2014.2.C.2

Question 2 Acute Pancreatitis (pp 893-896) **Subject:** Path LOA: 1	1. What are the potential causes of this man's pancreatitis? 2. What is the likely pathogenesis of acute pancreatitis?	1. **Gallstones, alcohol,** iatrogenic, viral, hyperlipoproteinaemia, hypercalcaemia, drugs, trauma, shock, vasculitis, genetic mutations, scorpion bite, atheroembolism, duct obstruction (tumour, parasites etc) 2. **Autodigestion of the pancreatic substance by inappropriately activated pancreatic enzymes,** eg trypsinogen	1. **Bold plus 1** 2. **Bold**
	3. What are the acute complications of severe pancreatitis?	Causes interstitial inflammation and oedema, proteolysis, fat necrosis and haemorrhage 3. Haemolysis, DIC, fluid sequestration, ARDS, diffuse fat necrosis. Peripheral vascular collapse; shock; acute renal tubular necrosis	3. **3 answers to pass**

2014.2.C.3

Question 4 Ischaemic Bowel (pp 791-793) **Subject:** Path LOA: 1	What conditions can lead to infarction of bowel	1. **Acute arterial obstruction** Atherosclerosis, Aortic aneurysm, Hypercoagulable state, OCP use, Embolism 2. **Intestinal hypoperfusion** cardiac failure, shock, dehydration, vasoactive drugs <u>systemic vasculitis</u> HSP, Wegeners granulomatosis <u>Mesenteric venous thrombosis</u> Hypercoagulable state, Invasive neoplasm, Cirrhosis, Trauma, Abdominal masses <u>Miscellaneous</u> Radiation, Volvulus, Stricture, Amyloid, diabetes	**BOLD to pass** **Minimum 2 from each bolded group** **2 from non-bolded**
	What are the clinical features of ischaemic bowel?	Severe **pain**, may be transient. Tenderness, peritonism, nausea, vomiting, bloody diarrhoea, melaena, shock, hyper/hypothermia, sepsis	**Bold + 3 features**
	What parts of the bowel are most susceptible to ischemic injury And why?	**Watershed zones** - Splenic flexure, sigmoid colon, rectum - Located at end of arterial supply Surface epithelium : Villi more at risk than crypts Intestinal capillaries run from crypts up villi to surface	**Must be able to explain why watershed zones are most at risk**

2014.2.D.3

Question 3	1. Describe the pathological effects on the liver long-term alcohol ingestion.	1. **Steatosis**: fatty change, perivenular fibrosis	Bold with 3 morphologic features of each to pass.
Alcoholic Liver Disease (pp 857-860) **Subject:** Path LOA: 1	PROMPT: please describe the morphological features	2. **Hepatitis**: liver cell necrosis, inflammation, Mallory bodies, fatty change, fibrosis 3. **Cirrhosis**: extensive fibrosis, hyperplastic nodules 4. (Hepatocellular carcinoma)	
	2. Which of these conditions reversible?	**Steatosis and Hepatitis are reversible**. Cirrhosis irreversible.	Bold to pass
	3. What are the possible sequelae of cirrhosis? Prompt: Complications?	**Portal Hypertension**, GIT Bleeding, Hepatic Failure, Coagulopathy, Hepatocellular Ca, Hepatorenal Syndrome, Hepatopulmonary Syndrome, Encephalopathy, Infection	Bold plus 3

2014.1.D.2

Question 3 Cholecystitis **Subject:** Path LOA: 1	(a) Describe the pathogenesis of acute calculous cholecystitis.	(a) **Chemical irritation of obstructed GB** • Mucosal phospholipases hydrolyse luminal lecithins to toxic lysolecithins • Protective glycoprotein mucus layer disrupted • Allows Bile salts to have detergent action on exposed mucosal epithelium • PGs contribute to inflammation • GB dysmotility develops • Distension and increased intraluminal pressure decreases mucosal blood flow	(a) Bold + 2/6
	(b) What are the complications of cholecystitis?	(b) **Bacterial infection - cholangitis / sepsis** • Perforation and localised abscess • Rupture and peritonitis • Biliary fistula • Porcelain gallbladder	(b) Bold + 2/4

