ADVANTAGES OF MODERN CLINICAL DIAGNOSTIC METHODS FOR CARDIOVASCULAR DISEASES

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Introduction

Cor pulmonale results from pathology of the lungs or their vessels; it does not include left ventricular (LV) failure, congenital heart defects (eg, ventricular septal defect), or secondary right ventricular (RV) dilatation due to acquired valvular pathology. Cor pulmonale is usually a chronic condition, but it can be acute and reversible. Primary pulmonary hypertension (i.e., not caused by lung or heart disease) is discussed elsewhere.

Research methods and materials: Pulmonary hypertension increases RV load, which leads to a cascade of events that occur in the left ventricular heart rate, including increased end-diastolic and central venous pressures, ventricular hypertrophy, and dilation. RV load may be increased by increased blood viscosity due to hypoxia-induced polycythemia. Sometimes RV failure leads to LV pathology, with the interventricular septum bulging into the LV cavity, preventing LV filling, thereby creating its diastolic dysfunction. The heart should be visualized with echocardiography or radionuclide scanning to assess LV and RV function. Echocardiography can help assess RV systolic pressure, but its feasibility is often limited in patients with pulmonary disease; cardiac MRI may be useful in some patients to assess the chambers of the heart and their function. Right heart catheterization may be necessary to confirm the diagnosis.

Research results:

Pulmonary heart disease is difficult to treat; it is aimed at the cause (see table of causes of heart failure), in particular, to alleviate or alleviate hypoxia. Early diagnosis and treatment are important, because Later structural changes in the lungs become irreversible.

Diuretics may be prescribed in the presence of peripheral edema, but they are effective only in the presence of LV failure and pulmonary fluid overload. Diuretics should be used with caution, as even a small reduction in preload often worsens cor pulmonale. Pulmonary vasodilators (eg, hydralazine, calcium channel blockers, nitric oxide, prostacyclin, phosphodiesterase inhibitors),

although effective in primary pulmonary hypertension, are ineffective in cor pulmonale. Bosentan (an endothelin receptor blocker) may be used in patients with primary pulmonary hypertension, although its use in cor pulmonale has not been adequately studied. Digoxin is effective only in the presence of concomitant LV dysfunction. This drug should be used with caution, as patients with COPD are particularly sensitive to its effects.

Phlebotomy has been suggested as necessary in hypoxic cor pulmonale, but the effect of reducing blood viscosity, except in cases of significant polycythemia, is unlikely to counteract the adverse effects of reduced oxygen carrying capacity. Long-term use of anticoagulants in patients with chronic cor pulmonale reduces the risk of venous thromboembolism.

Cor pulmonale (heart lung) is a lung disease characterized by right ventricular enlargement and subsequent failure, secondary to pulmonary artery hypertension.

Cor pulmonale is usually asymptomatic, but common physical findings include a left parasternal systolic rise, a loud pulmonary component of S2, functional tricuspid regurgitation, and pulmonary bruit; later, jugular vein edema, hepatomegaly, and lower extremity edema.

Diagnosis usually requires echocardiography or radionuclide imaging, and in some cases, right heart catheterization.

It is important to identify and treat the causes of the disease early, before systemic changes in the heart become irreversible.

Although patients may have significant peripheral edema, diuretic therapy is not indicated and may be harmful; even a modest reduction in preload often worsens cor pulmonale (pulmonary heart disease).

Heart failure (HF) is a syndrome of dysfunction of the ventricles of the heart. Left ventricular failure leads to the development of shortness of breath and rapid fatigue, while right ventricular failure leads to peripheral edema and fluid accumulation in the abdominal cavity. Both ventricles or each ventricle may be involved in the process. The diagnosis is made clinically and confirmed by chest radiography, echocardiography, and plasma natriuretic peptide levels. Treatment includes patient education, diuretics, angiotensin-converting enzyme (ACE) inhibitors, angiotensin II receptor blockers, beta-blockers, aldosterone antagonists, sodium-glucose cotransporter type 2 inhibitors, neprilysin inhibitors, sinus node inhibitors, or other specialized H/implantable pacemakers. Afterload is the force of resistance to contraction of myocardial fibers at the beginning of systole. It is determined by the pressure in the LV, the volume and thickness of the wall at the time of aortic valve opening. From a clinical point of view, the systemic systolic

blood pressure measured at or immediately after aortic valve opening is related to the peak systolic stress of the ventricular wall and approximates the afterload value.

