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Candida 2008-2

3. Candidiasis	1. What is the clinical spectrum of candida infection?	(Benign commensal) **Superficial mucosal infn** – mouth, vagina, oesophagus **Superficial cutaneous infn** – intertrigo, nappy rash, balanitis, folliculitis, paronychia, onychomycosis Chronic mucocutaneous (T-cell defects, endocrinopathy) **Invasive (disseminated)** – myocardial/ abscess/endocarditis, cerebral abscess/meningitis, renal/hepatic abscess, endophthalmitis, pneumonia	Highlighted – something from each category
	2. What mechanisms enable candida to cause disease? Prompt: What are the virulence factors?	1) **Phenotypic switching** to adapt rapidly to changes in host environment 2)**Adhesion to host cells** - imp. determ. of virulence –via adhesins (several types) 3) **Production of enzymes** (aspartyl proteases and catalases) degrade extracellular matrix proteins and may aid intracellular survival 4) secretion of adenosine – blocks neutrophil degranulation	1/3 Highlighted

Clostridia 2011-2

Question 3 LOA: 1	1. What type of organisms are the Clostridia? 2. Name the organisms and the diseases they cause in humans? 3. How does botulism toxin cause disease?	**1. Gm+ve, bacilli, anaerobic, spore-forming** **2. Gas Gangrene (Perfringens), Tetanus (tetani), Botulism (botulinum), Diarrhoea (difficile)** 3.. Normally ingested. In the cytoplasm, the "A" fragment cleaves the protein "synactobrevin". Synactobrevin is needed for fusion of neurotransmitter vesicles. Results in **flaccid paralysis**	**1.** needs 3 of 4 **2.** needs 3 of 4 3. must have some idea of this plus bold

Clostridia 2008-1

Q3. Clostridial infections	Name some clostridial diseases and causative organisms.	1) Tetanus (lockjaw) – Clostridium tetani 2) Botulism (paralytic food poisoning) – Clostridium botulinum 3) Gas gangrene, necrotizing cellulitis – Clostridium perfringens, C. septicum 4) Pseudomembranous colitis – Clostridium difficile	Pass: Require 2 out of 4
	What is the pathogenesis of gas gangrene (C. perfringens, C. septicum)	Release enzymes – hyaluronidase; collagenase Virulence factors – TOXINS <u>α-toxin</u> - multiple actions - phospholipase C: degrades membranes; muscle; RBC - release phospholipid derivatives: ITP; prostaglandins - these cause derangement in cell metabolism and cell death	At least 2 & α-toxin

Croup 2016-2-D

Question 4 Croup and Acute inflammation **Subject:** Path LOA: 1	a. What is croup? *Prompt: What is the effect on the airway* Prompt: *What viral agents cause croup*? b. Describe the main characteristics of acute inflammation. *Prompt: Describe the general characteristics of acute inflammation.*	1. **Acute laryngotracheobronchitis** in children: inflammatory/spasmodic **narrowing of the airway** produces barking cough, inspiratory stridor. Causes are predominantly viral, esp **Parainfluenza virus.** RSV, adenovirus and influenza are others. 2. Main characteristics of Acute Inflammation: A. Relatively rapid onset. B. Alterations in vascular calibre that **increase blood flow.** C. **Leaky microvasculature**: Structural changes in microvasculature that permit plasma proteins and leucocytes to leave circulation**.** This leads to oedema. D. **Emigration of leucocytes (esp neutrophils),** their accumulation at site of infection, and activation to eliminate offending agent. E. Duration of hours to days.	Bold Bold

E. coli 2017-2-B

3. Herpes simplex	1. Please give some examples of clinical Herpes Simplex Infection.	Cold sores, Gingivostomatitis, Encephalitis, Genital herpes, Keratitis (epithelial & stromal), Disseminated visceral herpes (esophagitis, bronchopneumonia, hepatitis, Kaposi varicelliform eruption, eczema herpeticum)	At least 3
	2. After primary herpes simplex infection, how does reactivation occur?	1. Viral nucleocapsids travel from the skin/ oropharynx/genitalia to the **nucleus in the sensory neurone**. 2. During **latent period**, only viral mRNA is produced, no viral proteins are made to escape immune recognition. 3. Reactivation from latency occurs by **avoiding immune recognition**, inhibiting the MHC class I recognition pathway, and elude humoral immune defences by producing receptors for the Fc domain of immunoglobulin and inhibitors of complement. Antidromically along sensory nerve.	

