

Документ подписан простой электронной подписью
 Информация о владельце:
 ФИО: Косенок Сергей Михайлович
 Должность: ректор
 Дата подписания: 2019.03.15
 Уникальный программный ключ:
 e3a68f3eaa1e62674b54f4998099d3d6bfdcf836

Assessment tools for midterm assessment

PATHOPHYSIOLOGICAL BASES OF EMERGENCY CONDITIONS IN THE CLINIC OF INTERNAL DISEASES

	31.05.01 General Medicine
Specialty	General Medicine
Form of education	full-time
Designer Department	Pathophysiology and general pathology
Graduate Department	Internal diseases

12 term

Sample tasks for Control work List of topics for abstracts

1. Pathophysiology of bacterial shock
2. Neonatal sepsis. Umbilical sepsis.
3. The role of chemokines and adhesion molecules in phagocytic reactions.
4. Intensive care of acute respiratory distress syndrome
5. Pharmacotherapy in pregnant women with respiratory pathologies
6. Features of changes in the mechanical properties of the lungs and gas exchange at different stages of acute respiratory distress syndrome at the stages of mechanical ventilation in patients.
7. Modern methods of stopping bleeding
8. Hemorrhagic syndrome following warfarin overdose.
9. Drug correction of posthemorrhagic anemia
10. Thrombophilias during pregnancy
11. Antiphospholipid syndrome;
12. Hereditary hemophilia.
13. Hemorrhagic vasculitis.
14. Collapse. Characteristics of the concept: types, causes, mechanisms of development. Manifestations, consequences. Principles of therapy.
15. Stress. The concept of stress as a nonspecific reaction of the body to the influence of various extreme stimuli
16. Hypoxia and disruption of energy supply processes as a link in the pathogenesis of coma.
17. Disorder of acid-base balance in coma.
18. Imbalance of ions and liquid as a link in the pathogenesis of coma.
19. Treatment strategy for comatose states.
20. Types of disturbances of consciousness.
21. Cirrhosis of the liver. Etiology, pathogenesis of liver cirrhosis. Signs and symptoms of liver cirrhosis. Stages of liver cirrhosis. Complications. Diagnostics.
22. Portal hypertension and varicose veins of the esophagus and stomach.

Sample tasks for Credit

List of oral questions

1. Classification of septic conditions and diagnostic criteria. General patterns of damage to vital organs in the development of sepsis. Main stages of SIRS development. The role of proinflammatory cytokines in the pathogenesis of SIRS. Clinical criteria of the syndrome.
2. Pathogenesis of perfusion, diffusion, and ventilation disorders in the development of respiratory distress syndrome in adults and newborns. Features of the clinical picture depending on the degree of gas exchange dysfunction and the stage of the process. Pathogenetic ways of correcting respiratory distress syndrome. TELA. Classification. Causes. Pathogenesis. Treatment. Pulmonary obstruction syndrome.
3. DIC syndrome. General characteristics. Etiology, stages. Pathogenesis and clinical manifestations of DIC syndrome. Laboratory diagnosis of stages of DIC syndrome. Principles of therapy. Forecast.
4. Thrombohemorrhagic syndrome. Thrombophilia. Etiology, pathogenesis, development mechanism, manifestations. Diagnostic methods and basic principles of treatment for these conditions. TELA. Classification. Causes. Pathogenesis. Treatment.
5. Blood loss. Pathogenesis and clinical manifestations of acute bleeding. Hemorrhagic shock. Pathogenesis of functional disorders with acute blood loss. Types, causes, pathogenesis and manifestations of chronic bleeding. Features of bleeding from the gastrointestinal tract. Features of massive obstetric hemorrhage. Adaptive mechanisms. Stages of blood loss compensation. Pathogenetic approach to the correction of blood loss. Infusion-transfusion therapy.
6. Epidemiology of hepatorenal syndrome. Classification of hepatorenal syndrome. Etiology. Clinical manifestations. Diagnostic principles. Principles of treatment. Disease prevention. Prognosis for hepatorenal syndrome.
7. Cirrhosis of the liver. Etiology, pathogenesis of liver cirrhosis. Signs and symptoms of liver cirrhosis. Stages of liver cirrhosis. Complications. Diagnostics. Hepatic coma. Etiology, pathogenesis, stages and clinical manifestations of coma. Pathogenetic approach to the correction of lumps.
8. Kidney failure. Etiology. Classification. Pathogenesis of renal failure. Specific and nonspecific symptoms of acute and chronic renal failure. Efferent methods of treatment of renal pathology.
9. The concept of shock. Classification. The main stages of the pathogenesis of shock. General manifestations of shock. Traumatic shock: Hemorrhagic or hypovolemic shock: Septic. Anaphylactic shock. Cardiogenic shock. Stage of compensation and decompensation. Principles of treatment of various types of shock.
10. The main links in the pathogenesis of comatose states. Diagnostic criteria for comatose states. Consequences of coma. Principles of examination and emergency care for patients with impaired consciousness. Key principles of intensive care for comatose states. Etiopathogenesis of multiple organ failure syndrome. Three main mechanisms for the development of MODS: mediator; microcirculatory and related reperfusion; infectious-mechanical route

Questions:

1. What form of pathology did N. develop?
2. What disease is this condition often a complication of?
3. What is the mechanism of development of this complication?
4. What factors can trigger its development?

