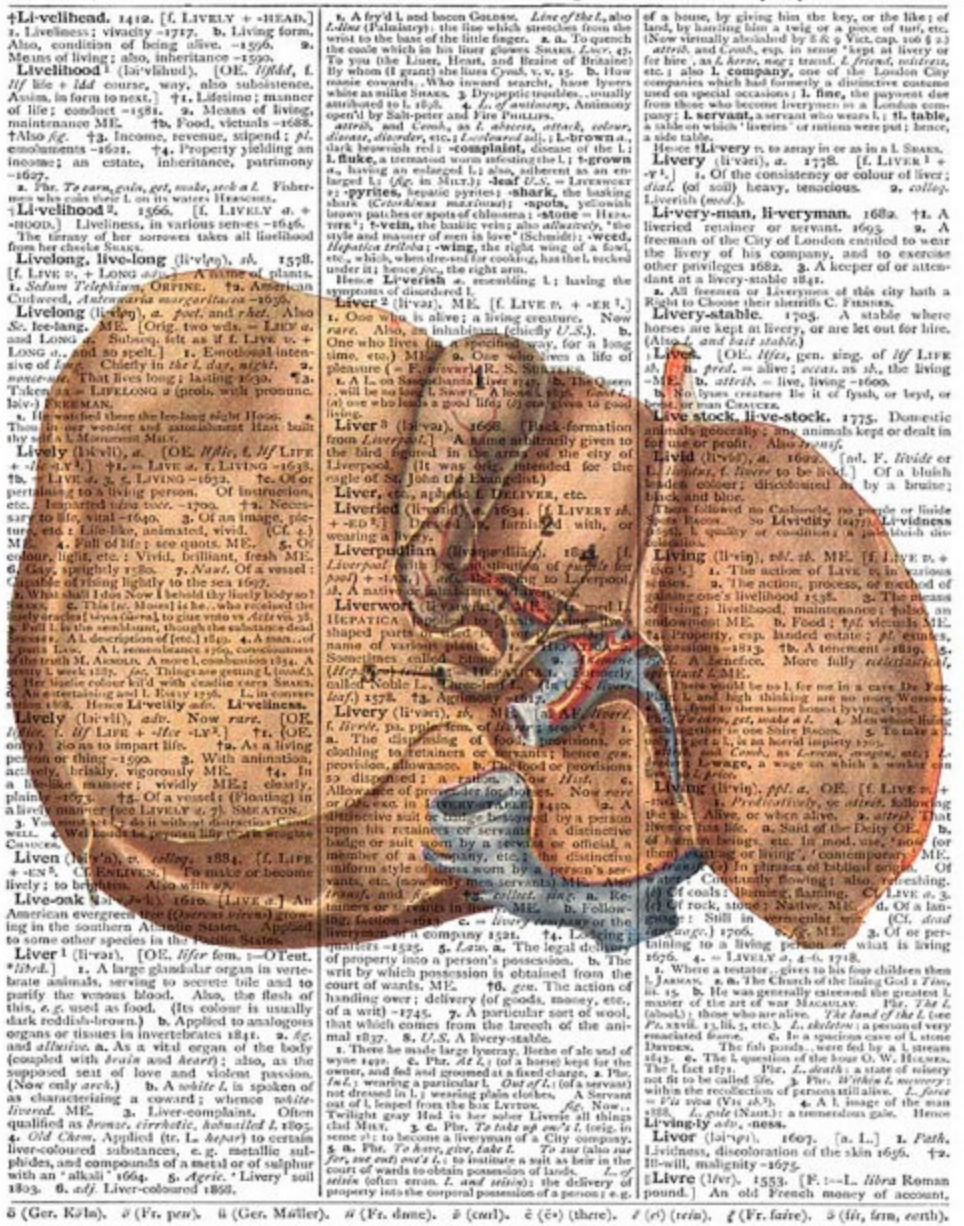
ACEM Primary Examination Vivas > Pathology > Liver and Pancreas Organises by edvivas.com	
Title	**Page**
Alcoholic Liver Disease 2017-1-C	3
Alcoholic Liver Disease 2014-2-D	4
Alcoholic Liver Disease 2011-2	5
Alcoholic Liver Disease 2007-1	6
Cholecystitis 2014-1-D	7
Cholecystitis 2010-2	8
Cholecystitis 2007-1	9
Cholelithiasis 2012-2	10
Cirrhosis 2015-2-D	11
Cirrhosis 2005-1	12
Hepatitis A 2015-2-D	13
Hepatitis A 2008-1	14
Hepatitis A 2005-2	15
Hepatitis B 2016-1-B	16
Hepatitis B 2013-1	17
Hepatitis B 2005-2	18
Hepatitis C 2012-2	19
Hepatitis C 2010-2	20
Hepatitis C 2009-1	21
Hepatitis C 2008-1	22
Hepatitis D 2010-1	23
Hepatitis D 2006-2	24
Hepatitis, General 2006-2	25
Jaundice 2017-2-D	26
Jaundice, Bilirubin 2012-1	27
Liver Failure 2011-1	28
Pancreatitis, Acute 2014-2-C	29
Pancreatitis, Acute 2010-2	30
Pancreatitis, Acute 2009-2	31
Pancreatitis, Acute 2006-2	32
Pancreatitis, Acute 2005-1	33



