ACEM Primary Examination Vivas > Pathology > Musculoskeletal
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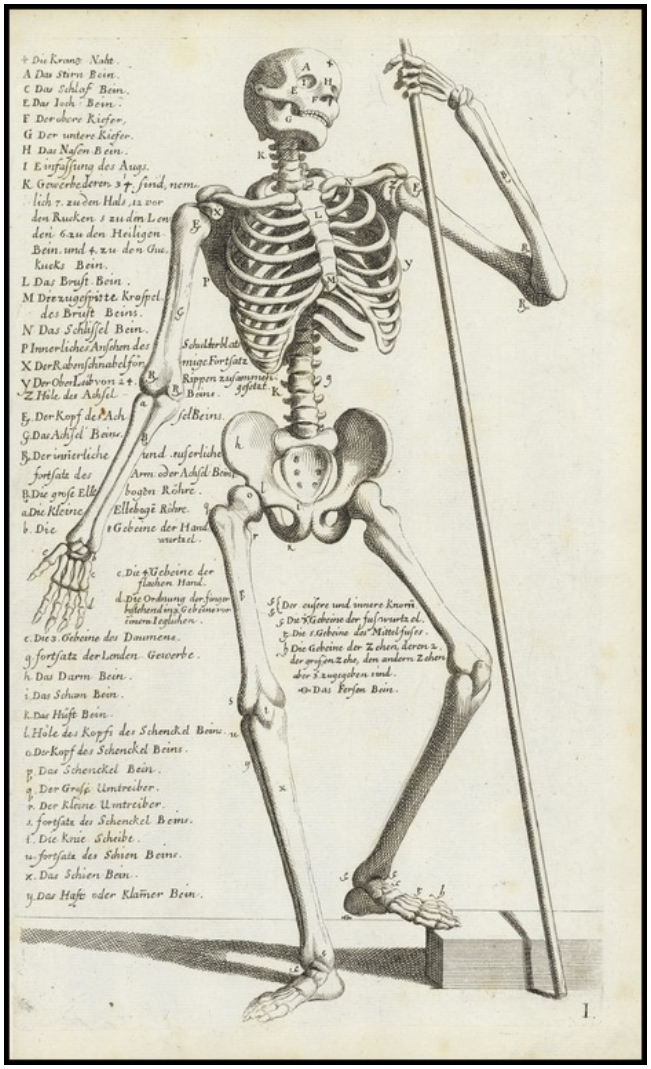

Fracture Healing 2016-2-C

Stem: He has multiple fractures. Moving onto Pathology.			
Question 5 Fracture healing **Subject:** Pathology LOA 1	1. How are fractures classified	• Complete/incomplete • Open/Closed • Comminuted • Displaced • Pathologic • Stress	3/6
	2. Describe the steps in fracture healing	1. **Haematoma** fills fracture gap – provides fibrin mesh framework (hours) 2. **Influx** of inflammatory cells, fibroblasts, new vessels (days) 3. Haematoma organising –> **Procallus** 4. **Ossification –> bony callus** (2-3/52) 5. Callus matures, **remodelling** (6 weeks)	• 4 of 5 steps • Logical sequence
	3. What factors can impede the healing of fractures	• **Inadequate immobilisation** • Marked displacement/soft tissues • Vascular compromise • **Infection** (open fractures/foreign bodies) • Systemic factors (nutrition, osteoporosis, smoking…)	BOLD and 1 other

Fracture Healing 2015-2-C

Stem: Moving on to Pathology.			
Question 4 Fracture Healing **Subject:** Path LOA: 1	1. How do fractures heal? Prompt: What are the stages of fracture healing?	1. **Haematoma** 2. **Influx of Inflammatory cells, platelets, fibroblasts, new vessels and osteoprogenitor cells = procallus** 3. **Fibrocartilagenous callus**: mesenchymal cells + new cartilage along fracture line undergoes **endochondral ossification =** 4. **Bony Callus** 5. **Remodelling**	3/5 to pass + detail
	2. What factors inhibit fracture healing	Inadequate immobilisation/severe displacement/poor reduction/soft tissue Vascular compromise Infection, foreign body Systemic – nutrition, osteoporosis etc.	3 to pass

