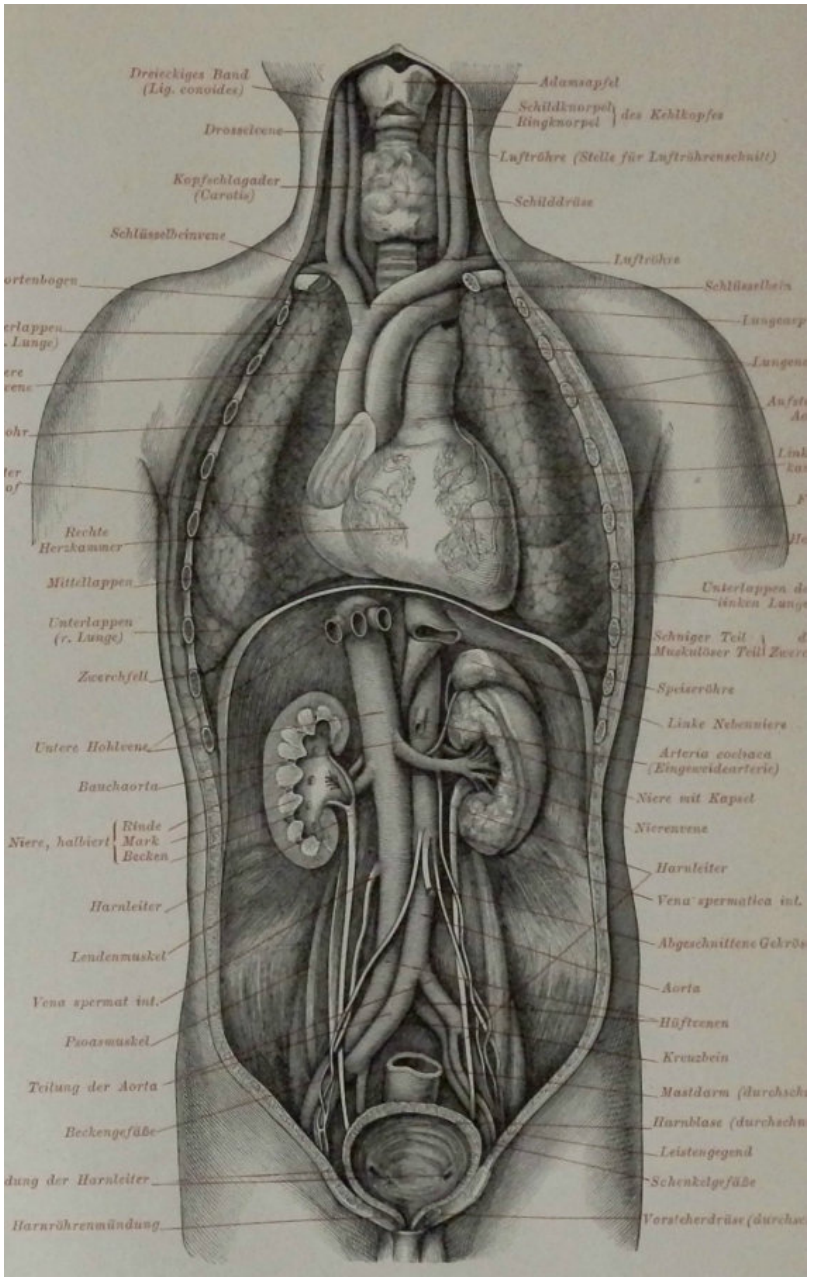
ACEM Primary Examination Vivas > Physiology > Renal Organised by edvivas.com	



Angiotensin II 2011-1

Question 3	3.1 Describe the factors influencing Angiotensin II production.	Is the effector protein in the renin-angiotensin system: integral to control of volume regulation So, principally those that influence renin secretion: Increased secretion due to: • Increased sympathetic activity • Increased circulating catecholamines • Prostaglandins (from Na^+ depletion, diuretics, hypotension, haemorrhage, dehydration, cardiac failure, cirrhosis, upright posture, renal artery and aortic constriction) Decreased secretion due to: • Increased Na^+ and Cl^- re-absorption across macula densa • Increased afferent arteriolar pressure • Vasopressin	Intravascular volume Renin + 2 others
	3.2 What are the physiological effects of Angiotensin II?	• Arteriolar constriction • Directly on adrenal cortex to increase aldosterone Facilitation release of norepinepherine release • Contraction of mesangial cells causing decreased GFR • Direct effect on renal tubules to increase Na+ re-absorption • On the brain to decrease sensitivity of baroreceptor reflex • On brain (circumventricular organs) to increase water intake and increase secretion vasopressin and ACTH	Vasoconstriction + 2 others

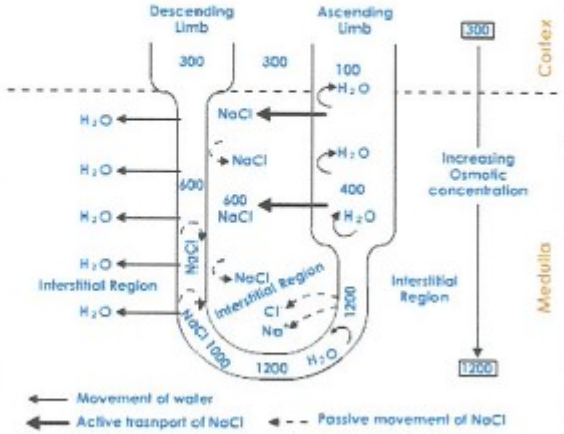
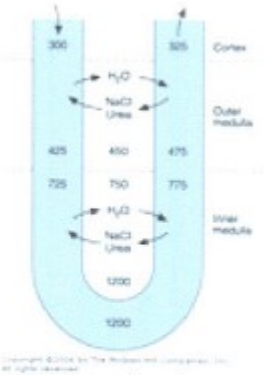
Counter Current Mechanism 2017-1-B

Stem: A 30 year old man collapsed while hiking. Starting with Physiology.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Counter current mechanism **Subject:** Physiology LOA: 1	How does the Countercurrent mechanism enable the kidney to concentrate urine? Prompt: what processes produce this gradient and where do they occur Candidates may choose to use diagram for demonstartion	Concentrating mechanism depends on maintaining a **gradient of increasing osmolality along medullary pyramids**. **Gradient is produced by Countercurrent multipliers** in the **LOH and maintained by Vasa recta acting as counter current exchangers**. 1) **Water moves out of the thin descending limb** (via aquaporin 1), **2) Active transport of Na and Cl out of thick ascending limb of LOH** 3) Continued inflow of isotonic fluid into the proximal tubule and out of desc tubule ..H2O moves out of collecting duct (into the hypertonic interstitium of the medullary pyramids) under the influence of ADH Vasa recta acts as countercurrent exchangers in the kidney in which NaCl & urea diffuse out of the ascending limb of the vessel & into the descending limb, while water diffuses out of the descending into the ascending limb of the vascular loop. As a result, the solute remains in the medulla pyramid & maintain the interstitial concentration.	Bold to pass. Need to demonstrate understanding.

Counter Current Mechanism 2012-1

Question 3 LOA: 1	3.1 What are the essential features of the loop of Henle countercurrent multiplier?	High permeability of the thin descending limb to water (via aquaporin-1) and active transport of Na^+ and Cl^- out of the thick ascending limb which is not permeable to water. A system in which Na K 2Cl are actively transported, and the inflow runs parallel to, counter to, and in close proximity to the outflow for some distance	Either version
	3.2What is the role of urea in the countercurrent mechanism?	Contributes to the **osmotic gradient** in the medullary pyramids	**Osmotic gradient**
	3.3 How does urea reach the interstitium?	Transported by urea transporters, by **facilitated diffusion** Amount of urea depends on the amount filtered which is influenced by dietary protein	**Facilitated diffusion**

Counter Current Mechanism 2009-1

Question 3: Counter-Current mechanism Ganong pp 716-8	i) Describe the counter-current mechanism in the kidney. Prompt: What is the role of the vasta recta?  **Countercurrent multipliers**	a) **Countercurrent multipliers** in the **LOH** through active transport of Na (& Cl^-) out of its thick ascending limb. Water moves out of the thin descending limb, with inflow of tubular fluid from the PCT. This increases the interstitial osmolarity. This results in hypotonic fluid flows into DCT, isotonic fluid flows into the asc thick LOH. The final result is a gradient conc from the top to the bottom of the LOH & **a gradient hyperosmolarity in the medulla interstitium**. b) **Vasta recta as countercurrent exchangers** in the kidney in which NaCl & urea diffuse out of the asc limb of the vessel & into the desc limb, while water diffuses out of the desc into the ascending limb of the vascular loop. As a result the solute remains in the medulla pyramid & maintain the interstitial conc.	 Countercurrent exchangers
Question 4:	i) Describe the steps in synthesis of thyroid	i) Thyroid epithelial cells secrete thyroglobulin	

Glomerular Filtration Rate 2017-2-B

Stem: Moving onto Physiology. He has decreased urine output.			
Question 2 GFR **Subject:** Physiology LOA: 1 Ganong's review of Medical Physiology. 25th edition. Chapter 37 Renal function and micturition. Glomerular filtration.	a) What is the definition of the glomerular filtration rate? b) What is the normal Glomerular filtration rate? c) List some factors that affect the GFR. Prompt " physiological factors" "are there any particular cells in the kidney that are involved in regulating GFR?"	a) Amount of fluid (plasma filtrate) filtered by the glomerulus per unit time b) Normal GFR – **125ml/min** (180L/24Hrs) in normal adult. 10% lower in females. c) -Size of capillary bed. Regulated by **mesangial cells** (contractile cells) located in the glomerulus (between the basal lamina and the endothelium). -Permeability of glomerular capillaries (50 x skeletal muscle capillaries) -Hydrostatic and osmotic pressure gradients Oncotic pressure (plasma protein concentration) Glomerular capillary hydrostatic pressure -Systemic blood pressure -Afferent arterial pressure (renal artery blood flow - kept stable by autoregulation 90-210mmHg) -Afferent or efferent arteriolar constriction -Hydrostatic pressure in Bowman's capsule -Intrarenal interstitial pressure (ureteral obstruction, renal oedema) -Age	Concept to pass +/- 10% to pass 110-140ml/min Bold to pass. Must mention mesangial cells and 3 other factors.
	d) What substances act on mesangial cells to change GFR?	d) Increased – ANP, dopamine, PGE2, cAMP Decreased – NA, vasopressin, AGII, Histamine, PGF2, endothelins, TXA2, leukotrienes	List at least one of each increased/decreased

Glomerular Filtration Rate 2016-2-D

Stem: Moving onto physiology.			
Question 4 Glomerular filtration **Subject:** Physiology LOA: 1	a. What is the definition of glomerular filtration rate (GFR)? b. What is the GFR in a normal average adult? c. What general factors within the glomerulus affect GFR? *Prompt: Other than hormones...*	1. Overall rate of fluid filtered through the renal corpuscles, into the renal tubules. **125ml/minute.** (Accept **100 to 150 ml/min)** Note it is distinct from renal blood flow (approx. 1250ml/min) or renal plasma flow (approx. 625ml/min). GFR/renal plasma flow is the filtration fraction (which is 10 to 20%). A. **Overall surface area of capillary bed within the glomerulus.** This is determined by glomerular mesangial cells (like smooth muscle cells). Contraction of these cells reduce surface area, and hence GFR. Conversely, relaxation of these cells increases GFR. B. **Permeability of glomerular capillaries.** C. **Hydrostatic pressure within glomerulus.** Increased by afferent arteriolar dilatation, efferent arteriolar constriction, increased renal blood flow. Systemic BP may be directly proportional if it's outside the range of auto-regulation. D. **Hydrostatic pressure within Bowman's capsule.** If increased, eg. ureteric obstruction, will reduce GFR. E. **Oncotic pressure within glomerulus.** If increased, will reduce GFR. F. **Number of functioning renal corpuscles.** Loss of corpuscles reduces GFR. This may result from many causes eg. atrophy, parenchymal disease, acute kidney injury, nephrectomy.	Concept to pass Bold 4 out of 6 bold