Epstein Barr Virus 2014-1-D

Stem: The next patient is a 20 yo woman who is dehydrated secondary to poor oral intake from glandular fever. This topic is **PATHOLOGY**.			
Question 3 EBV **Subject:** Path LOA: 2	(a) Describe the pathogenesis of glandular fever.	• **EBV** transmitted by close contact (saliva) • Envelope g/protein binds to B cells • Viral infection begins naso/oropharyngeal **lymphoid** tissues (esp. tonsils) • EBV accesses submucosal lymphoid tissues • B Cell infection 1) **lysis** infected cells and virion release (minority) or 2) **Latent** infection (EBV genes expressed) • Symptoms appear on initiation host immune response (cellular CD8+ cytotoxic T and NK cells) • Atypical lymphocytes (characteristic) • Reactive T cell proliferation lymphoid tissues – lymphandenopathy and splenomegaly. • IgM Ab (viral capsid Ag) and later IgG • Healthy – cease viral shedding with few resting B cells but Acquired defects may → B lymphomas	(a) To pass: EBV Lymphoid tissue Involves B (latent and lysis) and T cells
	b) What are the clinical features of glandular fever?	(b) Classically – Fever, sore throat, lymphadenitis splenomegaly Atypical presentation common – fatigue, lymphadenopathy, hepatitis, rubella-like rash	(b) 4 clinical features to pass
	(c) What are the outcomes of glandular fever?	4-6 weeks **most resolve** - some fatigue longer **Hepatic** dysfunction – j, abn. LFTs, appetite **Splenic** rupture Other systems – nervous, renal, lungs, heart. Transformation – **lymphomas**	(c) 3 outcomes to pass

Herpes Simplex 2007-1

3. Herpes simplex	1. Please give some examples of clinical Herpes Simplex Infection.	Cold sores, Gingivostomatitis, Encephalitis, Genital herpes, Keratitis (epithelial & stromal), Disseminated visceral herpes (esophagitis, bronchopneumonia, hepatitis, Kaposi varicelliform eruption, eczema herpeticum)	At least 3
	2. After primary herpes simplex infection, how does reactivation occur?	1. Viral nucleocapsids travel from the skin/ oropharynx/genitalia to the **nucleus in the sensory neurone**. 2. During **latent period**, only viral mRNA is produced, no viral proteins are made to escape immune recognition. 3. Reactivation from latency occurs by **avoiding immune recognition**, inhibiting the MHC class I recognition pathway, and elude humoral immune defences by producing receptors for the Fc domain of immunoglobulin and inhibitors of complement. Antidromically along sensory nerve.	

Herpes Zoster 2017-1-B

Stem: Moving onto Pathology.			
Question 4 Herpes Zoster **Subject:** Path LOA 2	Describe the pathogenesis of Herpes Zoster.	1 the patient has **had previous exposure** to herpes (chickenpox or subclinical) 2 VZV evades immune defenses & **infects sensory neurons** in and around dorsal root ganglia 3 Able to **remain latent** here for many years 4 Usually a single episode of recurrence in the form of zoster/ shingles 5 **Reactivation** often in the elderly or immuno-compromised **6** Vesicular eruption along **dermatome of one or more sensory nerves** Associated intense burning, itching and pain due to radiculoneuritis. May cause nerve dysfunction (e.g. Ramsay Hunt syndrome)	Bold to pass

