) and brain (1400g) in the cranium at any time remains relatively constant within a rigid structure.	Bold + 1 other to pass To pass: stating the above without the volumes / weights for a normal person

Cerebral Blood Flow 2009-1

QUESTION		ESSENTIAL KNOWLEDGE	NOTES
Question 1: Cerebral blood flow & its regulation. Ganong pp 611-620	i) What factors determine cerebral blood flow? Prompt: What about pressure or vascular factors? ii) Additional: Describe the process of autoregulation.	**(1) Pressures:** · **MAP** · **Intracranial pressure** / extra-cranial venous pressure (whichever is greatest). *Intracranial pressure is determined by intracranial blood volume, CSF volume, tissue oedema, SOL.* **(2) Cerebral arteriole tone** · **Autoregulation** Maintains normal CBF at MAPs of 65-140 mmHg (stretch response[myogenic], local [metabolic]); *Autoregulation may be lost/impaired by brain injury* · **pCO2** (effect on both arteriole tone and intracranial blood volume) · pO2 (at extremes) (3) Blood viscosity	Must get **bold** to pass.

Cerebral Blood Flow 2008-1

3.3 Cerebral blood flow. Brain metabolism & O2 requirements Ganong pp 616-620	What factors determine cerebral blood flow?	**(4 of 5)** • Intracranial pressure • Local constriction/dilation of cerebral arterioles, autoregulation etc • MAP at brain level • Blood viscosity • Mean venous press at brain level	
	What substances are important for brain metabolism?	• Oxygen ~49ml/min = 20% body O2 consumption • Glucose (major energy source) ~77mg/min • Glutamate (converted to glutamine as detox mech NH3) ~5.6mg/min	/2

Cerebral Blood Flow 2006-2

Question 4	Answer	Comments/***Marks***

What factors affect cerebral blood flow?	▪ MAP at brain level
	▪ MVP at brain level
	▪ ICP
	▪ Viscosity of the blood
	▪ Local constriction and dilatation of cerebral arterioles
	  ○ Post ganglionic sympathetic nerves
	  ○ Parasympathetic nerves
	  ○ Sensory nerves
	▪ Positive g ↓ MAP, ↓ MVP, ↓ ICP – therefore CBF unaltered
	▪ Negative g ↑ MAP, ↑ ICP – CBF unaltered
		4/7 = pass

What is Cushing's Reflex?	▪ Increased ICP > 33mmHg - ↓ CBF – ischaemia of VMA - ↑ systemic BP – stimulation of baroreceptors – stimulation of vagal outflow – bradycardia and slowing of respiration.	2/3 = pass
What is Munro-Kelly doctrine?	▪ VBr + VBL + VCSF = constant 1.4kgs + 75mls + 75mls (implications of addition of another compartment ie traumatic/non-traumatic blood/ SOL).	

Cerebral Blood Flow 2005-2

1.1 Factors affecting cerebral blood flow	Describe the factors involved in regulating cerebral blood flow Describe how blood flow can vary in different parts of the brain	**Arterial P, viscosity, Venous P**, local control of arterioles, CSF, **ICP**. **Monroe Kellie** doctrine. **Cushing** reflex. Local autoregulation **Active neurons attract blood flow** and oxygen in excess of needs; marked variation bl flow with activity. PET and fMRI imaging

Cerebral Metabolism 2017-2-B

Stem: Moving on to Physiology. He is hypertensive at 220/100.			
Question 3 Brain Metabolism and Energy Sources **Subject:** Physiology LOA: 1 Ganong 25th Edition pp 609-610	1. How is brain perfusion maintained in brain injury?	Aim is to maintain CPP With high ICP need to increase MAP to maintain CPP **CPP = MAP - ICP** Raised MAP results in systemic hypertension and reflex bradycardia with Vagal stimulation	BOLD or explanation of equation to pass
	2. What proportion of the total body Oxygen does the brain consume?	**20%** (despite brain weight 2% of body weight)	10-30%
	3. What energy substrates can be used by the brain?	**Glucose,** glutamate in prolonged starvation amino acids,	Glucose to pass