Task No. 2. Victim A. was taken to a surgical clinic from the scene of a car accident with multiple injuries to the chest, abdomen, legs and loss of a large amount of blood.

Objectively: consciousness is preserved, but the victim is not oriented in time and situation; pale skin, tachycardia, "thread-like" pulse, blood pressure 65/15 mm Hg. A. an operation was performed to ligate the bleeding blood vessels, 1200 ml of donor blood (shelf life from 2 to 17 days) and 2000 ml of blood substitutes were transfused.

In the intensive care unit: A.'s condition is serious; tachycardia, arterial hypotension, and shortness of breath persist; daily diuresis is significantly less than normal; bleeding occurred from small vessels of damaged tissues. Laboratory data indicate a decrease in blood clotting, hypoprothrombinemia, hypofibrinogenemia and thrombocytopenia.

On the second day, symptoms of acute renal failure developed. A.'s death occurred from progressive renal and cardiovascular failure. An autopsy revealed signs of multiple thrombosis of small vessels of internal organs.

Questions:

1. What pathological process developed in A.: a) soon after the injury; b) in the intensive care unit?
2. What is the pathogenesis of the pathological process that developed in the patient in the intensive care unit?
3. What are the mechanisms of development: a) renal failure; b) cardiovascular failure in the patient?
4. Transfusion therapy was ineffective. Make a guess - why?

Task No. 3. Patient Ch., 36 years old, a mining industry worker, was admitted to the clinic with suspected silicosis. Complains of shortness of breath, especially pronounced when walking and physical exertion, persistent cough (dry, sometimes with a small amount of sputum), chest pain. Arterial blood gas composition and spirometry data:

Gasarterial blood composition	
paO ₂	85 mmHg
After a test with voluntary hyperventilation of the lungs	88 mmHg
paCO ₂	40 mmHg
Oxygen capacity	19.2% by volume
SaO ₂	94. 3%
Spirometry	
Vital	4.2 l
FZHOL1	2.6 l
Vital capacity (% of the required value)	92
Tiffno coefficient	? (c al cu lat e)
MOD (% of proper value)	12 4
Additional data	
Breathing rate	19 in 1 minute

Questions:

1. Does Ch. have signs of a disorder in the gas exchange function of the lungs? If yes, please indicate them. Give reasons for your answer.
2. Does Ch. have signs of alveolar ventilation disorder? If yes, then determine its type (obstructive or restrictive).
3. Considering the possibility of developing pneumoconiosis, how do you propose to

assess the diffusion capacity of the lungs?

4. What is your general conclusion about the possible nature of disturbances in the gas exchange function of the external respiration system?

Task No. 4. Patient S., 50 years old, complains of dull aching pain in the epigastrium (more on the right) of a girdling nature, skin itching, shortness of breath with slight physical exertion, interruptions in heart function, and weakness.

From the anamnesis it is known that S. has been abusing alcohol for a long time. 10 years ago, cramping abdominal pain and dyspepsia (bitterness in the mouth, nausea) first appeared.

Subsequently, these symptoms occurred repeatedly after drinking alcohol, but he did not consult a doctor, since during periods of abstinence from alcohol, his health improved. About 6 years ago I began to notice unpleasant sensations in the chest during significant physical activity, which were relieved with rest. 3 years ago, for the first time, an attack of cardiac arrhythmia (paroxysmal form of atrial fibrillation) occurred, and therefore he stopped drinking alcohol. However, recently I began to drink alcohol again, after which I began to worry about “interruptions” in the work of my heart. The real deterioration lasts about a week.

On examination: the condition is of moderate severity. S. restless, irritable, low nutrition. The skin and visible mucous membranes are dry, icteric, and the gums bleed. There are multiple scratches on the skin, “spider veins”, and “liver palms” are visible. NPV 26 per minute. Heart sounds are muffled, the rhythm is irregular. Heart rate is 96 per minute, pulse deficit is 8 per minute. Blood pressure 110/70 mm Hg. Severe swelling of the legs and feet. The abdomen is increased in volume due to ascitic fluid. On the anterior abdominal wall there are dilated veins (“caput medusae”). Appetite is reduced; sleep inversion is noted (insomnia at night and drowsiness during the day). The feces are discolored and the urine is dark.

