Respiratory VIVAs (Pathology)

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2014.2.A.3

Question 2 Community Acquired Pneumonia (pp 710-716) **Subject:** Path LOA: 1	1. What organisms cause community acquired pneumonia? **Prompts** What organisms cause atypical pneumonia, and what viruses may cause atypical pneumonia?	1. **Bacterial – Step pneumonia**, H influenza, Moxarella catarrhalis, S.aureus, Kelbsiella, and pseudomonas 2. **Atypical orgs** Mycoplasma, chlamydiae spp, coxielle burnetti (Q fever), legionella pneum 3. **Viral** – RSV, parainf, influenza A and B, adenovirus, SARs, H1N1	Bacterial – bold plus 2 others Atypical – 1 to pass Viral – 1 to pass
	2. What are some potential complications of pneumonia? **Prompt** – pathological sequelae	Abscess formation, Empyema, Bacteraemia/bacterial dissemination (endocarditis, pericarditis, meningitis, kidney, brain abscess), sepsis, respiratory failure	3 complications to pass
	3. How do the clinical features of atypical pneumonias differ from classic (typical pneumonias)?	Moderate sputum, no physical findings of consolidation, only mod increase in WBC Cough not prominent, typical sx are fever, headache, myalgia. Lower mortality compared with classic pneumonia.	2 features to pass

2014.1.D.1

Question 3 Pneumonia including aspiration pneumonia **Subject:** Pathology LOA: 1	(a) Describe the pathogenesis of aspiration pneumonia. (Prompt: predisposing features, organisms, outcomes)	• Aspiration of **gastric** contents • Type of **patient** (↓conscious/debilitated/abnormal gag/repeated vomiting) • **Chemical and bacterial** • **>1 organism** (aerobes>anaerobes) • **Necrotizing** • Death / **abscess**	(a) 4 bold to pass
	(b) How are community-acquired pneumonias different?	• **Bacterial or viral** • **Variable** pneumonia dependent on – etiol., host response etc • **Predispose** – extremes age, chr disease etc • **Agents** – strep pneum, haem. Influenza, etc • Clinical course modified by **ABs** • **Low hosp, low death** • **Complications** – empyema, endo/pericarditis, meningitis	(b) 5 bold to pass

2013.2.B.1

Question 1 PATHOLOGY LOA: 1	*"An elderly man presents with an acute exacerbation of COPD."* What is the definition of emphysema?	• A condition of the lung characterised by **irreversible enlargement** of the airspaces distal to the terminal bronchiole accompanied by **destruction of their walls** without obvious fibrosis.	**BOLD TO PASS** • Irreversible • Destruction
	Describe the pathogenesis of emphysema. Prompt: What is the mechanism of the destruction?	• **Mild chronic inflammation (neutrophils + macrophages) - mediator release** (e.g. leukotriene B_4, IL-8, TNF) – causes damage and sustains inflammation • **Protease-antiprotease imbalance – destructive effect of high protease activity** in pts with **low anti-protease activity** - 1% of pts with emphysema have alpha1-antitrypsin deficiency (inhibits proteases, including elastase, secreted by neutrophils) • **Oxidant-antioxidant imbalance – abundant reactive oxygen species** (superoxide dismutase, glutathione)**in smoke depletes antioxidant mechanisms,** incite tissue damage	**TWO EFFECTS** • Chronic inflammation • High protease activity • Reactive oxygen species
	What are the possible complications of emphysema?	• Bullous lung disease • Expiratory airflow limitation • Infection • Respiratory failure • Pneumothorax • Cor pulmonale, congestive heart failure ("pink puffers")	**THREE COMPLICATIONS**

2013.1.1

Question 3 Community Acquired Pneumonia LOA:1	1.What organisms cause community acquired pneumonia? PROMPTS: What organisms cause atypical pneumonia? What viruses may cause atypical pneumonia?	**1 Bacterial** • **Strep pneumoniae** • Haemophilus influenza • Moraxella catarrhalis • Staph aureus • Legionella pneumophilia • Others eg klebsiella pneumonia, pseudomonas **Atypical pneumonia** • Mycoplasma pneumonia • Chlamydiae spp • Coxielle burnetti (Q fever) • RSV, parainfluenza, influenza A+B, adeno virus. SARS virus	Need • Bacteria bold +2 • Atypical 1
	2. What conditions predispose to the development of pneumonia?	2 Extremes of age, malnutrition, alcoholism Chronic conditions – CCF, COPD, DM Neurological/swallowing disorders-aspiration pneum Congenital or acquired immune deficiencies Decreased or absent splenic function- splenectomy, sickle cell disease Recent viral infection (esp staph). IVDU & staph	4 broad categories 2/3 bold
	3. What are the potential complications of pneumonia Prompt-Pathological sequelae	3 **Abscess formation** (type 3 pneumococcus, Kleb) **Empyema** **Bacteraemic dissemination** – endocarditis, pericarditis, meningitis, abscesses of kidney, spleen, brain, septic arthritis	