Compensation to Blood Loss 2017-1-C

Stem: A 40-year-old man presents with haematemesis. His pulse is 120 / minute and blood pressure is 90/60 mmHg. We will start with Physiology.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Cardiovascular response to moderate haemorrhage **Subject:** Phys LOA 1	a) Describe the factors affecting Cardiac Output	**CO=SVxHR** SV related to contractility, preload and afterload, HR controlled by intrinsic rate, autonomic, exogenous factors, heat, thyroid	Bold + 2 mechanisms from each SV and HR
	b) What are the physiological responses to losing 1L of blood in an adult?	Acutely: -↓venous return, **reduced stimulation of baroreceptors**, catecholamine release, tachycardia, vasoconstriction	Bold and 2
	Prompt: Are there any other non-cardiovascular responses	12 to 72 hours: - ↓ renal blood flow – activation of **renin angiotensin system** fluid shifts	Bold with some explanation
	Prompt: Are there any late compensatory responses?	3 to 4 days: - hepatic synthesis of proteins increasing PP	
		10 days+: - **increased RBC production** by ↑EPO	Bold

Compensation to Blood Loss 2005-1

Cardiovascular compensations for shock	Describe the cardiovascular compensations to acute blood loss. Describe the other physiologic compensations to acute blood loss.	Tachycardia; Vasoconstriction; Venoconstriction. Tachypnoea **PLUS** increases in any 3 from the following list: adrenaline/noradrenaline (sympathetic); vasopressin; glucocorticoids; renin/angiotensin/aldosterone;erythropoietin; plasma protein synthesis.	

‘Compensation to Blood Loss 2005-1

Cardiovascular compensations for shock	Describe the cardiovascular compensations to acute blood loss. Describe the other physiologic compensations to acute blood loss.	Tachycardia; Vasoconstriction; Venoconstriction. Tachypnoea **PLUS** increases in any 3 from the following list: adrenaline/noradrenaline (sympathetic); vasopressin; glucocorticoids; renin/angiotensin/aldosterone;erythropoietin; plasma protein synthesis.	

Coronary Blood Flow 2017-1-B

Stem: Moving onto Physiology			
Question 2 Coronary blood flow **Subject:** Phys LOA: 1	a) What is the coronary blood flow at rest?	***250ml/min or 5%*** *of the Cardiac Output.*	**Bold** to pass. a) Accept range of 200-300ml/min acceptable. 4-6%
	b) Describe coronary artery blood flow during the cardiac cycle. Prompt: Which part of the myocardium is most vulnerable to reduced coronary artery blood flow?	**Greater flow in diastole compared with systole. LV subendocardium most vulnerable.** RV flow continuous	b) Both bold
	c) What chemical factors may cause coronary vasodilation?	**Hypoxaemia,** local increase in **CO2, H+,** K+, **lactate,** PG, adenosine and adenine nucleotides.	c) 2/4 bold
	d) What receptors govern coronary blood flow?	Coronary arterioles have **alpha receptors – vasoconstriction**, **B receptors – vasodilation** Cholinergic receptors - vasodilation.	d) 1/2 bold

Coronary Blood Flow 2013-1

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1: CORONARY BLOOD FLOW LOA: 1	a. Describe coronary arterial blood flow during the cardiac cycle. Prompts: How is flow different in the left and right coronary arteries during systole and diastole? Which part of the heart is most at risk due to low coronary flow?	**Greater flow in diastole c/w systole in L coronary** due to higher pressures required in the LV to overcome aortic pressure in systole. **LV subendocardium** most vulnerable as only gets diastolic flow. **R coronary flow throughout systole and diastole** due to lower RV pressures	Three main features to pass
	b. What factors can decrease coronary artery blood flow?	**1.Physiologic**: **Tachycardia:** shorter diastole; reduced L coronary flow in particular **2.Pathologic**: AS: Increased LV pressures req. to overcome stenosis & decreased flow; Vasospasm; Coronary artery disease; Heart failure: increased venous pressure; reduced coronary perfusion press.	Tachycardia and 2 pathological

Coronary Blood Flow 2010-1

2 a). What **chemical** factors regulate coronary blood flow	Increases in blood flow occur secondary to coronary vasodilation due to:. • Hypoxia, • Locally increased CO2, • H+, • K+, • lactate, • PGs, • adenine nucleotides, • adenosine	Hypoxia plus 3 others
2 b). Describe the **neural** regulation of coronary blood flow	Alpha–adrenergic receptors mediate vasoconstriction Beta–adrenergic receptors mediate vasodilation Vagal nerve stimulation dilates coronaries. Noradrenaline constricts coronaries (although noradrenergic nerves cause increased HR and contractility, with resultant metabolite prod and vasodilation- this is the effect with hypotension that maintains coronary flow)	Alpha–adrenergic – Vc, and beta–adrenergic – Vd effects.

