**MICROCIRCULATORY DISORDERS**

1. What is the main pathogenic link of arterial hyperemia?

2. What is the correlation between influx and reflux in arterial hyperemia?

3. What is the pathogenic mechanism of neurotonic arterial hyperemia?

4. What is the pathogenic mechanism of neuroparalytic arterial hyperemia?

5. What is the pathogenic mechanism of neuromyoparalytic arterial hyperemia?

6. What is the pathogenic mechanism of functional arterial hyperemia?

7. What are the manifestations of arterial hyperemia?

8. What does venous hyperemia represent?

9. What is the cause of venous hyperemia?

10. What is the main pathogenic link of venous hyperemia?

11. What are the external manifestations of venous hyperemia?

12. What is the cause of increasing organ volume in venous hyperemia?

13. What is the cause of decreasing local temperature in venous hyperemia?

14. What are the consequences of venous hyperemia?

15. What are the local pathogenic mechanisms of ischemia?

16. What local hemodynamic changes are characteristic for the ischemia?

17. How does the cellular metabolism change in ischemia?

18. What are the external manifestations of ischemia?

19. What organ has collaterals total functional insufficient?

20. What type of embolism is considered to be as endogenous?

21. What type of embolism is considered to be as exogenous?

22. What are the causes of air embolism?

23. What are the causes of gaseous embolism?

24. What vessel is obturated in case of embolism with amniotic liquid?

25. What are the consequences of artery embolism?

26. What factor disturbs rheological properties of the blood?

27. What does the edema represent?

28. What are the pathogenic links of hydrostatic edemas?

29. What are the pathogenic links of hypooncotic edemas?

30. What are the pathogenic links of hyperosmotic edemas?

31. What are the pathogenic links of membranogenic edemas?

32. What are the pathogenic links of lymphogenic edemas?

33. What are the pathogenic links of edemas in circulatory failure?

34. What are the pathogenic links of edemas in nephritic syndrome?

35. What are the pathogenic links of edemas in nephrotic syndrome?

36. What are the pathogenic links of edemas in liver failure?

37. What are the pathogenic links of edemas in cachexia?

38. What are the pathogenic links of edemas in allergy?

**Inflammation**

1. What is the etiology of inflammation?

2. Which harmful factors can cause primary alteration?

3. What are the manifestations of primary alteration in the inflammatory site?

4. What are the factors that cause secondary alteration in the inflammatory site?

5. What carbohydrates metabolism disorders are found in the inflammatory site?

6. What pathogenic factors determine local acidosis in the inflammatory site?

7. What are the sources of accumulated histamine in the inflammatory site?

8. What is the key enzyme that causes prostaglandin synthesis?

9. What is the origin of inflammatory mediators?

10. What are the cells-sources(precursors) of inflammatory mediators? Biological effects of cell mediators

11. What are mast cells mediators with chemotactic effect?

12. What is the key enzyme for leukotrienes synthesis?

13. What are the biological effects of prostaglandins PGD2, PGE2, PGF2?

14. What mediators are derived from arachidonic acid? List their biological effects

15. What are the biological effects of IL-1?

16. What are derived mediators from neutrophil leukocytes? List their biological effects

17. What bactericidal factors are generated by neutrophil leukocytes?

18. What biologically active factors are formed due to activation of the complement?

19. What biological effects do the components of the activated complement have?

20. What are plasma-derived inflammatory mediators? List their biological effects

21. What is the consequence of vascular reaction in the inflammatory site?

22. What mediators determine the development of arterial hyperemia in the inflammatory site?

23. What are the peculiarities of arterial hyperemia in the inflammation?

24. Which pathogenetic factors determine the increase of vascular permeability in inflammation?

25. Which factors determine the occurrence of venous hyperemia in the inflammatory focus?

26. What is the biological significance of arterial and venous hyperemia and stasis in the inflammation?

27. What are the pathogenic factors of exudation in the inflammation?

28. Give the characteristic of different kinds of exudates?

29. How does the transudate differ from exudate?

30. What are the cells that have phagocytic activity?

31. Which cells are macrophages?

32. What factors contribute to the leukocyte migration in the inflammatory focus?

33. What is the biological significance of leukocyte emigration in the inflammatory focus?

34. What are the phagocytosis phases? List their succession

35. What is the succession of the phenomena involved in devitalizing of internalized microbe?

36. What are the sources of hydrolases in the inflammatory focus?

37. What is the sequence of white blood cell migration in the inflammatory focus?

