Pathology Definitions

Cellular Pathology

1. Aetiology – disease cause.
2. Pathogenesis – mechanism of disease development.
3. Morphologic changes – structural alterations in cells and organs.
4. Clinical significance – functional consequences of the morphological changes.
5. Cell injury – that which occurs after the limits of cell adaptation have been exceeded.
6. Morphological patterns of acute cell injury – apoptosis & necrosis.
7. Necrosis – the spectrum of morphological changes that follow cell death in living tissue. Due to enzymatic digestion & protein denaturation
8. Apoptosis – morphological pattern of cell death through activation of an internally controlled suicide program.
9. Cellular response to injury – acute cell injury, subcellular alterations, cellular adaptations, intracellular accumulations, pathological calcification & cell aging.
10. Hypoxia – low oxygen tension that impinges on aerobic oxidative respiration.
11. Ischaemia – lack of tissue blood supply due to impeded arterial or venous flow.
12. Physical agents – mechanical trauma, temperature extremes, atmospheric pressure changes, radiation and electric shock.
13. Chemical agents – therapeutic drugs, alcohol and drugs of abuse, industrial hazards, environmental pollutants, & poisons.
14. Oxidative phosphorylation – the production of ATP from ADP in a reaction that results in reduction of oxygen by the electron transfer system of mitochondria.
15. Glycolytic pathway – formation of ATP from ADP in the absence of oxygen using glucose or glycogen.
16. Reactive oxygen species –
17. Oxidative stress –
18. Ischaemia / Reperfusion injury – loss of cells in addition to those that are irreversibly damaged at the end of ischaemia.
19. Mitochondrial permeability transition – formation of a high conductance channel in the inner mitochondrial membrane, precluding maintenance of the proton motive potential, critical for oxidative phosphorylation.
20. Free radicals – chemical species that have a single unpaired electron in an outer orbit.
21. Antioxidant – substances that either block free radical formation or inactivate them.
22. Coagulative necrosis – denaturation of cytoplasmic proteins, breakdown of organelles, cell swelling with preservation of cellular outline for some days.

23. Autolysis – lysosomal derived enzymic digestion.
24. Heterolysis – enzyme derived from immigrant leukocytes.
25. Karyolysis – fading of chromatin basophilia thought due to DNase activity.
26. Pyknosis – nuclear shrinkage and increased basophilia. Seen in necrosis and apoptosis.
27. Karyorrhexis – pyknotic or partially pyknotic nucleus undergoes fragmentation.
28. Liquefactive necrosis – complete cellular digestion into a liquid viscous mass.
29. Pus – liquefactive necrosis due to acute inflammation with dead white cells.
30. Gangrenous necrosis – not a distinctive pattern of cell death. Due to coagulative necrosis.
31. Caseous necrosis – distinctive form of coagulative necrosis encountered most often in foci of TB infection. Amorphous granular debris composed of fragmented coagulated cells and debris with a distinctive inflammatory border. Obliteration of tissue architecture.
32. Fat necrosis – descriptive pattern of fat destruction typically occurring as a result of pancreatic lipase release into the pancreas and peritoneal cavity. Not a specific pattern of necrosis.
33. Caspases – cysteine proteases that cause protein hydrolysis. Specific feature of apoptosis.
34. Primary lysosome – membrane bound intracellular organelle containing hydrolytic enzymes.
35. Secondary lysosome (phagolysosome) – primary lysosome that has fused with membrane bound vacuoles that contain material to be digested.
36. Endocytosis – general uptake of materials from the external environment.
37. Phagocytosis – uptake of particulate matter.
38. Pinocytosis – uptake of soluble small macromolecules.
39. Autophagy – the internal uptake of intracellular products (organelles and cytosolic elements) in an autophagic vacuole formed by the ribosome free portion of RER which fuses with a primary lysosome or golgi apparatus.
40. Residual bodies – lysosome with undigested debris.
41. Lipofuscin pigment granules – undigested material resulting from intracellular lipid peroxidation. Cause brown atrophy / discolouration in large amounts.
42. Hyperplasia – increase in cell number in an organ or tissue which may then have increased in volume. [Physiologic (Hormonal eg. Breast in puberty & pregnancy), (Compensatory eg. Patrial hepatectomy) and (Pathologic eg HPV).
43. Hypertrophy – increase in cell size and subsequent increase in organ size. Physiologic (eg uterus in pregnancy), adaptive / pathologic (eg heart m.)
44. Atrophy – shrinkage in cell size by the loss of cell substance. Or a decrease in cell size and function. Physiologic (eg thyroglossal duct) or pathological (disuse, denervation, ischaemic, nutritional, endocrine, pressure & senile)
45. Metaplasia – reversible change in which one adult cell type (epithelial or mesenchymal) is replaced by another adult cell type. Eg columnar to stratified squamous change in resp tract due to cigarette smoke or Vit A deficiency.
46. Lipid classes – triglycerides, cholesterol, and phospholipids.
47. Steatosis (fatty change) – abnormal accumulation of TG's within parenchymal cells. Eg fatty liver in alcohol abuse or heart due to moderate hypoxia.
48. Cholesterolosis – focal accumulations of cholesterol laden macrophages in the lamina propria of the GB.
49. Pigment – coloured substance which may be endogenous (eg lipofuscin) or exogenous (eg carbon dust).
50. Haemosiderosis – systemic iron overload in which haemosiderin is deposited in many organs and tissues.
51. Pathological calcification – abnormal deposition of calcium salts in tissues. 2 forms.
52. Dystrophic calcification – deposition in dying tissues. Eg advanced atheroma or damaged heart valves.
53. Metastatic calcification – deposition of calcium salts in vital tissue due to abnormal calcium metabolism. Principally due to increased PTH, bone destruction, Vit D disorder or renal failure.
54. Hyaline change – alteration within cells or extracellular space which gives a homogenous, glassy, pink appearance.