Pancreatitis, Chronic 2012-1	34
Portal Hypertension 2013-1	35
Portal Hypertension 2010-1	36
Portal Hypertension 2005-1	37

Alcoholic Liver Disease 2017-1-C

Stem: He has a history of heavy alcohol use. Moving on to Pathology.			
Question 3 Alcoholic Liver Disease **Subject:** Pathology LOA 1	a) Describe the pathological features of the liver in alcoholic liver disease? PROMPT: please describe the morphological features	**1. Hepatic steatosis-** fatty change, perivenular fibrosis **2. Hepatitis:** liver cell necrosis, inflammation, Mallory bodies, fatty change, fibrosis **3. Cirrhosis:** extensive fibrosis, hyperplastic nodules **4. Hepatocellular carcinoma**	Pass
	b) Which of these features are reversible?	**Steatosis and Hepatitis are reversible**. Cirrhosis irreversible.	Bold to pass
	c) 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

Alcoholic Liver Disease 2014-2-D

Stem: Moving on to Pathology. She has a history of chronic alcohol abuse.			
Question 3 Alcoholic Liver Disease (pp 857-860) **Subject:** Path LOA: 1	1. Describe the pathological effects on the liver long-term alcohol ingestion. PROMPT: please describe the morphological features	1. **Steatosis:** fatty change, perivenular fibrosis 2. **Hepatitis:** liver cell necrosis, inflammation, Mallory bodies, fatty change, fibrosis 3. **Cirrhosis:** extensive fibrosis, hyperplastic nodules 4. (Hepatocellular carcinoma)	Bold with 3 morphologic features of each to pass.
	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

Alcoholic Liver Disease 2011-2

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

Alcoholic Liver Disease 2007-1

5. Alcoholic liver disease	1. What are the pathological features of Alcoholic Liver Disease? Prompt "What are the forms of alcoholic liver disease?"	Chronic maladaptive state in which cells of the liver respond in an increasingly pathologic manner to alcohol resulting in: 1. **Hepatic steatosis (fatty liver) 2. Hepatitis; then** **3. Progressive fibrosis to Cirrhosis**; Marked derangement of vascular perfusion with secondary portal hypertension	All three to pass
	2. What changes occur at the cellular level in Alcoholic Hepatitis?	1. Hepatocyte swelling and necrosis: Single or scattered foci, Swelling due to accumulation of fat, water and protein 2. Mallory Bodies: Eosinophilic cytoplasmic inclusions in degenerating hepatocytes, Characteristic but not specific feature 3. Neutrophilic reaction: Accumulate around degenerating hepatocytes 4. Fibrosis: Prominent activation of sinusoidal stellate cells and portal tract fibroblasts	At least 3
	3. In end stage alcoholic liver disease, what are the potential causes of death?	1. Hepatic failure and coma 2. Massive GIT bleeding 3. Intercurrent infection (to which these pts are predisposed) 4. Hepatorenal syndrome (following a bout of hepatitis) 5. Hepatocellular carcinoma (3-6%)	At least 3

Cholecystitis 2014-1-D

Stem: Your intern consults you on a 60 yo lady he suspects has acute cholecystitis. This topic is **PATHOLOGY**.			
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