2013.1.2

Question 4 Asthma LOA: 1	1. What are the pathological features of asthma? 2. Asthma may be categorized as atopic or non-atopic. What are the characteristics of each of these types? Prompt - What is the underlying mechanism of atopic asthma? What are some of the triggers? 3. In atopic asthma, what happens in the early-phase reaction?	**1. Increase airway responsiveness** to variety of stimuli; episodic **bronchoconstriction**; bronchial wall **inflammation**; incr mucus 2. Atopic- **IgE mediated type1 hypersensitivity** (allergen sensitisation); **environmental allergen triggers** e.g. dust, pollen, dander e.g. house dust mite, foods. Family Hx common; skin test positive to allergen; RAST shows allergen sensitivity Non-Atopic- **hyperirritability of bronchial tree**-no allergen sensitisation, skin tests usual negative; family Hx uncommon; triggers-resp infection secondary viruses common; inhaled air pollutants may contribute (SO_2, ozone, NO_2) **3. Allergen exposure**=> IgE. • Reexposure=> Mast cell degranulation with release of cytokines/mediators • =>bronchoconstriction (via subepithelial vagal/parasympathetic receptors), • mucus production, • vasodilation with increased vasc permeability	Bold Bold. One trigger for each Bold plus concept

2013.1.3

Question 3 Pulmonary Embolism LOA: 1	1. From where do pulmonary thromboemboli originate? 2. What are some risk factors for thrombus formation? 3. What are the clinical effects of pulmonary thromboemboli?	1/95% **arise in the deep veins of the leg** – pass up to R side of heart and into pulm vasculature. Size determines where they lodge. 2. **Primary** – (genetic factors) – factor 5 Leiden, protein C+S deficiency, antiphospholipid syn **Secondary-** (acquired) – stasis/immobilisation, long haul flights, active malignancy, trauma/burns/surgery, pregnancy, OCP. Indwelling catheters 3. most clinically silent 60-80%, Cough, SOB, fever, CP, haemoptysis, tachy-cardia/pnoea through to sudden death,cor pulmonale,CVS collapse Pulm haemorrhage / infarction, over time multiple emboli may cause pulm hypertension & cor pulmonale	Bold to pass (exact % not required but rough idea) At least one example from Primary, and 2 from secondary 5 features

2012.1.2

Question 4 ARDS LOA: 2	**Describe the pathogenesis of ARDS**	Initial **injury to alveolar capillary membrane** (endothelium); **acute inflammatory response** (neutrophil mediated); results in **increased vascular permeability** and alveolar flooding; fibrin deposition; formation of hyaline membranes; and widespread **surfactant abnormalities** (damage to Type II pneumocytes); eventually **–** organisation with scarring	**3 of 4 bold**
	What conditions are associated with the development of ARDS?	**Infection** (sepsis, diffuse pulmonary infection, gastric aspiration) **Physical / Injury** (trauma **–** head, pulmonary, fractures, near drowning, burns, radiation) **Inhaled irritants** (O2 toxicity, smoke, irritant gases and chemicals) **Chemical injury** (Heroin, barbituate, acetylsalicylic acid, paraquat) **Haematological conditions** (multiple transfusions, DIC) **Other** (pancreatitis, uraemia, cardiopulmonary bypass, hypersensitivity – organic solvents, drugs)	**Need 3 groups** (with **example** from each); **must include infection**

2012.1.3

Question 3 Cor	**What is cor pulmonale?**	Right sided heart failure that is **not secondary to left sided heart failure (pure RHF).** It can be acute (eg massive PE) or chronic (eg chronic lung disease).	**Bold** to pass
pulmonale	**What are the common causes of cor pulmonale?**	Diseases of pulmonary parenchyma (COPD; fibrosis; bronchiectasis). Diseases of pulmonary vessels (Primary pulmonary hypertension; recurrent PE; extensive pulmonary arteritis eg Wegener's granulomatosis). Disorders affecting chest movement (marked obesity; kyphoscoliosis; neuromuscular). Disorders causing pulmonary arterial constriction (hypoxaemia; metabolic acidosis; chronic sleep apnoea; altitude sickness). Common feature of all these is **pulmonary hypertension.**	**Bold** plus 3 other to pass
	What are the major morphological features of cor pulmonale? (Prompt: what are the organ features?)	Pulmonary congestion is minimal whereas engorgement of the systemic & portal venous systems may be pronounced. Heart: right ventricular hypertrophy and dilatation; leftward bulging of septum. Liver / portal system: congestive hepatomegaly; centrilobular necrosis; congestive splenomegaly Pleura, pericardial and peritoneal spaces: effusions; ascites. Subcutaneous tissues: oedema (dependent and peripheral portions of body; anasarca)	At least **three** to pass.

