

PRINCIPLES

1. cause of the disease
2. sequence/mechanism by which a disease occurs
3. How a tissue changes its looks/appearance
4. morbidity is a sickness, mortality is disease.
5. Reaction of the body to injury and or stress
6. continuously renewing cell populations such as epithelial cells, highly adaptable.
7. Expanding cell population such as hepatocytes, may expand if needed
8. static cell populations such as myocytes
9. severely, duration, condition, location, degree of cell specialization
10. atrophy, hypertrophy, hyperplasia, metaplasia, and dysplasia
11. shrinkage of cell due to loss of organelles.
12. Increase in cell size and functional capacity
13. when endocrine stimulation lessens
14. during diminished blood supply
15. pathological hypertrophy
16. increased cells :: hormonal stimulation (lactating breast), increased red blood cells at high altitudes
17. endometriosis, psoriasis, liver nodules
18. one terminally differentiated cell type is replaced by another terminally differentiated cell type
19. squamous epithelium.
20. Squamous metaplasia of bronchus or bladder, Barret, esophagus, myositis ossificans
21. stratified squamous is replaced by columnar epithelium to protect against stomach enzymes
22. loss of uniformity of cells and loss of architectural orientation
23. during persistent injury and is considered pre-neoplastic
24. hyperplasia and metaplasia.
25. Carcinoma in situ
26. normal cellular constituents, abnormal substance, a pigment
27. steatosis, water, pigments, proteins, glycogen, cholesterol
28. accumulation of fat w/in parenchymal cells :: liver :: protein malnutrition, toxins, obesity, anorexia
29. Signet ring
30. Failure of Na-K+ATPase pump, cytoplasmic organelles
31. abnormal accumulation of iron within parenchymal cells :: golden brown granular appearance
32. hemosiderosis (localized hemosiderin accumulation)
33. blood transfusions, autoimmune disease, hemolytic anemia, hemochromatosis
34. Lipofuscin :: yellow brown pigment of lipids and proteins
35. oxidative stress



36. Pompe, McArdle, Cori, Von Geirke
37. True
38. Macrophages, vascular smooth muscle cells (atherosclerosis)
39. Xanthomas
40. genetics, infections, nutritional imbalances, physical agents, immunological, chemicals/drugs, hypoxia
41. ATP
42. lack of oxygen, ↑ Intracellular calcium, depletion of ATP, defects in membrane permeability
43. cellular swelling, steatosis
44. Loss of Na+K+ATPase pump (sodium floods in, so water must too)
45. fat accumulation due to altered metabolism/transport of triglycerides
46. ↑ Lactic acid, ↓pH, ↓ATP, ↑Na, ↑H₂O, cell swelling, detachment of ribosomes, mito swelling
47. Mitochondrial vacuolization, lysosomal membrane rupture, nuclear change
48. Mitochondria fusing to a lysosome
49. Pyknotic (underwent pyknosis)
50. Karyorrhexic
51. Karyolytic
52. a free valence electron
53. final common pathway
54. chemical/radiation injury, cellular aging, oxygen toxicity, microbial killing by phagocytes
55. cell membranes (lipid peroxidation), cellular proteins (oxidative modification), DNA
56. SOD (super oxide dismutase), GSH (glutathione), Vitamin E, Catalase
57. Fenton Reaction & Haber-Weiss Reaction
58. H₃O₂ :: It's a free radical itself (so if stopped here SOD can actually be dangerous)
59. O₂ + H₂O
60. dystrophic calcification
61. systemic problem due to hypercalcemia resulting in calcium deposition in normal tissues

NECROSIS

62. A spectrum of morphological changes that occur in cells following cell death.
63. Denaturation of proteins: enzymatic digestion of organelles
64. Loss of blood supply to limbs extremities resulting in cell death.
65. Necrotizing fasciitis, diabetes, frostbite.
66. Caseous necrosis
67. Liquefactive
68. Hypoxic cell death, such as a myocardial infarct.
69. Liquefactive necrosis.



KRS STUDY GUIDES : Quiz Answers : Arar : "I regret the chicken BBQ."