2013.2.A.1

Pathology: Abdominal Aortic Aneurysms	1. What are the risk factors for development of abdominal aortic aneurysms?	Male; Smoking; Age > 60; Family History; Connective tissue disease (eg. Ehlers Danlos); Vasculitis; Hypertension, Diabetes; Atherosclerosis	5 to pass
	2. Describe the pathogenesis of AAA formation	**Atherosclerotic plaque** in intima compresses media with **degeneration and weakness of wall** and cystic medial degradation Local inflammation **Proteolytic enzymes** with collagen degradation -role of matrix metalloproteinases (MMP). Loss of vascular smooth muscle cells. Inappropriate Synthesis of non-elastic ECM	2 of 3 bold to pass
	3. What are the clinical consequences of an AAA?	**Rupture**: increase with diameter (higher if >5cm) & can be retroperitoneal OR intra peritoneal with rapid fatal haemorrhage Obstruction: ischaemia from branch vessel obstruction eg. mesenteric, vertebral, renal Embolism: plaque or thrombus Impingement or compression of adjacent structure (eg. ureter) Painless mass	Bold and 2 others.

2013.1.1

Question 4 Infective enterocolitis LOA: 2	1. What are the organisms that cause infectious enterocolitis?	**1. Bacterial**- E.coli, Salmonella, Shigella, Campylobacter, C.difficile, Cholera, Yersinia, Mycobacteria **Viral**- Norovirus, Rotavirus, Adenovirus Parasitic- Giardia, Amoeba, Cryptosporidium, other (nematodes, cestodes, trematodes)	Bold with 1 bact & 1 viral 3 examples total
	2. What is pseudomembranous colitis?	2. Colitis caused by **overgrowth of C. difficile** (also Salmonella, C.perfringens typeA, S.aureus) Associated with antibiotic use Forms a **pseudomembrane** made up of adherent layer of inflammatory cells and debris	Bold
	3. What are the risk factors for development of pseudomembranous colitis?	3. **Risk factors- advanced age, hospitalisation, antibiotic treatment**	2/3 Bold
	What are the clinical features of pseudomembranous colitis?	30% hospitalised patients colonised, but most aymptomatic **Fever**, leucocytosis, **abdominal pain**, cramps, hypoalbuminaemia, watery **diarrhoea**, dehydration, rarely gross bloody diarrhoea Diagnosis-usually detection of toxin Treat with metronidazole, vancomycin	Bold

2013.1.3

Question 4 Portal Hypertension LOA: 2	1/What are the causes of portal hypertension? May need to prompt for examples/classification.	1/ Incr resistance to portal blood flow Prehepatic – portal vein thrombosis or narrowing Hepatic – (most important)- **cirrhosis**, massive fatty change, schistosomiasis, granulomatous disease eg sarcoid/Tb Post hepatic - severe RHF, constrictive pericarditis hepatic vein occlusion	Bold. One from each other group
	2/What are the clinical consequences of portal hypertension?	2/ **Ascites** – with potential for infection **Porto-systemic shunts** : varices, haemorrhoids, spider naevi Congestive **splenomegaly** – thrombocytopaenia/pancytopaenia **Hepatic encephalopathy**	2/4 bold
	3/What mechanisms are involved in the formation of Ascites?	3/ Sinusoidal hypertension – Starling forces : Incr pressure and decr albumin Incr formation of hepatic lymph – exceeds capacity of thoracic duct- percolates into peritoneum Splanchnic vasodilation with dec BP=> Renal retention of sodium and water due to secondary hyperaldosteronism	2/3 concepts

2012.2.1

Q5 Cholecystitis LOA 2	1. Describe the pathogenesis of acute cholecystitis Prompt: what is the pathogenesis of acute calculous cholecystitis? *Prompt: What are the risk factors for acalculous cholecystitis?* 2. What is the role of bacterial infection in acute cholecystitis?	**1.** Disruption of protective mucous layer, bile salt detergent action -> irritation and inflammation (**occurs in absence of bacterial infection initially**) **90% due to gallstone obstruction** of neck or cystic duct; 10% acalculus cholecystitis Acalculous - Occurs in severely ill people, thought to be due to ischaemia (risk factors septic shock, immunosuppression, diabetes) burns, trauma 2. Often late	Concept and gallstones and acalculous to pass. Recognition of immunosuppression or critical illness to pass. Initial chemical irritation then bacterial superinfection.