Coronary Blood Flow 2006-1

TOPIC: Regulation of coronary blood flow NUMBER: 1

OPENING QUESTION	What are the factors which affect coronary blood flow?	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	Aortic pressure changes, chemical and neural factors Autoregulation Chemical- low O2, Increased CO2, H+, K+, Lactate, prostaglandins, adenine nucleosides, adenosine Neural- NAD-pos ionotropic and chronotropic effects → vasodaltor metabolites-> cor vasodlation B-Blocker for inotropic and chronotropic effects→ then Nad → cor vasoconstriction via a receptors B receptors and vagus → vasodilation Low Blood press→ metabolic changes in myocardium → cor art vasodilation. Phase of cardiac cycle: more flow in diastole esp left cor art>right, heart rate Disease states that reduce flow: cor art disease, valve lesions -AS, raised venous press - CHF Chemical vasodilators: O2 lack, CO2, H+, K+, lactate, PGs, adenine etc, adenosine *Neural: AdrenoRs - alpha-constrict, beta-dilate, vagal - dilate	Is there any difference in coronary blood flow to the right and left ventricles?	Need 3 factors and 1 example of each.

Flow 2008-1

1.3 Pressure, flow & resistance (Guyton pp 164-170)	What are the basic factors which determine the rate of flow of blood through a blood vessel?	Poiseulle's Law and formula describe these factors; **(Radius to 4th power + 2 others)** Where: F is the rate of flow; $P_A - P_B$ is the pressure differential; $F = \frac{P_A - P_B}{R}$ R is the resistance: r is the radius of the tube; $R = \frac{8 \eta L}{\pi r^4}$ η is the viscosity of the fluid L is the length of the tube $F = P_A - P_B \times \frac{\pi r^4}{8 \eta L}$	
	What factors cause turbulent flow in a blood vessel?	Expressed by Reynold's number; **(3 out of 4)** Where: ρ is the fluid density; D is the diameter of the tube; $Re = \frac{\rho D V}{\eta}$ V is the velocity of flow; η is the viscosity of the fluid. The higher the value of Reynold's number the greater the probability of turbulence' which usually occurs when Reynold's number is between 2000-3000.	/2

Flow 2005-2

3.2 Flow, pressure, resistance, blood flow	What factors cause turbulence in blood flow? Why is blood flow slower in capillaries? What is the relationship between pressure and wall tension in blood vessels of different sizes; What is the relationship between pressure and wall tension in the heart?	'Critical velocity'; smaller diameter, reduced viscosity. Velocity relates to total cross sectional area => capillaries, 1000x area aorta, low velocity same flow. P = T/r. Smaller = less tension in the wall for the same distending pressure. Eg aorta : vena cava : capillaries = 170,000 : 21,000 : 16 dynes/cm. Small vessels unlikely to rupture. Ventricular dilation means more tension required to generate same pressure = more work.	

Muscle Blood Flow 2007-2

QUESTION: 2. Cardiac output, muscle blood flow during exercise

Question	**Required response [Key items marked with*]**	**To Pass**
Describe the factors controlling blood flow through skeletal muscle during exercise.	*Increased flow mainly local regulation Due to chemical effects on muscle arterioles leading to vasodilatation. *Response to reduction in oxygen in muscle tissue. Hypoxic releases vasodilatory substances [especially adenosine], arterioles cannot maintain contraction in hypoxic conditions. Other vasodilatory chemicals: potassium ions, ATP, lactic acid and carbon dioxide. Other controlling factors: Sympathetic vasoconstrictor nerves, circulating adrenaline	Local regulation due to tissue hypoxia. At least 2 mediators
What other circulatory changes occur in the body during exercise and why?	*Increased cardiac output [rate and contractility] 2° sympathetic discharge. Contraction of peripheral arterioles not in skeletal muscle 2° sympathetic discharge. Coronary and cerebral systems spared. Contraction of capacitance vessels eg veins 2° sympathetic discharge giving increased venous return, filling pressure, cardiac output. Nett results, increased blood flow and increased arterial pressure.	Changes to cardiac output plus explanation Changes to venous return/ filling pressure

Renal Blood Flow 2013-2-D

Stem: Moving on to Physiology:

PHYSIOLOGY Question 4 LOA: 1	1. What percentage of cardiac output goes to the kidneys?	RBF = 1.2-1.3L/min or approx 25% CO (adult)	**RBF**
	2. How is renal blood flow regulated? Prompts: What other mechanisms are there?	**Substances/Chemicals** Norepeinephrine (noradrenaline) • Constricts renal vessels • Stimulates renal nerves to ↑ rennin secretion Dopamine – renal v/dilatation A II – arteriolar constrictor PG - ↑cortex flow, ↓medulla flow Ach – v/dilatation High protein - ↑ b/flow **Renal Nerves** • Stimulation nerves = ↑renin secretion , ↑JG sensitivity, ↑Na resorption, and renal vasoconstriction • Strong stimulation sympathetic (noradr) ↓flow • Fall in BP = v/constrict **Autoregulation** • Renal vasc resistance varies with pressure to keep RBF fairly constant • Present in denervated kidney, but not if drugs that paralyse vasc sm muscle • Factors = direct contractile response, NO, A II	**3**/6 substances **plus** nerve or auto – with example
	3. How can renal blood flow be measured?	• Fick principle – subs taken up/unit time • PAH used to measure renal plasma flow • Renal b/flow using plasma flow and Hct	**One** example
	4. Describe the differences in regional blood flow within the kidney.	- AV o2 difference for kidney = 14ml - Cortical b/flow = 5mL/g/min • Little o2 consumption - Medulla b/flow low (outer = 2.5ml, inner= 0.6ml) • Maintence of osmotic gradient	One aspect of regional blood flow to pass

Renal Blood Flow 2006-2

Question 4	Answer	Comments/Marks
Describe the physiological characteristics of renal blood flow?	Renal blood flow is 25% of the cardiac output. The glomerular capillary pressure is 40% of systemic arterial pressure. The peritubular capillary network and renal veins are low pressure systems. The renal cortex gets higher blood flow, but has low oxygen extraction (filtration). Renal medulla gets less blood flow, but high oxygen extraction (osmolality) and sensitive to hypoxia.	Pass – 2/3
What are the factors that affect renal blood flow?	↓ MAP - ↓ baroreceptor firing – renal vasoconstriction - ↓ RBF. Exercise ↓ RBF. Pg ↑ Rcbf ↓ Rmbf. Proteins ↑RBF ↑ GCP. Dopamine and ACh – vasodilatation - ↑ RBF. NA – vasoconstriction □1 > □2 constricts afferent arterioles and interlobular arteries ↓ RBF. Posture – lying to standing ↓ RBF. AgII constricts efferent arteriole ↑ perfusion pressure.	3/6 = pass
How can renal blood flow be calculated?	By determining clearance of PAH, its extraction ratio and the haematocrit.	1 mark

Renal Blood Flow 2005-2

2.2 Renal blood flow, normal values and regulation	Describe the control renal blood flow	- **Chemical: Noradrenaline** constricts interlobular and afferent arterioles. **Angiotensin 11** constricts efferent arterioles > afferent arterioles. Dopamine (made in kidney) vasodilates. Acetylcholine vasodilates. Prostaglandins inc. bl flow in cortex, dec. bl flow in medulla. - **Neural: SNS** -> dec bl flow. Fall of BP, vasoconstrictor response includes renal bl flow. - **Autoregulation**: contractile response of smooth muscle of afferent arteriole to stretch (BP). NO may be involved. Angiotensin 11 plays a role in constricting efferent arterioles, maintaining GFR.	

Shock 2017-2-D

Stem: Moving onto Physiology. She becomes hypotensive.			
Question 5 Shock **Subject**: Physiology LOA: 1	(a) Describe the receptors that respond to the fall in blood pressure. *Prompt: How do these receptors respond to this change?*	**Baroreceptors (**carotid sinus, aortic arch, atria) Reduced stretch -increased sympathetic stimulation - tachycardia and generalized vasoconstriction (sparing of brain and heart). With increased shock, paradoxical bradycardia (unmasking of vagal depressor reflex) then tachycardia again with further shock. **Chemoreceptors (**carotid body, aorta) Stimulated by reduced blood flow and acidosis. Stimulates vasomotor areas in medulla with increase vasoconstriction.	Receptors plus concepts
	(b) Describe the non-cardiovascular compensatory responses to shock. *Prompt: How do the kidneys compensate for shock?*	**Renal response**: Efferent arterioles constricted more than afferent. Renal plasma flow decreased more than GFR - filtration fraction (GFR/plasma flow). **Na+ retention**: Retained nitrogenous products of metabolism (uremia) **Angiotensin II:** plasma renin causing AG II, AG II maintains BP and causes stimulation thirst center in brain. **Vasopressin:** Retain Na+ and H2O. **Aldosterone**: Stimulated by circulating AG II and ACTH. Helps retain Na+ and H2O. **Adrenal stimulation:** Adrenal medulla secretion catecholamines **Increased circulation NA:** Increased discharge sympathetic NA nerves	Renal plus 2