Fracture Healing 2015-1-B

Stem: Moving onto Pathology. A junior doctor asks you about the healing process for the fracture			
Question 4 Fracture Healing **Subject:** Path LOA: 1	Describe the steps in fracture repair process	1 **haematoma** fills fracture gap – provides fibrin mesh framework (hrs) 2 **influx** inflam cells, fibroblasts, new vessels (days) 3 haematoma organising -> **procallus** 4 osteoprogenitors deposit trabeculae of woven bone – **ossification** -> **bony callus** (2-3 weeks) 5 callus matures, **remodelling** (6 weeks)	4 of 5 steps Logical sequence
	How does remodelling of callus occur?	Initial large volume of callus – portions not physically stressed are resorbed, reducing callus size/altering contour	Physical stress, resorption
	What factors can impede the healing of a fracture?	**Inadequate immobilisation**, marked displacement, **infection** (open fractures/FBs), systemic factors (nutrition, smoking…)	2 bold and 1 other
	(Supplementary – if time remaining) How are fractures classified?	Complete/incomplete, open/closed, comminuted, displaced, pathologic, stress	

Fracture Healing 2012-1

Question 2 Fracture healing LOA: 1	**How do fractures heal?** Prompt: What are the timeframes of these stages?	1 Haematoma formation/fibrin mesh - hrs 2 Inflammatory cell influx - days 3 Fibroblast/ Osteoprogenitor cells-procallus 4 Organised haematoma - 1wk, 5 Woven bone , bony callus - 2-3 wks 6 Callus maturation remodelling - 6 wks	Must have reasonable sequence and approximate times, at least 4 components to sequence
	What factors impair fracture healing?	Inadequate immobilisation, severe displacement, vascular compromise, infection /FBs, poor nutrition, systemic illnesses	**At least 3**

Fracture Healing 2007-2

TOPIC: Fracture Healing **NUMBER: Q1**

OPENING QUESTION	**Describe the process of fracture healing**	**COMMENTS**
POINTS REQUIRED	1 **Haematoma formation** - immediately after # / provides a fibrin mesh / framework for influx of cells	* Need 4/6 to pass
	2 **Influx of inflammatory cells** - plus ingrowth of fibroblasts + new capillary vessels	*
	3 **Stimulation of osteoclastic and osteoblastic activity** – by degranulated platelets and migratory inflammatory cells release of PDGF, TGF-B, FGF, other cytokines activation of posteoprogentitor cells in periosteum, medullary cavity, soft tissues, haematoma becomes organised	
	4 **Resultant soft tissue callus (procallus)** – by the end of the first week / fusiform uncalcified tissue unites fracture ends, but no structural rigidity	*
	5 **Bony callus - deposition of bone +/- cartilage** – activated osteoprogenitor cells deposit subperiosteal trabeculae +/- fibrocartilage and hyaline cartilage / repair tissue maximal girth 2nd-3rd week / mineralising and strengthening allows limited wt bearing	*
	6 **Callus maturation and remodelling of bone** – reestablishment of shape and outline of the bone, restoration of medullary cavity	
prompt	How is callus formed?	
SECOND QUESTION	**What factors may impede fracture healing?**	4 out of 6 to pass
POINTS REQUIRED	1 inadequate immobilisation	*
	2 severe displacement of fracture	
	3 devitalised tissue / vascular insufficiency	
	4 infection	*
	5 nutrition – inadequate Ca, PO4, vitamins	*
	6 Other eg diabetes, systemic infection	*

Fracture Healing 2005-1

Fracture healing	Describe the process of fracture healing. (3/4 stages required) What factors may interfere with fracture healing? *(3/6 required)*	1. Haematoma formation / fibrin mesh ✱ 2. Ingrowth of inflammatory cells / Activation of osteoprogenitor cells (procallus) ✱ 3. Woven Bone ✱ 4. Callus Formation ✱ 5. Remodelling times extra 1. Displaced / Comminuted 2. Foreign Bodies 3. Inadequate immobilization 4. Infection 5. Nutrition: Calcium / Phosphate 6. Diabetes / Systemic Illness	