Glomerular Filtration Rate 2015-2-D

Stem: Moving onto Physiology.			
Question 5 GFR **Subject:** Phys LOA: 1	What is the definition of the glomerular filtration rate?	The amount of fluid (plasma filtrate) filtered by the glomerulus per unit time	**Concept of filtration and time to pass.**
	What is the normal GFR?	Usually 125mL/min (180L/day) 10% less in women.	**+/- 20 % to pass (either per min or per day)**
	What are mesangial cells? (Prompt – Where are mesangial cells found? What do mesangial cells do?) (Prompt if "in nephron" stated – where in nephron?)	**Contractile cells** that help to **regulate GFR**. Located between the basal lamina and the endothelium, **in the glomerulus** Common between neighbouring capillaries, and in these locations the basal membrane forms a sheath shared by both capillaries Also secrete the extracellular matrix, take up immune complexes, and are involved in the progression of glomerular disease.	**Bold to pass**
	What factors influence GFR?	Age Afferent arterial (renal artery) pressure (however autoregulation keeps this stable between about 90-210mmHg) Afferent arteriolar pressure Efferent arteriolar pressure Efferent venous pressure Intra-renal (interstitial) pressure (obstruction, oedema) Oncotic pressure Glomerular filtration fraction	**Any 3 to pass**
	What substances act on mesangial cells to change GFR? (Prompt - What substances act on mesangial cells to alter their function?)	Glomerular filtration fraction (mesangial cell function) – influenced by: Increased – ANP, dopamine, PGE2, cAMP Decreased – noradrenaline, vasopressin, AII, PGF2, endothelins, TXA2, Leukotrienes	**BONUS!**

Glomerular Filtration Rate 2014-1-B

Stem: Moving now to your physiology question. The patient is noted to have a low eGFR.			
Question 2 GFR including hydrostatic and osmotic pressure.(Ganong 24th ed pp 678-680) **Subject:** Phys LOA: 1	1.What is normal Glomerular Filtration Rate (GFR) 2. What factors control GFR? Prompt: What agents, mediators & clinical factors affect GFR?	**125ml/min** in normal adult 180L/24h/10% lower in women **Hydrostatic Press/Osmotic press gradient, Size & permeability** of capillary bed (mesangial cell contraction/relaxation & loss of renal tissue) K in Starling Forces=GF coefficient=mesangial cell Increase –ANP Dopamine PGE2 cAMP Decrease – Endothelins, AGII, vasopressin, norepinephrine, PAF,PGF2, leukotrienes Ca/D4, histamine TxA2 Clinical: Systemic BP/Parenchymal odema/Ureteric obstruction/after-efferent arteriolar constriction/plasma proteins	Approx value 2/4 bold Role of mesangial cells Vaso active Agents - 2 Clinical examples - 2

Glomerular Filtration Rate 2013-2-A

The patient develops renal failure following his surgery			
Physiology: Renal regulation of K+ plus GFR	1. What is normal Glomerular Filtration Rate (GFR) and what factors regulate it? (Prompt: How does it change?) (Prompt: Identify **two clinical factors** that alter Starling Forces) 2. (Optional) How do the kidneys deal with Potassium?	1. **Normal GFR** = 125mls/min (180L/24hrs). 10% lower in females Controlled by **Starling Forces** ie: GFR = $K(P_{GC} - P_T) - (\pi_{GC} - \pi_T)$ P_{GC} = mean **hydrostatic** pressure in glomerular **capillaries**, PT = mean **hydrostatic** press in **tubule**. π_{GC} = **osmotic** press of plasma in glom **caps**, π_T **OP** of filtrate in **tubule**. K = GF coefficient; altered by **mesangial cell** contraction (-> dec area for filtration). Contraction = Angio II, ADH, NA, PAF, TxA2, hista Relaxation – ANP, dopamine, cAMP, PgE2 GFR changes along glomerular cap with Starling forces dropping from 15 mmHg to 0. **Clinical Factors** altering Starling Forces: Alterations in renal blood flow, systemic BP, ureteric obstruction, renal parenchymal oedema, changes in plasma protein concentration, changes in K as above 2. Freely filtered at glomerulus (600 mmol/d) Actively reabsorbed in PCT (560 mmol/d) Secreted in DT – rate proportional to flow Secreted in Collecting Ducts – Aldosterone Excreted = 90 mmol/d Total secreted load averages 50 mmol/d but varies with renal tubular flow and aldosterone lev.	***Pass/Fail*** 1.a) approx **value** for GFR b) Identify **Starling Forces** involved c) Identify central role of **mesangial cells and two factors which change their degree of contraction** d) Identify **two clinical factors** that alter Starling Forces 2. a) freely filtered at glomerulus, b) largely reabsorbed in PCT c) Sites of distal secretion plus influence of aldosterone

Glomerular Filtration Rate 2012-2

Question 3 LOA: 1	1.1 What is the normal Glomerular Filtration Rate? 1.2 What factors affect GFR? Prompt: what agents affect GFR and how?	Rate: ~125mL/min normal adult Factors: **Size** and **permeability** of capillary bed Primarily by mesangial cell contraction / relaxation [and loss of renal tissue] Agents: Increased – ANP, Dopamine, PGE2, cAMP Decreased – Endothelins, AG II, Vasopressin, Norepinephrie, PAF, Platelet-derived growth factor, TxA2, PGF2, Leukotrienes C4 & D4, histamine. **Hydrostatic and oncotic** pressure gradients. Renal blood flow, Systemic BP (esp below auto-reg range), afferent and efferent arteriolar constriction Ureteral obstruction, oedema of kidney, changes in plasma proteins (dehydration hypoproteinaemia), changes in capillary permeability	100-150 3 of 4 Bold

Glomerular Filtration Rate 2010-2

Question 2	1) What is normal GFR 2) what factors affect GFR 3)As well as filtration , by what other means does the kidney regulate the composition of urine –	1. @ 125ml/min in 70kg male, 10% less in female 2.a) Net filtration pressure= hydrostatic pressure in capillaries and bowmans capsule AND colloid osmotic pressure -afferent – efferent pressure- under control of – autoregulation, sympathetic, AgtII, dobutamine, PGs, serum Na - renin Pressure in bowmans capsule – renal obstruction 2. b)capillary filtration coefficient Relates to surface area and permeability of capillaries Surface area controlled by mesagial cells under control of AgtII, ADH, NA, PGs (constrict) ANP, dopamine, cAMP, PGE2(relax) 3.secretion and resorption	1) reasonable value 2) 3 to pass 3) 1 of 2

Glomerular Filtration Rate 2007-2

QUESTION: 3. GFR

Question	**Required response [Key items marked with*]**	**To Pass**
Describe a method for measuring the glomerular filtration rate Prompt Describe the properties of a suitable substance and give an example	Measure excretion of a substance which is ***freely filtered*** through the ***glomeruli neither secreted nor reabsorbed*** by the ***tubules*** Non toxic, not metablised Eg Inulin, NB Endogenous Creatinine has limitations $GFR = \frac{UX \times V}{PX}$ where Ux is the conc of X in the urine, P is the urine flow per unit time Px is the arterial plasma level of X. If X is not metabolized in the tissues then the peripheral venous plasma level can be substituted for the arterial plasma level.	Three of five One example Definition or description and basic formula
What is normal GFR and what are the factors which affect it	125ml/min in normal 70 kg male, 10% less for women, and correlates with surface area. Factors RBF, Systemic BP, Ureteric obstruction, compression by oedema within renal capsule, Plasma proteins, Permeability changes, Filtration surface area	Value (100 – 150) and three factors

Glomerular Filtration Rate 2006-1

TOPIC: Glomerular filtration **NUMBER: 3**

OPENING QUESTION	What factors control glomerular filtration?	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	Mention average 125ml/min or 0.16-0.2 of RPF and its derivation Ui x V/ Pi= Ci= GFR for inulin; Creatinine Clearance is approximation Control of GFR depends on 1. size of capillary bed*, 2. permeability of capills*, 3. hydrostatic pressure*, 4. oncotic pressure*. These influenced by changes in RBF, MAP,[plasma proteins}, effective surface area, changes in pressure across Bowman's capsule eg ureteric obstruction, renal oedema. Glomerular capills are 50x permeable as skeletal.		Need ¾ to pass.

Glomerular Filtration Rate 2005-1

Factors affecting GFR	What factors affect filtration across the glomerular capillary bed? How can GFR be measured?	• Permeability and area of the glomerular capillary bed. • Hydrostatic pressures in the capillary and the tubule. • Oncotic pressure in the plasma and the filtrate. $U_X \dot{V} / P_X$ or concepts.	

Glomerular Filtration Rate 2003-1

TOPIC: Glomerular filtration rate _____

OPENING QUESTION	What is a normal Glomerular Filtration Rate in humans?	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	1. 125 ml/min	1	
SECOND QUESTION (if needed)	What factors would cause a decrease in GFR?		3/5 to pass
POINTS REQUIRED	1. Hydrostatic pressure	1	
	2. Renal blood flow	2	
	3. Capillary permeability	3	
	4. Plasma protein osmotic pressure	4	
	5. Size of capillary beds	5	

Glucose Handling 2015-1-C

Stem: Moving onto Physiology. Urine analysis reveals glucose.			
Question 2 (Renal handling of glucose **Subject:** Phys LOA: 1	Describe the way the kidney handles glucose. What are the potential consequences of glycosuria	1. **Freely filtered** the glomerulus 2. **Resorbed in the early part of the PCT** by secondary active transport 3. **Na dependent** co transportation (SGLT2 into cells then GLUT 2 facilitated diffusion into interstitial fluid) 4. Excreted in the urine if **renal threshold** is exceeded. Osmotic diuresis – dehydration, electrolyte loss (Na, K)	1 & 2 plus understanding of 3 & 4 Understanding

H+ regulation 2017-1-A

Stem: Moving onto Physiology. His urinalysis shows a pH of 6.0.			
Question 3 Renal - H+ handling **Subject**: Phys LOA: 1	a) Where does the acidification of the urine occur? *(Prompt : where is hydrogen secreted in the kidneys?)*	a) Proximal and distal tubules, and collecting ducts	**2 of 3**
	b) How is H+ secreted in each of those areas?	b) **Proximal tubule – Na-H exchange transporter** (one Na and one HCO_3 reabsorbed for each H secreted) Distal tubule and collecting duct **–** the secretion of H+ is independent of Na. ATP driven proton pump - stimulated by aldosterone. Also H-K ATPase pump, and anion exchanger 1.	**Na-H and 1** mechanism in DCT/ CT
	c) What is the limiting pH of urine and where is it reached?	c) **The limiting pH is 4.5** (1000x concentration in plasma). It is the maximal H+ gradient that can be achieved in the tubules. It occurs in the **collecting duct**. Possible due to buffers (bicarb, dibasic phosphate and ammonia)	**Bold**

H+ regulation 2016-1-D

Stem: Moving onto Physiology			
Question 5 Renal buffers in acid-base regulation **Subject:** Phys LOA: 1	How will the kidneys respond to a metabolic acidosis?	Aims to **return serum pH to normal by increasing H+ excretion.** **Kidney reabsorbs HCO_3^- by actively secreting H+.** Renal tubule cells contain carbonic anhydrase converting CO_2 to H+ and HCO_3^-, then PCT cells secrete H+ in exchange for Na+ In the DCT, H+ is secreted by a proton pump, limited by urinary pH >4.5 (limiting pH).	Must know that H+ actively secreted into tubular fluid in exchange for Na. Must know about buffering and name 2 buffers.
	PROMPT: Describe the role of buffers in the kidney	**Buffering in tubular fluid** pH with H_2CO_3, HPO_4 and NH_3 **allows greater H+ secretion.**	

H+ regulation 2014-2-A

Stem: Blood gases show an acidosis. We will now move onto Physiology			
Question 3 H+ handling in metabolic & respiratory acidosis (pp 711-712) **Subject**: Phys LOA: 1	1.Describe the renal response to acidosis Prompt – Describe the role of buffers in the kidney	Aims to return serum pH to normal by increasing H+ excretion. Kidney retains HCO_3 by actively secreting H+ Renal tubule cells excrete carbonic anhydrase converting CO_2 to H+ and HCO_3, then tubule cells secrete H+ in exchange for Na+ Amount of secreted H+ limited by urinary pH >4.5 (limiting pH) Buffering in tubular fluid pH with HCO_2, HPO_4 and NH_3 allows greater H+ secretion.	Must know that H+ actively secreted into tubular fluid in exchange for Na. Must know about buffering and name 2 buffers.