2012.2.3

Q4 Cholelithiasis LOA: 2	1. What are the risk factors for the development of cholesterol stones?	**1.Age, Gender** – 25% in the > 80 yo, women > men; **Environmental factors** – OC, pregnancy – increase expression of hepatic lipoprotein receptors and stimulates hepatic HMG-CoA reductase – enhancing cholesterol uptake and synthesis. Obesity, rapid weight loss.; **Acquired disorders** – gallbladder stasis – neurogenic or hormonal; **Hereditary factors** – e.g. genetic factors encoding for hepatocyte proteins that transport biliary lipids - ATP-binding cassette (ABC) transporters.	3 of 5 bolded.
	2. Describe the pathogenesis of cholesterol stone formation.	2.Requires the following simultaneous conditions: **Bile supersaturated with cholesterol;** Hypomotility of gall bladder; **Cholesterol crystal nucleation** – accelerated; Hypersecretion of mucus in the gall bladder traps crystals – **aggregation into stones**	Bolded and displays understanding of concept

2012.1.2

Question 3 Jaundice LOA: 1	**Outline the normal metabolism and elimination of bilirubin?**	Bilirubin production from heme (breakdown of senescent **erythrocytes**) 2. Binds to serum **albumin** and delivered to liver. 3. **Hepatocellular uptake**. 4. **Glucuronidation** – bilirubin glucuronides excreted into bile. 5. Gut **deconjugation** – colourless urobilinogens. These and pigment residues excreted in faeces. ~20% urobilinogens **reabsorbed** in ileum and colon and returned to liver. Small amount of reabsorbed urobilinogen excreted in urine	**three of bold** to pass
	What are the common causes of jaundice? (Prompt for bold)	Disorders that affect the production and metabolism of bilirubin: 1. Predominantly unconjugated: **↑production** (haemolyisis; resorption of blood from internal haemorrhage; ineffective erythropoiesis); ↓hepatocyte uptake (drug interference with membrane carrier systems; Gilbert syndrome – some cases); impaired bilirubin conjugation (physiological jaundice of newborn - ↓UGTA1 activity; breast milk jaundice - β-glucuronidases; genetic deficiency of UGTA1 (Crigler-Najjar); Gilbert syndrome (autosomal recessive ↓UGTA1 activity); **hepatitis** (diffuse hepatocellular disease eg viral; drugs; cirrhosis). 2. Predominantly conjugated: **impaired bile flow;** deficiency of canalicular membrane transporters (Dubin-Johnson syndrome; Rotor syndrome)	**Bold to pass**

2012.1.3

Question 5 Chronic Pancreatitis	**What are the morphological features of chronic pancreatitis?**	Parenchymal fibrosis, reduced number and size of acini with relative sparing of islets of Langerhans. Variable dilation +- blockage of pancreatic ducts. Destruction of exocrine parenchyma and in later stages destruction of endocrine parenchyma. Calcification.	**Any 3.**
	What are the clinical consequences?	Irreversible impairment of pancreatic function including: Diabetes; Steatorrhea; Malabsorption chronic attack not immediately life threatening but long term outlook poor(50% 20-25 mortality) Disease may be silent. Amylase, lipase may not raise in chronic attack	**Any 3**
		Pseudocyst	

2011.2.2

Question 4 LOA: 2	1.Describe the potential effects on the liver of long-term excessive alcohol ingestion. PROMPT: Ask for morphological features if just list names of conditions	1. **Steatosis**: fatty change, perivascular fibrosis 2. **Hepatitis**: liver cell necrosis, inflammatory response, Mallory bodies, fatty change, fibrosis 3. **Cirrhosis**: extensive fibrosis, hyperplastic nodules 4. (Hepatocellular carcinoma)	Bold with some pathological features of each to pass. .
	2 Are any of these conditions reversible with abstinence from alcohol?	2 Steatosis and Hepatitis are reversible. **Cirrhosis irreversible.**	Bold Must know that cirrhosis is irreversible injury.
	3 What are the sequelae of liver cirrhosis?	**3 Portal hypertension, GIT bleeding, hepatocellular carcinoma, hepatorenal syndrome, coagulopathy Encephalopathy, infection**	Portal hypertension and 2 Bold