Cholecystitis 2010-2

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

Cholecystitis 2007-1

5. Cholecystitis	1. Please describe the pathogenesis of acute calculous cholecystitis Prompt "Describe the role of substances involved"	**Chemical irritation of obstructed GB** 1. Mucosal phopholipases hydroylyze luminal lecithins to toxic lysolecithins; 2. Protective glycoprotein mucus layer disrupted; 3. Bile salts – detergent action on mucosal epithelium; 4. PGs contrib. to inflam; 5. GB dysmotility develops; 6. distension and incr intraluminal press decr blood flow	1/1 2 others
	2. What conditions are associated with acute acalculous cholecystitis?	postop major surgery, severe trauma, severe burns, MOF, sepsis, prolonged IV hyperalimentation, postpartum	At least 4
	3. How do the clinical features of acute acalculous cholecystitis differ from calculous cholecystitis?	Acute: RUQ or epigastric pain & tenderness, mild fever, anorexia, tachycardia, sweating, nausea, vomiting *Calculous often more sudden, but can be mild & self-limiting ***Acalculous more insidious – may have no GB symptoms** - usually in pt with other illness –risk of complications higher eg perf/gangrene	Highlighted points
	4. What are the complications of acute and chronic cholecystitis?	*Bacterial infection: cholangitis, sepsis GB perf & abscess formation; GB rupture & peritonitis; Biliary enteric fistula; Aggravation pre-existing condition	1 + 2 others

Cholelithiasis 2012-2

Q4 Cholelithiasis LOA: 2	1. What are the risk factors for the development of cholesterol stones? 2. Describe the pathogenesis of cholesterol stone formation.	**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. 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**	3 of 5 bolded. Bolded and displays understanding of concept

Cirrhosis 2015-2-D

Stem: Moving onto Pathology			
Question 5 Cirrhosis **Subject:** Path LOA: 1	(a) What types of liver disease may result from chronic excessive alcohol consumption	Hepatocellular steatosis (fatty change) – reversible Alcoholic hepatitis – reversible Cirrhosis – non reversible Hepatocellular carcinoma – non reversible	1 reversible and 1 non-reversible
	(b) What are the morphological features of cirrhosis Prompt : what happens to liver cells when chronically exposed to toxins or injurious agent	Occurs **diffusely** throughout the liver, **parenchymal nodules (regenerating hepatocytes)** surrounded by dense **bands of fibrous scar, disorganised architecture,** variable degrees of **vascular / portosystemic shunting,** elements of progression and regression	3 out of 5 bold to pass
	(c) What are the possible sequelae of cirrhosis	**Portal Hypertension**, GIT Bleeding, Hepatic Failure, Coagulopathy, Hepatocellular Ca, Hepatorenal Syndrome, Hepatopulmonary Syndrome, Encephalopathy, Infection	Bold plus 3 others

Cirrhosis 2005-1

Cirrhosis	What are the causes of cirrhosis? (Alcohol essential + 2 others) Outline the pathogenesis of cirrhosis.	Alcoholic liver disease 60-70% ✱✱✱ Viral hepatitis 10%, biliary diseases 5-10%, primary haemochromatosis5%, Wilson disease, α_1-antitrypsin deficiency, idiopathic (cryptogenic) 10-15%, drug induced (α-methyldopa), cardiac disease, galactosaemia tyrinosis 1. fibrous septae ✱ 2. parenchymal nodules ✱ 3. disruption of the architecture of the ENTIRE liver✱ 4. Resulting in New vascular channels shunt blood around the parenchyma. Sinusoidal fenestrations are lost, hepatocyte secretion of proteins is impaired. Biliary channels are obliterated ✱ May also mention- Fibrosis and parenchymal injury are diffuse, nodularity is part of the diagnosis, vascular architecture is reorganised leading to a functional bypass of the hepatocytes. Fibrosis is the key feature of progressive liver damage and reversal is rare. Progressive fibrosis and reorganisation of the vascular microarchitecture of the liver leads to a fibrotic nodular liver where delivery of blood to hepatocytes is severely compromised, as is their ability to secrete substances into the plasma Type I and III collagen are laid down in the lobules by perisinusoidal stellate cells (usually fat storage cells). They are activated by cytokines from Kuppffer cells and other inflammatory cells. This leads to robust mitotic activity, a shift in phenotype to myofibroblast and increased capacity for synthesis of extracellular matrix. Activation occurs mainly areas of necrosis and inflammation. Myofibers increase vascular resistance in the parenchyma. Remaining hepatocytes proliferate as nodules within the fibrous septa.	