Inflammation Definitions

55. Inflammation – the response of vascularised living tissue to injury. Evoked by microbial infections, physical agents, chemicals, necrotic tissue & immunological reactions.
56. Acute inflammation – immediate and early response to injury consisting of blood flow changes, increased vascular permeability & leukocyte extravasation.
57. Exudate – inflammatory extravascular fluid with a high protein [], much cellular debris, and an SG > 1.020.
58. Transudate – extravascular fluid with a low protein [] and an SG < 1.012.
59. Oedema – excess fluid in the interstitial or serous spaces.
60. Pus (purulent exudate) – inflammatory exudate rich in leukocytes (mainly neutrophils) and parenchymal cell debris.
61. Immediate transient response – rapid, short-lived and reversible formation of endothelial gaps in venules due to histamine, bradykinin, LT's & substance P.
62. Endothelial contraction – separation of intercellular junctions due to contractile elements.
63. Endothelial retraction (cytoskeletal reorganisation) - formation of intercellular gaps due to reorganisation of the cytoskeleton. Mediated by IL-1, TNF & IFN, delayed onset (4-6hrs) and duration (24hrs).
64. Transcytosis –
65. Direct endothelial injury –
66. Delayed prolonged leakage –
67. Leukocyte mediated endothelial injury –
68. Leakage from new blood vessels –
69. Extravasation – the sequence of events in the movement of leukocytes from the bv lumen to the interstitium. Includes margination, rolling, activation, adhesion, diapedesis (transmigration) & chemotaxis.
70. Pavementing – lining of endothelium by leukocytes.
71. Chemotaxis – locomotion along a chemical gradient.
72. Types of chemotaxins – exogenous or endogenous (complement, leukotriene & cytokines)
73. Leukocyte activation – response following activation by chemotaxins, phagocytosis or Ag-Ab complexes resulting in production of arachidonic acid metabolites, lysosomal burst, modulation of adherence molecules and priming.
74. Priming – increased rate and extent of leukocyte activation to a mediator.
75. Collectins – carbohydrate binding proteins of plasma that bind microbial cell walls and are involved in innate immunity.
76. Phagocytosis – recognition, engulfment and killing or degradation of ingested material
77. Anaphylatoxins – C3a and C5a, both which cause mast cell release of histamine and lipoxygenase activation
78. Platelet Activating Factor – bioactive phospholipid derived mediator
79. Cytokines – proteins produced by many different cell types that modulate the function of other cell types. Includes monokine, lymphokines, CSF, IL's chemokines & growth factors
80. Outcome of acute inflammation – complete resolution, abscess formation, healing by CT replacement (fibrosis), and progression to chronic inflammation.
81. Chronic inflammation – inflammation of prolonged duration in which active inflammation, tissue destruction and attempts at repair are all proceeding simultaneously.
82. Granulomatous inflammation – pattern of chronic inflammation in which the predominant cell type is an activated macrophage with a modified epithelial appearance
83. Granuloma – focal area of granulomatous inflammation.
84. Morphologic patterns in acute and chronic inflammation – serous, fibrinous, suppurative and ulceration.
85. Abscess – focal localised collections of purulent inflammatory tissue.
86. Ulcer – local defect or excavation of the surface of an organ or tissue that is produced by the sloughing of necrotic inflammatory tissue.
87. Acute phase proteins – CRP, Serum amyloid A and P (SAA & SAP), complement and coagulation factors.

88. Acute phase reaction components – endocrine / metabolic (acute phase proteins, increased glucocorticoids, and decreased vasopressin

Tissue Repair

89. Repair – process involving both regeneration and fibrosis
90. Fibrosis – replacement of tissue with connective tissue. Aka – fibroplasia. Hall mark of chronic inflammation
91. Two processes of fibrosis – emigration and proliferation & deposition of ECM
92. Cell growth cycle phases – presynthetic (G1), DNA synthesis (S), premitotic (G2) & mitotic (M).
93. Extra-cellular matrix components:
    a. Fibrous structural proteins
    b. Adhesive glycoproteins
    c. Gel of proteoglycans & hyaluronan
94. Collagen - triple helix of 3 polypeptide alpha chains having a gly-x-y repeating sequence. Fibrillar (I, II & III) and non-fibrillar (IV, V & VI) types.
95. Repair by connective tissue - 4 components
    a. Angiogenesis
    b. Migration & proliferation of fibroblasts
    c. ECM deposition
    d. Remodelling
96. Vasculogenesis – assembly of primitive vascular network in embryonic development
97. Angiogenesis – process by which existing vessele endothelial cells send out capillary buds to produce new vessels.
98. Steps required for angiogenesis
    a. ECM proteolysis
    b. Migration & chemotaxis
    c. Proliferation
    d. Lumen formation, maturation & inhibition of growth
    e. Recruitment of peri-endothelial cells to support endothelium
99. Remodelling – process of concurrent ECM synthesis and degradation.
100. Dehiscence – wound rupture
101. Keloid (hypertrophic) scar – accumulation of excessive collagen in a wound giving a raised tumorous scar

Immunology definitions

102. Hapten - an antigen that can stimulate production of antibodies only in combination with a specific protein
103. Types of Leukocytes – neutrophils, lymphocytes, monocytes, eosinophils, basophils (occasionally plasma cells)
104. White cell concentration – 7,000 per ml (vs RBC 5 x 106 & platelets 300,000 per microlitre).
105. Diapedesis – movement of WC's through endothelium.
106. Chemotaxis – movement of cells along a chemical gradient
107. Phagocytosis – cellular ingestion of offending elements
108. Monocyte-macrophage system = reticulo-endothelial system – consists of monocytes, mobile macrophages, endothelial cells in bone marrow, spleen and lymph nodes.
109. Histiocyte – synonym for macrophage
110. Innate immunity – that portion if the immune system providing protection as a result of general processes rather than that aimed at specific diseases
111. Acquired immunity – that portion of the immune system, consisting of antibodies and activated lymphocytes that attack and destroy specific organisms or toxins.

