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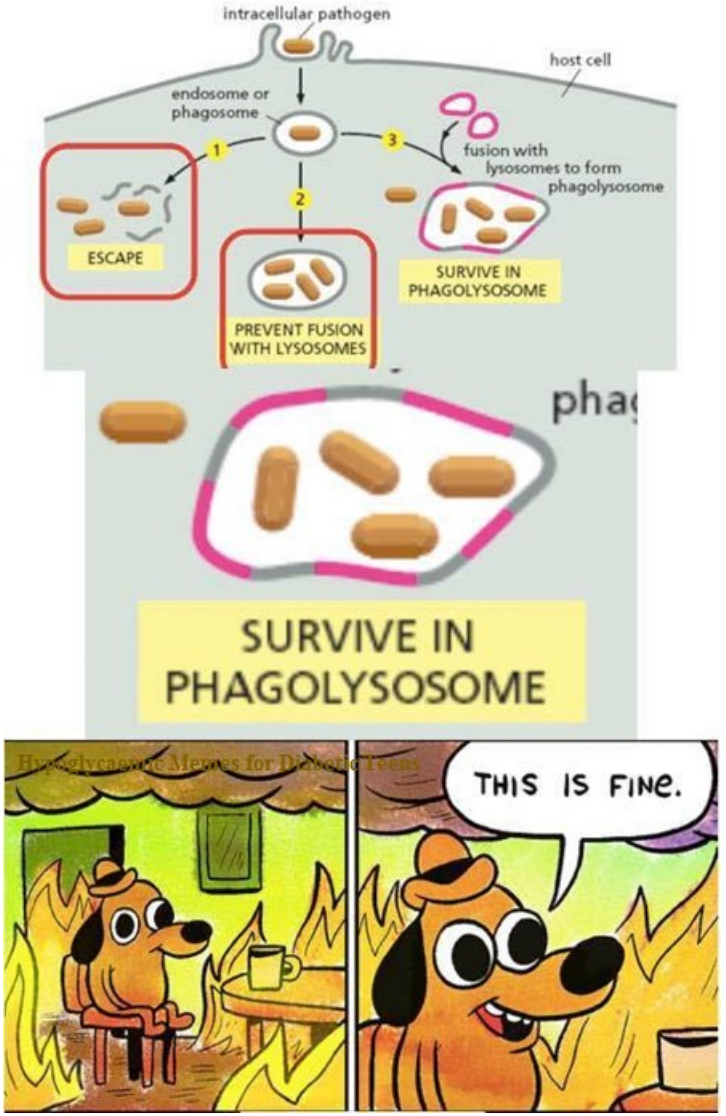

Apoptosis 2010-1

	precludes recovery of ATP / energy supplies for the cell	
a) What is apoptosis ?	**Programmed cell death** / " suicide programme"	
Prompt *Describe features and purpose of apoptosis*	Remove degraded- un needed cells, Stop excess growth, Tightly controlled Activates degradation enzymes, Intact membrane packaging(es), Phagocytosis encouraged = end point **Non inflammatory**,	Physiological or pathological initiators/ Capsases/ Intrinsic/ extrinsic paths Mitochondrial v death receptor
b) List some important stimuli for apoptosis ?	a) loss of growth/ stimulating hormones (e.g. GH, nerve growth, loss of sex hormones) b) excessive DNA damage (via p53 build up) c) unfolded protein build up d) developmental atrophy, (embryogenesis) e) proliferative tissues- homeostasis – non useful cells/ excess to function- loss of contact inhibition f) loss of useful cells after finished purpose (e.g. neutrophils/ lymph post infl) g) cells with harmful characteristics (e.g. autoimmune antigens /xs mutations) h) infections (viral leading to cell death) i) parenchymal damage after duct obstruction	3 concepts