70. liquefactive necrosis.
71. Enzymatic fat necrosis.
72. anucleated, fibroblasts.
73. When an exudate cannot be removed, such as in caseous necrosis.
74. Programmed cell death.
75. To protect against inflammation.
76. Cell shrinkage, chromatin condensation, apoptotic bodies, and phagocytosis
77. Organelle fragments.
78. Pathogenesis of neoplasms.
79. Heat shock proteins.
80. Induced or constitutively synthesized.
81. HSP 60/70, Protein folding.
82. ubiquitin
83. Attenuation of cell injury or death.
84. HSP 60/70
85. Degradation of proteins (sticks to damage cells for cleanup)

INFLAMMATION

86. complex reaction which leads to an accumulation of fluid & leukocytes / involved in the repair process.
87. Cell regeneration, scar formation.
88. Leukocytes, blood vessels, connective tissue cells, and extracellular matrix (collagen).
89. Physical, chemical, microbiological and immune responses.
90. Acute and chronic.
91. Primarily neutrophils also macrophages.
92. Primary lymphocytes also macrophages and fibroblasts.
93. Polymorphonucleotide
94. heat (Calor), swelling (tumor), redness (rubor), pain (dolor), loss of function (function laesa)
95. accumulation of fluid within the extra vascular tissue.
96. Excess fluid in body cavities.
97. Edema fluid with low-protein content, and no inflammatory cells. (specific gravity < 1.015)
98. Edema fluid with a high protein concentration containing inflammatory cells (specific gravity > 1.015)
99. watery edema lacks a cellular response
100. serous exudate that contains red blood cells
101. exudate containing a large amount of fibrin as activated by the coagulation system.
102. Edema that contains prominent cellular components, often pathological infections.
103. Purulent exudate accompanied by significant liquefactive necrosis.



- 104. vasopermeability change, vasodilation, extravasation of leukocytes
- 105. autonomic nervous system response to stop bleeding.
- 106. Causes stasis, which is increased viscosity.
- 107. Margination = movement of white blood cells toward the vascular wall.
- 108. Selectins (rolling), integrins (adhesion), immunoglobulins (allow leukocytes to pass through)
- 109. LAD 1 (no selectins), LAD 2 (no integrins, very rare)
- 110. severe recurrent bacterial infections.
- 111. Emigration/migration via chemotaxis
- 112. The opsonins (C3b & IgG) bind & coat the antigen marking it for phagocytosis
- 113. using free radicals, lysosomal enzyme, and production of amino acid metabolites

CHEMICAL MEDIATORS

- 114. substances, which act to initiate or enhance an inflammatory reaction
- 115. from plasma or cells, are short lived
- 116. harmful effects
- 117. short lifespan, inhibited by intrinsic mechanisms.
- 118. Kinin system, complement system, and clotting/coagulation system.
- 119. Bradykinin.
- 120. Pain, vasodilation, and increased vascular permeability.
- 121. Coagulation system, Hageman factor
- 122. when it comes into contact with none vasculature (collagen, negative charges, microbes, etc.).
- 123. c1, c3a, c3b, c5a, c5b, c9
- 124. bacterial LPS cell walls.
- 125. C1.
- 126. C3a, C5a
- 127. C3b
- 128. C5a
- 129. C5b, C9
- 130. proteins involved in *blood clot formation*, some also mediate inflammation
- 131. increase vascular permeability, adherence & fibroblast proliferation : activate fibrinogen.
- 132. Vascular permeability.
- 133. TPA (tissue plasminogen activator).
- 134. Clot/plug formation
- 135. break apart clots, activate complement system by cleaving C3 into C3a & C3b
- 136. cyclooxygenase pathway
- 137. lipoxygenase pathway



138. NSAIDs
139. *Histamine*, serotonin
140. ↑, ↑
141. PGI₂, TXA₂, PGD₂, PGE₂
142. Prostacyclin
143. Edema
144. Antagonistic prostaglandin, vasoconstrictive, ↑ platelet aggregation
145. smooth muscle contraction, vascular permeability, clinical symptoms of allergy reactions.
146. Produces chemotactic activity for neutrophils, monocytes, and macrophages.
147. Corticosteroids, NSAIDs, and fish oils
148. lymphocytes and macrophages :: endotoxins, immune complexes, and physical injury
149. interleukins (IL-1,2,6,8), tumor necrosis factor (TNF)
150. nitric oxide, endothelium adhesion molecules
151. IL-1 & IL-6, systemic acute phase reactions
152. neutrophils (IL-8 is the primary cause)
153. fever, ↓ Appetite, ↑ Protein synthesis, ↑ Leukocytes :: (FLULIKE SYMPTOMS)
154. endothelial -- derived relaxing factor (derived for endothelium & macrophages)
155. vasodilator, and cytotoxic as a free radical
156. inducible and constitutive
157. membrane phospholipids
158. all inflammatory cells, endothelial cells, and injured tissues
159. ↑ Permeability, vasodilation, platelet aggregation, ↑ Adherence, ↑ arachodonic acid metabolism
160. viral replication.
161. IL-6 (panacrine for b-lymphocytes)