112. Basic types of acquired immunity – B-cell (humoral) & activated T-cells (cell mediated) immunity.
113. Antibodies – globulin molecules in the plasma capable to attacking an invading agent
114. Epitope – regularly recurring molecular group
115. Hapten – low molecular weight substance which much first combine with another protein to become antigenic.
116. Primary response – the slow, weak and short-lived production of antibodies following initial contact with an antigen.
117. Secondary response – rapid, potent and long-lasting production of antibodies following subsequent antigenic exposure.
118. Complement system – collective term for an enzyme cascade, the proteins of which are in the plasma and lymph and consists of 11 proteins (C1 to 9, B and D).
119. Passive immunity – the transfer of antibodies or activated T cells.

Cardiovascular

120. Oedema – increased fluid in the interstitial tissue spaces or body cavities (Ro) OR the presence of excess fluid in the body tissues (Gu – divided into intra and extracellular)
121. Hyperaemia – an active process resulting from augmented tissue inflow because of arteriolar dilation
122. Congestion – a passive process resulting from impaired outflow from a tissue
123. Haemorrhage – extravasation of blood due to blood vessel rupture.
124. Haematoma – haemorrhage enclosed within tissues
125. Petechiae – 1-2mm haemorrhages into skin, mucous membrane or serosal surfaces
126. Purpura – haemorrhages >3mm
127. Ecchymoses - >1-2cm subcutaneous haemorrhages
128. Haemostasis – a normal physiological process maintaining blood in a clot free state in normal vessels and inducing a rapid and localised haemostatic plug at sited of vascular injury.
129. Hypercoagulability – any alteration of the coagulation pathways that predispose to thrombosis.
130. Thrombosis – inappropriate activation of the normal haemostatic processes in the uninjured or minimally injured vessels.
131. Coagulation cascade – a series of conversions of inactive proenzymes, culminating in the formation of thrombin. Thrombin then converts soluble fibrinogen into insoluble fibrin
132. Embolism – detached intravascular solid, liquid, or gas that is carried by the blood to a site distant from its point of origin
133. Infarct – an area of ischaemic necrosis usually caused by occlusion of the arterial blood supply. Other causes may include vasospasm, mechanical and venous obstruction.
134. Shock – state of systemic hypoperfusion owing to a reduction in either cardiac output or in the effective circulating blood volume. The end result is impaired tissue perfusion and cellular hypoxia.
135. Arteriosclerosis – generic term for 3 patterns of vascular disease that has in common thickening and loss of elasticity of arterial walls.
136. 16 Atherosclerosis – a slowly progressive disease of the arteries marked by fibrofatty intimal plaques, formed by lipid deposition, smooth muscle cell proliferation and the synthesis of ECM within the intima.
137. Monckeberg medial calcific sclerosis – form of atherosclerosis characterised by calcific deposits in medium sized muscular arteries in persons older than 50
138. Arteriolosclerosis – disease of small arteries and arterioles (with 2 anatomic variants – hyaline and hyperplastic) characterised by thickened vessel wall and luminal narrowing
139. Atheromatous plaque – raised intimal plaque with a core of lipid (consisting of cholesterol and cholesterol esters) and a covering fibrous cap. They have 3 principal components – cells, ECM and lipids.

140. Response to Injury hypothesis – atherosclerosis is a chronic inflammatory response of the arterial wall initiated by some form of injury to the endothelium.
141. Aneurysm – localised abnormal dilation of a blood vessel occurring most commonly in the aorta and heart
142. True aneurysm – bounded by complete but often attenuated arterial wall components
143. False aneurysm – an extravascular haematoma that communicates with the intravascular space
144. Arterial Dissection – arise when blood enters the wall of the artery, dissecting the layers between, and creating a cavity within the wall itself
145. Aortic dissection – a catastrophic illness characterised by the dissection of blood in between and along the laminar planes of the media, with the formation of a blood filled cavity within the aortic wall (dissecting intramural haematoma) that often ruptures, causing massive haemorrhage.
146. Congestive Heart Failure – the pathophysiologic state resulting from impaired cardiac function rendering the heart unable to maintain an output sufficient for the metabolic requirements of the tissues and organs of the body.
147. Angina pectoris – a symptom complex of IHD characterised by paroxysms and usually recurrent attacks of substernal or praecordial chest discomfort caused by transient myocardial ischaemia that falls short of inducing cellular necrosis that defines infarction.
148. Sudden cardiac death – unexpected death from cardiac causes early (< 1hr) after or without the onset of symptoms.
149. Hypertensive Heart Disease – LVH (without other cardiovascular pathology) & history or pathologic evidence for hypertension.
150. Pulmonary hypertensive heart disease (Cor Pulmonale) – RVH, dilation and RHF secondary to pulmonary hypertension caused by disorders of the lungs or pulmonary vasculature.