2011.1.2

Question 4. **Chronic gastritis**	1. What are the **causes of chronic gastritis?**	• **H Pylori** • Chronic bile reflux • NSAIDS • Autoimmune • Allergic response • Infections • Radiation	• Mechanical • Psychological stress • Chronic irritants (coffee, alcohol, caffeine) • Systemic disease • (Crohns, amyloid, graft vs host)	**H pylori** + 2 others
	2. Describe the features of **H pylori induced chronic gastritis**	• Most **common cause** • predominantly **antral** • **High acid** production • **Hypogastrinaemia** • Generates **ammonia** (specific test) • **Disruption normal mucosal defence** mechanisms		2/5
	3. What are the **complications of gastric ulcer?**	• Bleeding (15-20%) o Accounts for 25% of ulcer deaths • Perforation • Obstruction • Gastric adenocarcinoma (complication of chronic H. Pylori pangastritis)		2/3

2011.1.3

Question 4. **Ischaemic bowel disease**	**1. What conditions can lead to infarction of bowel?** **PROMPT; by what mechanisms do these conditions cause injury**	**Acute vascular obstruction** -atherosclerosis (esp. origin major vessels) -aortic aneurysm -hypercoagulable states -OC use -embolism **Intestinal hypoperfusion** -cardiac failure -shock -dehydration -vasoconstrictive drugs	Systemic vasculitis -Henoch-Scholein purpura -Wegener's granulomatosis Mesenteric venous thrombosis -hypercoagulable states -invasive neoplasms -cirrhosis -trauma -abdominal masses	Bolded headings with 4 clinical examples to pass
	2. **Describe the intestinal response to an acute ischaemic insult.** Prompt: what is the mechanism by which ischaemic bowel injury occurs?	• Initial hypoxic injury • **Secondary reperfusion injury** - major injury in this phase - free radical production, neutrophil infiltration, inflammatory mediator release • Magnitude of response determined by - vessels affected - timeframe over which ischaemia develops		Must know that it is predominantly a **reperfusion type injury**
	3. **Which parts of the bowel are most susceptible** to acute ischaemic injury and **why**?	**Watershed zones** -splenic flexure, sigmoid colon and rectum -located at end of arterial supply Surface epithelium: Villi more at risk than crypts -intestinal caps run from crypts up villi to surface		Must be able to explain why **watershed zones are most susceptible** to injury.

2011.1.3

Question 5. **Hepatic Failure**	1 What are the **causes of acute liver failure?**	• **Drugs and toxins:** Paracetamol, halothane, rifampicin, mushrooms, CCL4 • **Infections**: hepatitis A, B and (rarely) C. Mechanism: direct toxic eg paracetamol,mushrooms Or toxicity and/or immune mediated eg Hepatitis virus	**3 causes -** at least 1 drug and 1 infection
	2. What are the **clinical features of liver failure?**	• Jaundice • Ascites • Hypoalbuminaemia • Hyperammonemia→ encephalopathy • Coagulopathy • Portal hypertension • Foetor hepaticus • Spider naevi • Palmar erythema • Hypogonadadism + gynaecomastia	At least **5 features**
	OPTIONAL (Good candidates) What **do you understand by hepato-renal syndrome?**	• Renal failure in pt with severe chronic liver disease with no obvious cause for the renal failure. Features include: • Na retention • Impaired free water excretion • Decreased renal perfusion and GFR	Any features