HIV 2003-2

1.4 HIV, transmission	What are the major pathological sequelae of HIV infection? What are the modes of transmission of HIV?	**attacks CD4+ T cells -> profound immunosuppression -> opportunistic infections, neoplasms**, neurologic manifestations. 75% sexual, **heterosexual** globally more common; female partners of IVDUs. Female to male 1/20th as efficient in US, more in Thailand. Abetted by STDs. **Parenteral**: IVDUs major, blood products almost eliminated. **Mother-to-infant** in utero, at delivery, in breastmilk. 7-49%. Not by insect bites. Needle-stick 0.3%	

Influenza 2011-2

Question 3 LOA: 2	1. Describe the structure of the influenza virus. 2. What are the types and subtypes Prompt:- What do H and N stand for? 3. What is the pathological basis of pandemics and epidemics?	1. **Single stranded RNA** (8 helices) Spherical capsule 2. **ABC** (determined by a nucleoprotein) **Haemagglutinin and neuraminidase** (determined by proteins on the bilipid envelope 3 **Antigenic shift** for pandemics **Antigenic drift** for epidemics Both H and N are changed by recombination of RNA from animal viruses	1. Bold to pass 2. Bold Bold to pass

Influenza 2010-2

i) Describe the structure and classification of influenza viruses	**ssRNA, bound by nucleoprotein that determines type (A, B or C) and a lipid bilayer that contains both haemagglutinin and neuraminidase** (determining subtype eg H1N1)	Need RNA and major types
ii) What is the difference between antigenic drift and shift?	Only in influenza type A **Drift – mutation** of the haemaglutinin and neuraminidase antigens allowing escape from most host antibodies (epidemic) **Shift – antigens replaced via recombination of RNA** segments with those of animal viruses (pandemic) Types B and C do not show drift or shift, mostly infect children, who develop antibodies preventing re-infection	Bold
iii) How does the human body clear a primary influenza virus infection?	**2 mechanisms – cytotoxic T cells and macrophages** cytotoxic T cells kill virus infected cells, an intracellular antiinfluenza protein (MxI)is induced in macrophages by cytokines IFN-a and IFN-b. Future infection is prevented (haemagglutinin Ab) and ameliorated (neuraminidase Ab)	Bold to pass

Influenza 2009-1

Question 4: **Influenza**	**How does the Influenza virus cause pneumonia?**	1. **Attachment of virus to upper resp tract epithelium** 2. Necrosis of cells followed by inflammatory response 3. Interstitial inflammation with outpouring of fluid into alveoli 4. **Secondary infection by Staph / Strep**	Need bolded
	How does Influenza A cause epidemics and pandemics	Mutations of Influenza A haemagglutinin and neuraminidase allow virus to escape host antibodies (**antigenic drift**) and epidemics, whereas replacement of these with animal-derived RNA segments leads to new virus (**antigenic shift**) and pandemics	Needs bolded
[illegible]	[illegible]	[illegible]	Need bolded

Influenza 2005-1

Influenza	How are influenza viruses classified? Prompt: what is antigenic drift and shift? (require A, and some understanding of antigenic drift)	**A** – humans, pigs, horses, birds, major cause of pandemics/epidemics. Subtypes determined by hemmaglutinin and neuraminidase in envelope. Shows antigenic drift (H & N mutations) - epidemics, antigenic shift (H&N replaced by recomb RNA from animal viruses) - pandemic **B & C** no antigenic drift/shift – more in children – develop abs	
	How does the immune system specifically attack the influenza virus?	Abs to H & N if previous infection, cytotoxic T cells, anti-influenza protein in marcrophages(Mx1) induced by cytokines	

