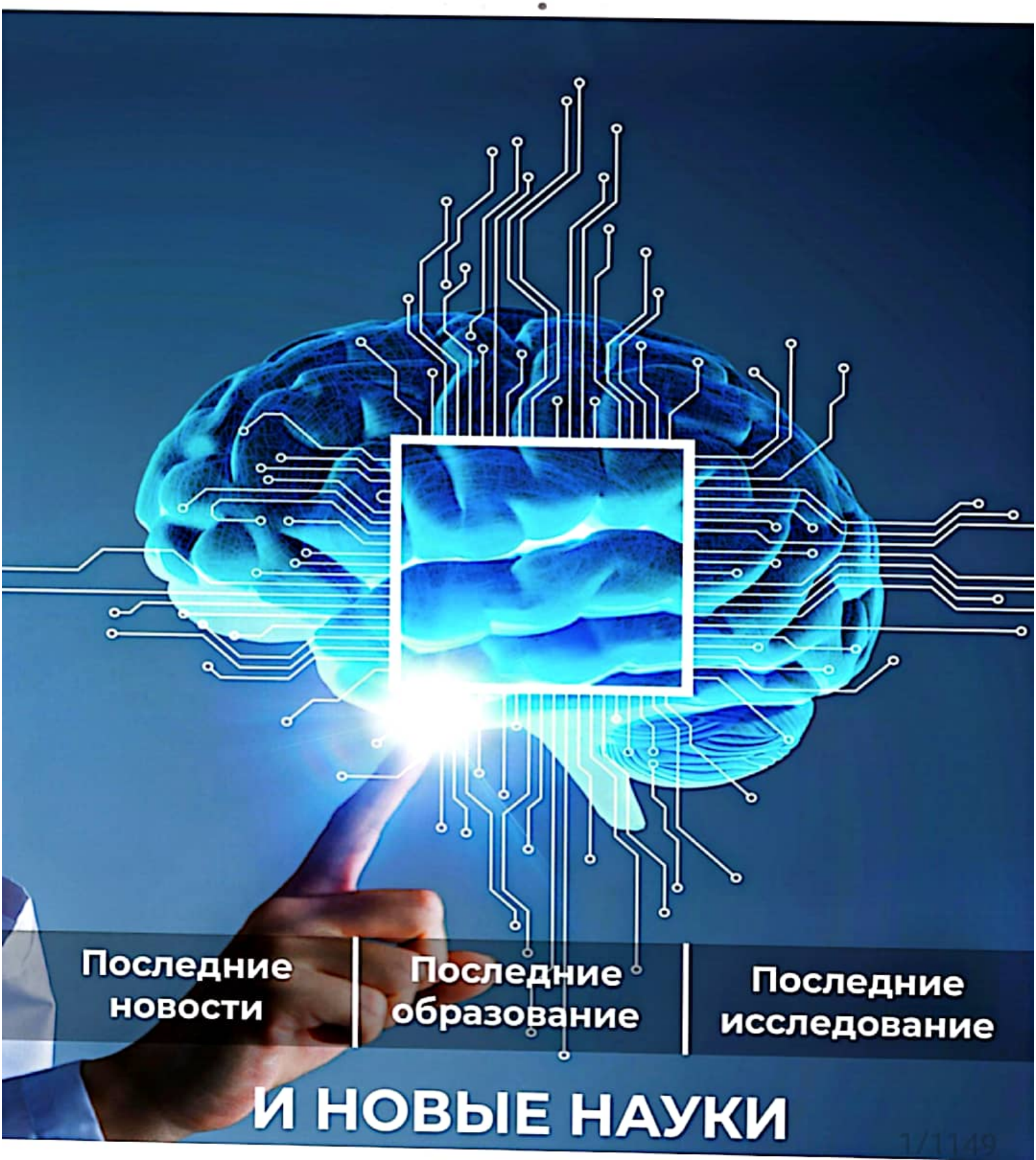
НАУЧНЫЙ ИМПУЛЬС
ЦЕНТР НАУЧНОЙ ПОДДЕРЖКИ

МЕЖДУНАРОДНЫЙ СОВРЕМЕННЫЙ НАУЧНО-ПРАКТИЧЕСКИЙ ЖУРНАЛ

НАУЧНЫЙ ИМПУЛЬС



Международный современный научно-практический журнал

Научный импульс

№ 5 (100)
Декабрь 2022 г.

