ACEM Primary Examination Vivas > Pathology > Respiratory

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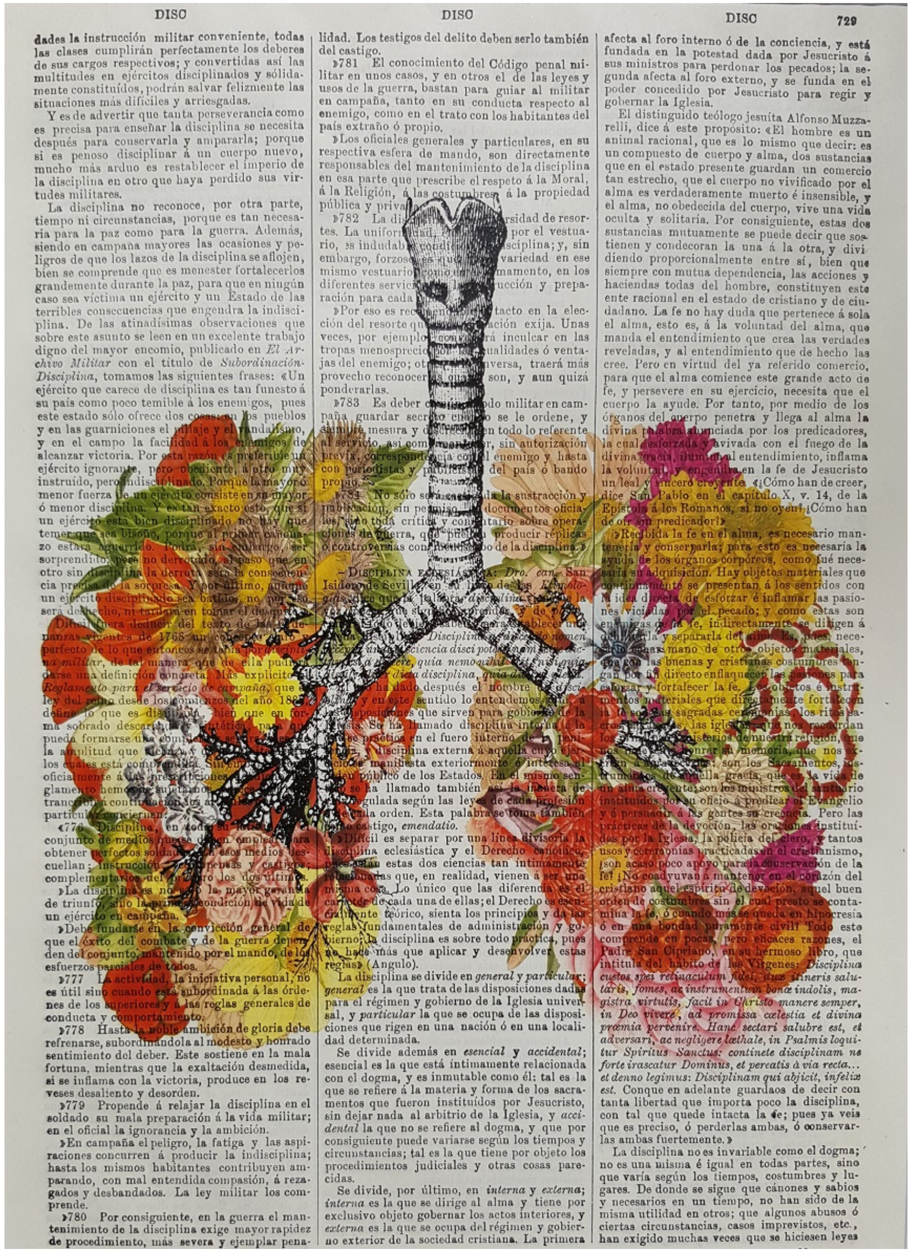

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ARDS 2017-1-C

Stem: The patient has Acute Respiratory Distress Syndrome. Moving on to Pathology.			
Question 3 ARDS **Subject:** Path LOA: 1	a) 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 Bold
	b) 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, barbiturate, acetylsalicylic acid, Paraquat) **Haematological conditions** (multiple transfusions, DIC) **Other** (pancreatitis, uraemia, cardiopulmonary bypass, hypersensitivity **–** organic solvents, drugs)	1 example from 3 groups

