Oedema, Shock and Burns VIVAs (Pathology)

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2015.1.A.1

Question 1 Septic Shock (pp 129-133) Subject: Path LOA: 1	What is Shock?	State where reduced cardiac output or effective blood volume results in **impaired tissue perfusion** and **cellular hypoxia**	Bold concepts
	How do microbes initiate septic shock? *Prompt: What are the mechanisms*	1 Interaction with innate cells of immune system – examples neutrophils, macrophages, monocytes 2 Interaction with Humoral cells of immune system to activate complement & coag pathways 3 Direct action on endothelium (complex, not fully understood) Toll-like receptors recognise microbial elements, and other mechs End result is **mediator release** examples TNF,IL-6, 8, 10, PAF PAI-1, HMGB1	2 of 3 plus examples of each (at least 1) + understand role of mediators
	When DIC develops, what is the process?	Induction of procoagulant state by: **1 Increased TF** production **2** Decreased production of **Protein C** **3** TF pathway inhibitor **Thrombomodulin** **4 Decreased fibrinolysis** by increasing plasminogen activator inhibitor, Combined with stasis (decr washout of activated coag factors) results in activation of thrombin & and fibrin rich thrombi	2 of 4 & understanding of process
	What factors determine the severity and outcome of septic shock in an individual?	Extent and virulence of infection Immune status of host Presence of other co-morbid conditions Pattern and level of mediator production	Bonus Q – no pass criteria

2013.2.D.1

PATHOLOGY Question 3 LOA: 1	1. What is hypovolaemic shock?	1. **Systemic hypoperfusion due to reduced effective circulating blood volume** resulting in **impaired tissue perfusion** and cellular hypoxia	Bold to pass
	2. Describe the stages of hypovolaemic shock Prompt: What compensatory mechanisms are involved?	2. A. **Non- Progressive phase** – reflex **compensatory mechanisms** activated to **maintain vital organ perfusion.** Variety of neurohumoral mechanisms activated to help maintain cardiac output and blood pressure (baroreceptors reflexes, release of catecholamines, activation of renin-angiotensin axis, ADH release and increased sympathetic output resulting in: tachycardia, peripheral vasoconstriction, and renal conservation of fluid with decreased urine output. Coronary and cerebral vessels less sensitive to sympathetic response and blood flow/ O_2 delivery spared.	All 3 phases to pass. 2A. Bold to pass + 3 features (prompt if necessary)
		B. **Progressive phase- tissue hypoperfusion** and worsening circulatory and metabolic imbalance including **acidosis.** Widespread tissue hypoxia resulting in anaerobic glycolysis with excess lactic acidosis production blunts vasomotor response → peripheral pooling, hypoxic injury, DIC, vital organs begin to failure	2B Bold to pass.
	3. What happens at the cellular and tissue level during the irreversible phase?	C. **Irreversible phase** - after body has incurred **cellular and tissue injury so severe** that even if haemodynamic defects are corrected, **survival is not possible** - Widespread cell injury - lysosomal enzyme release - nitric oxide → decreased myocardial contractility - acute tubular necrosis -> acute renal failure, - ischaemic gut→ bacteraemic shock - severe hypotension, unconscious, anuric - pre-cardiac arrest - > death	2C Bold to pass 3 features to pass

2013.1.2

		KNOWLEDGE (essential in bold)	NOTES
Question 1: **Oedema formation** LOA: 1	1. What are the mechanisms of oedema formation?	**1.↑ hydrostatic pressure –** impaired venous return, eg CHF,Constrictive pericarditis,ascites,venous obstruction(internal/external +Immobility), arteriolar dilatation eg heat **Decr plasm oncotic pressure (hypoproteinaemia)** – nephrotic syndrome, ,malnutrition, protein losing enteropathy. **Lymphatic obstruction** - inflammatory, neoplastic, post-surgery/radiation **Sodium and water retention** –XS salt with renal insufficiency, incr renin-angiotensin-aldosterone secretion **Inflammation** –acute/chronic, angiogenesis	3 out of 5 bold, example from each
	2. What is the pathogenesis of cardiogenic oedema?	**2.** Decreased cardiac output, decr renal perfusion, secondary aldosteronism, Incr blood volume, incr venous pressure	At least 3 steps.

2012.1.2

Question 2 Septic shock LOA: 1	**How do microbes initiate septic shock?**	1. Interaction with innate cells of immune system- neutrophils. macrophages and monocytes 2. Humoral interaction to activate complement and coagulation path 3. Direct endothelial action 4. End result is mediator release TNF,IL 6,8,10, NO,PAF, PAI-1	**at least 3 to pass**
	What are the effects of the mediators on the coagulation pathway?	Microvascular thrombosis, decreased fibrinolysis, DIC	**2/3 to pass**
	What are the consequent effects on tissues?	Tissue ischaemia, multi organ failure	**Either**

2011.2.2

Question 2 LOA: 1	1 What is hypovolaemic shock?	**Systemic hypoperfusion due to reduced effective circulating volume,** cellular hypoxia	Bold
	2 Describe the stages of hypovolaemic shock.	**Non Progressive phase**. Reflex compensation, vital organ perfusion. Baroreceptors, catechol, renin/angiotensin, ADH, sympathy stim.(↑HR, periph vasocons, ↓ urine) **Progressive Phase** Anaerobic glycolysis, lactic acidosis, ↓ vasomotor response, → periph pooling, hypoxic injury, DIC, vital organ failure **Irreversible Phase** lysosomal enz release., NO→ ↓ myocardial contractility, ATN, bacteraemic shock from isch gut.	3 phases to pass with details 4/9 3/7 2/4