The Frank-Starling law describes the relationship between preload and cardiac output. Typically, the systolic work (contractile capacity) of the heart, expressed as stroke volume or CO2, is proportional to preload within the normal physiologic range (see Frank-Starling law diagram). Contractility is difficult to measure clinically (because it requires cardiac catheterization with pressure-volume analysis), but is best reflected by the ejection fraction (EF), which is the ratio of stroke volume after ejection during contraction to end-diastolic volume (stroke volume/end-diastolic). Noninvasive techniques such as echocardiography, nuclear tomography, or MRI are usually used to adequately assess EF.

Force-frequency relationship refers to the phenomenon whereby repeated stimulation of a muscle over a given frequency range results in an increase in the force of contraction. Normal cardiac muscle at a normal heart rate exhibits a positive force-frequency relationship, so that higher heart rates result in more forceful contractions (and correspond to greater substrate demand). In some types of heart failure, the force-frequency relationship may be negative, so that myocardial contractility decreases when the heart rate exceeds a certain level.

Cardiac reserve is the ability of the heart to produce more than its resting level in response to emotional or physical stress. The body can increase oxygen consumption from 250 to ≥ 1500 ml/min at maximum exertion. Developmental mechanisms include:

Increased tissue oxygen extraction (the difference between the oxygen content in arterial blood and mixed venous or pulmonary artery blood)

In well-trained young adults, during maximal exercise, the heart rate can increase from 55–70 beats per minute (at rest) to 180 beats per minute, and CO 6 can increase to ≥ 25 l/min or more. At rest, arterial blood contains approximately 18 ml of oxygen per dL of blood, and mixed venous or pulmonary arterial blood contains approximately 14 ml/dL. Thus, oxygen extraction is approximately 4 ml/dL. As demand increases, oxygen extraction can increase to 12–14 ml/dL. This mechanism also helps to compensate for the decreased tissue blood flow in heart failure.

Conclusion : In heart failure associated with left ventricular dysfunction, CO is reduced and pulmonary venous pressure is increased. Since pulmonary capillary pressure exceeds the oncotic pressure of plasma proteins (approximately 24 mmHg), the liquid part of the blood flows from the capillaries into the interstitial space and alveoli, reducing lung function and increasing respiratory rate. In compensation, lymphatic drainage of the lungs increases, but this does not compensate for the increase in fluid volume in the lungs. A large accumulation of fluid in the

alveoli (pulmonary edema) significantly disrupts the ventilation-perfusion (V / Q) relationship: deoxygenated pulmonary arterial blood passes through poorly ventilated alveoli, which leads to a decrease in the partial pressure of oxygen (PaO2) in arterial blood and leads to the development of dysplasia. However, shortness of breath may occur before the V / P disorder, possibly due to an increase in pulmonary venous pressure and an increase in the work of breathing; The exact mechanism of this phenomenon is unclear.

REFERENCES

1. Andryev S. et al. Experience with the use of memantine in the treatment of cognitive disorders //Science and innovation. – 2023. – T. 2. – №. D11. – C. 282-288.
2. Antsiborov S. et al. Association of dopaminergic receptors of peripheral blood lymphocytes with a risk of developing antipsychotic extrapyramidal diseases //Science and innovation. – 2023. – T. 2. – №. D11. – C. 29-35.
3. Asanova R. et al. Features of the treatment of patients with mental disorders and cardiovascular pathology //Science and innovation. – 2023. – T. 2. – №. D12. – C. 545-550.
4. Begbudiyev M. et al. Integration of psychiatric care into primary care //Science and innovation. – 2023. – T. 2. – №. D12. – C. 551-557.
5. Bo'Riyev B. et al. Features of clinical and psychopathological examination of young children //Science and innovation. – 2023. – T. 2. – №. D12. – C. 558-563.
6. Borisova Y. et al. Concomitant mental disorders and social functioning of adults with high-functioning autism/asperger syndrome //Science and innovation. – 2023. – T. 2. – №. D11. – C. 36-41.
7. Ivanovich U. A. et al. Efficacy and tolerance of pharmacotherapy with antidepressants in non-psychotic depressions in combination with chronic brain ischemia //Science and Innovation. – 2023. – T. 2. – №. 12. – C. 409-414.
8. Nikolaevich R. A. et al. Comparative effectiveness of treatment of somatoform diseases in psychotherapeutic practice //Science and Innovation. – 2023. – T. 2. – №. 12. – C. 898-903.
9. Novikov A. et al. Alcohol dependence and manifestation of autoagressive behavior in patients of different types //Science and innovation. – 2023. – T. 2. – №. D11. – C. 413-419.