Fracture Healing 2003-1

TOPIC: Healing of Fractures **NUMBER:** ____

OPENING QUESTION	Describe the process of fracture healing	**COMMENTS**
POINTS REQUIRED	1 Haematoma formation / fibrin mesh	5 of 6
	2 Ingrowth of inflammatory cells	
	3 Activation of osteoprogenitor cells	
	4 Woven bone	
	5 Callus	
	6 Remodelling	
	7	
PROMPTS		
SECOND QUESTION (if needed)	What factors may interfere with this process	
POINTS REQUIRED	1 Displaced / Comminuted	5 to pass
	2 Foreign Bodies	
	3 Inadequate immobilisation	
	4 Infection	
	5 Nutrition Calcium / Phosphate	
	6 Diabetes / Systemic Illness	
PROMPTS		

Gout 2013-1

Question 5 Gout LOA: 2	1. What are the causes of gout?	**Hyperuricaemia:** 1. Primary Gout (90%; often idiopathic): Overproduction (diet, unknown enzyme defects); Reduced filtration/excretion with normal production. 2. Secondary Gout (10%; known cause, secondary effect is gout): Leukaemias/tumor lysis/psoriasis, inborn errors of metabolism (overproduction with increased excretion); Chronic renal disease (reduced excretion).	Hyperuricaemia + 1 Primary and 1 Secondary cause Or 1 overproduction and 1 decreased excretion
	2. Describe the pathogenesis of acute gouty arthritis. Prompt- What are the steps involved?	1. Hyperuricaemia 2. **Precipitation of urate crystals into joints** (in synovium / cartilage) 3. **Release of crystals into synovial fluid** (?trauma) 4. **Inflammatory response initiated** – crystals phagocytosed by macrophages and neutrophils; release of inflammatory mediators by macrophages (interleukins, cytokines (IL-1B)); resulting in further neutrophil chemotaxis; neutrophils also release inflammatory mediators (free radicals, leukotrienes (LT B4), lysosomal enzymes) – acute arthritis.	Bold to pass
	3. (only if needed) What factors contribute to the conversion of asymptomatic hyperuricaemia into gout	Age & duration of hyperuricaemia; genetic predisposition/etoh/obesity/drugs e.g. thiazides/lead toxicity	

Gout 2011-2

Question 5 Gout LOA: 2	Describe the morphological features of gout (Prompt pathological features)	**Acute arthritis-crystallisation of urates within or around joint** An event possibly trauma initiates the release of crystals into the synovial fluid. **Chronic arthritis** with repeated attacks- formation **tophi** in the inflamed synovial membrane and periarticular tissue **Nephropathy**- deposit urate crystals in kidney and formation **uric acid stones**	3 Bold to pass including arthritis
	What are the causes of gout?	**Hyperuricaemia** **Primary** (90%)-enzyme defects unknown (85-90%), known enzyme defect eg. HGPRT deficiency-rare overproduction of uric acid, under excretion or increased excretion **Secondary-**increased nucleic acid turnover eg. leukaemia(overproduction and excretion), CRF (reduced excretion with normal production), inborn errors of metabolism (over production and excretion)	Need hyperuricaemia plus 1 primary and 1 secondary cause to pass