Respiratory

151. Atelectasis – incomplete expansion of the lungs or collapse of previously inflated lung substance, producing areas of relatively airless pulmonary parenchyma.
152. Resorption atelectasis – that due to complete airway obstruction without impairment of blood flow.
153. Compression atelectasis – that due to pleural cavity, diaphragmatic or pulmonary mass effect impinging on lung function.
154. Patchy atelectasis – that due to loss of surfactant.
155. Contraction atelectasis – localised or generalised fibrotic changes in ling or pleura prevent full expansion.
156. ARDS = Diffuse alveolar damage – syndrome characterised by the rapid onset of severe life threatening respiratory insufficiency, cyanosis, and severe hypoxaemia that is refractory to oxygen therapy and may progress to extrapulmonary multi-system organ failure.
157. Pulmonary hypertension – mean pulmonary BP = ¼ mean systolic BP.
158. Obstructive airways disease – characterised by an increase in airflow resistance owing to partial or complete obstruction at any level (trachea to respiratory bronchioles).
159. Restrictive disease – reduced expansion of lung parenchyma with decreased total lung capacity.
160. Chronic obstructive pulmonary disease – group of conditions in which dyspnea is the major symptom, and is accompanied by chronic or recurrent obstruction to airflow in the lung.
161. Emphysema – lung condition characterised by abnormal permanent enlargement of the airway spaces distal to the terminal bronchiole, accompanied by destruction of their walls, and without obvious fibrosis.
162. Centri-acinar emphysema – anatomic pattern involving the central or proximal parts of the acini, formed by the respiratory bronchioles, sparing the distal alveoli. Eg. Tobacco smoking.
163. Pan-acinar emphysema – uniformly enlarged acini from the respiratory bronchiole to the terminal alveoli. Eg. Alpha-1 antitrypsin deficiency.

164. Distal acinar emphysema – proximal portion of the acinus is normal but the distal part is predominantly involved. Eg. Spontaneous pneumothorax.
165. Alpha-1 antitrypsin – a protease (esp elastase) inhibitor.
166. Compensatory emphysema – alveolar dilation without destruction of septal walls, in response to loss of lung tissue elsewhere.
167. Senile emphysema – over-distended, voluminous lungs in the aged, with larger alveolar ducts and smaller alveoli, without loss of elastic tissue.
168. Obstructive over-inflation – condition in which lung expands because of air trapped within it.
169. Bullous emphysema – any form of emphysema that produces large subpleural blebs or bullae (spaces > 1cm in distended state).
170. Interstitial emphysema – entrance of air into the connective tissue stroma of the lung, mediastinum, or subcutaneous tissue.
171. Chronic bronchitis – persistent cough with sputum for at least 3 months per year for 2 consecutive years.
172. Simple chronic bronchitis – productive cough without airflow obstruction.
173. Chronic asthmatic bronchitis – hyperactive airways with intermittent bronchospasm and wheezing.
174. Obstructive chronic bronchitis – chronic airflow obstruction usually with evidence of associated emphysema.
175. Asthma – chronic relapsing inflammatory disorder characterised by hyper-reactive airways, leading to episodic reversible bronchoconstriction, owing to increased responsiveness of the tracheobronchial tree to various stimuli.
176. Extrinsic asthma – that initiated by type 1 hypersensitivity reaction induced by exposure to an extrinsic antigen
177. Intrinsic asthma – that induce by diverse, non-immune mechanisms.
178. Acute response – bronchoconstriction, mucosal oedema and mucus secretion (+hypotension if severe).
179. Late-phase reaction –
180. Bronchiectasis – chronic necrotizing infection of the bronchi and bronchioles leading to or associated with abnormal dilation of these airways.
181. Kartagener's syndrome – autosomal recessive disorder consisting of bronchiectasis, sinusitis, situs inversus & infertility due to abnormal dynein arms of cilia.
182. Consolidation – exudative solidification of pulmonary tissue due to bacterial infection.
183. Lobar pneumonia – acute bacterial infection of a large portion of a lobe.
184. Atypical pneumonia – lacking alveolar exudate.
185. Primary atypical pneumonia = interstitial pneumonitis.
186. Cold agglutinins - autoantibodies that cause red blood cells to clump.
187. Lung abscess – local suppuration within the lung characterised by necrosis of lung tissue.
188. Ghon complex – parenchymal subpleural lesion and enlarged caseous lymph nodes draining the parenchymal focus.
189. Pneumoconiosis – diseases induced by particulates (both organic and inorganic), fumes and vapours.
190. Progressive massive fibrosis – confluent fibrosing reaction in the lung that can be the complication of any pneumoconiosis, although most common in coal worker pneumoconiosis & silicosis.
191. Caplan syndrome – coexistence of RA & pneumoconiosis, leading to the development of distinctive nodular pulmonary lesions that develop quickly.
192. Silicosis – lung disease caused by inhalation of crystalline silicon dioxide (silica).
193. Asbestos – family of crystalline hydrated silicates that form fibres.
194. Asbestosis – parenchymal interstitial fibrosis.
195. Sarcoidosis – systemic disease of unknown cause characterised by non-caseating granulomas in many tissues and organs.

196. Idiopathic pulmonary fibrosis (aka diffuse or cryptogenic fibrosing alveolitis) – diffuse interstitial inflammation and fibrosis of unknown cause, resulting in hypoxia and cyanosis in severe cases.
197. Hypersensitivity pneumonitis – spectrum of immunologically mediated, predominantly interstitial lung disorders caused by exposure to organic dusts and occupational antigens.
198. Bronchiolitis obliterans – filling of small airways by an inflammatory-fibrous exudate.
199. Horner's syndrome – enophthalmos, ptosis, miosis, & anhidrosis.
200. Neuroendocrine tumours – pulmonary neoplasms that share features with the dispersed neuroendocrine system. Eg. Small cell & carcinoid tumours.
201. Bronchiolitis obliterans-organising pneumonia (BOOP) – common response to infectious or inflammatory injury to the lungs.
202. Goodpasture's syndrome – characterised by the simultaneous appearance of proliferative, usually rapidly progressive GN and a necrotising haemorrhagic interstitial pneumonitis.
203. Carcinoid syndrome – diarrhoea, flushing and cyanosis.
204. Hydrothorax – non-inflammatory serous fluid collection within the pleura.
205. Meig's syndrome – hydrothorax, ascites and ovarian fibroma.
206. Chylothorax – accumulation of lymphatic fluid in the pleura.

