

Problems: “Cell injury”, “Inflammation”, “Acute phase response”, “Fever”, and “Hyperthermia”

#### List of control questions to the first colloquium

1. List the basic mechanisms of cell injury (4)
2. List the main mechanisms of cell membrane injury (4)
3. Name the major oxidants of non-enzyme origin which take part in free radicals and their products destruction (4)
4. Point to the mechanisms of major oxidants action (3)
5. Give a definition to term “inflammation”
6. Why an inflammation is called “typical pathological process?”
7. Point to the three main outcomes of inflammation
8. Call the signs of inflammation (5)
9. How can you explain such symptoms as redness and warmth in the site of inflammation?
10. Call 5 factors which can provoke a pain in the site of inflammation
11. Point to positive consequences of inflammation (3)
12. Point to negative consequences of inflammation (3)
13. Point to the factors which predispose to chronic inflammation development (3)
14. What is the role of mast cells in acute inflammation?
15. Call the preformed mediators of inflammation (4)
16. Call the inflammatory mediators which are formed “de novo” (4)
17. Call the inflammatory mediators originated from cells (4)
18. Call the mediators of inflammation originated from plasma (3)
19. Which substance triggers the classic pathway of complement system activation, and point to other pathways of its activation
20. What does “lysing complement complex” mean and its role in inflammation?
21. What are the roles of activated complement fractions in inflammation? (4)
22. What is the role of Hageman’s factor in an acute inflammation? Which 4 system it activates during inflammation?
23. List the effects of histamine in an acute inflammation (3)
24. Call three effects of bradykinin in the site of inflammation
25. Call the order of microcirculatory events in the site of inflammation
26. What events accompany inflammatory active hyperemia?
27. List the factors that are responsible for development of active hyperemia in acute inflammation (4)
28. What changes in microcirculation are characteristic of congestion?
29. Which factors predispose to conversion of active hyperemia into congestion in course of an acute inflammation (4)
30. Which processes promote blood condensation in the site of inflammation (4)
31. Which kind of stasis characteristic of inflammation? List the factors which predispose to stasis in acute inflammation (4)

32. Call 5 types of inflammatory exudate
33. List pathogenetic mechanisms which are involved in exudate formation (3)
34. Which types of microvessels become the most permeable in acute inflammation (2)
35. Which factors are responsible for the gaps formation between the endothelial cells? (3)
36. Which processes are responsible for increased tissue colloid- osmotic pressure?(2)
37. Which factors take part in increased interstitial osmotic pressure formation in inflammation?
38. Which factors promote leukocytes emigration in inflammation (4)?
39. What does positive chemotaxis mean?
40. Call polyvalent chemattractants (4)
41. Point to 3 categories of adhesion molecules which are involved in leukocyte emigration
42. What are the negative consequences of surplus expression of adhesion molecules which are responsible for leukocyte emigration?
43. Call the hereditary syndromes which are responsible for deficiency in leukocyte emigration and their phagocytic activity (4)
44. Call the stages of leukocyte emigration and explain their pathogenesis
45. Describe the order of different types of leukocyte emigration
46. Describe the steps of phagocytosis and explain their mechanisms
47. Which components of complement system take part in phagocytosis?
48. Call the opsonins (4) and describe the mechanisms of their activity in inflammation?
49. How can be explained a facilitation of phagocytosis after “enveloping of the microorganisms by IgG and C3b complement components?
50. Which processes are proceeding inside the phagosome?
51. Which bactericidal substances are found in neutrophil granules (4)
52. Which hydrolyzing enzymes are found in neutrophil granules? ((4)
53. What are the positive consequences of neutrophil degranulation?
54. What are the negative consequences of neutrophil degranulation ?
55. Point to the causes and consequences of non-completed phagocytosis
56. Call the main symptoms which reflect an activation of CNS in APR (4)
57. Which processes are proceeding in CNS during APR (4)?
58. Call 3 main cytokines which mostly provide the pathogenesis of APR
59. Point to the main 6 effects of IL-1 (local or distant)
60. List 5, the most characteristic of IL-6 effects in course of APR
61. Which hematological changes are characteristic of APR (4) ?
62. Which factors are mostly responsible for neutrophilia in APR (3)
63. Point to specific effects of IL-6 in APR (4)
64. Which of an acute phase response mediators mostly is responsible for APR proteins synthesizing?

65. Which of the mediators of APR are the endogenous pyrogens (2) ?

66. Concentration of which proteins in the blood becomes especially high during APR (2) ?

67. Which of the APR mediators could be named polivalent chemattractants (2) ?

68. Which mediator is directly responsible for such effects as fever, loss of weight and decreasing in bone and muscle mass under IL-1 and TNF-influence?

69. Call the protective mechanisms working during acute phase response (4)

70. List the mechanisms of loss of weight during acute phase response (3)

71. List the possible negatives consequences of APR (5)

72. Which changes the secondary pyrogen provokes in the termoregulation neurons of hypothalamus ?

73. Which absolute values of heat production and heat dissipation are characteristic of:

1.1<sup>st</sup>. stage , 2.2<sup>nd</sup>.stage, 3.3<sup>rd</sup>. stage? Give 3 answers reflected their rate and coordination

74. What causes may lead to the changes in termoregulation which result in hyperthermia (3)

75. Call the compensatory reactions of men when increased temperature of the surrounding (3)

76. Call two bacteriostatic substances which can be found n the neutrophil granules

77. Which cells possess by the receptors to C3b complement component and IgG (3)?