ARDS 2016-2-A

Stem: Moving onto Pathology. You suspect that she is developing Acute Respiratory Distress Syndrome.			
Question 3 ARDS **Subject:** Pathology LOA: 2	Describe the pathogenesis of ARDS	Type of acute lung injury. Initial **injury to alveolar capillary membrane** (epithelium) or vascular endothelium-> **acute inflammatory response** (neutrophil mediated), resulting in **increased vascular permeability** & alveolar flooding; fibrin deposition; formation of hyaline membranes; & widespread **surfactant abnormalities** (with damage to Type II pneumonocytes). Eventually organisation with scarring.	3 of 4 bold
	What conditions are associated with the development of ARDS?	**Infection** (sepsis, diffuse pulmonary infection) **Physical / Injury** (trauma – head, pulmonary, fractures, near drowning, burns, radiation). **Inhaled irritants** (O2 toxicity, smoke, irritant gases and chemicals). **Chemical injury** (Opiates, barbiturates, paraquat, acetylsalicylic acid, , gastric aspiration). **Haematological conditions** (multiple transfusions, DIC). **Other** (pancreatitis, uraemia, cardiopulmonary bypass, hypersensitivity – organic solvents, drugs)	Need 1 example from 3 categories + must include infection

ARDS 2012-1

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**

ARDS 2009-2

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
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

Asthma 2017-1-D

Stem: A 30-year-old male presents with a severe exacerbation of Asthma. We will start with Pathology

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Asthma **Subject:** Path LOA: 1	a) What is the pathological definition of asthma	a) Disorder of the conducting airways usually caused by an immunological reaction, marked by episodic **bronchoconstriction** due to airway sensitivity to a variety of stimuli, **inflammation** of the bronchial walls, and increased **mucus** secretion.	Bold to pass
	b) Name the main inflammatory cells involved	b) A wide range of inflammatory cells are involved **–** (lymphocytes, eosinophils, mast cells, macrophages, neutrophils)	Name 2
	c) How is asthma categorized pathologically?	c) Types of asthma: • **Atopic –** Most common. IgE (type 1) hypersensitivity reaction **–** T_H2 mediated. It is characterised by an immediate (bronchoconstriction) and late-phase (inflammation) reactions. TH2 cytokines, IL-4, IL-5 and IL-13 are important mediators (IL-17 & IL-9 in some). • **Non-Atopic –** No evidence of allergen sensitization (& negative skin test). Family history is rare. • **Drug induced (eg aspirin)** • **Occupational (eg epoxy fumes)**	Must list at least 2 types including atopic and trigger mechanism (Robbins, 9th ed. 679-682)

	d) Name some common triggers	d) Triggers:
		• **Atopic**
		  ○ Triggered by environmental factors (dust, pollens, food, etc.) in synergy with other pro-inflammatory cofactors such as respiratory viral infections. Positive family history and skin test for allergens.
		• **Non-Atopic** (Triggers are less clear)
		  ○ Viral respiratory infections (rhinovirus), parainfluenza, RSV)
		  ○ Inhaled air pollutants **–**smoking, sulfur dioxide, ozone, nitrogen dioxide
		  ○ Exercise induced
		  ○ Exposure to cold
			2 triggers

Asthma 2015-2-C

Stem: Moving onto Pathology.			
Question 5 Asthma **Subject:** Path LOA: 1	1. What are the pathological features of acute asthma?	1. **Increased airway responsiveness; episodic bronchoconstriction; bronchial wall inflammation; increased mucus**	**3/4 to pass**
	2. What is the underlying mechanism of atopic asthma? Prompt: What may trigger an exacerbation?	2. **IgE mediated type 1 hypersensitivity**; **Environmental allergens/triggers** (eg dust, pollens, foods, drugs)	**Bold and one trigger**
	3. What happens in the early-phase reaction in atopic asthma?	3. **Allergen exposure** produces IgE a. re-exposure triggers mast cell degranulation/cytokines b. bronchoconstriction c. mucus production d. vasodilation/incr vasc permeability	Bold & concept

Asthma 2013-1

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 (SO2, ozone, NO2) **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