Apoptosis 2008-1

Q1. Apoptosis	What is apoptosis?	1.Pathway of cell death. 2.Induced by tightly regulated intracellular programme 3. Cells that are destined to die activate enzymes that degrade the cells' own nuclear DNA and nuclear/cytoplasmic proteins. 4. The cell's plasma membrane remains intact. 5. Apoptotic cell becomes target for phagocytosis. 6. Dead cell rapidly cleared before contents leak out so this does not elicit an inflammatory reaction in the host. 7. Cell shrinks	Prompt: What are the features at a cellular level? Pass criteria: 3/6 to pass Must get no.1 and cell contents don't leak out.
	Describe the physiologic situations where apoptosis occurs.	1.Programmed destruction of cells during embryogenesis. 2. Hormone dependent involution in adult such as endometrial breakdown. 3. Cell deletion in proliferating cell populations e.g. intestinal crypt cells. 4. Death of host cells that have served their purpose e.g. neutrophils in acute inflammation. 5. Elimination of potentially harmful self reactive lymphocytes. 6. Cell death induced by cytotoxic T cells.	Pass criteria: 2/6 required

Apoptosis 2006-2

TOPIC: Apoptosis _____ **NUMBER:** _____

OPENING QUESTION	What is apoptosis?	**COMMENTS**
POINTS REQUIRED	1 Programmed death	Must give concept
PROMPTS		
SECOND QUESTION (if needed)	Under what conditions may apoptosis occur.	Must be able to give at least one example of each group to pass
POINTS REQUIRED	1 Physiological: embryogenesis, hormone-dependent involution in adult, cell deletion, elimination of potentially harmful self-reactive lymphocytes, cell death induced by cytotoxic T cells	
	2 Pathological: cell death secondary to radiation injury or cytotoxins, viral hepatitis, pathologic atrophy after duct obstruction in pancreas, parotid or kidney, cell death in tumours	
PROMPTS	Give an example of physiological (or pathological) apoptosis	
THIRD QUESTION (if needed)	What happens at a cellular level?	
POINTS REQUIRED	1 Cell shrinkage	
	2 Chromatin condensation	
	3 Formation of cytoplasmic blabs and apoptotic bodies	
	4 Phagocytosis of apoptotic cells or cell bodies, usually by macrophages	
PROMPTS		

Apoptosis 2005-2

TOPIC: Apoptosis_____ **NUMBER: 1c**_____

OPENING QUESTION	What is apoptosis	**COMMENTS**
POINTS REQUIRED	1 Programmed cell death	1 to pass
	4	
	5	
	6	
	7	
PROMPTS		
SECOND QUESTION (if needed)	Describe the mechanisms that result in apoptosis.	1 of 1 or 2 plus 3 or 4
POINTS REQUIRED	1 Extrinsic (Death Receptor-initiated) pathway TNF receptor family (TNFR1 & Fas) Activation of caspase leading to execution phase	
	2 Intrinsic (Mitochondrial pathway) Increased mitochondrial permeability releases pro-apoptotic molecules into cytoplasm which lead to caspase activation	
	3 Execution phase Proteolytic cascade mediated by activated caspases. Cleave cytoskeletal and nuclear matrix proteins Cleavage of DNA in nuclei	
	4 Removal of dead cells by phagocytosis prior to inflammation	
PROMPTS		

Apoptosis 2004-2

TOPIC: Features and mechanisms of apoptosis _____ **NUMBER: 1** _____

OPENING QUESTION	What is apoptosis?	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	Programmed cell death, occurs in single cells or clusters of cells, not associated with tissue inflammation		
SECOND QUESTION (if needed)	Give some examples of apoptosis.	Need 2 to pass	
POINTS REQUIRED	Endometrial cells during menstruation, GIT epithelium, Killer T Cell action, embryo development, tumours, neutrophils in acute inflammation, atrophy following duct obstruction, viral hepatitis, low does noxious stimuli : heat, radiation, hypoxia, cytotoxics, aging		