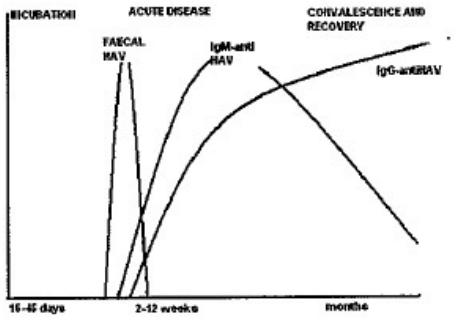
Hepatitis A 2015-2-D

Stem: Moving onto Pathology. You suspect Hepatitis A .			
Question 2 Hepatitis A **Subject:** Path LOA: 2	What is the causative agent of Hepatitis A?	**Hep A virus** – small unenveloped single stranded RNA picornavirus, icosahedral capsid	Bold to pass
	How is hepatitis A transmitted?	**Faecal oral spread**	Bold to pass
	How do the clinical outcomes of Hepatitis A differ from Hepatitis B? (Prompt- How are the long term outcomes different?)	Self-limiting illness no carrier state no chronic state no association with hepatocellular Ca rarely leads to fulminant disease low fatality rate of 0.1%	3/6 to pass
	How is Hepatitis A diagnosed serologically?	**Acutely IgM-anti- HAV**, followed by appearance / persistence of IgG-anti HAV	Bold to pass

Hepatitis A 2008-1

Q3. Hepatitis A	Describe the clinical course of Hepatitis A infection.	1) Oral faecal transmission. 2) Incubation period: 2-6 weeks. 3) No carrier state or chronic hep or cause hepatocellular Ca. 4) Rarely causes fulminant hepatitis, and so the fatality rate is about 0.1%.	Pass criteria: provide 3/4 Prompt: mode of transmission.
	How do the serological markers change with time in Hep A infection?	1) IgM anti HAV appears at the onset of symptoms. 2)Faecal shedding of the virus ends as IgM titre rises (2-12 weeks). 3) IgM Ab (months) 3) Replace by IgG anti HAV (years) . Encourage graph	Encourage graph.

TOPIC: Hepatitis A _____ **NUMBER: 3a** _____

OPENING QUESTION	Describe the features of the illness caused by Hepatitis A virus.	**COMMENTS**
POINTS REQUIRED	1 Faecal-oral transmission; consumption of raw/steamed shellfish which have concentrated the virus from sewage contaminated seawater.	4 of 7 to pass
	2 Incubation 2-6 weeks	
	3 in childhood symptoms mild or asymptomatic	
	4 adult life: febrile illness, nausea, lethargy, vomiting, possible dehydration.	
	5 rare (<0.1%) progression to fulminant hepatitis	
	6 acute disease more severe if superimposed on chronic hepatitis (B, C or alcoholic)	
	7 does not cause chronic hepatitis, nor carrier state	
PROMPTS	How is hepatitis A transmitted?	
SECOND QUESTION (if needed)	Describe the pattern of appearance of markers in Hepatitis A viral hepatitis. (with aid of a graph if you wish)	3 and 4 mandatory
POINTS REQUIRED	1 incubation: 15-45 days (2-6 weeks)	
	2 acute rise in Faecal HAV lasting 2-12 weeks (peaks within 1-2 weeks)	
	3 IgM anti-HAV begins to appear in blood as faecal HAV peaks, rises over 2-12 weeks	
	4 IgG-antiHAV begins to appear shortly after IgM and continues to rise more slowly over many months and remains elevated for many years.	
	 SEROLOGICAL MARKERS IN ACUTE HEP A	
PROMPTS		
THIRD QUESTION (if needed)	How does the community prevalence of Hepatitis A differ between developed and developing nations?	1 to pass Bonus question
POINTS REQUIRED	1 In developed nations ~ 50% at age 50 years have serological evidence of exposure	
	2 In developing countries – approaching 100% serological evidence by late teens	
PROMPTS		