H+ regulation 2014-1-A

Stem: We are now moving to physiology. Arterial blood gases show a metabolic acidosis			
Question 3 Renal role in the handling of H+ ions **Subject:** Phys LOA: 1	1. Describe how the kidney responds to metabolic acidosis 2. What substances act as urinary buffers for the excretion of H^+ 3. How else can the body compensate for a metabolic acidosis? Prompt: What other major system is involved in acidosis compensation?	**Renal tubule cells secrete H^+** into tubular fluid **in exchange for Na^+** **HCO_3^- is actively reabsorbed** into the peritubular capillary (for each H^+ secreted, $1Na^+$ and 1 HCO_3- are added into blood). **NH_3** forms NH_4^+; **HCO_3-**forms CO_2 and H_20; **HPO_4^{2-}** forms H_2PO_4 The **respiratory system** responds by **increasing ventilation** which results in a decrease in PCO2 which causes increase in pH (this is a rapid response)	Bold 2 of 3 Bold to pass

H+ regulation 2013-1

Question 3 Renal H+ regulation LOA: 1	Describe the renal response to metabolic acidosis Prompts: "What prevents H+ secretion stopping when a pH of 4.5 is reached?" "What substances act as buffers in the urine?"	• **Renal compensation aims to normalise blood pH by reabsorbing all filtered HCO_3^-, and generating new HCO_3^-** by titration of filtered acid. • Anions that replace HCO_3^- are filtered at the glomerulus along with corresponding cations • **Renal tubule cells secrete H+ into tubular fluid** in exchange for Na^+ and HCO_3^- • **Buffering in the urine gives greater capacity to this system** (otherwise limiting pH of 4.5 would stop further H^+ elimination) Urinary buffers include **HCO_3^-**, PO_4^-, and NH3	Pass criteria **bold** Buffers need bold and 1 other

H+ regulation 2012-2

Question 3 [Renal compensation acidaemia]	**3.1** Describe how the renal tubule cells respond to metabolic acidaemia.	a. Acidaemia: renal tubule cells **secrete H+** into tubular fluid, in **exchange for Na**	Bold to pass
LOA: 1	**3.2** In metabolic acidosis, describe which buffer systems in the urine are involved that allow excretion of large amounts of H+?	Secreted H+ reacts with buffers: a. **HCO3-** to form CO2 and H2O with bicarbonate absorption b. **HPO4 2-** to form H2PO4- c. **NH3** to form NH4+	Need two out of three bold
	3.2b What happens to glutamine synthesis in the liver in chronic metabolic acidosis?	a. Glutamine synthesis increased in liver, to provide kidney with additional source NH4+, as well as NH3 secretion increasing over days	Need to mention that **glutamine synthesis increased**

H+ regulation 2012-1

Question 3	3.1 By what mechanism is H^+ secreted in the distal tubules and collecting ducts of the kidney?	ATP driven proton pump. Aldosterone acts on this pump to increase H^+ excretion. Abundant carbonic anhydrase in the cells numerous tubulovesicular structures. Pumps in the vesicles $H - K^+$ ATPase	**ATP driven proton pump**
	3.2 In H^+ secretion, what is the limiting urine pH?	A urine pH of 4.5 is the maximal H^+ gradient against which transport mechanisms can secrete H^+	**pH 4-5**
	3.3 Describe the principal urinary buffers and what is their role?	HCO_3 buffer system particularly in the proximal tubules HPO_4^{2-} in the distal tubules NH_3 in the proximal and distal tubules	**2 examples + increased capacity to excrete H+**

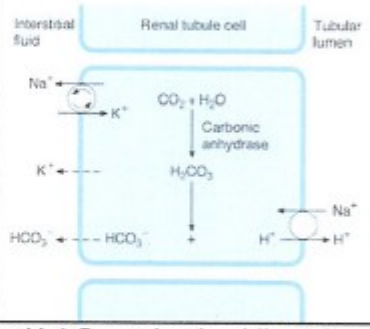
H+ regulation 2011-1

Question 3:	3.1 What are the principal buffering systems in the body? PROMPT: How about in other fluid compartments? 3.2 Outline how the body responds to a metabolic acid load.	Blood: Bicarbonate, Protein and Haemoglobin Interstitium: Bicarbonate Intracellular: Protein, Phosphate Urine: also uses ammonia a) Buffering in blood, interstitial and intracellular spaces b) Respiratory response: H_2CO_3 converted to H_2O and CO_2, CO_2 expired via lungs through increased minute ventilation. c) Renal: • Renal mechanisms operate to compensate for metabolic acidosis and return the serum pH towards normal • Anions that replace HCO_3^- are filtered at the glomerulus along with corresponding cations (mainly Na^+) • Renal tubule cells secrete H^+ into tubular fluid in exchange for Na^+ and HCO_3^- • Buffering in the urine gives greater capacity to this system (otherwise limiting pH of 4.5 would stop further H^+ secretion) • Buffering systems include: Bicarbonate, Phosphate, Ammonia	3 buffering systems to pass 2 fluids to pass Buffering in blood CO_2 expiration via lungs Acid secretion in kidney + buffering in urine All 3 to pass

H+ regulation 2010-1

5. Describe the renal response to metabolic acidosis.	• Renal mechanisms operate to compensate for metabolic acidosis and return the serum pH towards normal • Anions that replace $HCO3^-$ are filtered at the glomerulus along with corresponding cations (mainly Na^+) • Renal tubule cells secrete H^+ into tubular fluid in exchange for Na^+ and HCO_3^- • Buffering in the urine gives greater capacity to this system (otherwise limiting pH of 4.5 would stop futher H^+ secretion) • Buffering systems include: Bicarbonate, Phosphate, Ammonia Prompts: (i) What prevents H^+ secretion stopping when urine pH falls to 4.5? (ii) Can you name any of the buffers that operate?	Compensatory mechanisms identified Must know H^+ secreted into tubular fluid in exchange for Na^+ Must know about buffering and give two buffers

H+ regulation 2009-2

Question 3a: Score:	**Discuss how and where H+ is secreted in the kidney?** **Prompt: how is Bicarbonate involved.**	Prox Na Bic co tranport Distal H+ ATPase H+/K+ (I cells) with large C Anh conc + Cl/ HCO3 BM exchanger  	Active secretion H+ (H+/Na+ co transport- 2ary active secretion), allows reuptake of HCO3- fromC anhydrase brush border_- H20/ CO2- Bic then into interstitium with Na via Na/K ATPase) Bic in cell transferred to Interstitium along gdt_ In DCT/ Coll ducts- Principle cells/ Aldo- have H+ ATPAse channels + H/K ATPAse linked to Bic/ Cl- exchanger in BM **PASS-FAIL must know 2 diff mechanisms, and mention bicarb**
3b:	**What is the limiting pH of urine and how is this limitation dealt with?**	pH 4.5 maximal acidity urine much > er acidity required excreted 3 major BUFFER systems H2CO3 (proximal), NH4+(throughout) and HPo4 (distal)	H+ load would be 100-100 x greater than max pH, Buffers all inc (partic H2CO3 and NH4 when acidotic)- NH4 via glutamate in interstitium, H2CO3 inc with H+ extra = >substrate + > C anh. HPO4 v concentrated in DCT
Question		1) Concept: Balance between glucose	COMMENTS

H+ regulation 2009-1

Question 3: Acid secretion & absorption in kidney Ganong pp 720-1	i) How is H+ ion secreted in the proximal tubule of the kidney?	a) **Secondary active transport** (The renal tubular cells secrete **H^+** into the tubular fluid in **exchange for Na^+**; and for each H^+ secreted, one Na^+ and one HCO_3^- are added to the blood) b) Linked to **Na+/K+ ATPase**	
	ii) Outline the buffer systems that act to bind H+ ion in the tubular fluid	i) 3 systems – **HCO3, HPO4, NH3** ii) Major role of carbonic anhydrase/HCO3 system	To pass: 2/3, must have bicarbonate
	iii) What is the importance of H+ buffering systems in the urine ?	**Limiting pH (~4.5) would rapidly be reached unless free H+ is buffered**	
Question 4:	i) How does the body generate heat?	a) **Heat production: basal metabolic processes**	

H+ regulation 2006-2

Question 4	Answer	Comments/Marks
How does the kidney acidify the urine?	Secretion of hydrogen ions. Binding of the hydrogen ions with buffers. Secretion/absorption of bicarbonate ions.	2/3 = pass
Is there a difference between the proximal and distal tubules?	PCT/DCT/CD secrete H+. PCT via Na+/H+ exchange. Na+/K+ATPase – Na+ from cell to interstitium. DCT/CD H+ secretion ATP driven proton pump. .	Need to understand
What factors increase acid secretion?	Factors which increase acid secretion ↑ PCO_2 ↑ P_aCO_2 ↑ aldosterone ↓ K+, ↑ CA concentration ↑ K+ ↓ H+ secretion.	4/7 = pass

H+ regulation 2005-1

Renal regulation of H^+	What is the renal response to respiratory acidosis? What buffering systems are there for H^+ in renal tubular fluid?	Increased H^+ secretion and HCO_3^- absorption. At least HCO_3^- and one of HPO_4^{2-} or NH3 with explanation of buffering mechanism.	