2010.2.2

Question 2.3 Ulcerative Colitis	**1.** What are the pathological features of Ulcerative Colitis?	**1.1.** One of two disorders that compromise **inflammatory bowel disease (IBD)** **1.2.** Severe ulcerating inflammatory disease **1.3.** Limited to **colon and rectum.** 1.3.1. Continuous distribution (Starts in colon and extends continuously – No skip lesions) 1.3.2. Extends only into mucosa and submucosa (ie not trans mural) 1.3.3. Pancolitis if entire colon affected, limited distal disease eg ulcerative proctitis 1.4. Superficial broad based ulcers 1.5. Pseudopolyps 1.6. Malignant potential 1.7. Toxic megacolon	**1.** Bold (+ 2)
	2. What extra-intestinal manifestations occur in ulcerative colitis?	2. Extra-intestinal Manifestations 2.1. Polyarthritis, 2.2. sacroiliitis, ankylosing spondylitis 2.3. Uveitis 2.4. Skin lesions 2.5. Pericholangitis 2.6. Primary sclerosing cholangitis	2. 4 for pass

2010.2.3

Question 3.3 Pseudo-membranous Colitis	1. Describe the pathogenesis of pseudomembranous colitis. 2. What are the clinical features of pseudomembranous colitis? 3. What is the pseudomembrane?	**1.1. Disruption of normal bowel flora (ab's – esp. 3rd gen ceph) allowing overgrowth of *C. difficile*** **1.2. *C. difficile* elaborates toxins that cause:** 1.2.1. Ribosylation of small GTPases 1.2.2. Disruption of epithelial cytoskeleton 1.2.3. Tight junction barrier loss 1.2.4. Cytokine release 1.2.5. Apoptosis 1.3. Denuded surface epithelium 1.4. Superficial lamina propria contains dense infiltrate of neutrophils & occasional fibrin thrombi in capillaries 1.5. Damaged crypts are distended by mucopurulent exudates that erupt "volcanically" 1.6. Coalesce to form the pseudomembrane 2. Causes fever, (leukocytosis), profuse watery diarrhoea, abdo pain **3. Pseudomembrane is an adherent layer of inflammatory cells and debris at sites of colonic mucosal injury**	1. Toxin + one otherbold + 1 other (1.3 to 1.7) 2. 2/3 3. bold

2010.2.3

Question 3.4 Cholecystitis	1. Describe the pathogenesis of acute calculous cholecystitis 2. How does acalculous cholecystitis differ from this? 3. Describe the clinical features of acute cholecystitis.	1. Acute Calculous (90% of all) 1.1. **Obstruction by stones,** stasis- activates hydrolases 1.2. Lecithins -> (mucosal Phospholipases) **-> lysolecithins** 1.3. Disrupts glycoprotein mucous **-> epithelium exposed to bile salts** 1.4. Prostaglandin release -> **inflammation,** mucosal and mural 1.5. **Dysmotility** & **raised intraluminal pressure** **1.6. Bacterial infection secondary to stasis** **2. Acalculous (10%) – rarer, in predisposed individuals, slower often masked** **2.1. Ischaemia, end arteries (cystic)** **2.2. Other promoting features – sludging micro-crystals, stasis, local inflammation, distension** 2.3. Sepsis with hypotension, immunosuppression, major trauma and burns, diabetes, infection, severe atherosclerosis (drugs/ABs- ? vasculitic). 3. Right upper quadrant or epigastric pain, 3.1. Mild fever, anorexia, tachycardia, sweating, nausea, and vomiting, tender RUQ (Murphy's)	1. 3/6 2. 3/6 3. 4/7