Часть 2

Издается с августа 2022 года

Москва 2022

***Научный импульс:** научный журнал. – № 5(100). Часть 2. М., Изд. «МЦНО», 2022.*

Журнал **«Научный импульс»** освещает сферу духовно-просветительского мышления человека, общественно-политическую жизнь человека, институты гражданского общества, глобальные проблемы, проблемы образования, новые технологии, производимые сегодня, реформирование системы образования и публикуются научные статьи, посвященные открытому научно-популярному анализу.

СОДЕРЖАНИЕ:

CMODERN METHODS OF ANTICOAGULANT THERAPY

Mukhamedova N. Kh
Tursunov D.X
Alimov S. M
Tursunova O.D
Akhmatova K.A
Niyozova Sh.S
Sultonova D.Sh
Tashkent Medical Academy

Abstract: *Anticoagulant therapy (Greek anti - against, Latin coagulatio - clotting) -is a treatment that promotes slower blood clotting and prevents the formation of blood clots.The resulting blood clots are tightly attached to the wall of the veins or arteries. However high blood flow velocity, fever in infectious diseases, increase in blood pressure, physical overstrain become causes of thrombus rupture in humans. It happens suddenly and often the outcome of such a state largely depends on the speed of providing a person medical care.*

Keywords: *Anticoagulant therapy, heart defects, thrombin, heparin has a hypolipidemic effect.*

Anticoagulant therapy is one of the most popular approaches in practical medicine. Anticoagulants (AK; antithrombins, antithromboplastins) are highly effective drugs that are indicated in a wide variety of clinical situations, from emergency medical interventions to long-term maintenance and preventive therapy. Their effect is manifested in improving the rheological properties of blood and antithrombotic action, so the goal of anticoagulant therapy is to limit the onset of thrombosis and prevent thromboembolic complications. If thromboembolic complications have already occurred, then this therapy contributes to their elimination, reduces the number of relapses of this complication. In recent years, indications for anticoagulant therapy have been rapidly expanding. To a large extent, this is due to the results of international multicenter studies, the emergence of new acs and the creation of safer and more reliable test systems for monitoring the level of hypocoagulation. AK is the most important component of the basic therapy of acute coronary syndrome, acute cerebrovascular disorders, pulmonary embolism (PE), deep vein thrombosis of the lower extremities, heart defects, atrial fibrillation and other cardiovascular diseases. It is widely used in general surgery for the prevention of thrombosis in the postoperative period, as well as in hematology and nephrology for use with extracorporeal detoxification devices and automatic plasmapheresis, for the preparation of human blood components. INNOVATIVE APPROACHES TO SOLVING ACTUAL PROBLEMS OF MODERN BIOCHEMISTRY 2021 44 Classification of AK. AK is divided into 2