2011.1.1

Question 4. **Community acquired pneumonia**	**1.What organisms commonly cause community acquired pneumonia**? PROMPTS: What **organisms cause atypical** pneumonia? What **viruses may cause atypical pneumonia?**	**Bacterial** • Strep pneumoniae • Haemophilus influenza • Moraxella catarrhalis • Staph aureus • Legionella pneumophilia • Others eg klebsiella pneumonia, pseudomonas **Atypical pneumonia** • Mycoplasma pneumonia • Chlamydiae spp • Coxielle burnetti (Q fever) **Viral** • RSV, parainfluenza, influenza A+B, adeno virus. SARS virus	Need • Bacteria 3 • Atypical 1 • Viral 1
	2. How do **atypical pneumonias differ from classical (typical) bacterial** pneumonias PROMPT; how do the lung changes differ?	• **Moderate amount sputum** • **No physical findings of consolidation** • **Only moderate elevation of WCC** • **No alveolar infiltrate** • **Patchy inflammatory changes** largely confined to alveolar septa and pulmonary interstitium ie interstitial nature of the inflammation v alveolar exudates in classical pneumomia • **Different clinical presentation**; few localising signs, cough often absent, typical symptoms are fever, headache, myalgia, • **Lower mortality cf bact pneumonia** • (severe disease uncommon)	Lung changes to pass
	3. How is legionella pneumonia contracted?	• Artificial **aquatic environment** eg water cooling tower, water supply tubing • **Inhalation of aerosolised droplets** • Or **aspiration** of contaminated drinking water	• Water related

2010.1.1

Question 3: Emphysema	1. What is the pathological definition of emphysema?	"Emphysema is a condition of the lung characterised by abnormal permanent enlargement of the airspaces distal to the terminal bronchiole, accompanied by destruction of their walls, & without obvious fibrosis"	Bold to pass Permanent Enlargement + destruction
	2. Describe the pathogenesis of emphysema	**Protease – antiprotease theory** *Alveolar wall destruction results from an imbalance between proteases (mainly elastase) and antiproteases* **Elastases** from Neutrophils, also Macrophages, Mast cells, pancreas, bacteria **Anti-elastases**: α_1**AT**, secretory leukoprotease inhibitor, serum α_1 macroglobulin α_1AT inhibits neutrophil proteases. PiZZ variant predisposes to emphysema Neutrophils normally sequestered in lung (L > U) and a few gain access to the alveolar space. - Any stimulus that ↑ number of leukocytes (neutrophils / macrophages) in lung or release of their elastase containing granules ↑ elastocytic activity. - Stimulated neutrophils also release oxygen free radicals which inhibit α1-AT activity meaning process of elastic tissue destruction is unchecked	Know that key is imbalance between proteases (mainly elastase) and antiproteases
	3. What is the role of cigarette smoke?	• Smokers have ↑ **neutrophils & macrophages in alveoli**, - smoking stimulates neutrophil chemotactic factor (e.g. IL-8), nicotine chemotactic, smoke activates alternative complement pathway • **Smoking stimulates release of neutrophil elastase**, proteinase 3, Cathepsin G • Smoking ↑ elastase activity in macrophages (not inhib by α1-AT) • Reactive oxygen species in cigarette smoke deplete glutathione and superoxide dismutase Note centri-acinar distribution due to impaction of smoke particles in small bronchi / bronchioles with neutrophil influx. Differs to pan-acinar emphysema associated with a1-AT deficiency and chronic low level proteolysis from neutrophils in transit through the lung circulation.	2 effects

2009.2

Question 5 ARDS P715	What disorders can precipitate the Adult Respiratory Distress Syndrome, ARDS? *Prompt: What clinical conditions are associated with development of ARDS ?* *(same words as table)*	**Infection**: sepsis*, diffuse pulmonary infections*, gastric aspiration* **Trauma**: lung injury, head injury*, burns, radiation **Inhalation**: oxygen, smoke, irritants **Chemical injury**: heroin, salicylate, barbiturate, paraquat **Haematology**: transfusions, DIC **Other**: pancreatitis, uremia, CP bypass, hypersensitivity reactions (50% of ARDS cases associated with *)	4 groups, 1 example from each Need to include infection
	What is the pathogenesis of ARDS?	**Diffuse alveolar capillary damage**, variety of insults, initiated by different mechanisms. Capillary injury causes inc. vascular permeability, alveolar flooding & oedema, fibrin exudation, formation of hyaline membranes, **loss of diffusion capacity, abnormalities of surfactant.** Consequence of uncontrolled activation of acute inflammatory response; **most injury by neutrophils**. Macrophages alternative source of injury	3 out of 4 bold to pass
	What are the outcomes of ARDS?	Death, survival with organisation and **scarring.**	optional