2010.2.4

Question 4.2 Acute Pancreatitis	1. What is the aetiology of acute pancreatitis? 2. What is the suggested pathogenesis of acute pancreatitis? 3. What are the laboratory findings of acute pancreatitis?	1.1 Metabolic – **Alcohol** 5% (UK), 65% (US), M:F = 6:1, drugs eg. azothioprine, hyperlipoproteinemia, hypercalcaemia, 1.2 Genetic – trypsinogen and trypsin genes 1.3 Mechanical – **Gallstones** 35-60%, M:F = 1:3, trauma, iatrogenic/intraoperative/ERCP 1.4 Vascular – shock, atherosclerosis, vasculitis 1.5 Infectious – mumps 2. 2.1 **Autodigestion of pancreatic substance by inappropriately activated pancreatic enzymes** 2.2 3 mechanisms 2.2.1 **Pancreatic duct obstruction** eg. by impacted gallstone => accumulation of lipase in interstitium => local fat necrosis => release of proinflammatory cytokines => leaky vessels + oedema => vascular insufficiency and ischaemic damage to acinar cells 2.2.2 **Primary acinar cell injury** eg. alcohol, mumps, trauma, drugs, organ insufficiency aftershock/ischaemia 2.2.3 **Defective intracellular transport of proenzymes within acinar cells –** digestive enzymes and lysosomal hydrolases intermingled causing release of activated enzymes. Human mechanism not clear. 3 **3.1 Marked elevation of serum amylase** in first 24 hours **3.2 Rising serum lipase** within 72-96 hours **3.3** Glycosuria – 10% cases **3.4** Hypocalcaemia – poor prognostic sign if persistent **3.5** Leukocytosis **3.6** Acute renal failure	1. **Bold** + 2 of the other causes from different groups **2.1** **Bold** to pass **2.2** **2 of 3** bold **3.** **Bold** + 2 others to pass

2010.2.4

Question 4.3 Abdominal Aortic Aneurysm	1. Describe the pathogenesis of an aneurysm 2. What are the clinical consequences of an AAA? 3. What is the risk of rupture of an AAA?	1. **Structure or function of the vascular wall connective tissue is compromised** 1.1. **Poor intrinsic quality of the vascular wall connective tissue eg Marfan syndrome**, Ehlers-Danlos 1.2. **Collagen degradation vs synthesis by local inflammation (** proteolytic enzymes**) eg atherosclerotic plaque, vasculitis,** 1.3. **Loss of vascular smooth muscle cells** or the inappropriate synthesis of noncollagenous or nonelastic ECM (cystic medial degeneration) 2. 2.1. **Rupture** into the peritoneal cavity or retroperitoneal tissues with massive, potentially fatal haemorrhage 2.2. **Obstruction of a branch vessel** resulting in ischemic injury, eg. iliac, renal, mesenteric, or vertebral arteries 2.3. **Embolism** from atheroma or mural thrombus 2.4. **Impingement** on an adjacent structure, e.g. ureter, vertebrae 2.5. Nothing (if < 4cm and no embolic complic's) 3. Related to size - 3.1 4 cm or less in diameter nil 3.2 between 4 and 5 cm 1% per year 3.3 between 5 and 6 cm 11% per year 3.4 greater than 6 cm in diameter 25% per year	1. 2/3 **bold**, 2 examples 2. 3 out of 5 3. Low < 5cm, much higher > 5cm

2010.1.2

Question 4: Portal Hypertension	1. Classify portal hypertension giving examples for each. Prompt for most important hepatic cause.	**Increased resistance to portal blood flow classified as:** - **Pre hepatic**: portal vein thrombosis or narrowing - ***Hepatic: cirrhosis**, granulomatous disease, massive fatty change, schisto, nodular regenerative hyperplasia - **Post hepatic**, R heart failure, constrictive pericarditis, hepatic vein occlusion	3 groups including hepatic. Cirrhosis and one other cause
	2. What are the major clinical consequences of portal hypertension due to cirrhosis?	- Ascites: with potential for infection - Porto systemic venous shunts: varices > upper GI bleed. Other sites e.g caput, h'roids, retroperit. - Splenomegaly: thrombocytopenia - Hepatic encephalopathy > coma	At least 3 consequences
	3. What mechanism are involved in the formation of ascites?	- **Starlings forces**: increased pressure, decreased albumin - Increased formation of hepatic lymph overwhelms thoracic duct drainage > percolation into peritoneum - Intestinal fluid leak: ^pressure in intestinal capillaries and osmotic effect of protein rich ascitic fluid - Renal retention of Na and H2O due to 2ndary ^aldosterone.	Starlings forces and one other