Hepatitis B 2016-1-B

Stem: A 55 year-old man presents to the ED with haematemesis. Hepatitis B serology results from a previous admission are available.			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1: Hepatitis B Serology	*What is the most likely diagnosis, and why?* *Prompt: "Is this acute or chronic?"*	**HBsAg positive – indicates current infection.** Anti-HBc total positive – exposure to HBV. IgM anti-HBc negative – exposure not acute or recent. **Anti-HBs negative – no current immunity to HBV. Diagnosis: Chronic Hepatitis B.**	Bold to pass.
Stem: Moving onto Pathology.			
Question 2 Hepatitis B LOA: 1	*a. How may Hepatitis B lead to upper gastrointestinal bleeding?*	Cirrhosis and **portal hypertension** with development of **oesophageal varices.** **Coagulopathy** due to loss of synthetic function (unable to produce coag proteins)	2 out of 3 bold to pass
	b. What are the other complications of Hepatitis B-induced cirrhosis?	Jaundice; Hepatorenal syndrome; Hepatic encephalopathy; Ascites/pleural effusions Splenomegaly; Hypogonadism (testicular atrophy, amenorrhoea etc); Hepatocellular carcinoma	3 to pass
	c. In general, how may a patient acquire Hepatitis B?	Congenital (ie vertical; most common worldwide) Contaminated blood products – IVDU, transfusions (from many years ago), needlestick injury. Bodily fluids – eg sexual.	2 to pass
	d. What are the other possible outcomes of hepatitis B exposure?. *Prompt: 'Apart from progressive chronic hepatitis'*	Asymptomatic Acute hepatitis Non progressive chronic hepatitis Carrier state	2 to pass

Hepatitis B 2013-1

			At least 3 steps.
Question 2 Hep B LOA: 2	1. How can Hepatitis B infection be transmitted?	1. Vertical – perinatal during childbirth Horizontal – skin or mucosal breaches - intercourse - shared needles / syringes in IVDU - blood transfusion	**3/5**
	2. What are the potential outcomes following ACUTE Hepatitis B infection?	**2. Recovery** >90% Fulminant hepatitis necrosis <0.5% **Chronic Hepatitis** <5% - cirrhosis 12-20% +/- hepatocellular Ca - healthy **carrier state** - non progressive chronic hepatitis <2%	**Bold to pass**
	3. What are the serum markers of ACUTE infection with Hepatitis B? Prompt: What antigens and antibodies are present during acute hepatitis B?	**3. HBeAg, HBsAg** HBV-DNA, **Anti-HBc IgM** Anti-HBe, (not Anti-HBs)	**2/3 Bold**

TOPIC: Hepatitis B _____ **NUMBER:** 3b

OPENING QUESTION	How may Hepatitis B viral infection be transmitted?	**COMMENTS**
POINTS REQUIRED	1 Parenteral: contaminated blood product transfusion	Must have 1 plus 3 of rest
	2 sexual intercourse, esp. homosexual activity	40%
	3 IV drug use / dialysis	
	4 accidental needle stick injury with contaminated blood	
	5 vertical transmission (during delivery)	
	6 conjunctival splash of infected body fluid	
	7 one third of cases unknown transmission	
PROMPTS		
SECOND QUESTION (if needed)	Describe the patterns of disease progression following Hepatitis B viral infection.	3 to pass 40%
POINTS REQUIRED	1 subclinical disease (60-65%), with 100% recovery	
	2 acute hepatitis (20-25%) with 99% recovery, <1% fulminant hepatitis (potentially lethal)	
	3 'healthy carrier' state 5-10%	
	4 persistent infection 4%, with 67-90% recovery, 10-33% chronic hepatitis (20-50% develop cirrhosis→ 10% hepatocellular carcinoma or other lethal complication)	
PROMPTS		
THIRD QUESTION	What are the characteristic serological findings associated with the carrier state of HepB virus?	1 to pass 20%
	1. Presence of HBsAg in the serum for > 6 months	
	2. Chronic HepB viral ***replication*** has persistent HBsAg, as well as HBeAg, and HBV DNA, usually with anti-HBc and occasionally anti-HBs	
PROMPTS		

Hepatitis C 2012-2

Q4 Hepatitis C LOA: 2	1. What type of virus causes Hepatitis C? 2. What are the risk factors for acquiring Hepatitis C? 3. What is the natural course of Hepatitis C?	1. **Flaviviridae** family **RNA** virus 2. **IVDU** 54%; Multiple sex partners 36%; Recent surgery 16%; Needle stick 10%; Multiple contacts with HCV infected person10%; Health care workers 1.5% Unknown 32%; Children (perinatal) 6% (cf HBV 20%) 3. Incubation 2 – 26 weeks (mean 6 – 12); **Asymptomatic in 85%** HCV RNA detectable in 1 – 3 weeks Anti HCV Ab 50 – 70% while symptomatic Usually a mild disease **Persistent infection -> chronic hepatitis 80 – 85%** Cirrhosis 20 – 30% (5 – 20 years) Fulminant hepatitis rare	One of bold IVDU and 2 others Bolded