H+ regulation 2003-1

TOPIC: Renal regulation of acid excretion _____

OPENING QUESTION	What is the renal response to acidaemia?	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	1. Hydrogen ions actively secreted into the proximal tubule, thick ascending loop of Henle and distal tubules, facilitated the re-absorption of bicarbonate ions by forming the carbonic acid, which dissociates to form CO2 and water.	1	
	2.	2	
SECOND QUESTION (if needed)	Describe the buffer systems involved.		2/3
POINTS REQUIRED	1. Bicarbonate	1	
	2. Phosphate	2	
	3. Ammonia	3	

K+ Regulation 2017-1-D

Stem: Moving onto Physiology. His potassium is elevated at 7mmol/L.			
Question 5 Renal handling of K+ **Subject**: Phys LOA:1	a) How does the kidney handle potassium? **PROMPT**: What happens to potassium in different parts of the nephron?	**Filtered** in **glomerulus** (~600meq/24hrs) **Reabsorbed** in **proximal tubules** & thick ascending limb of loop of Henle (~560meq/24hrs : >90%) • active transport via Na-K-2Cl co-transporter **Secreted / excreted** by **distal tubules/collecting ducts** (~502meq/24hrs) • amount proportionate to flow rate through distal tubules (rapid flow rates reduces intratubular K^+ concentration, thus facilitating secretion) • under influence of aldosterone (induces K^+ secretion)	Bold to pass (need to have process and site)
	b) What other major ions are involved in potassium transport in the nephron?	Na^+, H^+	Both
	c) How do hydrogen ions influence potassium transport in the nephron?	Coupled to H^+ secretion [if H+ secretion increased, then K^+ excretion decreased as K+ is reabsorbed in exchange for H^+ (H,K-ATPase) in collecting duct cells]	Concept

K+ Regulation 2011-2

Question 3 LOA: 1	How does the kidney handle potassium?	**Potassium is filtered, reabsorbed and secreted** Per 24 hours ■ 600 mmol **filtered** ■ 560 mmol **actively reabsorbed** mainly in **PCT (65%)** but also 25% in **TALLOH (NaK2Cl co-transporter) and in CD** ■ 50 mmol **secreted by late DCT and cortical CT** cells proportional to flow via Principal Cells ■ With high/low potassium intakes the required extra secretion of K+ achieved by increased/decreased secretion in DCT/cortical CT; with extremely low K+ intake, there can be net reabsorbtion of K+ in DCT/cortical CT ■ 90 mmol excreted	Need bold to pass

K+ Regulation 2008-2

OPENING QUESTION	**Describe how the nephron handles potassium.**	**COMMENTS**
POINTS REQUIRED	**1. K+ is freely filtered at the glomerulus (~600 mEq/day).** **2. Most is reabsorbed by active transport in the proximal tubule (~560 mEq/day).** 3. K+ is then secreted by passive diffusion into the tubular fluid in the distal tubule. 4. K+ is also generally passively secreted into the tubular fluid in the collecting ducts. 5. The total K+ excretion is approximately equal to K+ intake (~90 mEq/day) and K+ balance is maintained. 6. There is no direct exchange of K+ for Na+ in the tubular fluid of the distal nephron. However reabsorption of Na+ into the tubular cell tends to promote secretion of K+ (or H+) to maintain the potential difference across the apical membrane.	Bolded + at least one other
PROMPTS		
SECOND QUESTION (if needed)	**What factors influence this?**	
POINTS REQUIRED	**1. The rate of secretion of K+ is proportional to the rate of flow of tubular fluid through the distal nephron. With rapid flow the concentration of K+ in the fluid remains lower and secretion continues.** 2. Increased delivery of Na+ to the collecting ducts promotes increased secretion of K+ (e.g. thiazide diuretics). 3. Conversely decreased delivery of Na+ to the collecting ducts promotes decreased secretion of K+. 4. Inhibition of K+ absorption in the proximal nephron (e.g. osmotic or loop diuretics) promotes excretion of K+. **5. In the distal nephron K+ and H+ compete for secretion in association with reabsorption of Na+. Therefore in acidosis when H+ excretion is increased, K+ secretion is decreased.** **6. Aldosterone increases reabsorption of Na+ in the collecting ducts and thereby promotes K+ secretion.**	At least two of the three bolded
PROMPTS		

COMMENTS

K+ Regulation 2008-1

1.2 Renal regulation K^+ (Ganong pp 724)	How does the kidney handle potassium? How do other ions affect potassium transport across the membranes in the nephron? Prompt: ***How is potassium transported into and out of the tubules?***	• K+ filtered ~600meq/24hrs • Active K+ reabsorption in prox tubules ~560meq/24hrs • K+ secretion ~502meq/24hrs at distal tubule – amount proportionate to flow rate through distal tubules • Secretion - Electrical coupling to Na+ reab, thus H+ also • Collecting tubules Na reab'd, K excreted, electrical coupling and passive K movement • Na reab'd in association with H secretion, K excretion decreased if Na low in distal tubule • Na/K 2Cl apical transporter/transport protein • 3Na/2K ATPase	/2

K+ Regulation 2004-2

TOPIC: Potassium handling by the kidney _____ **NUMBER:** _____

OPENING QUESTION	What happens to potassium as it passes through a nephron?	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	1 Freely filtered	1 Reabs/secret^{n}?	
	2 67% reabsorbed prox tub (with Na + H^2O)	2	
	3 20% reabsorbed asc limb (with Na and Cl)	3	
	4 Dist tub reabs or **secretes** (H/K/ATPase)	4	
	5 Reabs in alpha intercalated cells	5	
	6 Secretion by principal cells	6	
	7 Diet,aldosterone,A/B,lumen ions,diuretics	7	
	8		
SECOND QUESTION (if needed)	How does potassium handling by the kidney change in response to changes in pH?		
POINTS REQUIRED	1 H and K are exchanged	1	
	2 Acidosis dec K excretion	2	
	3 H makes K move into circ, less for excret^{n}	3	
	4 Alkalosis increases K excretion	4	
	5	5	
	6	6	
	7		
THIRD QUESTION (if needed)	How does aldosterone increase K secretion?		
POINTS REQUIRED	1 Increased Na entry into cells	1 Effect on Na?	
	2 Inc pumping out of Na by Na-K pump	2	
	3 Inc K uptake into principal cells	3	
	4 Inc K conc inc secretion driving force	4	
	5 Also inc luminal membrane K channels	5	
	6	6	
	7		

Loop Of Henle 2013-1

Question 3 Renal Tubular Function LOA:	a. How do the ascending and descending limbs of the Loop of Henle differ in function?	Thin **descending limb water permeable** (aquaporins) and tubular **fluid becomes hypertonic**. **Thick ascending limb impermeable to water, and Na^+, K^+,Cl^- actively transported out, so fluid ends up more hypotonic**. K^+ diffuses back passively	Bold, illustrate clear difference
	b. Describe the process of tubuloglomerular feedback in the nephron.	This process **aims to maintain the constancy of the load delivered to the distal tubule**. The macula densa **in the ascending limb of the loop of Henle senses the rate of flow and feeds back to either increase or decrease the rate of filtration** in the glomerulus	Correct concept

Loop of Henle 2011-2

Question 3 LOA: 1	1) Describe the differences between the ascending and descending loops of Henle. 2) Describe the changes in the tonicity of tubular fluid as it moves along the loop of Henle.	1)Descending – **thin** cells; **permeable** to water, due to the presence of aquaporin-1 in both the apical and basolateral membrane. Ascending- proximal (thin): as above Ascending - distal (thick): **thick** cells containing many mitochondria; **impermeable** to water; co-transport of Na^+, K^+, Cl^- out of lumen into interstitium. 2) Fluid in the descending limb of the loop of Henle becomes **hypertonic** as water moves out of the tubule into the hypertonic interstitium. In the ascending limb it becomes more dilute because of the movement of Na^+ and Cl^- out of the tubular lumen. When fluid reaches the top of the ascending limb (the diluting segment) it is now **hypotonic** to plasma.	*Prompt:* differences in structure and function a)Bold to pass b)Need to describe the changes in tonicity.

Loop of Henle 2008-1

3.2 Loop of Henle, structure & function Ganong pp 700, 714-718	Please outline the structure of the Lof H	• Thin/descending, Thick/ascending. Situated mostly in the renal medulla • **Origin from PCT** • Short (cortical) and long (juxta med.)loops • **Macula densa at distal end, where joins DCT**	
	What happens to electrolytes in the loop	• **(Thin) Descending limb water permeable** • **Fluid becomes hypertonic as descends loop** • **(Thick) Asc limb impermeable to water, NaK Cl transported out, hypotonic at end, so K+ diffuses back** • **Active trans. ATPase**	
	Explain the counter-current concentrating mechanism	• Gradient • Exchange (vasa recta)	/2

Loop of Henle 2005-2

1.4 Loop of Henle – structure and function	Describe the structure of the Loop of Henle Describe the function of the Loop of Henle	**Thin descending, thin ascending, thick ascending** limbs. Cortical nephrons with short loops (85%) & juxtamedullary nephrons with long loops into medullary pyramids (15%). **Counter current multiplier**: maintains gradient of osmolality; requires **vasa recta as countercurrent exchangers** **Thin descending: high permeability to water**; it moves out of tubule into interstitium **Thin ascending: high permeability to NaCl**; it moves out of tubule into interstitium **Thick ascending: active transport Na, K, Cl**, from tubule to interstitium; impermeable to H2O	

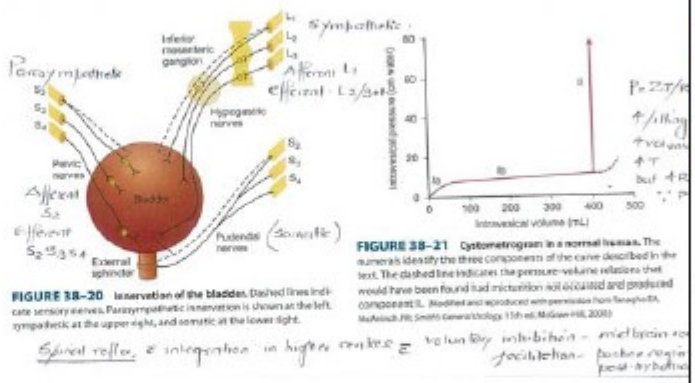
Micturition 2016-2-D

Stem: He has not passed urine for 12 hours. Moving onto Physiology.			
Question 5 Control of micturition. **Subject:** Physiology LOA: 2	a. Describe the neurological pathways involved in normal micturition.	**Sacral spinal reflex** mediated by S2, S3 and S4 nerve roots. **Facilitated and inhibited by higher centres;** **subject to voluntary control.** - First urge to void at 150ml. Marked fullness at 400ml - sudden rise in intra-vesical pressure triggers reflex contraction. Micturition reflex: - Stretch receptors in bladder wall. Afferent limb in pelvic nerves. **Parasympathetic efferent fibres** (via same pelvic nerves) mediate contraction of detrusor muscle. - Pudendal nerve (S2, S3 and S4) permits voluntary contraction of perineal muscles/external urethral sphincter, to slow or halt flow. - Sympathetic nerves to bladder play no role in micturition	To Pass: Spinal Reflex Parasympathetic control Higher centre control
	b. Describe the muscles involved in micturition. *Prompt: What is the bladder muscle called?*	1. **Bladder**: smooth muscle arranged in spiral, longitudinal and circular bundles. Circular bundle is called the **detrusor muscle.** Contraction of detrusor is responsible for involuntary emptying. **2. External urethral sphincter –** skeletal muscle sphincter of the membranous urethra. Relaxes during micturition. This is voluntarily controlled. 3. Perineal muscles. Relaxes during micturition. Also voluntarily controlled. 4. In males, urine left in urethra expelled by several contractions of bulbocavernosus muscle. 5. Contraction of abdominal wall muscles aids expulsion of urine. NB: Internal urethral sphincter (smooth muscle bundles passing on either side of urethra) plays no apparent role in micturition	Bold to pass