Atrophy 2017-2-C

Stem: Moving on to Pathology. His nerve injury does not resolve and many months later he has marked wasting of his hypothenar muscles.			
Question 5 Atrophy **Subject**: Pathology Robbins 9th Ed, pages 36-37 LOA: 1	(a) What is atrophy?	**Decrease in the size of an organ or tissue resulting from a decrease in cell** size and number. Can be physiological or pathological.	Bold to pass
	(b) What are the causes of atrophy?	**Decreased workload** (eg.# immobilized in plaster) **Denervation** **Diminished blood supply** (eg.due to arterial occlusion) **Inadequate nutrition** (eg. protein-calorie deficit - marasmus-> use of adipose stores + muscle for energy) **Loss of endocrine stimulation** (eg. endometrial atrophy after menopause). **Ageing** **Pressure**	4 of 7 bold
	(c)What are the mechanisms of atrophy?	**Decreased protein synthesis** **Increased protein degradation**. May be accompanied by increased autophagy (self-eating) - where a starved cell eats its own components in an attempt to find nutrients and survive.	One bold to pass

Atrophy 2011-1

Question 1.	**1. What is atrophy?**	**Shrinkage in the size of an organ or tissue** due to decrease in cell size and number.	Must know
Atrophy	**2. What are the causes of atrophy?**	• Disuse • Denervation • Diminished blood supply • Inadequate nutrition • Loss of endocrine stimulation • Pressure	At least 4
	3. Give some examples of atrophy	• Fracture disuse • damage to nerves causing muscle atrophy • breast/reproductive organs from oestrogen lack	At least 2

Atrophy 2007-1

1. Atrophy	1. What is atrophy?	**Shrinkage in the size of the cell** by loss of cell substance	
	2. What are the pathological types of atrophy? Prompt: What are the causes?	Disuse, Denervation, Diminished blood supply, Inadequate nutrition, Loss of endocrine stimulation, Ageing, Pressure	At least 4
	3. Give some examples of atrophy	Fracture disuse, damage to nerves causing muscle atrophy, breast/reproductive organs from oestrogen lack.	At least 2

Calcification 2008-1

Q 2. Pathological calcification	Please describe the 2 different forms of pathological calcification and give an example of each.	1. Dystrophic calcification – normal serum calcium - in necrotic or dying tissue 2. Metastatic calcification - normal tissue - abnormal (raised) calcium	Prompt: "What is meant by dystrophic calcification / metastatic calcification" ?
	Prompt: Please give an example(s) of dystrophic calcification, and metastatic calcification.	1. Dystrophic calcification – atherosclerosis; calcific aortic stenosis; tuberculous node 2. Metastatic calcification – nephrocalcinosis; pulmonary calcinosis; gastric mucosal	Prompt: "What type of abnormal calcification is nephrocalcinosis" ?
	"Describe the different principal pathological causes of hypercalcaemia, with some clinical examples.	1. Increased PTH secretion + bone resorption - hyperparathyroidism 2. Destruction of bone tissue – skeletal metastases, myeloma, Paget's 3. Vit-D related disorders – sarcoidosis, hypervitaminosis D 4. Renal failure – secondary hyperparathyroidism + phosphate retention	Prompt: "Hyperparathryoidism from increased PTH secretion is one example. Can you give another" ? Pass criteria: 2/4

Calcification, Metastatic 2004-2

TOPIC: Metastatic Calcification _____ **NUMBER: 5** _____

OPENING QUESTION	What is the difference between dystrophic and metastatic calcification?	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	Dystrophic calcification has normal calcium level in damaged tissue while metastatic calcification has high calcium level in damaged tissue.		
SECOND QUESTION	What are the causes of metastatic calcification?		
POINTS REQUIRED	hyperparathyroidism, vitamin D intoxication, systemic sarcoidosis, milk alkali syndrome, hyperthyroidism, idiopathic hypercalcaemia. Renal failure, destructive bone disease	Need 3 to pass	
THIRD QUESTION (if needed)	What tissues are most commonly affected by metastatic calcification?	Need 1 to pass	
POINTS REQUIRED	Gastric mucosa		
	Kidneys		
	Lungs		
	Systemic arteries		
	Pulmonary veins		