Asthma 2007-2

TOPIC: Atopic asthma **NUMBER: Q5**

OPENING QUESTION	**Describe the pathogenesis of atopic asthma**	**COMMENTS**
POINTS REQUIRED	1 **Initial sensitisation to inhaled allergen (antigen)** – favours IgE production and eosinophil recruitment eg dusts, pollens, animal dander, foods	* 4/5 to pass
	2 **(Immediate phase – minutes) Re-exposure to antigen triggers Ag induced cross linking of IgE bound to IgE receptors on mast cells in airways and release of chemical mediators.** This results in opening of tight junctions between epithelial cells **Antigen can then enter mucosa** to release further mediators (via mast cell and eosinophil activation) and cause **bronchoconstriction / oedema / mucus secretion +/- hypotension**	* Prompt: What are the major changes which occur the airways in asthma **bronchoconstriction oedema** **mucus secretion** Mandatory / prompt if needed
	3 **Mediators act directly or via neuronal reflexes to induce bronchospasm / increased vascular permeability / mucus production** / recruit mediator releasing cells form blood	*
	4 **(Late phase asthma - hours) Recruited leucocytes arrive** (neutrophils, eosinophils, basophils, lymphocytes, monocytes) initiates fresh round of mediator release and epithelial damage and airway constriction	*

Asthma 2007-1

4. Atopic asthma	1. Name some environmental triggers for atopic asthma Prompt: "One trigger is dust. Tell me another"	1. Dust 2. Pollen 3. Animal dander (old skin scales) 4. Foods	At least 2
	2. What are the pathological steps of the acute response? Prompt: "Various mediators are released. What do they do?"	**1. Ag cross links mast-cell bound IgE** 2. Release of preformed, and newly-formed **mediator**s, with opening of tight junctions between epithelial cells 3. **Cause bronchospasm**, mucus production, increased vascular permeability, recruitment of additional cells, vagal receptors	Each section
	3. What cells are involved in the late phase response;? Prompt: "Eosinophils are one important type. Name some others"	1. Neutrophils **2. Eosinophils – major basic protein** 3. Basophils 4. Monocytes	Eosinophils and 1 other
	4. Please describe some of the inflammatory mediators involved?	5 Eosinophil cationic protein; - eotaxin [?? same as eosinophil chemotactic factor (ECF)]; IL-1 and IL-6, TNF, nitric oxide, bradykinin,	Supp question

Asthma 2006-1

TOPIC: Atopic Asthma _____ **NUMBER:** _____

OPENING Qn	**What is meant by the term atopic asthma?**	**COMMENTS**
POINTS Req'd	1. Asthma triggered by environmental factors - dust, pollen, dander, food	Immune mechanism
	2. Type 1 IgE mediated hypersensitivity reaction	
PROMPTS		
SECOND Qn	**Outline the events occurring in the immediate phase.**	
POINTS Req'd	1. Immediate	3 to pass
	a. Antigen + IgE = mast cell degranulation	
	b. Release of preformed mediators – open epithelial tight junctions – allow antigen access to mucosa	
	c. Activates mucosal mast cells and eosinophils – more mediator release	
	d. Directly or thru' neuronal reflexes – bronchospasm, inc vascular permeability, mucus production, additional cell recruitment.	
PROMPTS		
THIRD Qn	**Which mediators are involved in atopic asthma?**	
POINTS Req'd	1. "Putative" mediators - effective pharmacological interventions	
	a. Leukotrienes C_4, D_4, E_4	
	b. Acetylcholine - bronchoconstriction.	
	2. Mediators found in atopic asthma, but with ? minor role - Histamine, PGD_2, PAF.	
	3. Suspected mediators - cytokines (IL-1, TNF, IL-6), chemokines, neuropeptides, NO, bradykinin, endothelins	
PROMPTS		

Bronchiectasis 2017-2-A

Stem: Moving onto Pathology. She has bronchiectasis.			
Question 3 Bronchiectasis **Subject:** Pathology LOA: 1	a) What is bronchiectasis? *Prompt: what are the major morphological features?* b) What conditions are associated with the development of bronchiectasis?	a) Bronchiectasis is a disease characterised by **permanent dilation of bronchi** and bronchioles caused by **destruction of the muscle and elastic tissue**, resulting from or associated with chronic necrotizing **infections.** Also scarring and persistent infections b) *Congenital/hereditary* = Cystic fibrosis, immunodeficiency, ciliary dyskinesia, Kartagener's *Post infectious (necrotising pneumonia)* = Staph aureus, Haemophilus; TB, Pseudomonas; adenovirus; HIV, Influenza, fungi, aspergillosis *Bronchial obstruction* = tumour, foreign body, mucous impaction *Other:* Rh Arthritis, SLE, Inflam bowel disease, post transplantation Idiopathic 25 -50%	Bold concepts to pass 4 causes