2009.2

Question 4: Ischemic bowel	1. What are the predisposing conditions for the development of ischemic bowel? Non-occlusive ischaemia • cardiac failure • shock • dehydration • vaso constrictive drugs Miscellaneous • radiation • volvulus • stricture • amyloid • diabetes • internal or external herniation	**Arterial thrombosis** • artherosclerosis. • vasculitis • aortic dissection • iatrogenic – angiography or aortic reconstruction • Hypercoagulable state. • Oral Contraceptive Pill **Arterial embolism.** • SBE • Angiography • Aortic atheroembolism **Venous Thombosis** • Hypercoagulation • OCP • AT III deficiency. • Intraperitoneal sepsis • Post-operative • Invasive neoplasms • cirrhosis • abdominal trauma	Simple list of 6 or more must contain examples of each of first 3 categories = straight pass headings + good examples of each = better pass.
	2. What are the clinical features of transmural infarction?	Pain Tenderness Nausea Vomiting Bloody diarrhoea, melanotic stool Shock Vascular collapse Absent bowel sounds Abdominal rigidity	Pain + any other 3 to pass

2009.1

Question 5: **Crohn disease**	**What are the pathological features of Crohn disease?**	**1. Transmural inflammation of bowel with skip lesions** **2. Noncaseating granulomata** **3. Fissures and fistulae**	2/3 Bold needed
	What are the extraintestinal manifestations of Crohn disease?	Migrating polyarthritis, sacroiliitis, ank spondylitis, erythema nodosa, finger clubbing, sclerosing cholangitis (uncommon), Uveitis, mild hepatic pericholangitis, renal disorders due to trapping of the ureters (uncommon). Systemic amyloidosis (rare) GI tract cancer (less common than UC). May occur prior to intestinal symptoms.	At least three systems Prompt: What other inflammatory conditions may be seen in Crohn disease?

2009.2

Question 4:	a. What are the causes of acute pancreatitis?
	
	b. Describe the pathogenesis of acute pancreatitis
		• Metabolic
		  ○ Includes alcohol
		• Mechanical
		  ○ gallstones
		  ○ trauma
		• Vascular
		• Infectious
		• Idiopathic (probably genetic basis)
		
		• Arises as a result of autodigestion by inappropriately activated pancreatic enzymes.
		• Trypsinogen is activated to trypsin. This in turn activates prophospholipase and proelastase, prekallikrein thus activating kinin system, and Hageman factor thus activated clotting and complement systems.
		
		Three potential pathways for initiation of pancreatic pathways:
		a. pancreatic duct obstruction
		b. primary acinar cell injury
		c. defective intracellular transport of proenzymes within acinar cells
			Identify alcohol and gallstones plus two others to pass.
			
			Autodigestion and key role of activation of trypsinogen as triggering factor to pass.

2008.1

Q4. Peptic ulcer disease.			
	By what mechanisms may Helicobacter pylori cause peptic ulcers?	*1.H. pylori* secretes **urease**, which generates free ammonia; and a **protease** which breaks down glycoproteins in the gastric mucosa. 2. *H.* pylori makes **phospholipases** →damage surface epithelial cells glycoprotein complexes. *3.H.* pylori enhances gastric secretion and impairs duodenal bicarbonate secretion. This enhances metaplasia. 4. Several H. pylori proteins are immunogenic→evokes **strong immune response** in the mucosa. Activated T and B cells are both seen in chronic gastritis caused by *H. pylori.* 5. Thrombotic occlusion of surface capillaries is promoted by a **bacterial platelet activating factor.** 6. Other antigens (including **lipopolysaccharide**) recruit inflammatory cells to the mucosa. 7. Damage to the mucosa is thought to permit **leakage of tissue nutrients** into the surface microenvironment, thereby sustaining the bacillus.	**Prompt:** What does *H. pylori* produce which can help cause ulceration? Pass criteria: need to say it involves immunogenic response.
	What complications may arise from peptic ulcer disease?	1. **Bleeding** (15-20%of patients), →25% of ulcer deaths 2. **Perforation** ~ 5% of patients →2/3 of ulcer deaths 3. **Obstruction** from oedema and or scarring ~ 2% of patients Mostly due to pyloric channel ulcers Rarely causes complete obstruction with intractable vomiting & incapacitating, crampy abdominal pain	Pass criteria: 2/3