Cell Injury 2012-2

Q2 Mechanisms of Cellular Injury LOA **1**	1.? What happens inside cells when they are injured? *Prompt: mechanisms of cell injury* 2. What is a free radical? 3. What are the pathologic effects of free radicals? *Prompt: At a cellular level.*	1. ATP depletion, mitochondrial damage, calcium influx, accumulation of free radicals or ROS, membrane damage, DNA/protein damage 2. Chemical species that have a **single unpaired electron** in outer orbit eg reactive oxygen species: superoxide, hydrogen peroxide, hydroxyl, ONOO- peroxynitrite 3. Overall can cause **necrosis** or apoptosis or can stimulate production of degrading enzymes Directly can cause: **Lipid peroxidation** (plasma or organelle membrane damage) **Oxidation of proteins** (affect protein structure eg enzymes) **DNA lesions** (breaks in DNA or cross-linkages)	3/6 Principal & one example to pass Necrosis & 1/3 bolded effects

Cell Injury 2004-2

TOPIC: Biochemical hallmarks of cell injury _____ **NUMBER: 1**

OPENING QUESTION	Describe the biochemical features of cell injury.	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	1. Depletion of ATP – sodium pump reduction – Na into cells, K+ out. Inc. catabolites in cells – inc. osmotic load – swelling. Anaerobic metabolism. – lactic acid, initially ↓ pH then normalisation or ↑ pH. 2. Free oxygen radical formation 3. ↑ intracellular Ca 4. Defects of membrane permeability- leakage of intracellular substances - myoglobin, CK, troponin, other enzymes 5. Mitochondrial damage Decreased protein synthesis ↑ Lipid breakdown products ↓ Intracellular glycine	Need at least 3 points to pass	

Hyperplasia 2017-2-A

Stem: Moving on to Pathology. His urinary retention is caused by benign prostatic hyperplasia.			
Question 2 Hyperplasia **Subject**: Pathology Robbins 9th edition pages 35-36 LOA: 1	a) What is hyperplasia? b) What are the different types of hyperplasia and give examples (Prompt: Types other than BPH) c) Apart from urinary retention, what are the clinical features of BPH?	a) ↑**number of cells** in organ/tissue → usually ↑mass b) **Physiologic** a. Hormonal: female breast at puberty & preg b. Compensatory: post-partial hepatectomy, skeletal muscle with increased workload **Pathologic** a. Excess hormones: BPH, DUB b. Viral infection – papillomavirus c) Frequency, nocturia, difficulty in starting and stopping stream, dribbling, dysuria, ↑risk of infections	Bold Bold plus one example in each category to pass Any 2.

Hyperplasia 2016-1-C

Stem: Moving onto Pathology. On examination her thyroid gland is enlarged.			
Question 5 Cellular injury – adaptation and hyperplasia **Subject:** Path LOA: 1	1. Define hyperplasia	Hyperplasia is **an increase in the number of cells** in an organ or tissue resulting in increased mass.	Bold to pass
	2. What are the different types of hyperplasia and give some examples for each of them	**Physiologic** hyperplasia a. hormonal-female breast at puberty and during pregnancy b. compensatory-post partial hepatectomy **Pathologic** hyperplasia a. excess hormones-Benign prostatic hypertrophy, dysfunctional uterine bleeding b. response to viral infection – papillomavirus	Bold and one example of each to pass
	3. Name some clinical manifestations of diffuse toxic hyperplasia of the thyroid (Prompt: Graves' disease)	Cardiac-tachycardia, palpitations, heart failure Eye-staring, lid lag, proptosis GI-malabsorption, diarrhoea Neuro-tremor, anxiety, poor concentration Other	4 to pass