Gout 2008-1

10/4/2008 Q4	Describe the pathological features of gout	**Hyperuricaemia**	
		Acute arthritis Precipitation of urate crystals into the joint/s An event (sometimes minor trauma) releases crystals into synovial fluid Cascade occurs resulting in intense inflammatory reaction (complement activated, chemotaxis of neutrophils and macrophages with phagocytosis and activation of lysosymal enzymes, leukotrienes, prostaglandins and free radicals **Chronic arthritis and formation of tophi** which are urate deposits in synovium and periarticular areas **Nephropathy** – deposition of urate in kidney as well as formation of **uric acid stones**	Pass criteria: 3/4
	What are the causes of gout?	**Primary** – enzyme defect unknown (90%) (overproduction, underexcretion or increased excretion) - rare enzyme defect (HGPRT deficiency) **Secondary** (10%) Increased nucleic acid turnover e.g leukaemias (overproduction and excretion) Chronic renal disease (decreased excretion) Inborn error metabolism (complete HGPRT deficiency – Lesch-Nyhan syndrome) overproduction and excretion	Need primary & 1 secondary to pass.

Gout 2005-2

TOPIC: GOUT **NUMBER:** 5c

OPENING QUESTION	Describe the pathogenesis of gouty arthritis.	**COMMENTS**
POINTS REQUIRED	1. Purine metabolism – 2 pathways.	3 to pass
	2. Hypoxanthine guanine phosphoribosyltransferase.	
	3. Supersaturation – synovial fluid.	
	4. Chemotactic, complement activation.	
	5. Macrophage → free radical, leukotrienes, lysosomal enzymes. Hagemann.	
	6. Acute arthritis – cartilage, joint damage	
PROMPTS		
SECOND QUESTION (if needed)	What are the risk factors for primary gout	4 to pass
POINTS REQUIRED	1. Age	
	2. Genetic predisposition	
	3. Alcohol	
	4. Obesity	
	5. Drugs	
	6. Lead toxicity	
PROMPTS		

Osteoarthritis 2010-2

	cholecystitis.	3.1. Mild fever, anorexia, tachycardia, sweating, nausea, and vomiting, tender RUQ (Murphy's)	3. 1/3
Question 3.5 Osteoarthritis	1. What factors lead to osteoarthritis 2. Describe the pathological changes that occur in an affected joint 3. Describe the major clinical features of osteoarthritis	1.1. Genetic & environmental (mechanical) 1.2. Age – virtually ubiquitous (80-90%) after 65 1.3. Other exacerbating diseases e.g. Obesity, diabetes, injury, abnormal joints, 2.**Chondrocyte injury** 1.3.1. Early OA: **chondrocytes proliferate (cloning) and secrete inflammatory mediators**, collagens, proteoglycans, and proteases which initiates secondary inflammatory changes. 1.3.2. Later OA: repetitive injury and chronic inflammation lead to chondrocyte drop out, marked loss of cartilage, and extensive **subchondral bone changes** 3.Mostly **asymptomatic** <50y.o. 1.4. Deep, achy **pain** worse with use, **morning stiffness, crepitus**, and **limited ROM** 1.5. **Oligoarthritis** 95% (occas generalized/early) 1.6. Impingement on spinal foramina by osteophytes results in cervical and lumbar nerve root compression and **radicular pain**, muscle spasms, **muscle atrophy**, and **neurologic deficits**. 1.7. Common: hips, knees, lower lumbar and cervical vertebrae, PIP, DIP of the fingers, 1st carpoMC joints, and 1st TarsoMT joints. Not wrists, elbows, shoulders	1. 2/4 answers 2. 2/3 bold, 3. 2/4

Osteoarthritis 2005-2

TOPIC: Osteoarthritis **NUMBER:** 5a

OPENING QUESTION	Describe the morphology of osteoarthritis affecting a major joint	**COMMENTS**
POINTS REQUIRED	1. Early - ↑ chondrocytes, ↑ water, ↓ proteoglycans	4 of 8 to pass
	2. Fibrillation/cracking/softening	
	3. Sloughing, bone exposure	
	4. Eburnation	
	5. Dislodged cartilage and bone (joint mice)	
	6. Fibrous subchondral bone cysts	
	7. Osteophytes	
	8. Synovium – fibrotic, inflammatory cells	
SECOND QUESTION (if needed)	Describe the causes of secondary osteoarthritis	2 of 4 to pass
POINTS REQUIRED	1. trauma	
	2 congenital or acquired deformity	
	3 Systemic disease eg diabetes	
	4 Obesity	
	5	
	6	
PROMPTS		

