**Problem questions from pathophysiology for 3rd year, winter (5th) semester**


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## A. PATHOPHYSIOLOGY OF PAIN

1. Definition of pain, classification of different types of pain, somatic, visceral, muscular, referred pain, nerve, neuropathic pain, allodynia, nociceptive, central, psychogenic and phantom pain.
2. Neuroanatomy of pain, afferent, central and efferent nervous structures involved in the origin of pain sensation.
3. Describe the difference between somatic and visceral pain, demonstrate these two types of pain using the example of a sudden abdominal episode.
4. Neuropathophysiology of pain - analyse the different levels in the pain pathway where neuromodulation of pain occurs. Characterize disorders of perception and nociception of painful stimuli.
5. Why does massaging a painful area reduce pain perception?
6. Explain why, in a local burn of moderate degree, we attenuate the pain by placing the injury with a cool object, ice or cold water?
7. Explain the mechanism of stress analgesia (hypesthesia)?
8. Silent myocardial ischemia.
9. Migraine.

## B. DIABETES MELLITUS

1. Define DM and classify the most common types of diabetes mellitus.
2. Diabetic - Type 1 patient is identified by history and basic examination with the following symptoms and signs: heart palpitations, tachycardia, feeling hungry, mydriasis, cold sweat. What do these symptoms and signs indicate? Explain their origin.
3. Diabetic - type 2 patient told you that he collapsed when getting out of bed, as well as from the chair (during the day) and also after eating. What could be the cause of the collapse? Explain the possible mechanisms of its occurrence.
4. List and explain the mechanisms of the symptoms and signs of DM. What findings do you expect to find on blood, urine and o - GTT examination?
5. What are the pathomechanisms of metabolic syndrome X?

6. A patient with type 1 DM has been repeatedly observed to have a coxsackievirus infection that preceded the onset of manifest DM by several weeks to months. Explain the possible pathogenetic relationship.
7. In an obese 40-year-old man, we found the presence of glucose in the urine. On examination, fasting glucose level was elevated and in OGTT we found impaired glucose tolerance. However, we detected antibodies against β cells of islets of Langerhans, GAD and insulin in the patient. What is the disorder of glucose metabolism? Define the terms MODY (maturity-onset diabetes of the young) and LADA (latent autoimmune diabetes of adults).
8. Hypoglycaemia and diabetes mellitus.
9. What organ damage can we detect in a patient with long-lasting diabetes mellitus?
10. A patient with decompensated diabetes mellitus is in a coma. What type of coma might this be?

## C. CEREBRAL ISCHEMIA

1. You characterize the cerebral circulation, its regulation in physiological circumstances.
2. Which pathomechanisms are involved in the origin and development of ischemic brain damage?
3. Define the degrees of brain cell damage by ischemia, explain the mechanisms and consequences.
4. You are called to an unconscious patient. Which symptoms and signs would alert you to local cerebral ischemia in the pyramidal pathway?
5. Why should penumbra be considered in an ischaemic cerebral lesion?
6. A patient has experienced transient cerebral ischemia during warm weather. Which pathomechanisms may have been involved in the occurrence of this episode
7. What is the relationship between cerebral ischemia, cerebral oedema, and intracranial hypertension?
8. A patient was brought to the emergency department over the morning with paralysis and decreased sensation in half of his body (right upper and lower extremity), he was confused and disoriented. After treatment, his condition corrected ad integrum within 24 hours with no neurological deficit. Explain what type of cerebral ischemia is manifesting in this case? Why didn't TIA (transient ischemic attack) leave a neurologic deficit?

## D. ACID-BASE BALANCE DISORDERS

1. Characterise the mechanisms compensating for the lack or excess of hydrogen ions in the body.
2. How does $CO_2$ interfere with ABR regulation?
3. A patient with duodenal ulcer disease was taking excessive NaHCO3. On examination, he had the following acid-base balance values: pH=7.5; [$HCO_3^-$] 33 mmol/l and $pCO_2$ 6.13 kPa. What type of acid-base disorder is manifesting in this case?
4. A patient with DM, with MAC present, has a pH of 7.2. We calculated, that to compensate for acid-base balance it is necessary to administer 80 ml of bicarbonate. We will administer only ½ of the calculated volume. Explain.
5. During the lecture, acid-base disturbances affecting the organism as a whole were mentioned. Do some localized disturbances (e.g. local acidosis) in organism tissues exist? Give an example. What is the practical application?