Shock 2016-2-A

Stem: Moving on to Physiology. His blood pressure falls to 80/40. Blood loss is a contributory factor			
Question 5 Physiological responses to shock **Subject:** Phys LOA: 1	1) What is hypovolaemic shock? 2) What are the physiological compensatory reactions to hypotension in acute blood loss? **Prompt:** What are the immediate responses? **Prompt:** What about intermediate or longer term?	**Systemic hypoperfusion due to reduced effective circulating blood volume** resulting in **impaired tissue perfusion** and cellular hypoxia. Rapid (Seconds/minutes) -**Baroreceptors** (decreased discharge with reduced arterial stretch, reducing the baroreceptor inhibition in medulla -> increased sympathetic discharge with vasoconstriction, venoconstriction and tachycardia) -**Chemoreceptors** (stimulation leads to peripheral vasoconstriction and rise in BP) -CNS receptors Early (Minutes/hrs): **Renin-angiotensin system activated** -Blood volume changes -Capillary fluid shifts (isolovaemic anaemia) Longer term: **Renal compensation via aldosterone** - Renin- angiotensin system - Blood volume changes -Salt intake	Bold to pass Bold to pass with understanding of baroreceptor and renin angiotensin function.

Venous Pressure and Flow 2014-1-A

Stem: We are now moving on to Physiology			
Question 3 Venous Pressure and flow (Ganong 24th ed pp 582-584) **Subject:** Phys LOA: 1	1. Describe the mechanisms of venous return to the heart	a) **Thoracic pump:** inspiration resulting in negative pressure in the thorax and positive pressure in the abdomen. Blood flow towards the heart because of venous valves b) Effect of heart beat: during systole, AV valves are pulled downward → increase the capacity of the atria c) Muscle pump: contraction of muscles around the veins in the limbs during activity d) Differential resistance: resistance of the large veins near the heart is less than peripheral veins	Thoracic pump plus one other
	2. What factors might effect the CVP of this patient?	a) **Decrease CVP**: Fluid loss; blood loss b) **Increase CVP**: Excessive fluid replacement; other pre-existing conditions eg CCF; positive pressure ventilation; increased thoracic pressures	1 example from each bold category
	3. What is the value of mean CVP in normal individuals	4.6-5.8 mmHg or 6-8 cm H2O	Reasonable value

Venous Return 2016-1-D

Stem: Moving onto Physiology.			
Question 5 Venous pressure and flow **Subject:** Phys LOA: 1	Describe the mechanism of venous return to the heart Prompt. In a healthy person	1. **Thoracic pump** : insp generates neg (-) intra-throracic P and pos (+) intra-abdo P. 2. Venous valves : one way flow 3. Heart beat : AV valves pulled downwards in systole – inc size atria – blood sucked into atria 4. Muscle/arterial pump : contraction musc/arteries adj to veins compresses veins 5. Differential resistance – less resistance in more prox (larger) veins	**Thoracic pump** plus one other
	What factors might affect CVP in this patient?	1. **Decrease CVP** - fluid loss (third spacing), loss arterial tone, loss muscle pump (ventilated), myocardial depression (acidosis), poor ventricular filling (tachycardia) 2. **Increase CVP** – positive P ventilation (but will decrease venous return), fluid replacement, vasopressor use	1 example from each bolded category
	What is the mean CVP in a healthy adult?	6-8 cm H2O or 4.6 to 5.8 mmHg	Reasonable value

Venous Return 2007-1

TOPIC: Venous return curve / mean systemic filling pressure **NUMBER:** _____

OPENING QUESTION	What is the normal value for venous return in the healthy human adult?	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	1 5-5.5 l/min		
SECOND QUESTION	What are the major factors that influence venous return to the heart?		¾ to pass
POINTS REQUIRED	1 Circulating blood volume		
	2 Sympathetic and parasympathetic tone		
	3 Muscle pump		
	4 Right atrial pressure (intrathoracic and intracardiac pressures and factors that influence them like phases of respiration, tamponade, PEEP		
THIRD QUESTION	What is the relationship between right atrial pressure and venous return?	What happens to venous return when right atrial pressure rises?	
POINTS REQUIRED	1 Downslope - reduced driving pressure		
	2 Plateau – collapse of vein walls		
	3 Normal value for MSPF = 7 mmHg		
	4 Normal value for mean RAP = 0 mmHg		