Neoplasia

207. Neoplasia – an abnormal mass of tissue, the growth of which exceeds and is uncoordinated with that of the normal tissues and persists in the same excessive manner after cessation of the stimuli which evoked the change.
208. Desmoplasia – formation of abundant collagenous stroma.
209. Adenoma – benign epithelial neoplasm that either forms glandular patterns or is derived from a gland.
210. Papilloma – epithelial tumour forming micro or macroscopic finger-like projections. Eg. Wart.
211. Polyp – benign neoplasm with a macroscopic projection above mucosa. Eg. TCC papilloma.
212. Malignant polyp – polypoid cancer.
213. Sarcoma - malignant mesenchymal tumour. Eg. Rhabdomyosarcoma.
214. Carcinoma – malignant neoplasm of epithelial origin. Eg. Malignant melanoma.
215. Teratoma – neoplasm originating from totipotential cells. May be malignant or benign. Eg. Dermoid cyst & immature teratoma.
216. Mixed tumour – tumour derived from one germ layer that differentiates into more than one parenchymal type. EG. Mixed salivary gland tumour.
217. Hamartoma – malformations that present as a mass of disorganised tissue indigenous to the particular site. Not a tumour. Eg. Lung hamartoma.
218. Choristoma – ectopic non-transformed tissue. Not a tumour. Eg pancreatic cells under small bowel.
219. Differentiation – the extent to which parenchymal cells resemble comparable normal cells (functionally and morphologically).
220. Anaplasia – lack of differentiation. Hallmark of malignancy.
221. Dysplasia – disorderly growth characterised by pleomorphism hyperchromatism and loss of normal cell orientation.
222. Carcinoma in situ – when dysplasia involves the entire epithelium.
223. Metastasis – tumour implants that are discontinuous with the primary tumour.
224. Tumour progression – multi-step process at the phenotype and genetic levels which results in excessive growth, local invasiveness and the ability to form distant metastasis.
225. Mutation – permanent change in DNA.

226. Paraneoplastic syndrome – symptoms not directly related to the spread of the tumour or elaboration of hormones indigenous to the tissue from which the tumour arose.

227. Cancer grading – based on the degree of differentiation of the tumour and the number of mitoses within the tumour as presumed correlates with aggressiveness.

228. Cancer staging – based on the size of the primary lesion, the extent of lymph node spread, and the presence or absence of blood-borne metastases.

Haematology

229. Myeloid – bone marrow and all cells derived from it.
230. Lymphoid – thymus, lymph nodes and spleen.
231. Anaemia – reduction below normal limits of the total circulating red cell mass (or a reduction in the oxygen carrying capacity of the blood).
232. Mean cell volume – the average volume of a red blood cell (in cubic micrometres).
233. 45 Mean cell Hb – the average content of Hb per RBC (picograms).
234. Mean cell Hb [] – the average [] of Hb in a given volume of packed RBC's (g/100ml).
235. Haemolytic Anaemia – features – shortened RBC life span, accumulation of Hb breakdown products, and marked increase in haematopoiesis. Intra vs. extravascular, hereditary vs. acquired, & intrinsic vs. extrinsic forms.
236. Hereditary spherocytosis – inherited disorder characterised by an intrinsic RBC membrane defect that renders it spheroidal, less deformable and vulnerable to splenic sequestration and destruction.
237. G6PD deficiency associated anaemia – haemolytic disease due to abnormalities in the hexose monophosphate shunt or glutathione metabolism resulting from impaired enzyme function reducing the RBC's ability to protect itself against oxidative injury.
238. Sickle cell disease – hereditary haemaglobinopathy characterised by the production of structurally abnormal Hb.
239. Thalassemia syndromes – heterogenous group of Mendelian disorders characterised by a lack or decreased synthesis of either the alpha or beta globin chain of HbA.
240. Beta-thalassemia – total lack or reduction in the synthesis of structurally normal beta-globin chain with unimpaired synthesis of alpha chains. Alpha thalassemia – reverse.
241. Megaloblastic anaemia – diverse group of anaemias characterised by impaired DNA synthesis and distinctive morphological changes in blood and bone marrow.
242. Pernicious anaemia – Vit B12 deficiency megaloblastic anaemia due to atrophic gastritis and failure of intrinsic factor production.
243. Ferritin – Iron-protein complex found in the tissues.
244. Anaemia of Chronic Disease – anaemia due to impaired red cell production due to chronic infection / immune disorder or neoplasm characterised by low serum iron, reduced TIBC, high ferritin and increased stored iron in the MNP cells.
245. Aplastic anaemia – pancytopenia characterised by anaemia, neutropenia and thrombocytopenia due to failure or suppression of multi-potent stem cells.
246. Myelophthisic anaemia – marrow failure due to space-occupying lesions.
247. Polycythemia (erythrocytosis) – relative or absolute increase in the concentration of red cells.
248. Haemorrhagic diathesis – excessive bleeding due to increased blood vessel fragility, platelet deficiency or dysfunction, coagulation mechanism dysfunction, or a combination of these.
249. Thrombocytopenia – plat count < 100,000/mm3. Post-traumatic bleeding 20 – 50. Spontaneous <20.
250. Thrombotic thrombocytopenic purpura – syndrome characterised by fever, thrombocytopenia, microangiopathic haemolytic anaemia, transient neurological deficits and renal failure.
251. Haemolytic uraemic syndrome – syndrome characterised by microangiopathic haemolytic anaemia, thrombocytopenia, and acute renal failure (dominates), absence of neurological deficits, and childhood onset.