Embolism, Fat 2008-1

Q2. Embolism	What clinical conditions may cause fat embolism?	1. (Microscopic) fat globules travelling in the circulation. 2. Long bone # 3. Soft tissue trauma/burns –rare 4. Very common with severe skeletal injury but rarely (<10%) of clinical significance	Pass criteria: 2 to pass.
	What is the pathogenesis of fat embolism syndrome?	1. Mechanical obstruction of microvasculature (lungs & brain): fat globules/aggregated platelet and RBCs. 2. Biochemical injury: FFAs from fat globules > endothelial injury, platelet activation & mediator release.	Main 2 points to pass
	What are the potential clinical sequelae of fat embolism?	1. Asymptomatic (Majority) 2. Neurological: altered LOC. 3. Pulmonary: Inc RR, SOB, hypoxia. 4. Haem: thrombocytopenia & anaemia.	2/4 to pass

Embolism, Pulmonary 2017-2-A

Stem: Moving on to Pathology. You suspect this patient has pulmonary emboli.			
Question 3 Pulmonary embolism **Subject:** Pathology LOA: 1 Robbins and Cottran P 127 to 129	a) Describe the pathogenesis of thrombotic pulmonary embolism (PE). *Prompt: Where do PEs originate?* *Prompt: Where do they lodge?* b) What are the symptoms and signs of pulmonary embolism? c) List 2 other types of emboli.	a) PEs originate from **deep vein thrombosis**. (~95% from lower limb). Fragmented thrombi from DVTs are carried through the venous system and into the right side of the heart before lodging in the **pulmonary arterial vasculature:** main pulmonary artery, pulmonary artery bifurcation or smaller branching arteries. b) Clinical manifestations depend on size and location of the thrombus in the pulmonary vasculature. -Most PEs (60-80%) are small and produce no symptoms nor signs A. Positive Symptoms - Chest pain - Dyspnoea - Collapse/syncope B. Positive Signs - Hypoxaemia/ tachypnoea - Tachycardia - Hypotension - Shock / sudden death - Acute right heart failure c). - Fat (bone marrow) - Air/other gas - Amniotic fluid - Foreign body (eg fragment of catheter)	Bold to pass 5 of list to pass 2 to pass

Embolism, Pulmonary 2016-1-A

Stem: Moving onto Pathology. A pulmonary embolus is diagnosed			
Question 3 Pulmonary Embolus **Subject:** Path LOA: 1	What are the clinical features of PE?	(60-80% are silent) Usually present with respiratory compromise – SOB hypoxia, dyspnoea, tachypnoea shock , collapse, hypotension Right heart failure, pleural rub/pleuritic pain, fever, cough, haemoptysis Death	5 to pass
	Name some risk factors for Pulmonary embolism?	Primary – factor V Leiden, Antiphospholipid syndrome, Prothrombin mutations Secondary- obesity, OCP, cancer, immobilisation, long haul flights , preg , indwelling CVL, hip fractures,	1 primary and 3 secondary factors
	What factors determine the severity of the pathophysiological response to pulmonary embolism? Prompt: features of the emboli…	1. **Extent of Pulmonary artery blood flow obstructed** 2. Size of the vessel occluded 3. Number of emboli 4. Overall CVS status 5. Release of vasoactive factors ie. (thromboxane A2)	Bold to pass Accept 2 of the others also as a pass

Embolism, Pulmonary 2009-2

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1: Embolism	What conditions predispose to the development of pulmonary thrombo-embolism?	**Hypercoagulable States:** 1. Primary- factor V Leiden, prohtrombin 20210 A, hyperhomocysteinaemia, antiphospholipid syndrome 2. Secondary – obesity, recent surgery, cancer, oral contraceptive pill, pregnancy **Other underlying medical conditions** – hip fracture, immobilization, cardiac disease, central venous lines	Simple list of 6 = straight pass Better pass with bold groups and examples of each
Question 2	What are the potential clinical sequelae of pulmonary thrombo-embolism?	Relates to size and number of emboli and overall status of cardiovascular system 1. Asymptomatic 2. Sudden death 3. Large PE –chest pain, dyspnoea, shock 4. Small PE-transient chest pain, cough and in predisposed individuals pulmonary infarct causing tachycardia, tachypnea, haemoptysis, fever,pleural rub. 5. Pulmonary hypertension	Any 3 to pass
Question 3:	What are the non-thrombotic types of pulmonary embolism?	1. Air 2. Bone marrow or Fat 3. Amniotic fluid 4. Tumour 5. Foreign bodies	3 to pass