Malaria 2016-2-C

Stem: A 36-year-old woman returns from Africa febrile. Starting with Pathology			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1: Malaria **Subject:** Path LOA: 1	What organisms cause malaria? Name the types [What sort of organism is that?] Describe the pathogenesis of malaria [What happens next?] [What does it do to the blood?] How does P falciparum present clinically?	Malaria is a **protozoal** infection, intracellular **parasite** *Plasmodium* **P falciparum**, P ovale, P vivax, P malariae Infectious stage (sporozoite) is found in saliva of female anopheles **mosquito.** Sporozoites released into blood and attach and **invade hepatocytes** . **Multiply** rapidly. **Hepatocyte ruptures**, releasing up to 30000 merozoites (NB P vivax and ovale have dormant hepatic stage therefore can relapse). Released merozoites from liver **bind to** surface of **RBC**, grow in vacuole. In RBC become trophozoite (single chromatin), then schizont (multiple chromatin masses) then each chromatin becomes merozoite again. **RBC lyses** and new merozoites infect additional RBCs. Only erythrocytic parasites cause illness **Fever,** severe **anaemia**, ARF, **cerebral symptoms**, pulm oedema, DIC Congestion and enlargement of spleen Infected RBCs clump → ischaemia due to poor perfusion→ manifestations of cerebral malaria (vessels plugged with parasitized RBCs, local venous stasis, local hypoxia and inflammatory infiltrate) ARF (Hb casts in tubules, pigment etc in glom) Stimulates cytokines, TNF, IFN, IL1 → pulm oed, fever, shock	**Parasite or protozoa** **P falciparum** + 1 **Bold and general idea** **Fever + 1**

Malaria 2008-2

3. Malaria	1. What micro-organisms cause malaria?	Parasitic protozoa **Plasmodium falciparum**, vivax, ovale, malarie	Falciparum +1
	2. How does Plasmodium falciparum infection differ from other forms of malaria? Prompts: How does it compare clinically? By what mechanism?	All do: sporozoite→liver→merozoites formed → release & bind to RBC→ Hb hydrolysed → trophozoite→ schizont → merozoite/gametocyte *P. falciparum:* **infects RBCs of any age**, causing **clumping/rosetting** so ischemia, high cytokine production, **high level parasitemia**, severe anemia, cerebral symps, renal failure, pul oedema, death *Others*: infect only new or old RBCs, P vivax & ovale form latent hynpnozoites (relapses), low parasitemia, mild anemia, rarely splenic rupture, nephrotic synd	2/3 Highlighted and 1 clinical feature
	3. What factors can make people less susceptible to malaria?	**Inherited alterations in RBCs: HbS** trait, HbC, Duffy Ag neg Repeated exposure stimulates immune response: Ab and T lymphocytes (P falc avoids this), HLAB53	Highlighted

Malaria 2003-2

3.4 Genesis of Plasmodium falciparum infection	Describe the sequence of events in Plasmodium falciparum infection What are the complications of P. falciparum infection?	**Anopheles** genus mosquito –>sporozoites –> liver cells. **Hepatocyte rupture** releases merozoites -> **RBC**s. In RBCs mainly merozoites **rupture cells**; some gametocytes form, are sexual, invade mosquito. Mature merozoites change in RBCs to shizont form, cell is rigid, **removed in spleen**. Anaemia, cerebral malaria, renal failure, pulmonary oedema, hepatosplenomegally, splenic rupture, DIC.	3/

Measles 2016-2-C

Stem: Moving onto Pathology. Measles is suspected.			
Question 5 Measles **Subject:** Pathology LOA: 2	What type of virus is Measles?	Single stranded **RNA virus,** a member of the **Paramyxovirus** family (Mumps, RSV and Paraflu). There is only one strain of virus – so preventable by vaccine.	1 Bold to pass
	How is it spread?	**Respiratory droplet spread.**	Bold
	Describe some of the clinical manifestations of Measles infection. [What are the serious manifestations?]	Viral pneumonia (60% of deaths) Conjunctivitis and Keratitis – scarring and blindness **Acute Measles Encephalitis** (1:1000) Adults> kids Subacute sclerosing panencephalitis (1:100000) Diarrhoea (enteropathy) Immunosuppression Croup	**Encephalitis** and 1 other
	What immune responses occur as a result of Measles infection?	T-cell mediated immunity controls the infection and produces the rash – a hypersensitivity reaction to viral antigens in the skin. (no rash if deficient cell mediated immunity) Antibody mediated immunity to Measles virus protects against reinfection	Bonus