252. Disseminated intravascular coagulation – acute, subacute or chronic thrombohaemorraghic disorder occurring as a secondary complication of a variety of diseases. Characterised by activation of the coagulation sequence that leads to the formation of microthrombi throughout the circulation of the body. As a consequence of the thrombotic diathesis, there is a consumption of platelets, fibrin and coagulation factors and, secondarily, activation of the fibrinolytic mechanisms.
253. Leukopenia – abnormally low white cell count usually due to reduced neutrophils (neutropenia or granulocytopenia) or lymphocytes (lymphopenia).
254. Agranulocytosis – neutropenia with a predisposition to infection (<1000 cells/mm3, serious <500).
255. Leukocytosis – elevated white cell count.
256. Leukemoid reaction – appearance of immature granulocytes in the blood simulating myelogenous leukaemia.
257. Haematocrit – the percentage of blood that is cells.
258. Pluripotent haematopoietic stem cells – bone marrow cells from which all cells in the circulating blood are derived.
259. Committed stem cell – early off-spring cells from PHSC that have become differentiated into a particular line of cells. Form colony forming units.
260. Growth inducers – growth and reproduction of stem cells.
261. Differentiation inducers – those that cause one type of stem cell to differentiate into a particular line of cells.
262. Erythroblastosis fetalis – disease of the fetus and neonate characterised by agglutinatination and phagocytosis of the foetus's RBC.

Infectious Diseases

263. Prion - protein particle that is capable of causing an infection or disease.
264. Virus - non-cellular biological entity that can reproduce only within a host cell. Viruses consist of nucleic acid covered by protein
265. Bacteriophage / Plasmid / Transposing – mobile genetic element that encodes bacterial virulence factors.
266. Bacteria – cells which lack nuclei but have rigid cell walls containing two phospholipid bilayers (Gram –ive) or a single bilayer (gran +ive).
267. Rickettsiae – vector-borne obligate intra-cellular bacteria.
268. Fungi – thick ergosterol-containing walled cells and grow in humans as budding yeast cells and slender tubes (hyphae).
269. Protozoa – single celled organism endowed with motility, pliable plasma membranes and complex cytoplasmic organelles.
270. Helminth – highly differentiated multicellular organism with complex life-cycles.
271. Ectoparasite – arthropods that attach to and live on the skin.
272. Viral tropism – the tendency to infect some cells but not others.
273. Contagious – direct transmission from person to person by direct contact or aerosol.
274. Latency – inability to recover infectious particles from cells that harbour the virus.
275. Boil (furuncle) – focal suppurative inflammation of skin and subcutaneous tissue, single or multiple, or recurrent in successive crops.
276. Carbuncle – deep suppuration that spreads laterally beneath subcutaneous fascia and then burrows superficially to erupt in multiple adjacent skin sinuses.
277. Hydradenitis Suppuritiva – persistent abscess formation of apocrine gland regions, esp in axilla.

Liver

278. Hepatic stellate cell – fat containing cells involved in the storage and metabolism of Vit A, found in the space of Disse and which transform into myofibroblasts in inflammation.

279. Ballooning degeneration – swollen, oedematous hepatocytes with clumped cytoplasm and large clear spaces.
280. Councilman body – an apoptotic hepatocyte
281. Foamy degeneration – retained bile imparts a foamy appearance.
282. Steatosis – accumulation of fat droplets within hepatocytes. (Micro and macrovesicular forms)
283. Centrilobular necrosis – hepatic necrosis around the terminal hepatic vein.
284. Hepatitis – injury to the liver associated with an influx of acute or chronic inflammatory cells.
285. Icterus – yellow discoloration of the sclerae.
286. Physiologic jaundice of rhe newborn – transient, mild unconjugated hyperbilirubinaemia in neonates due to immature hepatic conjugating and excretion machinery.
287. Gilbert syndrome – common (6%) benign inherited disorder characterised by mild fluctuating unconjugated hyperbilirubinaemia due to a reduction in glucuronidation activity.
288. Conjugated hyperbilirubinaemia – abnormal elevation of bilirubin with >50% conjugated, typically associated with cholestasis.
289. Xanthoma – cutaneous deposits of cholesterol.
290. Cholestasis – blockage of bile excretion causing retention of bilirubin and bile salts. Or – retention of bilirubin, other biliary solutes and cholesterol. Hepatocellular dysfunction or biliary obstruction.
291. Hepatic encephalopathy – life threatening disorder of CNS and N-M transmission accompanied by only minor changes in the brain.
292. Hepatorenal syndrome – appearance of ARF in patients with severe liver disuse in whom there are no morphological or functional causes.
293. Cirrhosis morphology – fibrosis, nodules and disruption of parenchymal architecture.
294. Portal hypertension – increased resistance to portal blood flow.
295. Ascites – the excess accumulation of fluid in the peritoneal cavity.
296. HBcAg – Hep B nucleocapsid core protein / antigen.
297. HBeAg – Hep B longer nucleocapsid polypeptide with a core and pre-core region.
298. HBsAg – Hep B envelope / surface glycoprotein.
299. Hep B DNA polymerase – reverse transcriptase which allows genomic replication.
300. Carrier – individual without manifest symptoms who harbours and therefore can transmit an organism.
301. Chronic hepatitis – symptomatic, biochemical, or serological evidence of continuing or relapsing hepatic disease for more than 6 months, with histologically documented inflammation and necrosis.
302. Fulminant hepatic failure – progression of hepatic insufficiency from onset of symptoms to encephalopathy within 2 to 3 weeks. Sub-fulminant – up to 3 months.
303. Xenobiotic - A xenobiotic is a chemical which is not a natural component of the organism exposed to it. Synonyms: drug, foreign substance or compound, exogenous substance or compound.
304. Reye's syndrome – a potentially fatal syndrome of mitochondrial dysfunction in the liver and brain occurring predominantly in children exposed to aspirin for the relief of virus induced fever.
305. Idiosyncratic drug reaction – unpredictable reaction to a drug usually related to the propensity of the host to mount an immune response to the antigenic stimulus (eg halothane) or the rate of drug metabolism (eg. Chlorpromazine).
306. Haemochromatosis – syndrome characterised by excessive accumulation of iron in the body, most deposited in parenchymal organs.
307. Wilson's disease – autosomal recessive disorder of copper metabolism marked by the accumulation of copper in the eye, brain and liver.
308. Alpha-1 Antitrypsin deficiency – autosomal recessive disorder characterised by abnormally low levels of this important protease inhibitor.