Emphysema 2016-1-B

Question 2 Chronic Obstructive Pulmonary Disease LOA: 1	*a. What is emphysema?*	Chronic lung condition characterised by **irreversible** enlargement of the airspaces **distal to the terminal bronchiole,** accompanied by destruction of alveolar walls **without fibrosis.**	2 out of 3 bold
	b. Describe the pathogenesis of emphysema.	- Loss of cellular homeostasis - caused by **exposure to toxic substances** such as tobacco smoke and inhaled pollutants which induces **ongoing inflammation, epithelial cell death and extracellular matrix proteolysis.** - Accumulation of neutrophils, macrophages and lymphocytes results in release of **elastases, cytokines** (including IL-8) and **oxidants** that cause epithelial injury and proteolysis of the extracellular matrix. - **Elastin degradation** products further increase the inflammation. - End result is destruction of the alveolar walls without fibrosis.	2 bold
	c. How do the clinical features of emphysema differ from those with chronic bronchitis?	- "Pink Puffer" = emphysema. Barrel chested, dyspnoeic, prolonged expiration, hyperventilation. Relatively normal gas exchange until late in disease. - "Blue Bloater" = chronic bronchitis. Hx of recurrent chest infections with purulent sputum, less dyspnoea, decreased respiratory drive. Patient is hypoxic and cyanotic. Peripheral oedema results from *cor pulmonale* and RV failure.	2 distinguishing clinical features.

Emphysema 2013-2-B

TOPIC	QUESTIONS	KNOWLEDGE (essential in bold)	NOTES
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**

Emphysema 2010-1

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_1AT**, 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, Catepsin 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

Emphysema 2007-2

TOPIC: Emphysema _____ **NUMBER: Q3** _____

OPENING QUESTION	What is emphysema?	**COMMENTS**
POINTS REQUIRED	Abnormal permanent enlargement of air spaces distal to terminal bronchioles with alveolar wall destruction and minimal fibrosis.	Required
SECOND QUESTION	What are the anatomical types of emphysema?	
POINTS REQUIRED	• *Centriacinar* – involves central or proximal parts of respiratory unit – acinus – sparing distal alveolar, involvement of upper lobes and apices, primarily in male smokers associated with chronic bronchitis. • *Panacinar* – uniform destruction and enlargement of acinus, predominantly in lower basal zones, associated with alpha 1 antitrypsin deficiency. **Extra** • *Distal acinar* (paraseptal) • *Irregular emphysema* (air space enlargement with fibrosis). • Other types of emphysema – compensatory emphysema, senile emphysema, obstructive over inflation, bullous emphysema and interstitial emphysema *(bonus marks for the high performing candidate)*.	***Must mention the above 2 types.***
	What is the pathogenesis of emphysema?	
	Protease – antiprotease hypothesis – imbalance between pulmonary proteases (elastase) and their inhibitors. ***Alpha 1 antitrypsin*** present in serum tissue fluids and macrophages is a major protease inhibitor. Individuals with hereditary deficiency of alpha 1 antitrypsin develop emphysema and at a younger age if they smoke. ***Tobacco smoking*** activates alveolar macrophages – recruit neutrophils into lung. Enhance neutrophil and macrophage elastase activity. Inactivate alpha 1 antitrypsin by oxidants in tobacco smoke following by free radicals released by activated neutrophils.	***Must mention alpha 1 antitrypsin and tobacco smoking.***

Emphysema 2006-1

TOPIC: EMPHYSEMA _____ **NUMBER:** _____

OPENING Qn	**What is meant by the term 'emphysema' in relation to the lung?**	**COMMENTS**
POINTS Req'd	1. Abnormal permanent enlargement of the air spaces distal to the terminal bronchiole.	Need 3
	2. Destruction of alveolar walls.	
	3. No obvious fibrosis	
SECOND Qn	**What are the major types of emphysema?**	Need 1st 2 + explanation
POINTS Req'd	1. Centriacinar (centilobular) 95%	
	a. Central/proximal parts of acini affected	
	b. Distal alveoli spared except in severe disease	
	c. More common/severe in upper lobes/apices	
	d. Occurs in heavy smokers, often associated with chronic bronchitis	
	2. Panacinar	
	a. Uniform enlargement from resp bronchiole distally	
	b. Tends to occur at lower zones	
	c. Association with α_1-antitrypsin deficiency	
	3. Distal acinar (paraseptal)	
	4. Irregular emphysema	
THIRD Qn	**Describe the pathogenesis of emphysema**	
POINTS Req'd	1. mild chronic inflammation – inflammatory mediators	
	2. protease-antiprotease theory	
	a. protease from neutrophils/macrophages vs α_1-AT	
	b. α_1-AT activity ↓ by oxygen free radicals (smoke)	
	c. neutrophil etc elastase ↑ by oxygen free radicals	