6. $CO_2$ poisoning is accompanied by RAC. What is the acid-base balance in CO poisoning?
7. Patient with pleural effusion had acid-base-related values pH=7.35; [$HCO_3^-$] 27 mmol/l and $pCO_2$ 6.99 kPa. What type of acid-base balance disturbance are you considering?
8. What type of acid-base balance disorder puts individuals at high altitudes at risk?
9. The patient was admitted to the hospital after a trauma. Intraabdominal haemorrhage was diagnosed with the development of shock and respiratory insufficiency. What type of acid-base disorder are you considering in laboratory values pH=7.03; [$HCO_3^-$] 17.1 mmol/l and $pCO_2$ 8.76 kPa?
10. Why does the kidney exacerbate metabolic alkalosis in a patient with excessive vomiting?

## E. DISTURBANCES OF VOLUME AND COMPOSITION OF BODY FLUIDS

1. What is the role of sodium in the regulation of individual body fluid compartments?
2. The role of osmotic gradient, oncotic pressure in the regulation of body fluid volume.
3. Why are the principles of treatment of hypovolaemia and dehydration different?
4. Can haematocrit, red blood cell count, and blood protein concentration be used in the diagnosis of body fluid volume and composition disorders?
5. A patient has had elevated intracranial pressure for several days and is at risk of developing an occipital conus. From a pathophysiological point of view, what should be done to reduce the intracranial pressure?
6. A patient is in a hyperglycaemic-hyperosmolar non-ketogenic coma for several hours. State the pathophysiological principles of managing this process.
7. Explain the case report: a 58 years old male observed swelling around the ankles on the lower extremities during the day. He also states that he has shortness of breath and his chest sometimes stings on physical exertion; at rest, the symptoms subside. Oedema worsens towards the evening but in the morning the patient is free of oedema. At night he urinates frequently.
8. What are the possible mechanisms of ascites in the chronic abuser of alcohol?
9. What is the relationship between dehydration and frostbite? Which type of dehydration (hypo-, iso-, hyperosmotic) is most dangerous for their development?
10. What role do changes in effective arterial volume play in the development of oedema? Why does oedema occur in nephrotic syndrome with increased arterial blood volume?

## F. STRESS

1. What is the relationship between the reactivity of the organism and the response of the organism to the load?
2. Which afferent pathways provide the "activation of the stress response centre" when exposed to different stressors (physical, chemical, biological, psychological)?
3. Why is it important to know the metabolic stress response?
4. What is the positive and negative significance of the stress response?
5. What are the so-called adrenaline sports? What do you think motivates people who engage in them to pursue these often-life-threatening activities?
6. The patient has diabetes mellitus:
    a. What effect will repeated psychological stress have on the course of his/her disease?
    b. Explain the mechanisms by which psychological stress can affect the course of diabetes.

7. A patient is diagnosed with partial adrenal cortical hypofunction, which is caused by progressive degeneration of cortical cells. Surgery is planned for this patient.
    a. Should special care be given to this patient in the preoperative period?
    b. If yes - why (explain and state the focus of this care)
8. Regular physical activity reduces the risk of developing so-called civilisation diseases. Explain this statement based on the stress response.
9. A patient has elevated BP readings during repeated blood pressure measurements in the GP's office. After starting pharmacological treatment, he repeatedly collapsed, especially in the morning. Explain.
10. Characterize the relationship between stress reaction and shock.

## G. SHOCK

1. Define shock, classify shock into types based on the triggering cause of shock.
2. Characterize the compensatory mechanisms that occur after a decrease in blood pressure.
3. What is the role of the various components of the cardiovascular system in the onset and development of circulatory shock?
4. Which processes in the body represent the main characteristics of the different phases of circulatory shock?
5. Which pathomechanisms lead to the progression of circulatory shock?
6. Which hemodynamic processes distinguish the different types of circulatory shock and which are common?
7. The characteristic disturbance in shock is an inadequately low supply of oxygen and nutrients to cells in tissues. Try to define the positive feedback loops that are activated by this process and contribute to the progression of shock (these represents the activation of multiple vicious circles).
8. Characterize the changes in the kidney and lung that occur as a result of shock. What can lead to prolonged renal hypoperfusion and why?
9. Why does multiorgan failure occur in the progression of shock?
10. Which organ represents the “engine” of progression of circulatory shock and why?