Measles 2014-1-B

Stem: A 3 year old boy presents to ED with measles.			
TOPIC	QUESTIONS	KNOWLEDGE (**essential in bold**)	NOTES
The first questions is in regard to pathology			
Question 1 Measles **Subject:** Path LOA: 2	1.What organism is responsible for measles infections and how is it transmitted? 2.What type of immune response occurs in measles? 3.What are the clinical features of measles? 4. What are the complications of measles?	1.**Virus,** RNA, Paramyxo >>**respiratory transmission** 2.T cell mediated controls infection and causes rash Antibody mediated protects against reinfection 3. **fever, rash**, **conjunctivitis, cough/coryza**, Koplik spots, lymph nodes. 4. pneumonia, secondary bacterial infection, delayed – encephalitis, SSPE	Bold to pass Antibody mediated 3 bold to pass 2 as minimum.

Measles 2011-1

Question 3. **Measles**	1. **Describe the pathogenesis of measles** PROMPTS: What type of virus is measles? What is the mode of transmission?	1. **Paramyxovirus (single stranded RNA)** 2. **Respiratory droplet spread** 3. Multiplies in upper respiratory tract epithelial cells 4. >lymphoid tissue where it replicates in mononuclear cells 5. haematogenous spread 6. Preventable by vaccination as only single strain. 7. Epidemics amongst un-vaccinated individuals	• **Virus** • **Respiratory droplet spread** • **+ 1 other**
	2. What type of immune responses occur in measles?	• **T cell mediated immunity** controls infection + causes rash • **Antibody mediated** protects against re-infection • epidemics in unvaccinated hosts	• cell mediated • antibody mediated
	3. **Describe some of the systemic features of measles virus infection.** Prompt: What are some complications of measles infection?	• **Rash**–blotchy, red/brown. Skin hypersensitivity reaction • Oral mucosal ulceration – **Koplik's spots** • **Croup** • **Interstitial pneumonia** • **Conjunctivitis, Keratitis**, with scarring and visual loss • **Encephalitis**; - plus SSPE, measles inclusion-body encephalitis • **Diarrhoea** with protein losing enteropathy • **Immunosuppression** • **Secondary bacterial infection**	• Rash • + 3 others

Measles 2007-1

3. Measles	1. What is the mode of transmission of the measles virus?	**1. Respiratory droplets** 2. Upper respiratory epithelial cells	
	2. Describe some of the systemic features of measles virus infection. Prompt: "One feature is croup or pneumonia. Tell me others"	1. **Rash**–blotchy, red/brown. Skin hypersensitivity reaction 2. Oral mucosal ulceration – Koplik's spots 3. Croup, or interstitial pneumonia 4. Keratitis, with scarring and visual loss 5. Encephalitis; - plus SSPE, measles inclusion-body encephalitis (immunocompromised) 6. Diarrhoea with protein-losing enteropathy 7. Secondary bacterial infection	At least rash and 3 others
	3. What are the cell-surface receptors for the measles virus Prompt: "One example is CD46. Tell me about this"	1. CD 46 (complement regulatory protein): inactivates C3 convertases; present on all nucleated cells; binds viral haemagglutinin protein 2. SLAM (Signalling Lymphocytic Activation Molecule): involved in T cell activation; only present on cells of the immune system; binds viral haemagglutinin protein	Supp question