Biliary

309. Acute calculous cholecystitis – acute inflammation of the GB, precipitated 90% of the time by obstruction of the neck or cystic duct.
310. Cholangitis – bacterial infection of the bile ducts.
311. Ascending cholangitis – infection of the intra-hepatic biliary radicles.
312. Cholesterol stone tetralogy – 1. Supersaturation, 2. GB hypomotility promotes nucleation, 3. Cholesterol nucleation in bile is accelerated, & 4. GB mucus hypersecretion traps crystals permitting agglomeration.

Pancreas

313. Acute pancreatitis – an acute condition typically presenting with abdominal pain associated with raised serum pancreatic enzymes.
314. Chronic pancreatitis – a disease characterised by repeated bouts of pancreatic inflammation with continued loss of parenchyma and fibrous tissue replacement.
315. Pseudocyst – localised collections of pancreatic secretions that develop after pancreatitis.
316. Pancreatic abscess – (sterile) liquefactive necrosis of severely damaged pancreatic parenchyma.
317. Diabetes mellitus – a heterogenous group of disorders with defective or deficient insulin secretory response, glucose under utilisation, and hyperglycaemia as a common feature.
318. Ketone bodies – butyric acid and acetoacetic acid.
319. AGE – advanced glycosylation end products.

Bone & Joint

320. Osteoid – unmineralised bone.
321. Osteoprogenitor cells – pluripotential mesenchymal stem cells allocated at bone surfaces with the capacity to undergo cell division and differentiate into osteoblasts.
322. Osteocyte – osteoblast surrounded by matrix.
323. Osteoblast – cell on bone surface which synthesises bone matrix and initiate mineralisation.
324. Osteoclast – cell which is responsible for bone resorption and derived from haematopoietic stem cells.
325. Woven bone – collagen deposited in a random weave, quickly produced and able to resist multidirectional strain.
326. Lamellar bone – collagen layered in an orderly manner, is slowly formed but is stronger than woven bone.
327. Remodelling – the breakdown and renewal of bone.
328. Osteoporosis – increased skeletal porosity due to a reduction in bone mass, results in a predisposition to fracture.
329. Stress fracture – a slowly developing fracture that follows a period of increased physical activity in which new loads are placed upon the bone.
330. Pathological # - a break through a bone already altered by a disease process.
331. Synovial fluid – plasma filtrate containing hyaluronic acid.
332. Ankylosis – loss of joint movement.
333. Osteoarthritis – degenerative joint disease characterised by progressive deterioration and breakdown of articular cartilage, mainly in the weight bearing joints.
334. Rheumatoid arthritis – chronic systemic inflammatory disorder that affects many tissues and organs (skin, bv's, heart lungs, & m's) but principally affects the joints, producing a non-suppurative proliferative synovitis that often progresses to a destruction of the cartilage and joint ankylosis. Or basically a severe form of chronic synovitis that can lead to destruction and ankylosis of affected joints.
335. Pannus – fibrocellular mass of synovium and synovial stroma consisting of inflammatory cells, granulomatous tissue and fibroblasts that cause erosion of the underlying cartilage. Or exuberant synovium.

336. Arthus reaction – localised are of tissue necrosis resulting from acute immune complex vasculitis, usually in the skin.

Endocrine

337. Hormone – secreted molecules involved in endocrine signalling.
338. Cushing's syndrome – hypercortisolism.
339. Cushing's disease – hypercortisolism due to excessive ACTH production by the pituitary.
340. Nelson's syndrome – large destructive pituitary adenomas due to adrenal resection.
341. Pituitary apoplexy – sudden haemorrhage into the pituitary gland (often an adenoma) causing sudden onset of a headache, diplopia and hypopituitarism.
342. Sheehan's syndrome – post-partum anterior pituitary ischaemic necrosis.
343. Empty sella – any condition that destroys part or all of the pituitary gland
344. Empty sella syndrome – presence of an enlarged empty sella turcica not filled with pituitary tissue.
345. Goitrogen – substance which inhibits thyroid function.
346. Thyrotoxicosis – hypermetabolic state caused by elevated levels of T3 &4.
347. Hyperthyroidism – thyrotoxicosis due to hyperfunction of the thyroid gland.
348. Hypothyroidism – any structural or functional derangement that interferes with the production of adequate levels of thyroid hormone.
349. Cretinism – hypothyroidism developing in infancy or early childhood.
350. Myxoedema – hypothyroidism developing in the older child or adult.
351. Thyroiditis – diverse group of disorders characterised by some form of thyroid inflammation.
352. Graves's disease – hyperthyroidism with diffuse thyroid enlargement, infiltrative ophthalmopathy (exophthalmos) and dermopathy (pre-tibial myxoedema).
353. Goitre – thyroid enlargement.
354. Plummer syndrome – development of a hyperfunctioning nodule in a multi-nodular goitre.
355. Primary hyperparathyroidism – autonomous over-production of PTH
356. Secondary hyperparathyroidism – PTH over-production due to any condition which causes the chronic suppression of serum calcium.
357. Hypercalcaemia – increase in ionised (free) calcium in the serum.
358. Tertiary hyperparathyroidism – PTH over-production due to secondary causes which becomes excessive and autonomous.
359. Pseudohyperparathyroidism – resistance of organs to normal or elevated PTH levels.
360. Primary hyperaldosteronism – generic term for syndromes characterised by chronic excess aldosterone production.
361. Conn's syndrome – primary hyperaldosteronism caused by an aldosterone producing adenoma.
362. Adrenocortical insufficiency – hypofunction due to primary adrenal disease or ACTH deficiency.
363. Waterhouse-Friderichsen syndrome – primary acute adrenocortical insufficiency due to an overwhelming bacterial infection, hypotension and DIC.
364. Addison's disease – primary chronic adrenocortical insufficiency due to adrenal cortex destruction.
365. Phaeochromocytoma – chromaffin cell neoplasm which synthesises and release catecholamines and sometimes peptides.
366. MEN syndromes – group of familial autosomal-dominant diseases associated with hyperplasias or neoplasms.