## H. THERMOREGULATION

1. What is the role of thermoregulation in maintaining the stability of the inner environment?
2. A patient has a body temperature of 40°C.
   **Problem No. 1:** What can be the cause of this temperature?
   **Problem No. 2:** If we find the causes of elevated body temperature, is it necessary to lower it? If so, why? If not, why? If you decide to lower the high body temperature, what options do you have from a pathophysiological point of view?
3. The patient has a body temperature of 34°C.
   **Problem No. 1:** What can be the cause of this temperature?
   **Problem No. 2:** If we find the causes of decreased body temperature, is it necessary to increase it? If so, why? If not, why? If you decide to increase the low body temperature, what options do you have from a pathophysiological point of view?
4. Does alcohol contribute to an increase in body temperature in hypothermic individuals?
5. In infectious diseases, fever develops in most of those affected. Why do some sufferers develop no fever or only a small increase in body temperature?
6. Are there differences in the mechanisms of fever in infectious diseases, tissue necrosis, hematomas, and stress?

7. What is your opinion on the control of fever by administering baths, and/or showers with lukewarm water?

## I. GENERAL ETIOPATHOGENESIS OF DISEASES

1. What are the main mechanisms and consequences of the action of mechanical noxa on the organism?
2. The main pathogenetic mechanisms of the origin and development of blast and crush syndrome, pressure sores (decubiti).
3. What are the mechanisms of the development of kinetosis and its manifestations?
4. What are the mechanisms by which vibration and noise affect the human organism?
5. Explain the mechanisms of action of ionizing radiation on the human organism - on its tissues.
6. What are the main effects of ionizing radiation on the human organism?
7. Explain with an example the occurrence of manifestations of damage to the tissues of the organism by ionizing radiation.
8. Explain the genesis of the main forms of acute radiation sickness.
9. What are the main consequences of the action of electric current on the organism?
10. What are the main consequences of chronic noxae action on cells?
11. Which endogenous chemicals can harm the human body?
12. Mechanisms of damage to the tissues of the body by corrosive substances.
13. By what mechanisms do organophosphates act on the human organism?
14. What are the main effects of animal toxins on the human organism?
15. The importance of apoptosis in the pathogenesis of diseases.
16. Disruption of cell volume regulation - its significance in the pathogenesis of diseases.

## J. SIRS

1. Define inflammatory response, describe the difference between local and systemic inflammatory response.
2. What is the difference between the following processes - bacteraemia, sepsis, SIRS, septic shock, and multiorgan failure
3. How does the initiation of SIRS occur in infectious and non-infectious processes?
4. What are the symptoms and signs of SIRS and what mechanisms are involved in their initiation?
5. Describe the mechanism leading to the production of pro-inflammatory cytokines via transcription factor κB.
6. How does the natural system antagonizing SIRS work?
7. Which mechanisms are involved in the development of multiorgan failure in SIRS?

## K. CAUSES AND MECHANISMS OF DYSFUNCTION OF ARTERIAL AND VENOUS SYSTEM OF LOWER EXTREMITIES AND VISCERAL CIRCULATION

1. Explain the mechanism of individual symptoms and signs in acute arterial occlusion of the lower extremities.

2. Explain the mechanism of the development of individual symptoms and signs in progressive arterial occlusion of the lower extremities.
3. Explain the functioning of venous circulation in the lower extremities of a healthy person.
4. List the main risk factors for venous thrombosis and explain how they contribute to the pathogenesis of venous thrombosis.
5. Explain the mechanisms of the development of chronic venous insufficiency and its symptoms.
6. Explain the difference between thrombophlebitis and DVT, analyse which condition is dangerous in terms of pulmonary embolism and explain why.
7. You are investigating the case of a 40-years-old patient who was brought in with severe pain in the LE, the limb is pale, the limb volume is unchanged, the venous pattern is weak, pulses are absent in the periphery. The patient is afebrile, on auscultation, you find a diastolic murmur at the apex, the pulse is rapid with unequal filling, irregular, and pulse deficit is present.
    a. Analyse the situation in the vasculature of the LE.
    b. Analyse which pathological process could be its cause.
8. You are investigating the case of a 40-years-old patient who was brought in with severe LE pain, limb is pale, the volume of the limb is increased, the venous pattern is increased, pulses are present. The patient has tachycardia and fever. The colour of the limb gradually changes to cyanotic.
    a. What pathological process might be involved?
    b. Explain the possibilities of skin colour changes on the limb.
    c. What could be the predisposing factors for the development of this condition in a relatively young patient?
9. A patient complains of sudden onset of intense severe pain in the abdominal region, the nature of which varies. Explain whether this is occlusive or non-occlusive intestinal ischaemia.
10. Consequences of ischemia-reperfusion injury to the intestinal wall.
11. The gastrointestinal tract as an “engine” of multiorgan failure.

*Translation: Tomáš Buday, MD, PhD.*