General blood test unchanged. Urinalysis: urine is dark, foams strongly when shaken. Biochemical blood test: total bilirubin 599 $\mu\text{mol/l}$ (normal 5-21 $\mu\text{mol/l}$), direct bilirubin 462 $\mu\text{mol/l}$ (normal 0-4.5 $\mu\text{mol/l}$), indirect bilirubin 137 $\mu\text{mol/l}$ (normal 2-17 $\mu\text{mol/l}$), ALT 124 U/l (norm 0-45 U/l), AST 267 U/l (norm 0-35 U/l), albumin 29 g/l (norm 35-55 g/l), γ -globulins 26 g/l (normal 10-19 g/l), urea 1.86 mmol/l (normal 1.5-8 mmol/l), cholesterol 5.89 mmol/l (normal 3.0-5, 2 mmol/l), thymol test 8 U/l (normal up to 6 U/l), α -amylase –143 U/l (normal 28-100 U/l). Antibodies to HBs, HCV are negative. Prothrombin index 70% (normal 80-100%). ECG: atrial fibrillation. ECHO-CG shows moderate dilatation of both atria, impaired diastolic function of the left ventricle. Ejection fraction 48%.

Questions:

1. What forms of pathology does S. have?
2. What are the causes and mechanisms of development of each of the forms of pathologies you named?
3. What are the main syndromes and symptoms characteristic of these forms of pathology?

Task No. 5. Victim A. was taken to the clinic 5 hours after the traffic accident. The ambulance doctor discovered multiple rib fractures, bruises of the soft tissues of the pelvis and lower extremities with the formation of extensive hematomas. At the time of admission: confusion, pale skin, thready pulse, blood pressure 60/20 mm Hg, periodic breathing. One day after intensive plasma replacement therapy (3 liters of polyglucin and rheopolyglucin were infused) and 0.5 liters of blood transfusion, blood pressure rose to 110/60 mm Hg.

During the first 24 hours, there was no diuresis. Over the next three days, the condition continued to remain serious. A. complained of severe headache, dizziness, frequent, uncontrollable vomiting, general lethargy, short-term convulsions, development of edema of the subcutaneous tissue, bradycardia, and occasional extrasystole were observed. Diuresis did not exceed 150–250 ml per day, blood pressure was 160/90 mm Hg. Blood test: residual nitrogen 90 mg%, hyperkalemia, hypermagnesemia, hyponatremia and hypochloremia, pH 7.30; Urinalysis: specific gravity 1.040, slight proteinuria and cylindruria, single leukocytes in the field of view, myoglobinuria.

On the 7th day, A. registered an increase in diuresis (up to 2500 ml/day), an improvement in her general condition (vomiting, cramps, headaches stopped), and the severity of edema

decreased.

Urinalysis: specific gravity 1.010–1.012, moderate proteinuria, a large number of granular casts.

Questions:

1. What kind of renal syndrome did A. develop and what are its causes?
2. What are the causes of anuria during the period of shock before blood pressure restoration?
3. Why did diuresis not recover after intensive transfusion therapy?
4. What are the mechanisms of development of symptoms in A. on the 2nd–4th day after the injury?

Task No. 6. Patient K., 31 years old, was taken to the clinic by ambulance. Upon admission: passive, inhibited, apathetic, does not always immediately and adequately answer questions. The tongue is coated. Temperature 36.5 °C. The skin and mucous membranes are jaundiced, there is telangiectasia on the skin of the upper body, and erythema of the palms is detected. The abdomen is enlarged due to ascites fluid, which makes palpation of the liver difficult. Edema of the lower extremities. The border of the left ventricle of the heart is slightly enlarged. Blood pressure 160/95 mm Hg, heart rate 90, pulse rhythmic

Laboratory examination results:

General blood analysis:

Hb - 108 g/l; erythrocytes $4.0 \cdot 10^{12}/l$, leukocytes $4.8 \cdot 10^9/l$; ESR 35 mm per hour.

Biochemical blood test

total bilirubin 7.1 mg%

glucose 80 mg%

CT scan - above normal

urea content - reduced

prothrombin index - decreased

cholinesterase activity - reduced

Australian Ag - not detected

Questions:

1. What are the mechanisms of development of these changes in the skin vessels and persistent erythema of the palms in K.? What other symptoms are caused by this same effect?
2. What are the causes of portal hypertension and ascites? What is the role of ascites in secondary disorders of body functions?
3. Does K. have signs of liver failure? If yes, what is the mechanism of their development?
4. Taking into account the clinical and laboratory data, what forms of pathology can we reasonably assume to be developing in K.: diabetes mellitus? Acute hepatitis? Cirrhosis of the liver? Why?
5. What additional data do you need to accurately answer the last two questions?