Hepatitis C 2010-2

Question 1.3 Hepatitis C Infection	1. What causes Hepatitis C infection? 2. Describe the clinical course of Hepatitis C infection 3. What are the risk factors for acquiring Hepatitis C? *Additional question for good candidates. After completion of 5 questions* 4. What features of the Hepatitis C virus make vaccine development difficult?	**1.1. Flaviviridae family RNA Virus** 2.1 Incubation period 2-26 wks (mean 6-12 wks) 2.2 Acute infection usually mild or asymptomatic (1-3 weeks) 2.3 Persistent and Chronic hepatitis with exacerbations in 80% 2.4 Cirrhosis in 20-30% 2.5 Fulminant hepatic failure rare **3.1 IVDU (54%)** **3.2 Multiple sex partners (36%)** 3.3 Needle stick (10%) (risk of HCV is 1.8% v 0.3% for HIV) 3.4 HCW (1.5%) 3.5 Blood Transfusion (in the 1980's), 3.6 Vertical, 3.7 Unknown (32%) 4.1 Highly stable core, **extremely variable envelope (E protein)** 4.2 RNA polymerase inherently unstable; frequent mutations, multiple *quasispecies* found in any one pt 4.3 Genomic and Antigenic variability 4.4 Actively inhibits interferon mediated cellular response at many levels	1. Bold 2. 3/5 3. 3/7 4. 2/4

Hepatitis C 2009-1

Question 3: **Hepatitis C**	**What are the routes of transmission of Hepatitis C virus?**	1. **Blood inoculation with IV drug use** 60%. 2. Unknown 3. Transmission via blood products pre1991 10% 4. Dialysis < 5% 5. Occupational exposure < 5% 6. Sexual transmission infrequent 7. Vertical transmission very low.	Need bold and 2 other?
	What are the potential outcomes of infection?	1. Acute Infection generally asymptomatic, rarely fulminant hepatitis 2. **85% progress to chronic persistent hepatitis** 3. 15% resolve completely 4. **20% of chronic infection progress to cirrhosis** 5. Some cirrhotics develop **Hepatocellular carcinoma**	Need bold

Hepatitis C 2008-1

Q3. Hepatitis C	Describe the potential outcomes of acute hepatitis C infections in adults.	1)Acute fulminant rare 2)15% resolve 3)85% chronic - >80% stable/20% cirrhosis (50% mortality) hepatocellular Ca	3 major points with most > chronic fulminant) and potential for cirrhosis/Ca
	How does the serology for Hepatitis C infection change in case of resolution?	1)Incubation period (2-26 weeks) 2)HCV-RNA (detectable for 1-3 weeks co-incident with transaminitis) 3)Anti HCV antibodies emerge. Only about 50% detectable during symptomatic acute infection. Remainder after 3-6 weeks. IgG/IgM. IgG persists.	All major points & 'window' when both virus & Ab may be – ve. Diagram encouraged.

Hepatitis D 2010-1

i) Describe how the Hepatitis D virus infects the human body	RNA virus **Must always be in conjunction with Hep B** 1) **acute infection –** indistinguishable from classical acute Hep B.) Exposure to serum containing both Hep B and D. HBV must establish first to provide HBsAg necessary for development of complete HDV viridons 2) **superinfection.** -chronic HBV carrier exposed to new inoculum of HDV. Disease develops 30-40 days later 3) helper-independent latent infection- in liver transplantation patients	Bold to pass
ii) Prompt: *Superinfection is one of the ways that Hepatitis D can infect the human host.* How does superinfection with HDV manifest?	1) severe acute hepatitis in previously unrecognised HBV carrier 2) exacerbation of preexisting mild chronic hepatitis B 3) 80-90% chronic progressive disease and cirrhosis	Need one
iii) How is Hepatitis D infection diagnosed?	**IgM anti-HDV** – most reliable marker of recent HDV exposure but late and short lived HBV an HDV coinfection – best with IgM against both HDAg and HBcAg *2 phases –* *acute phase – active HDV replication, suppression of HBV, high ALT levels* *chronic phase – HDV replication decreases, HBV replication increases. ALT levels fluctuate, progression to cirrhosis and hepatocellular cancer* *HDV RNA detectable in blood and liver just prior and in early days of acute symptomatic disease* *In chronic delta hepatitis, HBsAg is present and IgM and IgG anti-HDV antibodies persist for months*	At least one