Micturition 2014-2-B

Stem: We will now move on to Physiology.			
Question 4 Micturition (pp 693-695) **Subject:** Phys LOA: 2	1. Describe the neurological pathways involved in normal micturition.	**Spinal reflex mediated by S2, S3 and S4 nerve roots. Facilitated and inhibited by higher centres; subject to voluntary control.** - First urge to void at 150ml. Marked fullness at 400ml - sudden rise in intra-vesical pressure triggers reflex contraction. **Micturition reflex**: - Stretch receptors in bladder wall. Afferent limb in pelvic nerves. **Parasympathetic efferent fibres** (via same pelvic nerves) mediate contraction of detrusor muscle. **- Pudendal nerve (S2, S3 and S4)** permits voluntary contraction of perineal muscles/external urethral sphincter, to slow or halt flow. - Sympathetic nerves to bladder play no role in micturition	To Pass: Spinal Reflex Parasympathetic control higher centre control
	2. Describe the muscles involved in micturition.	**1. Bladder**: smooth muscle arranged in spiral, longitudinal and circular bundles. Circular bundle is called the **detrusor muscle. Contraction of detrusor is responsible for involuntary emptying.** **2. External urethral sphincter – skeletal muscle sphincter of the membranous urethra. Relaxes during micturition. This is voluntarily controlled.** 3. Perineal muscles. Relaxes during micturition. Also voluntarily controlled. 4. In males, urine left in urethra expelled by several contractions of bulbocavernosus muscle. 5. Contraction of abdominal wall muscles aids expulsion of urine. NB: Internal urethral sphincter (smooth muscle bundles passing on either side of urethra) plays no apparent role in micturition.	Bold to pass
	3. What prevents vesico-ureteric reflux?	**Oblique passage of ureters through bladder wall** keeps ureters closed except during peristaltic waves.	Bold to pass

Micturition 2012-1

Question 3 LOA: 1	3.1 Describe the micturition reflex.	**Spinal reflex, voluntary facilitation/inhibition** from the higher centres. Micturition centre in the brain stem. Bladder innervation - **sympathetic** L1,2,3; **parasympathetic** S2,3,4; **somatic** S2,3,4.  **FIGURE 38–20** Innervation of the bladder. Dashed lines indicate sensory nerves. Parasympathetic innervation is shown at the left, sympathetic at the upper right, and somatic at the lower right. **FIGURE 38–21** Cystometrogram in a normal human. The numerals identify the three components of the curve described in the text. The dashed line indicates the pressure-volume relations that would have been found had micturition not occurred and produced component IL. (Modified and reproduced with permission from Tanagho EA, McAninch JW: Smith's General Urology, 15th ed. McGraw-Hill, 2000.) **Bladder muscle smooth** and plastic (explanation) **Initial urge at 150mls, fullness 400 mls.** **Detrusor muscle contracts.** **Perineal muscles/external urethral sphincter relax.** In females aided by gravity; in males contraction of bulbocavernous muscle	Need bold to pass – Innervation , sympathetic – inhibitory, parasympathetic- excitatory. Bladder distention, excitation of the mechanoreceptors, afferent projection to the brain stem and efferents via sympathetic, parasympathetic and somatic nerves. cystogram for additional marks Plastic – tension initially produced by filling (distension) is not maintained. P = 2T/R as T increases so does R, i.e. filling and distension therefore P remains constant

Micturition 2010-2

Question 3	Describe the Physiological process of Micturition Prompts: What muscles and nerves are involved?	• Spinal reflex facilitated and inhibited by higher centres • First urge to void at 150ml • Marked fullness at 400ml • During micturition, the Detruser muscle contracts and perineal muscles/external urethral sphincter relax • Parasympathetic(S2,3,4) afferents respond to stretch receptors in bladder wall to initiate reflex contraction via parasympathetic efferents. • Pudendal nerve to External Urethral Sphicter causes relaxation. • Spinal reflex integrated in sacral portion of spinal cord • Sympathetic (L1,2,3) play no role in micturition but only in prevention. EUS and perineal muscles can be controlled voluntary for a period of time but eventually void reflex overcomes voluntary control.	To Pass Spinal Reflex Parasympathetic control Voluntary Control

Micturition 2008-2

OPENING QUESTION	Describe the physiologic process of Micturition	COMMENTS
POINTS REQUIRED	1 **A spinal reflex inhibited and facilitated by higher centres** Intravesical pressure rises only after 400mls urine in bladder **Anatomy: Detrusor m, int and ext urethral sphincters** **During micturition Detrusor contracts, and perineal muscles and EUS relax.**	
	2.Nerve Supply **Parasympathetic (S 2,3,4,) via pelvic nn (afferent and efferents) to/from detrusor (efferent contraction) and pudendal nn to EUS (relaxation)** **Sympathetic** (L1,2,3) – Hypogastric nn via Inf Mesenteric Ganglion play no role in active micturition per se but role in prevention. (cause contraction of bladder muscle to prevent reflux of semen into bladder during ejaculation)	
	4. Initiation – remains unsettled, pelvic floor muscle relaxation initiates. Perineal muscles and EUS can be contracted voluntarily for prolonged periods. Bladder SM has intrinsic contractile activity Post urination, female urethra empties by gravity. Male expels by contraction of bulbocavernosus m	(optional/extra detail)
PROMPTS	What muscles and nerves are involved?	
SECOND QUESTION (if needed)	**List other factors that stimulate and inhibit micturition**	
POINTS REQUIRED	**1. Stimulants –** **a) Stretch/pressure** (intravesical volume > 400mls) **b) Higher centre input** **c) Parasympathetics** (eg organophosphates) **d) Sympathetic inhibiting drug**(eg a-blockers) **e)** Voluntary abdominal muscle contraction augments stream but does not initiate micturition per se	**(3 of 5 to pass)**
	2. Inhibitors **a) Parasympathetic inhibitors (atropine)** **b) Higher centres** **c) Sympathomimetics**	**(b) + one other**
PROMPTS	a) What is the effect of autonomic agents on micturition ? b) What non autonomic precipitants and inhibitors do you know?	(optional)

Micturition 2007-2

Question	Required response [Key items marked with*]	To Pass
Please describe how the urinary bladder empties Prompt ; Could you describe the relationship between pressure and volume in the bladder as it relates to bladder emptying	1)Smooth muscle of the bladder is arranged in spiral, longitudinal, and circular bundles 2)Contraction of the circular muscle, **(detrusor muscle),** is mainly responsible for emptying the bladder during urination ** 3)Micturition is fundamentally a **spinal reflex** facilitated and inhibited by higher brain centers and, like defecation, subject to voluntary facilitation and inhibition ** 4)Urine enters the bladder without producing much increase in intravesical pressure until the viscus is well filled 5) The bladder muscle has the property of plasticity; when it is stretched, the tension initially produced is not maintained 6) The curve shows an initial slight rise in pressure when the first increments in volume are produced; a long, nearly flat segment as further increments are produced; and a sudden, sharp rise in pressure as the micturition reflex is triggered 7) The first urge to void is felt at a bladder volume of about 150 mL, and a marked sense of fullness at about 400 mL. 9) The flatness of segment Ib is a manifestation of the law of Laplace which states that the pressure in a spherical viscus is equal to twice the wall tension divided by the radius. In the case of the bladder, the tension increases as the organ fills, but so does the radius. Therefore, the pressure increase is slight until the organ is relatively full . 11) **During micturition, the perineal muscles and external urethral sphincter are relaxed; the detrusor muscle contracts; and urine passes out through the urethra. **** 12)The mechanism by which voluntary urination is initiated remains unsettled. One of the initial events is relaxation of the muscles of the pelvic floor, and this may cause a sufficient downward tug on the detrusor muscle to initiate its contraction. 14)The perineal muscles and external sphincter can be contracted voluntarily, preventing urine from passing down the urethra or interrupting the flow once urination has begun.	2 3 11 Basic understanding the process in an organised fashion
Describe the reflex control associated with voiding	1)The bladder smooth muscle has some inherent contractile activity; however, when its nerve supply is intact, stretch receptors in the bladder wall initiate a reflex contraction that has a lower threshold than the inherent contractile response of the muscle. **2)Fibers in the pelvic nerves are the afferent limb of the voiding reflex, and the parasympathetic fibers to the bladder that constitute the efferent limb also travel in these nerves. **3)The reflex is integrated in the sacral portion of the spinal cord. **4) In the adult, the volume of urine in the bladder that normally initiates a reflex contraction is about 300–400 mL. **5) The sympathetic nerves to the bladder play no part in micturition, 6) They do mediate the contraction of the bladder muscle that prevents semen from entering the bladder during ejaculation	Parasympathethetic reflex Sacral portion of cord Vol to trigger 300 – 400mls

Nephron 2010-2

Question 2	Can you draw a nephron and describe the functions of each part	• Glomerulus - filtration • Afferent arteriole(contain juxtaglomerular cells – secrete renin) then capillary tuft then efferent arteriole encapsulate in bowmans capsule • PCT- resorption of most solute – Na, glucose, aa, reclaim HCO3 • Desc limb of LOH – thin, water permeable • Thick Asc LOH – site of Na K 2 Cl – generates concentration gradient • DCT – site of Na K Cl pump • Proximal part is the macula densa forms juxtaglomerular apparatus - • CD- p cells - under control of ADH and aldosterone(water and Na resorption) • I cells – involved in H+ excretion	To Pass: Draw basic shape and label 1 function of each part

Nephron 2007-1

TOPIC: Functional anatomy of the nephron _____ **NUMBER:** _____

OPENING QUESTION	Describe the cell types in the glomerulus and their functions ?	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	1 Capillary endothelial cells – Afferent arteriole becomes a tuft of capillaries invaginated into Bowman's capsule. Endothelium fenestrated with 70-90 nm pores. Separated from capsule epithelium by basal lamina.	What types of cells lie between blood and the capillary and filtrate in Bowman's capsule? What are their functions?	Need fenestrated capillary membrane.
	2 Epithelial cells of Bowman's capsule: (a) Podocytes possess pseudopodia that interdigitate to form 25 nm wide filtration slits over capillary endothelium. Each slit is closed by a thin membrane. (b) Mesangial cells are stellate and lie between capillary endothelium and basal lamina. Involved in regulation of filtration, secretion of various substances and absorption of immune complexes.		Need podocytes with pseudopodia forming filtration slits.
SECOND QUESTION	What properties of substances in the blood prevent free passage across the glomerular membrane?		Need both
POINTS REQUIRED	1 Larger diameter > 8 nm		
	2 Lack of neutrality (charged)		

Regulation of ECF Volume 2014-2-A

Stem: Her BP is low.			
Question 3 Renal response to hypovolaemia (pp 701-706) **Subject**: Phys LOA: 1	1. Explain how hypotension activates the renin-angiotensin system.	1. Hypotension leads to **reduced perfusion pressure of the afferent glomerular arteriole**, stimulating release of **renin** by the juxtaglomerular cells.	1. Bold to pass.
	2. How does the renin-angiotensin system contribute to the restoration of the blood volume?	2. Renin **converts angiotensionogen to angiotensin I. Angiotensin converting enzyme** converts AG1 to **angiotensin II.** Ang II acts on the adrenal cortex's zona glomerulosa cells to **release aldosterone**. Aldosterone acts on the renal distal tubules to **retain Na and water**, thus increases intravascular volume. Ang II also a potent arteriolar constrictor and contributes to a rise in blood pressure.	2. 4/5 bold to pass.
	3. What other factors increase renin secretion?	3. Renin (protease) release is stimulated by increases in: **catecholamines, sympathetic activity** through renal nerves, **prostaglandins**, low Na states: cardiac failure, liver failure and Na depletion.	3. 1/3 bold to pass.