Hyperplasia 2012-2

TOPIC	QUESTIONS	KNOWLEDGE (**essential in bold**)	NOTES
Q1 Hyperplasia LOA: 1	1. What is hyperplasia? 2. What are the causes of hyperplasia? 3. Give some examples of hyperplasia *Prompt: can you give me a physiological/pathological example?*	Hyperplasia is an **increase in the number of cells** in an organ/tissue, usually get increased mass of organ/tissue a. Hormonal effects – reversible with withdrawal of hormonal stimulation b. Tissue damage or resection - compensatory hyperplasia c. Growth factors - pathological hyperplasia d. Increased workload (muscle) - as for hypertrophy Physiological: female breast at puberty and during pregnancy, partial hepatectomy, Pathological: endometrium – hyperplasia, dysfunctional uterine bleeding; BPH; Papilloma virus	Bold to pass 2/4 required to pass 1 physiological and 1 pathological cause to pass

Hyperplasia 2007-1

1. Hyperplasia	1. What is hyperplasia?	**Increase in number of cells in organ/tissue**, occurs if cellular population can synth DNA so get mitotic division	
	2. What are the cellular mechanisms of physiological hyperplasia?	**1. Increased local production of growth factors,** **2.incr growth factor receptors on cells, or** **3. activation of intracell signalling pathway.** Leads to incr transcript factors & cellular prolif. Eg Hormonal: hormones act as growth factors and trigger gene transcription (breast, uterus in pregnancy) Compensatory: unclear, ?stem cells (liver)	At least 2
	3. Please give examples of pathological hyperplasia.	Endometrial hyperplasia & abnormal menstrual bleeding Benign prostatic hyperplasia, Skin warts	At least 1

Hypertrophy 2012-2

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Q1 Hypertrophy LOA: 1	1. What is hypertrophy? 2. What are the types of hypertrophy? 3. Describe examples of each type hypertrophy? *Prompt: Can you give examples of physiologic and pathologic hypertrophy?*	**Increased size of a tissue due to increased cell size);** Due to synthesis of structural components. May be **physiological or pathological** depending upon increased functional demand or specific hormonal stimulation. Cell hypertrophy can occur in dividing or non-dividing cells Physiological: skeletal muscles with exercise, uterus in pregnancy (hormonal), breasts in lactation. Pathological: prostate in BPH, heart in chronic hypertension.	Bold One example of each

Hypertrophy 2010-2

Question 2.1 Hypertrophy	1. What is tissue hypertrophy? 2. What are examples of hypertrophy (Prompt: How is it classified??) 3. How is hyperplasia different form hypertrophy?	1.1. **Increase in cellular size not number** leading to overall organ/tissue size increase 1.2. Cell size increased by **more structural components and** increased synthesis of cellular proteins 1.3. Triggered by increased functional demand or stimulation by hormones or growth factors 1.4. Can be selective hypertrophy of specific sub-organelles 2. Examples 2.1. **Physiological** skeletal muscle enhancement through training or uterus under influence of hormones such as oestrogen 2.2. **Pathological** such as cardiomegaly in hypertension and CCF (has an **upper limit** after which regression occurs -> cell injury -> apoptosis/necrosis) 3. Hyperplasia involves an **increase in the number** of cells.	1. Bold 2. Bold (+ 1 example of each) 3. Bold

Hypertrophy 2010-1

(a) What is hypertrophy?	**Increase in the size of cells** – due to the **synthesis of more structural components** – resulting in an increase in the size of the organ; caused by increased functional demand or by hormonal stimulation. – pathological or physiological.	a) Highlighted
(b) Give examples of physiological and pathological hypertrophy	Physiological – Skeletal muscle (gym etc – workload); Uterus in pregnancy (hormonal) Pathological – Myocardium (due to hypertension, aortic stenosis – workload); BPH	b) One example of each