Hepatitis D 2006-2

TOPIC: Hepatitis D _____ **NUMBER: 5** _____

OPENING QUESTION	***Describe the hepatitis D virus***	**COMMENTS**
POINTS REQUIRED	1 Unique RNA virus that is replication defective, causing infection only when it is encapusulated by HBsAg	
	2 A 35nm double shelled particle with a HBsAg outer coat and an internal HD Ag polypeptide with an associated single strand of circular RNA.	
PROMPTS	What is unusual about this virus	
SECOND QUESTION (if needed)	***How does HD cause hepatitis?***	Coinfection and superinfection to pass
POINTS REQUIRED	1 Needs Hep B S Ag	
	2 Coinfection with HBV	
	3 Superinfection of HBV carrier	
	4 Mild to fulminant (5%). More risk than Hep B alone. Can present as an acute severe hepatitis or can make a mild existing Hep B severe. Can become chronic progressive (mostly and the usual with a superinfection). . HD RNA found when symptoms start. Ig M anti HDV is reliable but occurs late and is short lived.	
PROMPTS		

COMMENTS

TOPIC: Sequelae of viral hepatitis _____ NUMBER: 5 _____

OPENING QUESTION	Outline the clinical syndromes which may develop following exposure to hepatitis viruses in an individual not immune to that virus.	COMMENTS
POINTS REQUIRED	**1. Acute asymptomatic infection with recovery** Incidental finding on serology testing 2. **Acute symptomatic infection with recovery.** Any hepatitis virus (although rarely Hep C) Four phases – Incubation, Symptomatic Pre-icteric, Symptomatic Icteric, Convalescence. Symptomatic – constitutional, Serum sickness in10% (esp Hep B), Liver symptoms Icteric (conjugated) Adults with Hep A, about 50% of Hep B, unusual in Hep C. Recovery in weeks to months with T cell response.	Permissible to miss number 1.
	2 Chronic Hepatitis Symptomatic, biochemical or serological continuing or relapsing disease for six months or more. Aetiology is the most important predictor of likelihood of progression to cirrhosis. Carrier states eg after vertical childhood Hep B (95%). Variable course, hepatocellular failure, cirrhosis, hepatoma.	
PROMPTS	**3 Fulminant Hepatitis** Over 2-3 weeks with encephalopathy. Usually A or B (can be reactivation). No stigmata of chronic liver disease, coagulopathy, cardiovascular, renal, ARDS, biochemical disease.	
SECOND QUESTION (if needed)	Describe the morphology of acute hepatitis	
POINTS REQUIRED	1 Acute enlarged red (green) liver, ballooning degeneration, cholestasis and plugs, isolated cells or clusters necrose, cytolysis / apoptosis, bridging necrosis, architecture disarray	Reasonable description incl. regeneration
	2 Regeneration hepatocyte proliferation, sinusoidal cell reactive changes (debris in Kupfer cells, influx of monocytes) Portal tract inflammation.	
PROMPTS	Organ and cellular	

Jaundice 2017-2-D

Stem: Moving onto Pathology.			
Question 4 Jaundice **Subject:** Pathology LOA: 1	a) What are the causes of jaundice? *Prompt: are there different types of jaundice? (hyperbilirubinaemia)*	**Predom unconjugated** 1. ↑ production of bili: haemolysis, resorption of haemorrhage, thalassaemia 2.↓hepatic intake: drug interference with membrane carrier systems, Gilbert sy 3. impaired conj: phys J of newborn, breast milk J, Crigler-Najjar sy, Gilbert sy, hepatitis (viral, drugs, auto-immune, cirrhosis) **Predom conjugated** 1.Impaired bile flow (cholangiopathy, biliary stricture, malignancy, choledocholithiasis) 2.Deficiency canalicular membrane transporters (Dubin Johnson sy, Rotor sy)	Bold plus 1 cause each
	b) Apart from jaundice, what are the clinical features of liver failure?	Icterus, pruritis, fetor hepaticus, palmar erythema, spider angiomata, hypogonadism, gynaecomastia, encephalopathy(asterixis), coagulopathy, hepatorenal syndrome, hepatopulmonary syndrome, portal HT(varices, ascites, caput medusae)	5 features.