Renal

367. Juxtaglomerular apparatus – juxtaglomerular cells (modified smooth muscle cells), macula densa (distal tubule) & lacis cells (?modified JG cells).
368. Azotemia – elevation of urea and creatinine usually due to a decreased GFR.
369. Pre-renal azotemia – hypoperfusion that impairs renal function in the absence of parenchymal damage.
370. Post-renal azotemia – impaired renal function due to obstruction of urine flow below the level of the kidney.
371. Uraemia – azotemia with clinical signs and symptoms & biochemical abnormalities.
372. Acute nephritic syndrome – glomerular syndrome characterised by an acute onset, haematuria, proteinuria & hypertension. (Eg post-streptococcal GN).
373. Nephrotic syndrome – proteinuria (>3.5gm/day), hypoalbuminaemia, oedema, hyperlipidaemia, and lipiduria.
374. Acute renal failure – oliguria or anuria with recent onset of azotemia. Due to glomerular, vascular, interstitial or ATN.
375. Chronic renal failure – symptoms and signs of uraemia and is the result of chronic renal diseases.
376. Renal tubular defects – polyuria, nocturia, & electrolyte disorders.
377. UTI – bacteriuria and pyuria. Asymptomatic vs symptomatic. Cystitis vs pyelonephritis.
378. Nephrolithiasis –
379. Stages of renal failure – diminished renal reserve (GFR ~ 50%), renal insufficiency (GFR 20 to 50%), renal failure (GFR < 20 to 25%) & end-stage renal disease (GFR < 5%).
380. ATN – clinicopathological entity characterised by destruction of tubular epithelial cells and acute suppression of renal function (abnormal U/Cr and UO < 400ml/d).
381. Pyelonephritis – disorder affecting the tubules, interstitium and renal pelvis.
382. Acute pyelonephritis – acute suppurative inflammation of the kidney caused by bacterial infection due to ascending or haematogenous spread.
383. Chronic pyelonephritis – chronic tubulo-interstitial renal disorder in which chronic tubulointerstitial inflammation and renal scarring are associated with pathologic involvement of the calyces and pelvis.
384. Benign nephrosclerosis – sclerosis of renal arterioles and small arteries.
385. Malignant nephrosclerosis – form of renal disease associated with the accelerated phase of hypertension.
386. Malignant hypertension syndrome – diastolic BP > 130mmHg, papilloedema retinopathy, encephalopathy, cardiovascular abnormalities and renal failure.
387. Obstructive uropathy / hydronephrosis – unrelieved urinary obstruction leading to permanent renal atrophy and associated with infections and calculi.
388. Hydronephrosis – dilation of the renal pelvis and calyces associated with progressive atrophy of the kidney due to the obstruction of the outflow of urine.

Diseases of Immunity

389. AIDS – a retroviral disease characterised by profound immunosuppression that leads to opportunistic infections, secondary neoplasms and neurologic manifestations.
390. Cytokine – short acting soluble mediator of intercellular communication.
391. 5 categories of cytokines:
    a. Natural immunity mediators – eg IL1,6, TNF & type 1 IFN
    b. Lymphocyte growth regulators – eg IL2,4, 5, 12 & 15, and TGF
    c. Inflammatory cell activators – IFN gamma, TNF
    d. Chemokines – eg IL8
    e. Haematopoiesis stimulators eg CSF's
392. 4 general properties of cytokines
    a. May be produced by several different cells types

b. Effect many different cell types
c. Effects via
    i. Autocrine – IL2
    ii. Paracrine – IL7
    iii. Endocrine – IL1 and TNF
d. Mediate their effects by specific receptors eg IL2 receptors.

393. Physiologic function of MHC – bind peptide fragments of foreign proteins for presentation to appropriate antigen specific T cells.

394. Antigen presenting cells – macrophages, B lymphocytes and dendritic cells but may be induced on other cells by IFN.

395. Type 1 hypersensitivity – rapidly developing immunologic reaction occurring within minutes after the combination of an antigen with antibody bound to mast cells or basophils in individuals previously sensitized to the antigen.

396. Atopy – a genetically determined predisposition to develop localised anaphylactic reactions to inhaled or ingested allergens.

397. Type II hypersensitivity – mediated by antibodies directed towards antigens present on the surface of cells or tissue components.

398. Sub-types of Type II hypersensitivity
a. Complement dependent reactions – eg transfusion reaction
b. Antibody dependent cell mediated cytotoxicity – eg parasitic infections
c. Antibody mediated cellular dysfunction – eg myasthenia gravis

399. Type III hypersensitivity – induced by antigen antibody complexes that produce tissue damage as a result of their capacity to activate the complement system.

400. Patterns of Type III hypersensitivity:
a. Systemic immune complex disease – eg serum sickness. 3 phases:
    i. Formation of Ag-Ab complexes
    ii. Deposition of complexes
    iii. Inflammatory reaction to the complexes via complement and direct Ag-Ab effects.
b. Local immune complex disease (Arthus reaction)

401. Arthus reaction – localised area of tissue necrosis resulting from acute immune complex vasculitis usually in the skin. Eg hyperacute rejection.

402. Type IV hypersensitivity – cell-mediated type of hypersensitivity initiated by specifically sensitised T lymphocytes. Includes the classic delayed type hypersensitivity initiated by CD4 cells (eg tuberculin reaction) and direct cell cytotoxicity mediated by CD8 cells (eg TB, contact dermatitis, virally infected cells and graft rejection).

403.