Hypertrophy 2007-1

1. Hypertrophy	1. What is hypertrophy?	**Increase in the size of the cells, due to synthesis of more structural components**, resulting in an increase in the size of the organ (no new cells, just larger cells) Physiological or pathological in response to increased functional demand or specific hormonal stimulation Can occur in both dividing and non-dividing cells	Highlighted point
	2. What are the types of hypertrophy?	Physiological Pathological	Both
	3. Give examples of physiologic and pathologic hypertrophy.	1. Physiologic: Enlarged skeletal muscles in labourers (workload), Enlarged uterus in pregnancy (hormonal), Enlarged breasts in lactation 2. Pathological: Enlarged prostate in BPH, Enlarged heart in valve disease or chronic hypertension (workload)	1 example of each

Hypertrophy and Hyperplasia 2008-1

	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Q1. Hypertrophy vs hyperplasia.	What are the differences between hyperplasia and hypertrophy?	1 Hyperplasia - increase in number of cells in organ/tissue - usually resulting in increase in volume - occurs if cellular population capable of synthesising DNA thus permitting mitotic division. 2 Hypertrophy - increase in size of cells - causes increase in size of organs. 3 Hypertrophy and hyperplasia often co-exist.	Prompt: What are the differences at a cellular level Pass criteria: 2/3 to pass
	Describe the different types of hyperplasia and give an example of each.	1 Physiologic: Hormonal, Compensatory. 2 Pathological - hormonal stimulation excessive e.g. oestrogen and effect on uterus, benign prostatic hypertrophy caused by androgens - growth factors e.g. proliferation of connective tissue cells and blood vessels in aiding wound repair.	Pass criteria: Need basic classification to pass

Ischaemic Cell Injury 2016-1-D

Stem: Moving onto Pathology. He is found to have absent right pedal pulses.			
Question 3 Ischaemic injury **Subject:** Path LOA: 1	Describe the sequence of events that occur in **reversible ischaemic** cellular injury PROMPT: What occurs within the cell after delivery of oxygen and substrate is compromised?	↓ox phos & **↓ATP prodⁿ** → **Failure Na pump**: K efflux, Na influx, Cell swelling →**Ca influx:** Further ↓ATP prodⁿ; enzymes activated→ ↓glycogen & ↓protein synthesis → **Cytoskeleton Δs**: loss of microvilli, "bleb" formation, "myelin figures" from degenerating cell membranes → Mitochondrial swelling	3 of 4 **bold**
	List the morphological changes of **irreversible** cellular injury	1.Severe mitochondrial swelling 2.Extensive damage to plasma membrane (including "myelin figures") 3.Lysosomal swelling 4.Necrosis or apoptosis	Any 2
	Describe reperfusion injury (time permitting)	**Increase injury to ischaemic cells with restoration of perfusion**. Due to generation of **reactive O_2** and nitrogen species (NO), Ca++ re-entering cell, **activation inflamm and complement cascades**	At least one concept

Ischaemic Cell Injury 2011-1

Topic	Question	Essential Knowledge	Pass criteria / Comments
Question 1. **Ischaemic cell injury**	1.What are the **stages of ischaemic cell injury?**	• **Initial Reversible** • **Irreversible (prolonged ischaemia injury and necrosis)**	2/2
	2. Describe the **sequence of events that occurs in reversible ischaemic cellular injury**. PROMPTS What occurs in the cell? What happens to pH?	• Due to loss of oxidative phosphorylation → **decreased ATP →failure of sodium pump** → loss of K+; influx of Na+ and H2O → iso-osmotic cell swelling. • **Increase in Ca++** initially release from intracellular stores then influx of Ca++ across plasma membrane → failure of ATP generation, activation of enzymes, induction of apoptosis → membrane and nuclear damage • **Decreased cellular pH due to increased lactate (increased anaerobic metabolism)** • Loss of glycogen, decreased protein synthesis • Loss of microvilli, formation of cell surface blebs, myelin figures, mitochondria + ER swelling, ribosome detachment clumping of nuclear chromatin fatty change	Bold (3 items)
	3. Describe **the morphological changes of irreversible ischaemic** injury	• Severe swelling of mitochondria • Extensive damage to plasma membrane • Swelling of lysosomes • Cell death by necrosis/apoptosis	2/4

Ischaemic Cell Injury 2009-1

Question 1: **Cellular changes following ischaemia**	**Describe the types of damage that occur inside a cell after severe ischaemia**	1. **ATP depletion** leading to NaK pump failure, anaerobic metabolism, Ca pump failure, reduced protein synthesis and protein misfolding 2. **Membrane damage** - mitochondria, lysosomes and plasma membrane 3. **Increased intracellular Ca^{++}** / loss **Ca^{++} homeostasis** 4. **Accumulation of reactive O_2 species** **5. Defects in membrane permeability**	Need 3/5 bold Prompt: What would happen to energy production in the cell?