Regulation of ECF Volume 2014-1-D

Stem: Moving onto **PHYSIOLOGY**			
Question 4 Renal response to dehydration **Subject:** Phys LOA: 1	(a) What is the renal response to dehydration?	(a) **Renin release, converts a-gin to AT1** **ACE converts AT1 to AT2** **AT2 increases aldosterone synthesis, vasoconstriction of aff arteriole** **Aldo - Na and water retention**	(a) Need details re secretion i.e. reduced pressure at JG cells of renin and actions of A-2
	(b) What is the role of vasopressin in dehydration?	(b) Promotes **water resorption** in CD via **aquaporins** insertion. **Vasoconstriction**	(b) Bold to pass

Regulation of ECF Volume 2005-1

Cardiovascular compensations for fluid overload	What hormone systems are involved in the maintenance of Extracellular fluid volume? What are the effects of Atrial Naturetic Peptide in response to fluid overload ?	Renin, angiotensin aldosterone/vasopressin Increase sodium secretion from the kidneys Diuresis	

Renal Blood Flow 2017-2-D

Stem: Moving on to Physiology. He has reduced urinary output.			
Question 4 Renal blood flow **Subject:** Phys LOA: 1	(a) What is the normal renal blood flow?	**1.2 – 1.3 l/min (~25% of C.O.)**	Either bold
	(b) Describe the factors which determine renal blood flow.	**Perfusion pressure** (systemic MAP) **Renal arterial effects** (local constriction from NA and Ang II, dilation from ACh, PGs, dopamine) **Renal nerves** (symp/constrict/decr RBF) **Autoregulation** (myogenic, NO, Ang II), BONUS Regional differences cortex to medulla	3 of 4 bold
	(c) How does hypotension activate the renin-angiotensin system? *Prompt: what stimulates renin release?*	Hypotension leads to **reduced perfusion pressure of the afferent glomerular arteriole**, stimulating release of **renin** by the JG cells	Bold concept

Renal Blood Flow 2012-2

Question 3 RBF LOA: 1 RBF	1.What is normal renal blood flow (L/min)? 2. Describe the mechanisms which determine renal blood flow.	**1.2 – 1.3 L/min** (25% of C.O.) at rest Perfusion pressure (**systemic MAP**); **renal arterial flow** (local constriction from NA & Ang II, dilatation from Ach, PGs, dopamine); **Renal nerves** (stim of sympath → NA → decreased RBF); **Autoregulation** (in part due to direct smooth muscle contractile response to stretch of the afferent arteriole; NO; Ang II has a role at low perfusion pressures); **Regional** differences in RBF (greatest at cortex, less in inner medulla)	Must say 3 of 5

Renal Blood Flow 2010-2

Question 3: Renal blood flow	1) what is the normal RBF 2) How is renal blood flow regulated	1) RBF @ 1250ml/min, 25% CO 2) a)neuroendocrine; NA constrictor, DA dilator, Angio2 constrictor, PG's (incr cortical decrease medulla), ACH dilates b) autoregulation, probably vessel wall stretch reflex as occurs in denervated isolated vessels c) renal nerves.	1) approximate value 2) Must have 2 of DA, NA, AgtII, autoreg

Renal Blood Flow 2008-2

OPENING QUESTION	**What is normal renal blood flow and how can it be measured?**	**COMMENTS**
POINTS REQUIRED	1. **Fick principle** (amount of a substance taken up per unit time divided by arterio-venous concentration difference)	
	2. **PAH (excreted, not metabolised or stored, doesn't affect flow) is used to measure effective renal plasma flow (90% cleared)** ERPF = Clearance of PAH = UV/P = 630 mL/min	2 of 3
	3. Actual renal plasma flow = ERPF/0.9 = 700 mL/min	
	4. Renal blood flow = RPF x 1/1-Hct (Hct = 0.45)	
	5. **Renal blood flow = approx 1250 mL/min**	
PROMPTS	What substance can be used to measure renal plasma flow?	
SECOND QUESTION (if needed)	**How do blood flow and oxygen extraction vary in different parts of the kidney?**	
POINTS REQUIRED	1. **Cortical flow is high** (5 mL/gm of tissue) and oxygen extraction is low	
	2. **Medullary blood flow is low** (2.5 mL/gm in outer cortex, 0.6 mL/gm in inner cortex) and oxygen extraction is higher (more metabolic work done)	2 of 3
	3. **Medulla is vulnerable to hypoxic damage** if flow is reduced (low flow, high oxygen usage)	
PROMPTS	How much blood flows to the renal medulla?	

COMMENTS

Renal Blood Flow 2008-1

2.2 Renal blood flow Ganong pp 702-705	What is a typical value for renal blood flow in an adult at rest? What factors regulate renal blood flow?	**~25% of cardiac output or 1250 ml/min** **Chemical**: Noradrenaline constricts interlobular and afferent arterioles. **(3 of 5)** Dopamine causes renal vasodilation. Angiotensin II constricts efferent arterioles to a greater extent than the afferent arterioles. Prostaglandins increase blood flow in the cortex and decrease blood flow in the medulla. Acetylcholine produces renal vasodilation. **Neural**: Strong stimulation of the sympathetic nervous system produces renal vasoconstriction. **Autoregulation**: Direct contractile response of smooth muscle of afferent arteriole to stretch. NO may be involved. **At low perfusion pressures angiotensin II plays a role in constricting efferent arterioles.**	/2

Renal Blood Flow 2004-2

TOPIC: The factors in the control of renal blood flow ____ **NUMBER:** _____

OPENING QUESTION	What determines renal blood flow?	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	Systemic blood pressure		
	Renal vascular resistance, which is in turn influenced by:		
	Catecholamines (nerves & systemic)		
	Angiotensin II (JG cells -> renin)		
	Prostaglandins		
	Control systems:		
	Renal autoregulation (myogenic-stretch response, vasodilator metabolites, ?NO, ?prostaglandins)	What control systems influence renal blood flow?	
	JG apparatus		
	Renal sympathetic nerves		
SECOND QUESTION (if needed)	What are the consequences of a sustained reduction of renal blood flow?		
POINTS REQUIRED	(Renal blood flow maintained MBP >70)		
	Medulla is vulnerable to hypoxia (high MR)		
	ATN		
	Uraemia		
THIRD QUESTION (if needed)			
POINTS REQUIRED			

COMMENTS

Renal Circulation 2015-2-A

Stem: Moving onto Physiology			
Question 3 Renal Circulation **Subject:** Phys LOA: 1	What is normal renal blood flow?	**Renal blood flow = approx 1250 mL/min**	Bold (accept 1000 – 1500)
	What substances influence renal blood flow and how?	Noradrenaline-constriction, Dopamine, ACh -dilatation Angiotensin II – constricts afferent and efferent arterioles PGs-increase flow in cortex and decrease in medulla	2/5 substances + correct action
	How can renal blood flow be measured? Prompt: What substance can be used to measure renal plasma flow?	1. Fick principle (amount of a substance taken up per unit time divided by arterio-venous concentration difference) 2. PAH (or any substance that is excreted, not metabolised or stored, doesn't affect flow) is used to measure effective renal plasma flow (90% cleared) ERPF = Clearance of PAH = UV/P = 630 mL/min 3. Actual renal plasma flow = ERPF/0.9 = 700 mL/min 4. Renal blood flow = RPF x 1/1-Hct (Hct = 0.45)	Concept/Principle

Renal Failure 2015-1-A

Stem: We will now move to Physiology. Initial urea and electrolytes show renal failure			
Question 3 Effects of disordered renal function (pp 692-693) Subject: Phys LOA: 1	What are the major physiological features of acute intrinsic renal failure? *(prompt: what happens to urine concentration?)*	**Loss of urine concentrating and diluting** capacity due to loss of countercurrent mechanism and nephron number. **Polyuria →oliguria → anuria** **Uraemia** due to urea and creatinine and toxins (phenol and acids) build up. **Acidosis.** Anaemia **Na+ retention** and oedema and heart failure	3/5 bold ones
	What are common findings in urinalysis of acute intrinsic renal failure?	**Proteinuria, leucocytes, red cells and casts**	3 bold
	What are urinary casts?	**Proteinaceous** material precipitated in tubules washed into bladder.	Bold

Renin 2013-1

Question 3 Renin- Angiotensin System LOA: 1	a. What are the actions of the renin-angiotensin system?	**Mediated through AT II;** - arteriolar constriction with **rise in SBP and DBP; increases secretion of aldosterone;** facilitates release of NAd acting on post-ganglionic neurones; positive feedback loop on brain by decreasing sens. to baroreflex and increase effect of AT II, and secretion of vasopressin and ACTH	**Bold**
	b. What factors affect renin secretion?	**Stimulation: sympathetic activity** via renal nerves; increased circ. **Catecholamines;** prostaglandins **Inhibition:** - **increased Na and Cl reabsorption** across macula densa; **- increased afferent arteriolar pressure;** AT II; vasopressin	**Bold**

Renin 2012-2

Question 3 Renin secretion LOA: 1	1.What physiological factors are involved in regulating renin secretion?	1. **Intrarenal baroreceptors**- An increase of afferent arteriolar pressure at the JG cells causes a decrease in rennin secretion (and vice versa) 2. **Amount of Na and Cl** entering the distal tubules in the macula densa cells(increase in NaCl causes a decrease in rennin secretion (? NO mediated)) 3. Plasma K level (probably thru NaCl effect) 4. Angiotensin II/Vasopressin (inhibitory) 5. **Increase in sympathetic Nervous system** 6. Catecholamines and norepinephrine 7. Prostaglandins	1-4 inhibit rennin secretion 5-7 stimulate renin secretion
	2. What conditions increase renin secretion?	Sodium depletion Dehydration Diuretics Cardiac failure Hypotension Cirrhosis Haemorrhage Constriction renal Artery Upright position Constriction of aorta Various psychological stimuli	**3 conditions to pass**

Renin 2004-2

TOPIC: Renin-angiotensin system _____ **NUMBER:** _____

OPENING QUESTION	Describe how the renin-angiotensin system regulates blood pressure and flow.	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	1 Describes pathway	1	
	2 Fall in renal blood flow leads to renin	2	
	3 Renin, angiotensin I > II	3	
	4 Vasoconstrictor	4	
	5	5	
	6	6	
SECOND QUESTION (if needed)	What factors regulate renin secretion?		
POINTS REQUIRED	1 Stim: sympathetic nervous system, catechols, prostaglandins	1	
	2 Inhib: Na and Cl reabsorp, inc BP, angio II, vasopressin	2	
	3	3	
	4	4	
	5	5	
THIRD QUESTION (if needed)	6	6	
POINTS REQUIRED	1	1	
	2	2	
	3	3	
	4	4	
	5	5	
	7		

Renin 2003-2

TOPIC: Renin Secretion _____ **NUMBER:** _____ 3b

OPENING QUESTION	**What physiological factors affect renin secretion**	**How do they affect secretion?**	Steer away from the clinical conditions – may need prompt to do this
POINTS REQUIRED	1 **Afferent arteriolar pressure** – increased pressure at the level of JG cells in kidney causes decrease in renin secretion & vice versa	1	**Must have to pass**
	2 **Na & K transport across macula densa** – increased reabsorption leads to decreased renin secretion & vice versa	2	**Must have to pass**
	3 **Angiotensin II** – inhibitory feedback to JG cells	3	**Must have to pass**
	4 **Circulating catecholamines** – increased SNS activity increases renin	4 How does SNS activity affect renin secretion	
	5 **Other – Prostaglandins** – increases renin; **vasopressin** – decreases renin	5	
	6	6	
SECOND QUESTION (optional)	Please give 2 clinical conditions which increase renin secretion and by what mechanism they work		

Renin Angiotensin System 2016-2-B

Stem: Moving onto Physiology			
Question 3 Renin angiotensin system **Subject** Phys LOA: 1	1 What leads to activation of the renin-angiotensin system. Prompt "List some conditions which activate the renin-angiotensin system" 2 What are the principal effects of angiotensin II? Prompt Where does angiotensin II act?	**Activated in response to decrease in BP/ ECF or increased sympathetic activity** eg hypotension, haemorrhage, dehydration, cardiac failure, cirrhosis, Na depletion, diuretics, upright posture, pain, fear, arousal **Arterioles** (AT1 receptor) – **vasoconstriction** – increases TPR **Adrenal cortex - increase aldosterone production – increased Na and H2O resorption** Kidney – direct effect to decrease GFR and increase Na reabsorption Brain – decreased sensitivity of brain baroreceptor reflex – potentiates pressor effect Pituitary – increase ADH and increase ACTH scretion	Bold + 4 conditions Bold to pass Prompt "What causes that or what effect does that have?"