10. Pachulia Y. et al. Assessment of the effect of psychopathic disorders on the dynamics of withdrawal syndrome in synthetic cannabinoid addiction //Science and innovation. – 2023. – T. 2. – №. D12. – C. 240-244.
11. Pachulia Y. et al. Neurobiological indicators of clinical status and prognosis of therapeutic response in patients with paroxysmal schizophrenia //Science and innovation. – 2023. – T. 2. – №. D12. – C. 385-391.
12. Pogosov A. et al. Multidisciplinary approach to the rehabilitation of patients with somatized personality development //Science and innovation. – 2023. – T. 2. – №. D12. – C. 245-251.
13. Pogosov A. et al. Rational choice of pharmacotherapy for senile dementia //Science and innovation. – 2023. – T. 2. – №. D12. – C. 230-235.
14. Pogosov S. et al. Gnostic disorders and their compensation in neuropsychological syndrome of vascular cognitive disorders in old age //Science and innovation. – 2023. – T. 2. – №. D12. – C. 258-264.
15. Pogosov S. et al. Prevention of adolescent drug abuse and prevention of yatrogenia during prophylaxis //Science and innovation. – 2023. – T. 2. – №. D12. – C. 392-397.
16. Pogosov S. et al. Psychogenetic properties of drug patients as risk factors for the formation of addiction //Science and innovation. – 2023. – T. 2. – №. D12. – C. 186-191.
17. Prostyakova N. et al. Changes in the postpsychotic period after acute polymorphic disorder //Science and innovation. – 2023. – T. 2. – №. D12. – C. 356-360.
18. Prostyakova N. et al. Issues of professional ethics in the treatment and management of patients with late dementia //Science and innovation. – 2023. – T. 2. – №. D12. – C. 158-165.
19. Prostyakova N. et al. Sadness and loss reactions as a risk of forming a relationship together //Science and innovation. – 2023. – T. 2. – №. D12. – C. 252-257.
20. Prostyakova N. et al. Strategy for early diagnosis with cardiovascular diseaseisomatized mental disorders //Science and innovation. – 2023. – T. 2. – №. D12. – C. 166-172.
21. Rotanov A. et al. Comparative effectiveness of treatment of somatoform diseases in psychotherapeutic practice //Science and innovation. – 2023. – T. 2. – №. D12. – C. 267-272.
22. Rotanov A. et al. Diagnosis of depressive and suicidal spectrum disorders in students of a secondary special education institution //Science and innovation. – 2023. – T. 2. – №. D11. – C. 309-315.

23. Rotanov A. et al. Elderly epilepsy: neurophysiological aspects of non-psychotic mental disorders //Science and innovation. – 2023. – T. 2. – №. D12. – C. 192-197.
24. Rotanov A. et al. Social, socio-cultural and behavioral risk factors for the spread of hiv infection //Science and innovation. – 2023. – T. 2. – №. D11. – C. 49-55.
25. Rotanov A. et al. Suicide and epidemiology and risk factors in oncological diseases //Science and innovation. – 2023. – T. 2. – №. D12. – C. 398-403.
26. Sedenkov V. et al. Clinical and socio-demographic characteristics of elderly patients with suicide attempts //Science and innovation. – 2023. – T. 2. – №. D12. – C. 273-277.
27. Sedenkov V. et al. Modern methods of diagnosing depressive disorders in neurotic and affective disorders //Science and innovation. – 2023. – T. 2. – №. D12. – C. 361-366.
28. Sedenkova M. et al. Basic principles of organizing gerontopsychiatric assistance and their advantages //Science and innovation. – 2023. – T. 2. – №. D11. – C. 63-69.
29. Sedenkova M. et al. Features of primary and secondary cognitive functions characteristic of dementia with delirium //Science and innovation. – 2023. – T. 2. – №. D11. – C. 56-62.
30. Sedenkova M. et al. The possibility of predicting the time of formation and development of alcohol dependence: the role of genetic risk, family weight and its level //Science and innovation. – 2023. – T. 2. – №. D12. – C. 173-178.
31. Shamilov V. et al. Disorders of decision-making in the case of depression: clinical evaluation and correlation with eeg indicators //Science and innovation. – 2023. – T. 2. – №. D12. – C. 198-204.
32. Solovyova Y. et al. Protective-adaptive complexes with codependency //Science and innovation. – 2023. – T. 2. – №. D11. – C. 70-75.
33. Solovyova Y. et al. Suicide prevention in adolescents with mental disorders //Science and innovation. – 2023. – T. 2. – №. D11. – C. 303-308.
34. Solovyova Y. et al. The relevance of psychotic disorders in the acute period of a stroke //Science and innovation. – 2023. – T. 2. – №. D12. – C. 212-217.
35. Spirkina M. et al. Integrated approach to correcting neurocognitive defects in schizophrenia //Science and innovation. – 2023. – T. 2. – №. D11. – C. 76-81.