Jaundice, Bilirubin 2012-1

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**

Liver Failure 2011-1

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

Pancreatitis, Acute 2014-2-C

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

Pancreatitis, Acute 2010-2

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

Pancreatitis, Acute 2009-2

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES

Question 4:	a. What are the causes of acute pancreatitis?	• Metabolic
		  ○ Includes alcohol
		• Mechanical
		  ○ gallstones
		  ○ trauma
		• Vascular
		• Infectious
		• Idiopathic (probably genetic basis)
			Identify alcohol and gallstones plus two others to pass.

	b. Describe the pathogenesis of acute pancreatitis	• 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	Autodigestion and key role of activation of trypsinogen as triggering factor to pass.

Pancreatitis, Acute 2006-2

TOPIC: Acute Pancreatitis _____ **NUMBER: 3** _____

OPENING QUESTION	***What are the aetiological factors in acute pancreatitis?***	
POINTS REQUIRED	1 Metabolic: alcoholism, hyperlipoproteinemia, hypercalcaemia, drugs, genetic	Require Alcohol, gallstones and 2 others
	2 Mechanical: trauma, gallstones, iatrogenic injury	
	3 Vascular: shock, atheroembolism, polyarteritis nodosa	
	4 Infectious: mumps, coxsackie, mycoplasma	
PROMPTS	Any other causes?	
SECOND QUESTION	***Describe the cellular morphology of acute pancreatitis.***	
POINTS REQUIRED	1 Range from trivial inflammation and oedema to extensive necrosis and haemorrhage. Microvascular leakage with oedema, lipolysis by enzymes, acute inflammatory reaction, proteolysis of parenchyma, destruction of blood vessels. Fatty acids released combine with calcium to precipitate as salts, acini, ducts and islets can all be involved. Red black haemorrhage mixed with yellow white fat necrosis.	Require Inflammatory changes and lipolysis
	2 Extra pancreatic fat necrosis eg in omentum.	
	3 Peritoneal fluid with fat globules	
PROMPTS		

Pancreatitis, Acute 2005-1

Acute pancreatitis	List the causes of acute pancreatitis. (Require EtOH, gallstones + 2 others)	Metabolic- alcohol*, hyperlipidaemia, hypercalcaemia, drugs, genetic Mechanical- trauma, gallstones*, iatrogenic (eg ERCP, perioperative) Vascular- shock, atheroembolism, polyarteritis nodosa Infectious- Mumps, Coxsackievirus, Mycoplasma Idiopathic	
	Outline the pathogenesis of acute pancreatitis. * required	Final common pathway of Autodigestion of pancreas by inappropriately activated pancreatic enzymes (esp trypsinogen to trypsin *) 1. Activation of enzymes - caused by pancreatic duct obstruction with accumulation of enzyme rich fluid. Interstitial oedema, ischaemia and acinar cell injury 2. Primary acinar cell injury esp in infectious and traumatic causes, drugs and alcohol- release of proenzymes and lysosomal hydrolases 3. Defective intracellular transport of proenzymes to lysosomal compartment within acinar cells esp in metabolic, alcohol and duct obstruction Alcohol- causative mechanism not established- increases in secretion, spasm of sphincter of Oddi, direct toxic effect on acinar cells, or causes protein rich fluid that plugs and obstructs small ducts	

Pancreatitis, Chronic 2012-1

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 Pseudocyst	**Any 3**

Portal Hypertension 2013-1

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

Portal Hypertension 2010-1

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

Portal Hypertension 2005-1

Portal HPT	What causes portal hypertension? (At least one of each required)	Pre-hepatic: obstructive thrombosis, portal vein narrowing, splenomegaly Intrahepatic: cirrhosis (most common), schisto, fatty change, sarcoid/TB, nod hyperplasia Post-hepatic: RVF, constrictive pericarditis, hepatic vein outflow obstruction	
	What are the clinical consequences of portal hypertension? (Shunts + 1 other required)	Ascites, portosystemic venous shunts (eg varices, haemorrhoids, spiders), congestive splenomegaly, hepatic encephalopathy	