Renin Angiotensin System 2016-1-B

Stem: Moving onto Physiology. His blood pressure is low.			
Question 5 Renin- angiotensin system. LOA: 1	*a. List some conditions which activate the renin-angiotensin system.*	Activated in response to decrease in BP / ECF volume or increased sympathetic activity. Examples: Hypotension, haemorrhage, dehydration, cardiac failure, cirrhosis, Na^+ depletion / diuretics, upright posture. Pain, fear and arousal (fight, fright, flight) may trigger it too.	4 conditions
	b. What are the principal effects of angiotension II? *Prompt: "Where does angiotensin II act?"*	**Arterioles (AT_1 receptor)** : **vasoconstriction** → ↑TPR. **Adrenal cortex (AT_1 receptor)** : **↑aldosterone** production → **↑Na^+ / H_2O reabsorption** Kidney : direct effect to ↓GFR & ↑Na^+ reabsorption. Brain : ↓sensitivity of brain baroreceptor reflex → potentiates pressor effect Pituitary : ↑ADH & ↑ACTH secretion.	Bold to pass

Renin Angiotensin System 2003-1

TOPIC: Renin-angiotensin system **NUMBER:**

OPENING QUESTION	How does the renin-angiotensin system respond to hypotension?	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	1. With a drop in BP, renin is released from the JG cells and act on a renin substrate to form angiotensin I, which is converted to angiotensin II in the lung. Angiotensin II causes vasoconstriction and decrease the excretion of both salt and water (long term effect).	1	
	2.	2	
SECOND QUESTION (if needed)	What are the other effects of the renin-angiotensin system?		2/5
POINTS REQUIRED	1. Salt and water retention	1	
	2. Stimulate aldosterone secretion	2	
	3. Faciliate the release of noradrenaline	3	
	4. Downgrade the baro-receptors	4	
	5. Increase the secretion of vasopressin	5	

Response to fluid 2016-1-A

Stem: A 78 year old lady is found collapsed at home on the floor. She is found to be dehydrated. Starting with Physiology.....			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Response to fluid bolus **Subject:** Phys LOA: 1	What are the physiological effects of dehydration?	Water loss lowers ECF and ICF leading to **↓BP, ↑HR, ↑ADH, ↓UO**, ↓GFR, ↑Renin/Angiotensin, ↑Thirst aiming to maintain IV volume. In adrenal insufficiency Na is lost not only in urine but also into cells.	Na movement is key. Bold elements.
	Describe the effects of a rapid IV infusion of 1000 ml of Normal Saline. Prompt: what is the cardiovascular effect you would see?	↑Cl and acidosis, ↑Baroreceptor firing, **↓HR, ↑BP,** increased UO, ↓Renin/Angiotensin and improved capillary return. (Bainbridge reflex described initial increase HR if slow initially)	Bold plus one more.
	What is an alternative physiological fluid replacement?	**Hartmann's** (lactated Ringer's) or **Plasmalyte**.	1 of these.

Sodium Absorption 2017-2-A

Stem: Moving on to Physiology of the kidney.			
Question 5 Renal regulation sodium **Subject**: Physiology LOA 1	a) Where does Na reabsorption occur in the nephron?	a) **Primarily** (60%) **PCT** by Na-H exchange but also a range of co-transport (gluc, AA, lactate) - 30% thick asc limb of LoH (Na –K-2Cl co-transporter, Na-H exchange) - Nil at thin part of LoH - 7% DCT (NaCl co-transporter) - 3% collecting ducts through Na channels (ENaC)	2 out of 5 described correctly including bold to pass
	b) How is Na transported from the tubular cell into the interstitium?	b) **Na/K ATPase active transport.** (3Na/2K) across basolateral membrane predominantly into the lateral intercellular spaces.	Bold
	c) Following high Na intake, what mechanisms act to enhance Na excretion? *Prompt: What mechanisms reduce Na reabsorption?* *Prompt: Can you describe any mechanisms mediated outside the kidney?*	c) A slight increase in ECF occurs triggering various mechanisms: - stretch receptors in RA and pulm veins → inhibits sympathetic outflow to kidneys → decreased Na reabs - small increase in arterial pressure → pressure natriuresis - suppression AT-II formation - reduced aldosterone secretion secondary to reduced AT-II formation - stimulation of ANP	2 mechanisms

Sodium Absorption 2011-1

Question 3	3.1 Describe how Na^+ is handled by the kidney.	• Glomerular filtration • Actively transported out of the tubule in all sections EXCEPT descending thin limb of loop of Henle. • 96-99% reabsorbed overall • Reabsorption influenced by changes in GFR and in tubular reabsorption (MAINLY in the 3% reaching collecting ducts) – circulating aldosterone, other adrenocoticoids, circulating ANP and other natriuretic hormones and rate of tubular secretion H+ and K+.	Filtration Active re-absorption Need to understand concepts
	3.2 How does aldosterone influence renal sodium handling?	• Increased tubular reabsorption of Na+, with secretion of K+ and H+ • Latent period of 10 – 30 minutes before effect (time delay due to need to alter protein synthesis via action on DNA) • Act principally on the collecting ducts to increase number of active epithelial sodium channels	Increased reabsorption

Sodium Absorption 2010-1

<table>
<tr>
<td>3 a). What general mechanisms are involved in renal tubular reabsorption and secretion?</td>
<td>Mechanisms involved in re-absorption and secretion include endocytosis, passive diffusion and facilitated diffusion and active transport.</td>
<td>2 of Bold to Pass</td>
</tr>
<tr>
<td>3 b). How is Sodium reabsorped in the various parts of the nephron?</td>
<td>No sodium transport in Thin descending Loop of Henle.
In rest of system, sodium moves by co-transport, exchange or down concentration gradient.
Sodium pumped out of cell by Active Sodium-Cl-Potassium pump in basolateral membrane.
60% in PCT by Sodium-Hydrogen exchange.
30% in thick ascending Limb via Sodium –Potassium co-transport.
7% in DCT via Sodium-Chloride exchange
<table>
<tr><th>Site</th><th>Apical Transporter</th><th>Function</th></tr>
<tr><td>Proximal tubule</td><td>Na/. . . . CT</td><td>Na$^+$ uptake, . . . uptake</td></tr>
<tr><td></td><td>Na$^+$/P$_i$ CT</td><td>Na$^+$ uptake, Pi uptak</td></tr>
<tr><td></td><td>Na$^+$ . . . . CT</td><td>Na$^+$ uptake, ... uptake</td></tr>
<tr><td></td><td>Na/lactate CT</td><td>Na$^+$ uptake, lactate uptake</td></tr>
<tr><td></td><td>Na/H exchanger</td><td>Na$^+$ uptake, H$^+$ extrusi</td></tr>
<tr><td></td><td>Cl/base exchanger</td><td>Cl$^-$ uptake</td></tr>
<tr><td>Thick ascending limb</td><td>Na–K–2Cl CT</td><td>Na$^+$ uptake, Cl$^-$ uptake, K$^+$ uptake</td></tr>
<tr><td></td><td>Na/H exchanger</td><td>Na$^+$ uptake, H$^+$ extrusio</td></tr>
<tr><td></td><td>K$^+$ channels</td><td>K$^+$ extrusion (recycling)</td></tr>
<tr><td>Distal convoluted tubule</td><td>. . CT</td><td>Na$^+$ uptake, Cl$^-$ uptake</td></tr>
<tr><td>Collecting duct</td><td>Na$^+$ channel (ENaC)</td><td>Na$^+$ uptake</td></tr>
</table>
</td>
<td>Bold to pass, demonstrating reasonable understanding of different processes</td>
</tr>
</table>

Sodium Absorption 2009-2

Question 3: a)	**Describe how sodium is handled in the glomerulus and the PCT**	**Most Filtered** out with solutes/ AAs (90%) **Most (60%)** Na-H counter-transport, **Bicarbonate** is main anion reabsorbed with Na Absolutely depends on Na K ATP ase (Basement M)/ C Anhydrase-tub cell to generate H+/ Bic Small co-transport with nutrients /anions/ Cl latter part Approx 60%	NB – good candidates will volunteer Na resorbtion through out except TALH, 60/30/7/3 % - all Na excretion last 3%
b)	**List the mechanisms that effect Na reabsorption**	1)Tubulo-glom - Macula Densa,↑Na↑adenos/ Ca, aff vasocon 2)Glomer/tub balance- > filtered = > resorbed (good capacity)- mainly oncotic p in eff capillaries 3) Humeral Aldosterone- distal CT / ENaC, K+/H+ PGE2 – pron Na K ATP ase block/ Ca ++ > Ouabain endog- ATP ase block effect Endothelin and IL-1 cause natriuresis (prob > PGE2) ANP-↑ cGMP – less ENaC Angio 2- renal ACE ↑circ Ang 1 + renal -↑ PCT > reabs	**Reqd :1 humeral / 1other**

Sodium Absorption 2009-1

Question 3: Renal regulation of sodium Ganong pp 709-10, 723-4	i) Where does sodium reabsorption occur in the nephron?	a) All parts of the nephron except thin part of the LoH (+Specify at least two of:) b) 60% **PCT primarily** by Na^+-H^+ exchange but also a range of cotransport (glc, Pi, AA, lactate) c) 30% thick ascending limb of LoH (Na^+-$2Cl^-$-K^+ cotransporter) d) 7% DCT LoH (Na^+-Cl^- cotransporter) e) 3% collecting ducts through Na+ channels (ENaC)	Must get **bold** to pass.
	ii) What are the mechanisms of sodium re-absorption in the nephron?	• **Na/K ATPase active transport.** Moves (by gradient thus generated) across apical membranes from tubular lumen into cell via cotransport & exchanger proteins. Driven by active transport by Na-K ATPase (3Na/2K) from tubular cell into interstitium (mainly into lateral interstitial spaced)	
	iii) What mechanisms in the kidney reduce sodium excretion? Prompt (if they get it ar**-about-t**) *What mechanisms in the kidney cause the body to retain sodium by reducing sodium excretion?*	Multiple regulatory mechanisms (reflects importance of Na as the prime determinant of ECF volume) • **Reduced GFR** • Increased tubular reabsorption ○ ↑**adrenocortical hormones esp. aldosterone** - act primarily on collecting ducts (activation of ENaC) ○ ↓ANP (inhibit ENaC) ○ AT-II (PCT) ○ [↓secretion of K^+ and H^+]	Must get **bold** to pass.

