

1st Colloquium Questions
(cell injury, inflammation, APR)

1. How and why do the cellular contents of K^+ and Na^+ change in the damaged cell?
2. How and why does the cellular content of Ca^{++} change in the damaged cell?
3. List the general mechanisms of cell injury.
4. List the general mechanisms of cell membrane damage.
5. Name the main non-enzymatic antioxidants involved in the inactivation of free radicals and lipid peroxidation products.
6. Name the main enzymatic antioxidants involved in the inactivation of free radicals and lipid peroxidation products.
7. What are the mechanisms of action of the main enzymes - antioxidants (for catalase and SOD)?
8. Consequences of excessive intracellular accumulation of ionised calcium.
9. Reperfusion injury. Mechanisms of tissue injury (e. g. in myocard).

10. Give a definition of inflammation.
11. Why inflammation is a typical pathological process?
12. Indicate the positive effects of inflammation.
13. Indicate the negative effects of inflammation.
14. What are the 3 main outcomes of acute inflammation?
15. List the classic signs of inflammation.
16. List the factors that cause pain in the site of inflammation.
17. Name the changes in microcirculation in the site of inflammation in the correct sequence.
18. Name the mechanisms involved in the development of arterial hyperemia in acute inflammation.
19. List the factors that contribute to the transition of arterial hyperemia into venous in acute inflammation.
20. List the factors that contribute to the development of true capillary stasis in acute inflammation.
21. Name the inflammatory mediators related to pre-formed mediators.
22. Name the inflammatory mediators related to the newly formed mediators of cellular origin.
23. Give a scheme of cell lipid mediators formation.
24. Name the inflammatory mediators related to the newly formed extracellular mediators (derived from plasma sources).
25. What is the mechanism of complement activation in the classic activation pathway? What other pathways to activate complement are known?
26. Name the complement fractions that play the role of a) anaphylatoxins, b) chemoattractants and opsonins (c).
27. Name 4 «watchdog» (patrol, protective) systems that are activated by Hageman factor.
28. Name the effects of histamine in the site of inflammation.

29. What is the role of mast cells in the development of acute inflammation?
30. Name the effects of bradykinin in the site of inflammation.
31. Give a scheme of bradykinin formation.
32. What is exudation? Name the types of inflammatory exudates.
33. What pathogenetic factors contribute to the formation of exudates in the site of inflammation?
34. Name the mechanisms that contribute to an increase in vascular permeability in the site of inflammation.
35. What processes underline the increase in oncotic pressure in the interstitium in the site of inflammation?
36. What processes underline the increase in osmotic pressure in the interstitium in the site of inflammation?
37. The cause and consequences of increasing hydrostatic pressure in the microvessels in the site of acute inflammation.
38. Name the mechanisms of leukocytes emigration into the site of inflammation.
39. Name the types of leukocytes and their correct order of emigration into the site of acute inflammation (the law of Mechnikov).
40. Name the substances that can act as positive chemoattractants during inflammation.
41. Indicate the main types of adhesion molecules causing the emigration of leukocytes during acute inflammation.
42. What are the consequences of «leukocyte adhesion deficiency» syndrome?
43. Name the stages of phagocytosis. What kinds of leukocytes are the «professional» phagocytes?
44. What are the role of opsonins? What substances belong to opsonins?
45. What and how the different complement fractions can promote phagocytosis?
46. What processes occur inside the phagosome and phagolysosome? What are the «perulent bodies»?
47. List the bactericidal substances and hydrolytic enzymes in neutrophil granules.
48. Mechanism and effects of «respiratory burst» of neutrophils in the site of acute inflammation.
49. Indicate positive and negative significance of leukocytes degranulation and formation of neutrophil extracellular traps in the site of acute inflammation.
50. What is the primary and secondary alteration?
51. Indicate the factors contributing to development a chronic inflammation.
52. What is a role of macrophages in the development of chronic inflammation?
53. What is a role of the granuloma in chronic inflammation?
54. Give the definition of APR.
55. Indicate a positive role of APR.
56. What systems and organs play the main role in the pathogenesis of APR?
57. Name the main signs from the central nervous system during APR.
58. What main changes occur in the immune system during APR?

59. What changes in peripheral blood cells be observed during nonspecific or aseptic inflammation and APR?
60. What mediators are responsible for development of neutrophilia during APR?
61. Name the cytokines that are main mediators of APR.
62. Indicate the sources of APR cytokines synthesis.
63. Name the effects of IL-1 during APR.
64. Name the effects of IL-6 during APR.
65. Name the effects of TNF during APR.
66. Which of the APR mediators most stimulates the synthesis of AP proteins in the liver?
67. Describe positive role of «positive» AP proteins (including complement) in the body during acute inflammation.
68. Indicate positive and negative effects of the «negative» proteins in the body during APR.
69. Concentration of what plasma proteins is increased most significantly during APR.
70. How and why the hemostasis changes during APR?
71. Which mediator mediate the effects of IL-1 and TNF α in the development of fever, weight loss, cartilage and bone tissue damage?
72. What mechanisms related to APR associated with increased body resistance?
73. Which APR mediators are chemoattractants for neutrophils and monocytes?
74. Name the possible negative effects of APR.
75. Indicate the mechanisms of cachexia in case of APR.
76. What and why can happen to cartilage and bone tissue in case of APR?
77. Name the main mechanisms of body weight loss during APR.
78. What is a Fever?
79. How is fever different from hyperthermia?
80. Indicate the pathogenetic types of hyperthermia.
81. List the human compensatory reactions by significant increase in environment temperature.
82. What mediators of APR are endogenous pyrogens?
83. What changes are caused by endogenous pyrogens in the neurons of thermoregulatory center in hypothalamus?
84. List the stages of a) fever and b) exogenous hyperthermia.
85. How do absolute values of heat production and heat loss in the a) first, b) second and c) third stage of fever related to? Answer to give separately for a, b, c.
86. Indicate what reactions of the body cause heat loss, and what – heat production.
87. Indicate what physical processes underlie the physiological reactions that cause the heat loss.