Neiserria 2016-1-D

Stem: Moving onto Pathology			
Question 4 Neisseria meningitidis **Subject:** Path LOA: 1	1. How does Neisseria meningitidis cause infection	1. Common coloniser of oropharynx - 10 % of popn at any one time, carry it for months 2. Spread by resp droplets 3. Most people develop immune response and clear it – protected against later infection from this serotype (>/13 serotypes). 4. Invasive dx when encounter new serotype 5. Invades resp epithelium, then blood stream 6. Capsule allows evade immune response by prevention opsonisation and complement destruction 7. Mortality still approx. 10% despite AB Rx.	Need 4/7 to pass
	2. What are the clinical consequences of N. meningitidis infection	Death, gen sepsis, necrotising vasculitis, seizures, SIADH, CVA, hydrocephalus, meningitis, sensorineural hearing loss, cognitive impairment	Need 4 to pass
	3. Apart from Neisseria, what else can cause meningitis?	Other bacteria: E coli & Group B Strep (infants), Strep pneumonia, Listeria, Haemophilus, Listeria Viral: Enterovirus, measles Other: TB, Rickettsial, Carcinoma, Auto-immune, chemical	Must have two specific bacteria plus viral as a group

Neiserria 2009-1

Question 3:	**What are the microbiological features of *Neisseria*?**	1. Aerobic 2. Gram negative diplococci 3. Coffee bean shaped 4. Require chocolate blood agar 5. 13 serotypes of *N. meningitidis*	Prompt: What are the staining characteristics of *Neisseria?*

Neiserria 2009-1

Question 3: **Neisserial infections**	**What are the two clinically significant *Neisseria*?** **Describe the pathogenesis of a *N. meningitidis* infection**	1. *meningitidis* 2. *gonorrhoeae* 1. Respiratory spread 2. Common coloniser of the oropharynx 3. (10% of the population at any one time) 4. Colonisation lasts for months 5. Immune response leads to protection against that strain 6. Invasive disease crosses respiratory epithelium to enter blood 7. Capsule of *Neisseria* reduces opsonisation & protects against destruction by complement proteins 8. Outbreaks in young people living in crowded quarters who encounter new strains	Both Need 5/8 Prompt: How does it spread?

Staph Aureus 2017-2-D

Stem: Moving onto Pathology. You suspect Toxic Shock Syndrome.			
Question 2 Staph Aureus **Subject:** Pathology LOA: 1	(a) Describe the virulence factors of Staph aureus *Prompt: How does Staph aureus cause disease*	**Surface proteins:** involved in adherence- (express receptors for fibrinogen (and others) to bind to host endothelial cells, and artificial materials), and evade host immune response **Secreted enzymes**: degrade proteins (promoting invasion and destruction) e.g. lipase degrades skin lipid associated with ability to produce abscess **Secreted toxins**: that damage host cells: - Alpha toxin- membrane depolarisation/ damage - Beta toxin- sphingomyelinase - Exfoliative A and B toxin - Gamma toxin and leukocidin - Superantigens –TSS and food poisoning	Toxins with example plus 1 other bold.
	(b) What are the risk factors for Toxic Shock Syndrome?	Use of tampons, Post op wound infection, Post partum, Nasal Packs, Staph or strep skin infection	Any 2
	(c) What are the clinical features of Toxic Shock Syndrome?	Hypotension (shock), acute renal failure, coagulopathy, respiratory failure, soft tissue necrosis at site of infection, a generalized erythematous rash,	Any 3

Staph Aureus 2015-2-B

Stem: Moving onto Pathology. On examination there is a purulent discharge coming from the wound.			
Question 3 Staph aureus **Subject:** Path LOA: 1	(a) Name some common bacteria that cause wound infections	**Staphylococcus aureus** **Streptococcus pyogenes** Clostridium perfringens Aerobic Gram negative bacilli Pseudomonas aeruginosa Clostridium tetani	Staph aureus, Strep and 1 other
	(b) What diseases are caused by Staphylococcus aureus?	Skin / soft tissue : cellulitis, impetigo, abscess (furuncle, carbuncle), folliculitis, paronychia, felon, lymphadenitis, necrotising soft tissue infection, scalded skin syndrome Pneumonia Endocarditis Osteomyelitis / septic arthritis Food poisoning Toxic shock syndrome	3 skin and 3 non-skin infections
	(c) Describe the clinical features of Staph. Aureus toxic shock syndrome.	Hypotension (shock), renal failure, coagulopathy, liver disease, respiratory failure, generalised erythematous rash, soft tissue necrosis at site of infection	4 out of 7 (must have specific organs)