Sodium Absorption 2006-1

TOPIC: Renal sodium & potassium excretion **NUMBER: 3**

OPENING QUESTION	What are the major physiological factors affecting sodium excretion from the kidney?	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	1. Amount filtered versus amount reabsorbed, therefore 2. ECF, 3. GFR, 4. Na intake, 5. hormonal e.g. aldosterone angiotensin and K and H excretion	How does the kidney regulate sodium excretion?	4 to pass
SECOND QUESTION (if needed)	What are the major physiological factors affecting potassium excretion from the kidney?		2 to pass
POINTS REQUIRED	1. K is reabsorbed in PTs and secreted in distal tubule, 2. Amount secreted relates to tubular flow, 3. Na excretion or reabsorption, 4. K intake	How does the kidney regulate potassium excretion?	

COMMENTS

Sodium Absorption 2005-1

Renal regulation of sodium balance.	How is the secretion of renin regulated?	**Stimulatory:** Increased sympathetic nervous activity ; Increased circulating catecholamines; Prostaglandins **Inhibitory:** Increased Na^+ and Cl^- reabsorption across the macula densa; Increased afferent arteriolar pressure; Angiotensin II; Vasopressin. Three of the above with one from each.	
	Describe the juxtaglomerular apparatus	Afferent and efferent arterioles and tubule touch at one point.. Macula densa and juxtaglomerular cells.	

Thirst, ADH, tonicity 2016-1-C

Stem: Moving onto Physiology			
Question 2 Tonicity, vasopressin **Subject:** Phys LOA: 1	1. Describe the process by which extracellular fluid tonicity is regulated. *(Prompt lead in: where is plasma osmotic pressure sensed?)*	As plasma osmotic pressure rises → **thirst ↑** + sensed via **osmoreceptors** in anterior hypothalamus (mainly organum vasculosum of the lamina terminalis OVLT) → **vasopressin (ADH) secretion rises** (from posterior pituitary) → renal **V_2 receptor stimulation** → insertion of water channels (**aquaporins)** in luminal membranes of renal collecting tubules, allowing more water to return to body. Conversely as plasma osmotic pressure falls (285mosm/kg is the critical point) ADH secretion suppressed.	Thirst increases water intake. ADH reduces water excretion by kidneys -> dilution of ECF Bold + correct understanding of concept to pass
	2. What factors *other* than rising osmotic pressure increase vasopressin secretion?	**Decreased ECF vol**, **pain**, emotion, surgical stress, exercise, nausea & vomiting, standing, angiotensin II, meds (clofibrate & carbamazepine).	Bold + 1 more to pass

Tubular function 2016-2-D

Stem: The patient has an acute kidney injury from his sepsis. Moving onto Physiology.			
Question 5 Renal tubular function **Subject:** Physiology LOA 1	a. In the renal tubules, what are the mechanisms of reabsorption & secretion? b. What are the main mechanisms for Na reabsorption in the renal tubule? *Prompt: In the proximal renal tubule, what other transport proteins are involved in the movement of sodium and chloride across the apical membrane?*	**Co-transporters** (sec active transport), **exchangers**, ion channels, pumps, endocytosis, passive diffusion, facilitated diffusion, active transport **Prox tubule** 60% reab, mostly **Na/H exchanger** Thick AL 30% reab, mostly Na K 2Cl Co-transporter DCT: 7% reab, mostly Na Cl CT Prox: **Na/glucose** CT, Na/phosphate CT, Na/lactate CT, Na/amino acid CT, **Na/H** exchanger, Cl/base exchanger	Bold + 1 other to pass Bold to pass Bold to pass (Note Na/K ATPase is in basolateral membrane, & Na channel is in collecting duct)

Tubular function 2007-1

TOPIC: Tubular function_____ **NUMBER:** _____

OPENING QUESTION	What factors influence clearance of substances by the kidney?	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	1 Amount of substance excreted = amount filtered + net amount transferred		Need 3.
	2 Changes in RBF and systemic BP		
	3 Active transport (primary and secondary)		
	4 Hormonal (aldosterone, angiotensin, endothelin)		
SECOND QUESTION	Explain the mechanism of tubuloglomerular feedback.		Explain feedback mechanism
POINTS REQUIRED	1 Increased rate of flow in LoH and DCT increases GFR and local Na^+		
	2 Macula densa adenosine A1 receptors activated by increased Na^+/K^+ activity, causing increased Ca^{2+}, vasoconstriction and decreased GFR		
	3 % solute reabsorbed remains constant (glomerulotubular balance)		

Vasopressin 2011-2

Question 3 LOA: 1	1) How does vasopressin act on the kidney? 2) What hormonal changes occur after drinking a large amount of water?	1) In the **collecting duct**, ADH binds to G-receptor V2 activates adenylate cyclase ↑ IC c-AMP → migration of IC endosomes H_2O channels (**aquaporin-2**) inserted into luminal membrane **↑H_2O permeability**, with ↑H_2O reabsorption 2) Begins about 15 min after ingestion. Maximum in about 45 min. The act of drinking produces a small decrease in **ADH** (vasopressin) secretion **resulting in dieresis**. Most of the inhibition is produced by the **decrease in plasma osmolality** after the water is absorbed.	Need bold to pass

Vasopressin 2004-2

TOPIC: Control of osmolality _____ **NUMBER:** _____

OPENING QUESTION	What is THIRST, and what causes it?	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	An appetite, under hypothalamic control		
	Increased plasma osmolality ▪ osmoreceptors in anterior hypothalamus		
	Hypovolaemia ▪ Renin-angiotensin system ▪ Baroreceptors in heart and blood vessels	Are there others that might involve higher centres?	
	Prandial ▪ Learned or habit response ▪ Osmolality & GI hormone effects		
	Psychogenic		
	Dry pharyngeal mucous membranes		
SECOND QUESTION (if needed)	What are the actions of vasopressin (ADH), and what influences secretion of this hormone?		
POINTS REQUIRED	ACTIONS: ▪ Retention of water by kidney (collecting duct permeability), thus decreasing blood osmolality ▪ V2 receptors -> insertion of aquaporin-2 (water channel proteins stored in endosomes) into cell membranes ▪ Decreased cardiac output (via area postrema) ▪ Vasoconstriction via V1 receptors ▪ Glycogenolysis ▪ ACTH secretion from ant pituitary	What is the cellular mechanism of action in the kidney? Anything else?	
	SECRETION INFLUENCED BY: ▪ Osmolality ▪ ECF volume (low pressure receptors in great veins, atria and pulmonary vessels, high pressure receptors in carotid sinuses and aortic arch) ▪ Increased secretion with high osmolality &/or low ECF Volume, and visa versa ▪ Pain, nausea, surgical stress and some emotions increase secretion ▪ Alcohol decreases secretion	How does the body detect and respond to changes in ECF volume? Anything else?	

Vasopressin 2003-2

TOPIC: ADH release _____ **NUMBER:** _____ 3a

OPENING QUESTION	What factors affect Antidiuretic Hormone (ADH) secretion	PROMPTS ADH is also known as vasopressin. What stimulates vasopressin secretion? Prompt to get the list	COMMENTS Must be able to distinguish the most important, esp. osmotic pressure
POINTS REQUIRED	1 **Plasma osmotic pressure** – increase POP leads to increase ADH secretion. Sensitive to changes around 285mosm/kn\g	1 how does that affect it?	Must have to pass
	2 **Extracellular fluid volume** – inverse relationship between ADH secretion and ECF volume. Primary mediators are the low pressure receptors in great veins, atria and pulmonary vessels	2	Must have to pass
	3 **Pain, emotion, stress, nausea vomiting**– all increase ADH	3	Must be able to name and explain 2 of the 4 last group to pass
	4 **Standing**	4	
	5 **Drugs** – eg, carbamazepine, clofibrate increase, alcohol decreases	5	
	6 **Angiotensin II** - increases	6	
	7	7	
	8		

QUESTION: 3 Water excretion.

Question	Required response [Key items marked with*]	To Pass
Describe water handling in the collecting ducts of the kidneys Prompt ; How does vasopressin affect water handling in the collecting ducts ?	1) The collecting ducts (CD) have two portions: a cortical portion and a medullary portion **2) Changes in osmolality and volume in the CDs depend on amount of **vasopressin** acting on ducts **3) This antidiuretic hormone from the post pituitary gland **increases the permeability of CDs to H2O** 4) Key to action of vasopressin on the CDs is aquaporin-2. Unlike other aquaporins, this is stored in vesicles in cytoplasm of principal cells. 5) Vasopressin causes rapid insertion of these vesicles into apical membrane of cells. Effect is mediated via the vasopressin V_2 receptor, cyclic AMP, protein kinase A, and a molecular motor, one of the dyneins **6) In presence of enough vasopressin to produce maximal antidiuresis, **H2O moves out of hypotonic fluid entering cortical CDs into interstitium** of cortex, and the tubular fluid becomes isotonic 7) As much as 10% of the filtered H2O is removed 8) When vasopressin is absent, the collecting duct epithelium is relatively impermeable to water and the fluid therefore remains hypotonic, and large amounts flow into renal pelvis.	2 3 6
What is an osmotic diuresis ? *Prompt:* *Describe how it occurs.* *Prompt:* *Can you give me an example*	** 1) Presence of large quantities of unreabsorbed solutes in renal tubules causes an ↑ in urine volume called **osmotic diuresis** ** 2) Solutes that are not reabsorbed in the proximal tubules exert an appreciable osmotic effect as volume of tubular fluid ↓ and their concentration ↑ ** 3) Therefore, they "hold water in the tubules 4) Concentration gradient against which Na^+ can be pumped out of proximal tubules is limited. Normally, movement of H2O out of proximal tubule prevents any appreciable gradient from developing, but Na^+ concentration in fluid ↓ when H2O reabsorption is ↓ because of presence in tubular fluid of ↑ amounts of unreabsorbable solutes. Limiting concentration gradient is reached, and further proximal reabsorption of Na^+ is prevented; more Na^+ remains in tubule, and H2O stays with it 5) The result is that loop of Henle is presented with a greatly ↑ volume of isotonic fluid. 6) This fluid has a ↓ Na^+ concentration, but total amount of Na^+ reaching the loop per unit time is ↑ 7) In loop, reabsorption of water and Na^+ is ↓ because the medullary hypertonicity is ↓. The ↓ is due primarily to ↓ reabsorption of Na^+, K^+, and Cl^- in the ascending limb of loop because limiting concentration gradient for Na^+ reabsorption is reached. More fluid passes through the distal tubule, and because of the ↓ in osmotic gradient along the medullary pyramids, less water is reabsorbed in collecting ducts. Result is a marked ↑ in urine volume and excretion of Na^+ and other electrolytes. 8) Osmotic diuresis is produced by administration of compounds such as mannitol and related polysaccharides that are filtered but not reabsorbed. It is also produced by naturally occurring substances when present in amounts exceeding the capacity of the tubules to reabsorb them. E.g. diabetes mellitus, glucose that remains in tubules when filtered load exceeds TmG causes polyuria. Osmotic diuresis can also be produced by infusion of large amounts of sodium chloride or urea.	1 2 3

Water Excretion 2006-1

TOPIC: Water excretion by the kidney NUMBER: 3

OPENING QUESTION	Describe how water is reabsorbed in the different parts of the nephron.	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	1. 60-70% in the Proximal tubule		Need to understand that water is reabsorbed in different parts & the role of vasopressin in the collecting duct.
	2. 15% in the loop of Henle		
	3. 5% in the distal tubule		
	4. Up to 10% in the collecting duct depending on the presence of antidiuretic hormone.		
SECOND QUESTION (if needed)	What hormonal factor influences water excretion?	What does vasopressin do?	
POINTS REQUIRED	1. Vasopressin increases the permeability of the collecting duct to water & allow water to be reabsorbed.		