Osteomyelitis 2017-1-D

Stem: A 50-year-old man who is on dialysis is being treated for right hip osteomyelitis. We will start with Pathology.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Osteomyelitis **Subject:** Path LOA: 1	a) Describe the pathogenesis of acute osteomyelitis	Haematogenous spread of organism to bone Extension from a contiguous site Local bone injury and direct organism entry	2/3
	b) What organisms cause osteomyelitis?	**Staphylococcus aureus** >80% of pyogenic ones Others: Escherichia coli, Klebsiella pneumoniae, Pseudomonas aeruginosa from IVDU and GU; Haemophilus influenzae, Group B streptococcus – in neonates	Bold + 1 other organism to pass
	c) What pathological changes occur to the bone?	• **Acute inflammation** - neutrophilic • **Abscess** – sub-periosteal / surrounding soft tissue • **Necrosis** – dead bone - sequestrum • Involucrum (fibrous tissue and reactive bone deposition) forms around devitalized infected bone	Bold to pass

	d) What are the possible sequelae of osteomyelitis?	• **Resolution**
		• **Chronic – up to 25%**
		  ○ Acute flare-ups
		  ○ Pathological fracture
		  ○ Endocarditis
		  ○ Severe sepsis
		  ○ SCC in draining sinus tracts
		  ○ Sarcoma in infected bone
			Bold + 1 complication of chronic osteomyelitis to pass

Osteomyelitis 2014-1-C

Stem: Several months after discharge, he develops osteomyelitis. Moving onto pathology.			
Question 4 Osteomyelitis **Subject:** Path LOA: 1	1.Describe pathogenesis of osteomyelitis. (Prompt what organisms cause osteomyelitis?)	***Local bone injury** and organism entry, **blood borne** organisms, neighbouring source entry. ***Staph Aureus** > 80% of pyogenic ones Others E coli, Kl Pneum, Ps Aerug from IVDU and GU, haemophilus influenza, Gp B Streptococcus. 50% no orgs found.	1.Bold + 1 to pass / other organism
	2.What changes occur to the bone?	***Acute inflammation, necrosis, abscess** Sclerosis, involucrum and sequestrum, lytic focus and surrounding necrosis- periosteal elevation	2.Bold to pass
	3.What are the pathological sequelae of osteomyelitis?	***** Chronic up to 25%, resolve, deformity and bone destruction**, severe sepsis, pathological fracture, endocarditis, SCC, sarcoma.	3.Bold

Osteomyelitis 2013-2-D

Stem: Moving on to Pathology: Her Xray reveals evidence of bony destruction in the mandible.			
PATHOLOGY Question 3 LOA: 1	1. Describe the pathogenesis of osteomyelitis. Prompt: How would this patient have suffered a bony infection of his jaw?	**Local infection** related to extraction of tooth **Blood borne** **Spread from neigbouring** gingival source.	2/3
	2. What organisms cause osteomyelitis?	**Staph Aureus majority** >80% pyogenic E Coli, Kl Pneum, Pseudo A, from GU tract or IVDU H Infl and GBS in neonates Viruses, Fungi, Parasites, TB, syphilis also About 50% no orgs found.	Staph A and 1 other
	3. What changes occur in the bone?	**Acute inflammation and necrosis**, abscess formation Sclerosis and **involucrum** formation Deformity and sequestrum formation, Draining sinus Characteristic lytic focus surrounded by zone of necrosis on X ray, lifting of periosteum 5-25% become chronic inflammation	Bold
	4. What are the clinical consequences of osteomyelitis?	Resolution after Rx with IV antibiotics and drainage Conversion to chronic O myelitis Deformity and bony destruction Severe sepsis syndrome, ARF etc.	2 to pass