38. What are the cellular sources of regeneration and proliferation in the inflammatory focus?

39. What processes include physiological regeneration in the inflammatory focus?

40. What are the disorders in the body that reflect the acute phase response to inflammation?

41. What are systemic manifestations in the organism during the inflammation?

42. What disorders of the internal environment attest the presence of an inflammatory process in the organism?

43. Which hormones have pro-inflammatory action?

44. Which hormones have anti-inflammatory action?

**Hypersensibility disorders**

1. What does represent allergy?
2. What is the difference between immune reactions and allergic reactions?
3. What is the name of the factors which trigger allergic reactions?
4. What substances can be considered complete allergen?
5. What substances can be considered haptenes?
6. In what conditions the haptenes can trigger allergic reactions?
7. What are the conditions which favorise the development of cross-linked allergic reactions?
8. What endogenous allergen are natural natives allergenes?
9. What antigenes and tissues are considered „sequestred antigenes”?
10. What endogenous allergen are infectious aquired allergens?
11. What endogenous allergen are non-infectious aquired allergens?
12. What does represent exogenous allergenes?
13. What phenomena underlie on the basis of allergic reactions?
14. What are the stages of allergic reactions? The characteristics of stages
15. In what conditions there is considered that the body is sensitized?
16. In what conditions can develop passive sensitization?
17. What immunoglobulin is involved in type I allergic reaction?

18. On what immune cell the immunoglobulin E is attached in type I hypersensibility?

19. Where does occur the interaction between immunoglobulin and antigen in type I hypersensibility?

20.What is the characteristic of immunologial phase in hypersensibility disorders?

21. What are the sources of primary anaphylaxia mediators?

22.What mediators are released in the result of mast cell degranulation?

23. What are the biological effects of histamin?

24. What are the sources of secondary anaphylaxia mediators?

25. What mediators are derived from arachydonic acid?

26.What are mediators with brnchoconstrictor and bronchodilator effects?

27. What are mediators with vasoconstrictor and bronchoconstrictor effects?

28.What phenomena are characteristic for pathophysiological stage in hypersensibility disorders?

29. What are the characteristics of immunological stage in reaginic allergic reactions?

30. What are the mediators of anaphylactic reaction?

31. What mediator triggers bronchial spam in allergic bronnchial asthma?

32. What disorders underlie on the basis of type I hypersensibility?

33.What disorders underlie on the basis of cytotoxic-cytolytic allergic reaction?

34.What immunoglobulines are involved in type II hypersensibility?

35.What are mediators involved in type II hypersensibility?

36.What complement fractions represents anaphylatoxins?

37. What does represent anaphylatoxin?

38. What are the mechanisms of cytotoxicity in type II hypersensibility?

39.What are the characteristics of pathochemical stage in type II hypersensibility?

40. What is the common pathogenetic mechanism in type III hypersensibility?

41. In what conditions the circulating immune complexes become aggresive?

42. What are the conditions which favor the long-lasting circulation of immune complexes?

43.What are the mechanisms of tissue injury in type III hypersensibility?

44. What is the sequence of phenomena which lead to injuries in type III hypersensibility?

45. What diseases underlie on the basis of type III hypersensibility?

46. What are the characteristics of immunological stage in delayed hypersensibility?

47. What cells are involved in type IV allergic reactions?

48. In what type of hypersensibility there is production of sensitized lymphocytes?

49. What are the mediators of pathochemical stage in delayed hypersensivbility?

50.What are the mechanisms of tissue injury in type IV allergic reaction?

51. What disorders underlie on the basis of delayed hypersensibility?

52. What does represent antibody mediated cellular dysfunction? What does represent the allergen in this reaction?

53. What immunoglobulins are involved in antibody mediated cellular dysfunction?

54. Where does occur the interaction between the antigen and antibodies in Graves disease?

55. What is the difference between antibody mediated cellular dysfunction and classical type II allergic reactions with cell injury?

56.What is the main pathophysiological mechanism on which underlie the autoimmune reactions?

57. In what autoimmune disorders the auto-antibodies are organ-specific?

58. In what autoimmune disorders the auto-antibodies are not organ-specific?

59. What type of allergic reaction underlies on the basis of autoimmune thyrotoxicosis?

60. In what type of allergic reaction there is recommended specific hyposensibilization?

61.What substances are used to perform specific hyposensibilization?

62. What does represent primary and secondary immunodefficiency?