Staphlococci 2012-2

Q3 Staph infections LOA: 1	1. Describe the virulence factors of Staph aureus. What infections do the different species of Staphylococci cause? 2. *Prompt: Name the Staphylococcal species*	a. **Surface proteins involved in adherence** – expresses receptors for fibrinogen (and others) to bind to host endothelial cells. b. **Secreted enzymes that degrade proteins** (promoting invasion and destruction) e.g. lipase degrades skin lipids associated with ability to produce abscesses **c. Secreted toxins that damage host cells** alpha toxin – membrane depolarisation/damage; beta toxin – sphingomyelinase; Exfoliative A & B toxin; Superantigens – TSS and food poisoning **S. aureus** – skin, pneumonia, osteomyelitis etc **S. epidermidis** – opportunistic eg prosthetic valves **S. saprophyticus** – UTI in women	2/3 bolded sections including toxin 2 of the 3 bolded

Streptococci 2016-2-C

Stem: Moving onto Pathology. Blood cultures grow a Streptococcus			
Question 5 Streptococcal infection **Subject:** Path LOA: 1	1. What is the microscopic appearance of streptococci? 2. What are some post-infectious syndromes caused by streptococcal infections? 3. List some infections caused by streptococcus	1. **Gram positive cocci in pairs** or chains 2. -**Rheumatic fever** (+/- complications, chorea) -**Immune complex glomerulonephritis** -Erythema nodosum, rash, myoclonus, myalgia, arthritis, neuropsychiatric sequelae, tics 3. Mouth – dental caries – *S.mutans* Skin – erysipelas – *S.pyogenes (grp A strep)* Skin – scarlet fever – *S.pyogenes* ENT – pharyngitis - *S.pyogenes* Lungs – pneumonia – *S.pneumoniae / pneumococcus* CNS - meningitis – *S.agalactiae (grp B strep)* Neonatal sepsis CV – endocarditis – *S.viridans*	1. **Bold** **2. 1 bold +1** **3. Any 4**

Streptococci 2012-2

Q3 Strep infections LOA: 1	1. What types of infections do Streptococcal bacteria cause? *Prompt: Give examples of the different strep subtypes and the infections they cause?*	1. Acute **suppurative: skin, throat, lungs and heart valves.** Group A S.pyogenes (throat, skin), Group B S.agalactiae (female genitial, neonate sepsis), α Haemolytic, S.pneumoniae (CAP), meningitis S.viridans (mouth, SABE), S.mutans (teeth)	Pus >= 2 to pass
Fri PM Q3 Strep (con'td)	2. What post infectious syndromes do streptococci cause?	2. GN, rheumatic fever, erythema nodosum	1 to pass

Streptococci 2009-2

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Qn 1	What are streptococci?	**Gram-positive cocci** growing in pairs or chains. Facultative or obligate anaerobes. Cause variety of suppurative infections and immunologically mediated post-streptococcal syndromes.	Bold to pass
Qn 2	Name some of the different types of streptococci and give examples of diseases they cause.	Alpha haemolytic - **S. pneumonia - pneumonia** - meningitis S viridans - endocarditis β Haemolytic - **Group A. (Pyogenes)** pharyngitis - scarlet fever - erysipelas - Impetigo - Rheumatic fever - Toxic Shock Syndrome - Glomerulonephritis Group B. (Agalactiae) - neonatal sepsis and meningitis - chorioamnionitis Strept. mutans - dental caries	3 major type/group + 6 diseases to pass
Qn 3	What factors in streptococci contribute to their virulence? .	Capsules pyogenes, pneumoniae M Protein prevents phagocytosis (anti M protein AL → Rh.F.) Complement C5a peptidase Pneumolysin lyses target cells (S pneumoniae) activates complement Pyrogenic exotoxin- rash and fever High MW glucans plaque formation aggregation of platelets Sucrose → lactic acid (S. mutans).	Any 3 to pass Capsule important.