CELLULAR PARTICIPANTS OF INFLAMMATION

162. neutrophils
163. PAF, Leukotrienes, prostaglandins, and lysosomal enzymes :: bone marrow
164. 48 hours
165. Eosinophils :: Histamine
166. in allergic reactions, parasitic infections, and Hodgkin's disease
167. release histamine and heparin :: hypersensitivity reactions
168. mast cell
169. late stages of acute inflammation and all chronic inflammation
170. kupffer (liver), alveolar macrophage (lung), microglial (nervous), osteoclasts (bone), histocyte (generic)
171. PAF, leukotrienes, prostaglandins, cytokines, lysosomal enzymes



- 172. increased metabolism and size, & change shape (improves phagocytosis)
- 173. lymphocytes (20-30% of circulating leukocytes)
- 174. MHC = major histocompatibility complex
- 175. cell mediated
- 176. orchestrates humoral immunity, helper/inducer T cells, secrete cytokinin
- 177. cytotoxic/suppressor T cells, secrete cytokines to cause cell lysis
- 178. CD4
- 179. Humoral
- 180. plasma cells and memory cells
- 181. IgG, IgE, IgA, IgM, IgD
- 182. terminally differentiated b-lymphocyte.
- 183. Immunity not requiring prior exposure to the antigen, nonspecific & lacks memory
- 184. Inflammatory response
- 185. specific response requiring antigen exposure. Subsequent exposures magnify response
- 186. body surfaces that are externally exposed
- 187. all body fluids, bacterial and viral infections
- 188. they are the smallest (BTW, most abundant too)
- 189. IgM, blood and lymph fluid
- 190. IgD (responds to milk, poison, medications)
- 191. IgM
- 192. lung, skin, and mucous membranes :: people with allergies (responds to dander, pollen, etc...)

HYPERSENSITIVITY REACTIONS

- 193. type 1 immediate hypersensitivity, type 2 cytotoxic, type 3 immune complex mediated, type 4 delayed
- 194. IgE, binds to mast cells and basophils
- 195. Histamine (+ leukotrienes & eosinophil chemotactic factor)
- 196. leads to arterial dilation (hypotension), venules (vascular leakage)
- 197. allergies, poison ivy, anaphylactic reactions
- 198. 1st exposure → increased IgE → IgE binds to mast cells → 2nd exposure → degranulation of mast cells
- 199. Stationary antigens
- 200. Complement protein, Ig-Fc receptor bearing cells.
- 201. Autoimmune
- 202. Graves' disease, Myasthenia gravis
- 203. anti-glomerular basement membrane antibodies
- 204. anti-beta cell islet antibodies, glucose intolerance and vasculopathy
- 205. none hyperthyroid, large goiter, anti-thyroglobulin antibodies.



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- 206. Autoimmune thyroiditis, with hyperthyroid. Anti-TSH, receptor antibodies.
- 207. Anti-acetylcholine receptor antibodies leads to paralysis
- 208. Immune complexes, huge
- 209. complement system.
- 210. Systemic (lupus), blood vessels (vascular disease), kidney (glomerulonephritis), joint space (arthritis)
- 211. inflammatory response, the complement system, leukocytes.
- 212. Two weeks
- 213. Antigen elicited response (no antibody needed)
- 214. Cytokine, CD4+ T-helper cells
- 215. Macrophage, lymphocyte, 24-48 hours
- 216. T-DTH :: during the second exposure
- 217. poison ivy, cosmetics, TB, Crohn's, sarcoidosis, Spirochete diseases, fungal infections

CHRONIC INFLAMMATION

- 218. persistent microbial infection, prolonged toxin exposure, autoimmune diseases.
- 219. Chronic inflammation with activated macrophages and possessing a epithelioid appearance
- 220. multi-nucleated giant cells, lymphocytes, fibroblasts
- 221. Central core of necrosis surrounded by epithelioid cells, lymphocytes, and fibroblasts
- 222. Langerhans giant cell (horseshoe arranged peripheral nuclei)
- 223. coagulation system
- 224. angiogenesis, migration/proliferation of fiberblasts, adding of extracellular matrix, remodeling CT
- 225. migration and proliferation of endothelial cells (building the road)
- 226. type 3 collagen, type I collagen
- 227. granulation tissue.
- 228. Proliferating capillaries/fibroblasts/myofibroblasts, extracellular fluid, macrophages
- 229. fill in tissue spaces, remove dead cells, wound contraction, form the pre-scar
- 230. blood clot, neutrophil infiltration, epithelial continuity restored, macrophage infiltration, granulation tissue fills space, progressive collagenization, remodeling
- 231. granulation tissue
- 232. prolonged inflammatory reaction, debris, inflammatory exudate, pronounced scar formation
- 233. myofibroblasts
- 234. type, size, location, vascular supply, type of infection, movement
- 235. circulatory status, infection, malnutrition
- 236. proud flesh
- 237. keloid formation
- 238. wound dehiscence
- 239. Contracture, dimpling of the skin