Metaplasia 2012-2

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Q1 Metaplasia LOA 1	1. What is metaplasia? 2. Describe some examples 3. What are the possible outcomes of metaplasia? 4.	1. Replacement of one normal cell type with another normal cell type; can be adaptive or pathological. 2. Columnar to squamous (respiratory-chronic irritation eg smoking; excretory ducts due to stones eg salivary, bile). Squamous to columnar (Barrett oesophagus). Connective tissue (myositis ossificans). 3. Malignant transformation, reversibility/resolution, ongoing	Correct definition and 2 examples to pass 2 to pass

Metaplasia 2010-1

QUESTION	ESSENTIAL KNOWLEDGE	NOTES
a) What is metaplasia and give some examples?	• **Reversible change** (Among differentiated cells such as epithelial or mesenchymal) • Where **one cell type is replaced by another** by reprogramming of precursor stem cells or undifferentiated mesenchymal cells Examples: • Respiratory tract: trachea and bronchi in respiratory tract – due to chronic irritation such as smoking; ciliated columnar to stratified squamous • GIT: oesophagus – due to chronic gastric acid reflux; squamous to intestinal-like columnar "Barrett's oesophagus"	a) Highlighted and 1/2 examples
(b) How may metaplasia progress? (Prompt: What is the potential undesirable outcome of metaplasia?)	Cells lose normal protective function Persistence of influence that initiated the metaplasia **initiates malignant transformation** (e.g. squamous cell lung ca; adenocarcinoma oesophagus) Reverses	(b) Highlighted

Metaplasia 2007-2

TOPIC: Metaplasia _____ **NUMBER: Q1** _____

OPENING QUESTION	**What is metaplasia?**	**COMMENTS**
POINTS REQUIRED	1 A reversible change in which one adult cell type (epithelial or mesenchymal) is replaced by another. Prompt: Describe metaplasia	
	2 An adaptive change brought on by chronic stress such as chemical or physical irritation, so that cells change to other cell types better able to withstand the adverse environment. Prompt: Why does it occur ?	
SECOND QUESTION	**Can you give examples ?**	
POINTS REQUIRED	1 Most common is change of columnar to squamous epithelium eg • In trachea and bronchi in response to chronic smoking • In salivary, pancreatic & biliary ducts by stones, when secretory columnar epithelium replaced by non-functioning squamous epithelium. • Vitamin A deficiency may cause squamous metaplasia in respiratory, corneal, renal epit. • Vitamin A excess may stimulate osteoclast formation > bone resorption, osteoporosis & fractures.	At least 2 to pass
	2 Metaplasia of squamous to columnar can also occur • In Barrett's oesophagus due to acid irritation, predisposing to adenocarcinoma	
	3 Metaplasia can occur from one connective tissue / mesenchymal tissue to another eg muscle to bone or cartilage eg • In myositis ossificans.	
THIRD QUESTION	**What is the mechanism causing metaplasia ?**	**If needed**
POINTS REQUIRED	• A reprogramming of epithelial stem cells or undifferentiated mesenchymal cells Involving signals from cytokines, growth factors, extracellular matrix components, genes, DNA methylation.	Additional information