Varicella Zoster 2014-2-C

Stem: We are now moving to pathology. The rash is diagnosed as Varicella Zoster			
Question 3 Varicella Zoster (p 353) **Subject:** Path LOA: 1	1. What are the 2 clinical conditions caused by this virus	Chicken pox and shingles	**Both**
	2. Describe the pathogenesis and clinical course of infection with this virus Prompt: start with how the virus is transmitted	Starts with aerosol or direct contact spread →haematogenous dissemination → vesicular skin lesions → vesicles rupture, crust over then heal Some virus lies dormant in dorsal root ganglia and reactivated later with immunosuppression	**Reasonable sequence**
	3. What are the complications of chicken pox	Lung → interstitial pneumonia Nervous system - encephalitis, transverse myelitis Skin and mucous membranes → shingles, bacteria superinfection Gut – necrotising visceral lesions	**3 to pass**

Varicella Zoster 2006-1

TOPIC: Thursday AM Q 5 - Varicella Zoster_____ **NUMBER:** _____

OPENING QUESTION	What tissues may be involved in a primary varicella zoster infection?	**COMMENTS**
POINTS REQUIRED	1. Mucous membranes	
	2. Skin	
	3. Neurones	
PROMPTS		
SECOND QUESTION (if needed)	What is shingles?	
POINTS REQUIRED	1. Reactivation of latent virus	
	2. Erodes immune response	
	3. Dormant in sensory ganglion	
	4. Interstitial pneumonia	
	5. Visceral lesions	
	6. Encephalitis etc	
PROMPTS		

Varicella Zoster 2005-2

TOPIC: Varicella _____ **NUMBER:** 3c

OPENING QUESTION	Describe the illness caused by acute varicella infection.	**COMMENTS**
POINTS REQUIRED	1 Transmitted in epidemic fashion by aerosols, dissemination haematogenously	3 to pass
	2 Rash occurs about 2 weeks after respiratory aerosol exposure.	
	3 Rash begins centrally & spreads centifugally in multiple waves. Rash initially macular with rapid progression to a vesicle	
	4 After a few days the vesicle ruptures, and crusts over. Most heal with no scarring (unless bacterial superinfection)	
	5 Involves skin & mucous membranes.	
	6 A milder illness in childhood cf. adults	
PROMPTS	How is it transmitted?	
SECOND QUESTION (if needed)	What are the complications of acute varicella infection?	
POINTS REQUIRED	1 Secondary bacterial skin infection	1 plus one other
	2 encephalitis/cerebellitis	
	3 interstitial pneumonitis	
	4 transverse myelitis	
	5 necrotizing visceral lesions (esp. in the immunosuppressed)	
PROMPTS	What possible complications from the skin lesions	
THIRD QUESTION (if needed)	Describe the relationship between varicella infection and subsequent zoster eruption.	Optional for good candidate
POINTS REQUIRED	1VZV evades immune defences & infects neurons in and around dorsal root ganglia	
	2 Able to remain latent here for many years	
	3 Usually a single episode of recurrence in the form of zoster	
	4 Reactivation often in the elderly or immuno-compromised	
	5Vesicular eruption along dermatome of one or more sensory nerves	
	6Associated intense burning, itching and pain due to radiculoneuritis. May cause nerve dysfunction (eg Ramsay Hunt syndrome)	
PROMPTS	Describe the clinical picture of zoster eruption.	