Lung Cancer 2017-2-B

Stem: Moving on to Pathology. He has multiple metastases from his lung cancer			
Question 4 Lung Cancer **Subject:** Pathology LOA: 1 *Robbins 9th edition Pg. 712-719*	a) What factors predispose to lung carcinoma?	a) **Tobacco smoking** **Environmental exposures**: radiation, asbestos, air pollution (particulates), occupational inhaled substances (Nickel, Chromates, Arsenic) **Genetic mechanisms**: dominant oncogenes (c-MYC, k-RAS) & loss of tumour suppressor genes (e.g. p53, RB) **Precursor lesions**: squamous dysplasia, carcinoma in situ, atypical adenomatous hyperplasia	Tobacco smoke + 2 concept areas to pass
	b) What are the classic clinical features of lung carcinoma	b) Cough (75%), weight loss (40%), chest pain (40%), dyspnoea (20%).	3 of 4
	c) What paraneoplastic syndromes are associated with lung carcinoma? *Prompt: what hormones can be released by tumours?*	c) SIADH - hyponatraemia; ACTH - Cushings syndrome; hypercalcaemia - parathormone, parathyroid hormone-related peptide or prostaglandin E; hypocalcaemia - calcitonin; gynaecomastia - gonadotropins; carcinoid syndrome -serotonin, bradykinin . Others: Lambert-Eaton Myaesthenic sy, peripheral neuropathy, acanthosis nigricans, clubbing (hypertrophic pulm. osteoarthropathy).	At least 3

Lung Cancer 2016-1-C

Stem: Moving onto Pathology			
Question 5 Lung Neoplasia and principles of neoplasia **Subject:** Path LOA: 2	1. What are the main categories of primary lung cancer?	**Adenocarcinoma** (more common in F) **Squamous cell carcinoma** (more common in M) **Small cell carcinoma** (v. malignant) Large cell carcinoma (undifferentiated)	Bold
	2. What are the pathways by which a malignant tumour may spread?	**Local invasion** **Direct seeding** of cavities/surfaces **Lymphatics** **Haematogenous** Surgical instruments/nerves	3 of 4 Bold
	3. What paraneoplastic syndromes can be associated with lung carcinomas?	**SIADH** (HypoNa as per CBB, small cell Ca) ACTH (Cushing's) PTH, PTH-related peptide, PGE (HyperCa) Calcitonin (HypoCa) Gonadotropins (gynaecomastia) Serotonin/bradykinin (carcinoid syndrome)	SIADH and 1 other

Lung Cancer 2014-2-B

Stem: A 70 yo woman with metastatic lung cancer presents with polydipsia and polyuria. We will start with Pathology.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Lung Tumours (pp 721-731) **Subject:** Path LOA: 2	1. What are recognised aetiological factors in lung cancer? Prompt for detail: Are you aware of any environmental factors that place you at greater risk for lung cancer?	**Tobacco smoking -** 87% of cancers in recent or current smokers- 10x increase in risk, Statistically associated with daily amount; inhalation tendency; duration of habit, Histologic changes in respiratory epithelium in smokers **Industrial Hazards** Ionising radiation, Uranium, Asbestos **Air pollution -** Radon **Molecular genetics -** Familial clustering **Precursor lesions -** Squamous dysplasia and CIS, Atypical adenomatous hyperplasia, Diffuse idiopathic pulm neuroendocrine cell hyperplasia	Tobacco smoking and 2 other bold to pass
	2. What are the most common presenting symptoms of lung cancer?	**Cough** (75%), Loss of weight(40%), Chest pain (40%), Dyspnoea (20%), Haemoptysis	3 to pass
	3. What are the clinical effects of local lung tumour spread?	• **Airway obstruction ->pneumonia, abscess, lobar collapse**, Lipoid pneumonia, • **Obstruction of SVC** leading to SVC syndrome • **Pleural effusion**, • **Pericarditis or tamponade**, • **Hoarseness** (r/c laryngeal n), • **Dysphagia** (oesophagus), Rib destruction, • **Diaphragmatic paralysis** (phrenic nerve) • **Horner syndrome** (sympathetic ganglia)	5 of 8 bold to pass
	4. What paraneoplastic syndromes are associated with lung cancer? PROMPT: What hormones might be produced?	Clinically significant in 1-10% of patients ACTH- **Cushing's** (predominantly small cell) ADH—**hyponatraemia** (predominantly small cell) PTH, PTH related peptide, PGE and some cytokines**- hypercalcaemia** (predominantly small cell/squamous cell), Calcitonin-hypocalcaemia, Gonadotrphins-gynaecomastia, 5HT and bradykinin-wheeze/flushing	2/3 bold + 1 other to pass