Metaplasia 2004-2

OPENING QUESTION	What is metaplasia?	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	Reversible change where one adult cell type is replaced by another adult cell type	Must say this point to pass	
	May be adaptive- sensitive cell type replaced by resistant cell type		
	The influences that predispose to metaplasia can induce cancerous transformation		
SECOND QUESTION (if needed)	Please give examples of metaplasia.	Give 1 example to pass	
POINTS REQUIRED	Columnar to squamous epithelium in the resp tract secondary to chronic irritation		
	Stones in pancreas, salivary or bile ducts ⇒ squamous epithelium from columnar		
	Barrett esophagitis squamous ⇒ columnar epithelium		
	Connective tissue metaplasia. Cartilage, bone or adipose tissue forms in other tissues		
THIRD QUESTION (if needed)	What causes metaplasia?	Optional	
POINTS REQUIRED	Reprogramming of stem cells		
	May be due to cytokines, growth factors, and extracellular matrix components.		
	Tissue specific and differentiation genes influenced to lead to cellular differentiation		
	Known factors include, chronic irritation, Vit A deficiency and excess, cytostatic drugs		

Necrosis 2011-1

Question 1. **Cell Death / Necrosis**	**1. Describe the cellular changes in necrosis** PROMPT Start with the cellular features.	• Usually irreversible injury • Often adjacent inflammation • **Swollen cells** • Increased eosinophilia • Myelin figures (whorls of cell membrane bits) • Nucleus fades (karyolysis), may shrink (pyknosis) and then fragments (karyorrhexis) • Organelle disruption → amorphous mass • **Cell membrane disrupted**, contents released	• Swelling • Disruption of cell integrity.
	2. What are the patterns of tissue necrosis? PROMPT What are the different macroscopic appearances of necrotic tissues?	• **Coagulative** (architecture preserved) • **Liquefactive** (digestion → liquid viscous mass) • Caseous (friable white) • *Gangrenous (usually applied to limb. Typically coagulative. Superimposed liquefaction from infection → 'wet gangrene') • *Fat necrosis (focal areas of fat destruction) • Fibrinoid (microscopic feature of Ag-Ab complexes in vessel walls from immune mediated)	• Coagulative • Liquefactive Prompt with names needs to describe difference *these terms clinical not true pathology terms

Reversible Cell Injury 2009-2

TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 1: Reversible Cell Injury	What are the morphological and chemical changes associated with early cell injury.	1. Decreased generation of ATP 2. Loss of cell membrane integrity 3. Defects in protein synthesis 4. Cytoskeletal damage 5. DNA damage	3 out of 5 to pass
Question 2:	What are the phenomena that characterize irreversible cell injury	The first is the inability to reverse **mitochondrial dysfunction** (lack of oxidative phosphorylation and ATP generation) even after resolution of the original injury. The second is the development of profound **disturbances in membrane** function.	Bold to pass
Question 3:	Can you give an example of a protein that leaks across degraded cell membranes? Prompt – "specific organs"	1. Cardiac muscle – contains a specific isoform of the enzyme creatine kinase and of the contractile protein troponin. 2. Liver (and specifically bile duct epithelium) – contains a temperature-resistent isoform of the enzyme alkaline phosphatase. 3. Hepatocytes – contain transaminases.	1 example to pass

Steatosis 2004-2

TOPIC: Discuss steatosis (fatty Change) **NUMBER: 5**

OPENING QUESTION	What is steatosis?	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	Abnormal accumulations of triglycerides within parenchymal cells		
SECOND QUESTION (if needed)	Which organs are commonly involved in steatosis?	Need 2 to pass	
POINTS REQUIRED	Liver		
	Heart, muscle, kidneys		
THIRD QUESTION (if needed)	What are the causes of hepatic steatosis?	Need 2 to pass	
POINTS REQUIRED	Alcohol abuse		
	Toxins (CCl4), protein malnutrition, diabetes mellitus, obesity, anoxia, starvation		
	In the liver it results from defects in any one of the events in the sequence from fatty acid entry to lipoprotein exit (FFA-esterified to triglycerides- converted into cholesterol and phospholipids or oxidized to ketone bodies- associated with apoproteins to form lipoproteins and released into the circulation)		