2011.2.3

Question 1: LOA: 1	1) What is the pathogenesis of oedema?	**1.Hydrostatic pressure** and **osmotic pressure** normally balance to ensure that net fluid into and out of capillaries remains relatively equal with the little over removed by **lymphatics. Increased HP or diminished OP or overload of the lymphatics** will result in oedema.	Bold to pass
	2) How is oedema categorised and provide some examples?	**2.Increased hydrostatic pressure** - impaired venous return, eg. CCF, constrictive pericarditis, ascites, venous obstruction (internal or external + immobility); arteriolar dilatation eg. heat, neurohumeral dysregulation **Reduced plasma osmotic pressure (hypoproteinaemia)** - nephrotic syndrome, ascites, malnutrition, protein losing gastroenteropathy **Lymphatic obstruction** - inflammatory, neoplastic, postsurgical, postirradiation **Sodium retention** - excessive salt with renal insufficiency, increased tubular reabsorption of sodium (renal hypoperfusion, increased renin-angiotensin-aldosterone secretion) **Inflammation** - acute, chronic, angiogenesis	3 of 5 bold to pass with one example each category quoted

2011.1.1

Question 2. **Septic Shock**	1. How do microbial constituents initiate septic shock?	1. Interact with **cells of the innate immune system** (Neutrophils/Macrophages/Others) to release inflammatory mediators (& immunosuppressants) 2. Interact with **humoral elements of innate immunity** to activate complement and coagulation pathways 3. Act on **endothelium**	2 of 3 bold
	2. What is the **effect of endothelial cell activation and injury during septic shock**? PROMPT; What happens in the vessel?	**1. Thrombosis** 2. Increased **vascular permeability** **3. Vasodilation**	2 of 3
	3. **How does endothelial activation result in DIC (disseminated intravascular coagulation)?** **PROMPT; what mechanisms contribute to the coagulapathy in DIC**	1. Sepsis favours coagulation a. Increased tissue factor production b. Decreased fibrinolysis c. Stasis **d.** Decreased washout of activated coagulation factors e. Results in multiple fibrin rich thrombi 2. Increased hypoperfusion **Consumption Coagulopathy = DIC**	Consumptive and some detail

2010.1.3

Question 2: Oedema	a) What factors govern the movement of fluid between the vascular and interstitial spaces? (30%)	Hydrostatic Pressure – Osmotic Pressure- protein/ Na Normal capillary walls- most protein retained Small fluid out art end Most back venous end Small amount back via lymphatics	3 concepts mentioned A > c > V May know some Pressures, may mention gravity/ leg v head. Capillaries are fluid leak vessels. Normal tissue flow important. Thoracic duct return of lymphatics
	b) What are the major mechanisms of oedema formation (with examples)? 70%	**>Increased Hydrostatic Pressure** (local- DVT/ systemic- CCF)/venous obstruction **< Oncotic P** (mainly prot loss e.g. Nephrotic syndrome or poor production eg cirrhosis/ malnutrition or loss via gut) **Capillary leak-** (inflammatory injury/ systemic / infection) **Obstructive lymphatics-** e.g. lymphodema/ tumour/ op etc **Na retention with H2O** (renal insuff/ renin angio)- mainly dilutional	3 key features + a couple of examples

2009.1

Question 2: **Septic shock**	**What is an endotoxin?**	**Bacterial cell wall Lipopolysaccharides** usually from Gram -bacilli. Consists of a generic fatty acid core and a complex polysaccharide coat unique for each species.	**Bold**
	How does an endotoxin cause septic shock?	**Dose dependent activation of neutrophils, macrophages and monocytes** → mediator release → local/systemic inflam. response. Activation via: LPS binding prot. + CD14 receptor via IC toll I receptor. Mediators: TNF, IL-1, 6, 8, chemokines → cytokine release Low dose: enhanced local inflammatory response and clearance of infection. Moderate dose: fever, procoagulant activity. **High dose: Syndrome of septic shock** • Systemic vasodilatation • Decreased myocardial contractility • Widespread endothelial injury → alveolar capillary damage (ARDS) • Activation coag system → DIC	**3/4 needed**

2014.1.A.1

Question 2 Thermal Injury (Robbins pp 421-422) **Subject:** Path LOA: 1	How are thermal burns classified?	According to depth of injury: • **Superficial** – confined to epidermis • **Partial thickness** – extends to dermis • **Full thickness** – involves subcutaneous tissue	Bold required
	What are the potential complications of thermal burns?	**Early**: • Hypovolaemic shock (especially with >20% BSA) • Compartment syndrome (circumferential LL burn) • Associated injuries (eg inhalational burn, CO poisoning) • Airway compromise • Hypermetabolic state **Late**: • Infection / sepsis (Pseudomonas) • ARDS • Multi organ failure • Skin grafting, scarring / cosmetic • Psychological	2 early and 2 late
	How do you determine the extent of burns?	TBSA calculation notoriously inaccurate. Does not include superficial burns • Wallace "rule of nines"/Lund & Browder diagram	Mention 1 method

2011.2.2

Question 5 Thermal injury LOA: 2	How are thermal burns classified? (Prompt as to morphological depth classification?) What are the complications of a thermal burn? (Prompt for late)	**Superficial**-confined to epidermis **Partial thickness**-involves dermis **Full thickness**-extend to the subcutaneous tissue **Early vs late** **Early-hypovolaemic shock** with >20% BSA, pain, inhalational lung injury + airway oedema **Late- sepsis** (pseudomonas), MSOF, acute lung injury, scarring, cosmetic deformity, psychological	Bold Need 2 early & 2 late complication to pass