Mesothelioma 2009-1

Question 5: **Malignant mesothelioma (pleural)**	**Describe the relationship between asbestos exposure and malignant mesothelioma** **Where can malignant mesothelioma arise?**	1. **Increased incidence among people with heavy exposure to asbestos.** Lifetime risk up to 7-10%. 2. **Asbestos bodies** found in increased numbers in lungs of patients with mesothelioma. 3. **Long latent period** for mesothelioma (25-45 yrs). 4. No increased risk in asbestos workers who smoke (in contrast to asbestos related lung carcinoma). Asbestos workers more at risk of dying from lung carcinoma (especially if they smoke). 1. **Pleura** 2. **Peritoneum** 3. pericardium 4. tunica vaginalis 5. genital tract	**2/4** **Bold**

Pneumonia 2017-2-B

Stem: We will start with Pathology.			
Question 2 Robbins – 9th Edition. Page 704-6 **Subject: Path** LOA: 1	a) What are the most common causes of bacterial community acquired pneumonia?	a) The most common bacterial causes are: - **Strep pneumoniae** - mycoplasma pneumoniae - Haemophilus influenzae - Moraxella catarrhalis - Staph aureus - Klebsiella pneumoniae - Pseudomonas aeruginosa - Legionella pneumoniae	Strep plus 2 others
	b) What factors predispose patients to the development of acute bacterial pneumonia?	b) 1. Extremes of age. 2. Underlying chronic disease such as COPD, Diabetes mellitus, Congestive cardiac failure, 3. Immunodeficiency: congenital or acquired, Abnormal splenic function: decreased splenic function or asplenia.	3 examples from 2 groups to pass

Pneumonia, aspiration vs CAP 2014-1-D

Stem: During the reduction she becomes persistently hypoxic. This topic is **PATHOLOGY.**			
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

Pneumonia, atypical 2003-1

TOPIC: Atypical Pneumonia **NUMBER:** _____

OPENING QUESTION	What is "Atypical Pneumonia"	**COMMENTS**
POINTS REQUIRED	1 Interstitial Pneumonitis	
	2 Lack of exudate	
	3 Different Clinical Picture	
	4	
	5	
	6	
	7	
PROMPTS	Tell us about the pathological changes	
SECOND QUESTION (if needed)	What organisms are usually involved	
POINTS REQUIRED	1 Mycoplasma, Q fever, Legionella, psittacosis (3 to pass)	
	2 Viruses (name at least one)	
	3	
	4	
	5	
	6	
PROMPTS		
THIRD QUESTION (if needed)	Describe the clinical features	
POINTS REQUIRED	1	
	2	
	3	
	4	
	5	
	6	
PROMPTS	How is it different from "typical pneumonia"	

Pneumonia, Community Acquired 2014-2-D

Stem: A CXR shows pneumonia. We will now move onto Pathology			
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

Pneumonia, Community Acquired 2013-1

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	

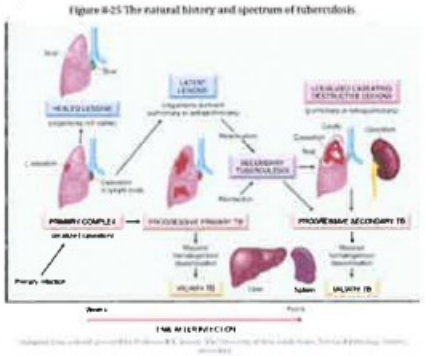
Pneumonia, Community Acquired 2011-1

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

Pneumonia, Legionella 2005-1

Legionella pneumonia	How is Legionella acquired?	Aerosols/artificial aquatic environments – cooling towers, water supplies etc. Also in other moist conditions Aspiration of aerosolised organisms or aspiration of contaminated water. (L.longbeachae)	
	What groups of patients are at risk of Legionella infection?	Underlying co-morbidities – cardiac, renal, immune, hematologic. Transplant pts especially. (Smokers, chronic lung and elderly)	
	How is Legionella diagnosed?	Urinary antigen or fluorescent Ab test on sputum. Culture is gold standard. – special medium. (PCR)	