groups: direct and indirect Direct anticoagulants are fast-acting drugs (sodium heparin, надропаринсalcium nadroparin, эноксапаринsodium enoxaparin, etc.) that are effective in in vitro and in in vivo. They have a direct effect on the activity of circulating clotting factors in the blood. A common feature of currently used direct AA is their ability to inhibit the enzymatic activity of thrombin (or factor IIa), which plays a key role in thrombosis. Therefore, according to the main mechanism of antithrombotic action, all direct AK can be considered as thrombin inhibitors. There are two main groups of direct AK depending on how they inhibit the activity of thrombin. The first group includes heparin, its derivatives and some other glycosaminoglycans (heparan and dermatan), which can inhibit the activity of thrombin only in the presence of plasma cofactors, and especially antithrombin III. These are so-called antithrombin III-dependent thrombin inhibitors, or indirect thrombin inhibitors. Another group of direct AK consists of hirudin, its synthetic analogues (girugen, hirulog, etc.) and some oligopeptides (argatroban, rivaroxaban, dabigatran), which neutralize thrombin by directly binding to its active center. This group of direct AK is called antithrombin III independent thrombin inhibitors, or direct (selective, specific) thrombin inhibitors. Heparin was discovered by American medical student J. McLean in 1916. It is a glycosaminoglycanconsisting of several chains of sulfated mucopolysaccharides of various lengths and molecular weights. Heparin is produced mainly by mast cells (mastocytes) located in all tissues of the body: its highest concentration is found in the lungs, intestines and liver. There are various heparin salts (sodium, calcium, potassium, magnesium, barium). The most widely used sodium and calcium salts of heparin. Due to the fact that commercial preparations of conventional heparin differ in origin, degree of purification and water content, their doses should be expressed not in milligrams, but in international units (units). The mechanism of anticoagulant action of heparin is well studied and consists in inhibiting the activity of thrombin, which catalyzes the conversion of fibrinogen to fibrin and some other reactions in the hemostatic system. In addition to inactivating thrombin and other serine proteases, heparin has a hypolipidemic effect, inhibits the proliferation and migration of endothelial and smooth muscle cells of the vascular wall. By suppressing the proliferation and migration of smooth muscle cells, heparin can potentially slow the progression of atherosclerotic lesions, i.e., with prolonged use, it can have an anti-atherogenic effect. The effect of heparin on platelet aggregation is ambiguous. On the one hand, by inactivating thrombin, it can reduce or prevent platelet aggregation. On the other hand, heparin can enhance platelet aggregation caused by other inducers (in addition to thrombin). Oral anticoagulants—antivitamins K (UAC—AVK) are the cornerstone of modern antithrombotic therapy. Evidence based studies have shown that UAC-AVK is highly effective in preventing and treating venous thromboembolism, preventing systemic embolism associated with atrial fibrillation or artificial heart valves, and preventing stroke and recurrent myocardial infarction. Warfarin therapy during its implementation prevents the possibility of recurrent venous thrombosis by 90-95%, reduces the risk of stroke in non-valvular atrial fibrillation by 68%; the appointment of UAC-AVC after mechanical valve

transplantation reduces the occurrence of systemic thromboembolism by 75%; in patients with peripheral artery diseases and in persons with significant risk factors, oral anticoagulants are effective for prevention of acute myocardial infarction. The use of Warfarin in an adequate dose can reduce not only the frequency of stroke, but also the severity of its clinical manifestations and mortality. According to Gage B. F. and INNOVATIVE APPROACHES TO SOLVING CURRENT PROBLEMS OF MODERN BIOCHEMISTRY 2021 45 etal., the treatment of patients with atrial fibrillation (AF) UAC-AVC can prevent about 40,000 strokes per year and save $ 600 million. Warfarin It is the most commonly used anticoagulant in this group of drugs (2 million prescriptions per year in the United States). Nevertheless, therapy aimed at preventing the above-mentioned thrombotic complications is far from optimal. Despite the fact that acetylsalicylic acid (ASA) is less effective than Warfarin in preventing thrombosis, ASA therapy continues to occupy a leading position, and many patients do not receive any therapy at all. According to Carlson J. etal., 78% of patients with stroke and transient ischemic attack and previous AF did not receive Warfarin, and only 40% of patients were prescribed UAC—AVC at discharge for secondary stroke prevention. The reasons for inadequate use of Warfarin include, first of all, the fear of serious bleeding, the need for constant laboratory monitoring, doctors ' ignorance of the specifics of selecting and regulating the dose of the drug, interaction with concomitant drug therapy and food.

REFERENCES:

1. Schuster V, Hügle B, Tefs K. Plasminogen deficiency. J Thromb Haemost. 2007;5:2315–22.

2. Martinelli I, Bucciarelli P, Mannucci PM. Thrombotic risk factors: Basic pathophysiology. Crit Care Med. 2010;38:S3–9. 3. Balabolkin MI. Diabetology. Moscow; 2000. 672s.

3. Polderman HK. Hypothermia and coagulation. Crit Care. 2012;16:A20. 5. Belyakova N.A., Mikhailova D.G., Tsvetkova I.G. Adaptation reactions, arteriolar blood flow and factors influencing them in patients with type 2 diabetes mellitus // Siberian Medical Journal (Irkutsk). - 2010. - T. 115. No. 8. – P.61-64