Osteomyelitis 2011-1

Question 5. **Osteomyelitis**	1. **Describe the pathogenesis of osteomyelitis** PROMPT; how do organisms reach the bone?	3 basic methods of infection • **blood born** (haematogenous) • **local infection** (extension contiguous site) • **trauma /surgery** (direct implantation)	**2/3**
	3. What Bacterial **organisms cause osteomyelitis?** (good candidates differentiate by age; Neonatal versus adults)	• **S Aureus** • Gp B strep (neonatal) • S Aureus (> 80%) Surgery/open fractures mixed Patient with UTI or IV drug user • E. Coli, Pseudomonas, Klebsiella	**S Aureus and 1 other**
	2. What are the changes in the bone that occur in **osteomyelitis**	• New bone around area of necrosis • Involucrum • Abscesses • Sclerosis • Deformity • Sequestrum • Draining sinus	3 items

Rheumatoid Arthritis 2011-2

Question 5 Rheumatoid Arthritis LOA: 2	1.What is the pathogenesis of Rheumatoid arthritis?	Triggered by exposure of genetically susceptible host to an arthritogenic antigen resulting in chronic inflammatory change. Continuing autoimmune reaction with activation CD4 helper T cells and inflammatory mediators and cytokines that destroy the joint **Genetic susceptibility**- associations with HLA-DRB1 alleles **Environmental arthritogens-** unclear what-various microbial agents implicated- none proven **Autoimmunity-**once inflammatory synovitis initiated-autoimmune reaction T cells result in chronic destruction.	Auto immune plus one other
	2.What are the extra articular manifestations of rheumatoid arthritis	**2 Rheumatoid nodules –**elbows forearms, lumbar **Fibrinoid necrosis** of lymphocytes **Vasculitis –** purpuric, nail bed, neuropathy, ulcers	At least 3
	3 What are the long term complications of RA?	3 Joint destruction, renal failure,	Any details

Rheumatoid Arthritis 2005-2

TOPIC: Rheumatoid Arthritis **NUMBER:** 5b

OPENING QUESTION	Describe the morphology of the joint lesion in Rheumatoid Arthritis	**COMMENTS**
POINTS REQUIRED	1. Joints – perivascular inflammatory infiltration CD4+ helper T cells, plasma cells, Macrophages.	3 to pass
	2. ↑ vascularity	
	3. Organising fibrin, rice bodies	
	4. Neutrophil accumulation	
	5. Osteoclastic - juxta articular erosion, subchondral cysts, osteoporosis	
	6. Pannus	
	7. Fibrous ankylosis → bony ankylosis	
PROMPTS		
SECOND QUESTION (if needed)	What are the extra articular manifestations of RA	2 to pass
POINTS REQUIRED	1. Rheumatoid nodules - 25% forearm/elbow/occiput/lumbosacral. Less frequent – lungs/spleen/heart/pericardium/valves/aorta.	
	2. Fibrinoid necrosis – epithelioid cells/Lymphocytes/ Macrophage.	
	3. Vasculitis- purpura, ulcers, nail bed infarcts	
	Digital - endarteritis (neuropathy/ulcers/gangrene)	
PROMPTS		

Septic Arthritis 2016-2-D

Stem: You are concerned about Septic Arthritis. Moving onto Pathology			
Question 4 Septic Arthritis; Staphlyococcal infections Subject: Pathology LOA 1	a. Which organisms may cause septic arthritis?	**Staph**, **Strep,** Gonococcus, H influenza, Gram neg (E coli, Salmonella, Pseudomonas)	Bold to pass
	b. What are some predisposing conditions for septic arthritis?	- Immunosuppression – DM, steroids, other; - Joint trauma/surgery/prosthesis, - Chronic arthritis, IVDU	2 to pass
	c. Name two different species of Staphylococci and give examples of infections they cause?	**S. Aureus** – skin (furuncle, boil, carbuncle, impetigo, abscess, wound), pneumonia, osteomyelitis, GI/gastro, TSS S. Epidermidis – opportunistic, eg catheterized, IVDU, prosthetic valves S. Saprophyticus – UTI in young females	S.aureus and 2 examples + 1 other Staph and example to pass