TB 2016-1-D

Stem: Moving onto Pathology. TB is suspected			
Question 2 TB **Subject:** Path LOA: 1	1. Outline the natural history and spectrum of TB. PROMPT: What can happen after primary infection?	1. Primary infection 2. **Primary complex** (localised caseation) (Ghon complex is primary TB with mediastinal nodes) 3. Primary complex may heal (orgs not viable) or lead to latent lesion (orgs viable) 4. **Latent period** OR progressive primary TB (latter of which may lead to miliary TB) 5. Latent lesion reactivated leading to **secondary TB**. (Reinfection may also lead to secondary TB) 6. Secondary TB occurs as localised (pulm or extra-pulm) caseating destructive lesions OR progressive secondary TB 7. Progressive secondary TB may lead to **miliary TB**	4/4 in bold 
	2. How is TB diagnosed?	1. **Clinical features** in at risk patients (Hx and Ex) and apical lung consolidation/cavitation on **CXR** **2. Microbiological confirmation** a. Acid fast smears and cultures (3-6 wks solid agar media, 2 weeks liquid media) b. PCR 3. Other eg Mantoux test (TST)	Must have bold

TB, Primary 2008-2

3. Tuberculosis	Describe the **pathogenesis** of tuberculosis in a previously **unexposed immunocompetent** person Prompt if doesn't mention airborne.	**Infection by M. tuberculosis airborne** • M. tuberculosis usually person to person airborne droplet spread **M tuberculosis enters alveolar macrophages and replicates** • Enters alveolar macrophages and replicates by blocking phagosome/lysosome fusion leading to bacteraemia (person generally asymptomatic or mild flu like illness **Immunity through T cell mediated delayed type hypersensitivity reaction that also causes hypersensitivity and tissue destruction- in particular granuloma formation and caseation** • About 3 weeks later T cell activation via MHC antigens on macrophages and IL-2 leading to macrophage becoming bactericidal (thru IFN-γ) This macrophage response also causes tuberculin positivity and formation of granuloma and caseation by recruiting monocytes ("epithelioid histiocytes") **Re- exposure or reactivation causes heightened immune reaction as well as tissue destruction** • Infection may be contained or may progress and may reactivate with immunosuppression from any cause	Highlighted

TB, Primary 2003-2

2.4 Genesis of Primary TB	Describe the pathogenesis of primary tuberculosis	Relates to ability to **escape macrophages, induce delayed hypersensitivity**: cord factor, LAM (lipo arabino mannan), complement activation, heat-shock protein. Cell mediated (Type VI) hypersensitivity causes destructiveness and resistance. Naïve macrophages (hilar lymph nodes) are unable to digest bacilli, which multiply, lyse cell, invade others, may disseminate intravascularly. 3weeks, **T-cell mediated reaction –> epithelioid cell granuloma**s, then caseating granulomas –> calcified scar = Gohn complex.	

TB, Secondary 2011-1

Question 3. Tuberculosis	**1. What is secondary tuberculosis?**	Pattern of disease that arises in a previously sensitised host	**previously sensitised host**
	2. How may infection occur in secondary tuberculosis?	1. May follow shortly after primary infection (<5%) 2. **Reactivation of latent organisms** • Typically in areas of low disease prevalence 3. **Reinfection** • Typical in regions of high prevalence	**Items 2 and 3**
	3. Describe the pathological features in the lung of secondary infection with TB.	• Locale - **apical UL in secondary** • **Area of inflammation / granuloma** / multinucleate giant cells • Central **caseous necrosis** • **cavitation** • **Healing with fibrosis and calcification** • +/- Complications include tissue destruction, **erosion of blood vessels, miliary spread, pleural effusion, empyema, fibrous pleuritis**	**Need 3 of:** • Apical site • Inflammation / granuloma • Caseous necrosis • Cavitation • Fibrosis / calcification

TB, Secondary 2005-1

Mycobacterium tuberculosis	What is secondary tuberculosis? What are the main features of secondary pulmonary infection. 3 required	Secondary TB is the pattern of disease that arises in a previously sensitised host. ✱ May follow shortly after primary infection, but commonly arises from reactivation of dormant primary lesions when host resistance is weakened, or from reinfection. Characteristic feature is gross tissue necrosis, especially at apices- cavitation secondary to cytokine/TNF and IL-1 etc Large lesions with caseous necrotic tissue = tuberculoma - Cavity formation✱ - oxygen and CO_2 present, bacilli replicate freely, - erosion of blood vessels.....Hemoptysis - Infect other parts of lung....local spread ....Bacilli in sputum - Patient infectious Fibrosis and scarring with resolution	