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MODERN VIEW ON THE MORPHOFUNCTION OF THE HEART AND INTRACARDIAC NERVOUS STRUCTURES

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Abstract

Introduction. Over the past 10 years, there has been a high mortality rate from cardiovascular system pathologies associated with the autonomic nervous system. Mortality from heart pathology and its complications is mainly observed among the working-age population. At the same time, the relationship between the cardiac conduction system and the autonomic nervous system, as well as their role in regulating the stable functioning of the heart, occupy a significant place in modern scientific hypotheses proposed by a number of researchers. In this regard, research in this area remains highly relevant.

Aim: To analyze the scientific literature on the morphofunctional basis of the regulation of cardiac activity and intracardiac nervous structures.

Search Strategy: The search for articles was conducted using the databases of PubMed, Google Scholar, Embase, and the Cochrane Library, as well as data from domestic scientific publications. The search covered a 10-year period (2014–2024). A total of 71 scientific publications were analyzed.

Results. As a result of the study, explanations are provided regarding the relevance of morphological research on the cardiovascular system, along with modern scientific conclusions on the morphological function of the heart's nervous structures.

Conclusion. The significance of morphological structures, including intracardiac nerve plexuses, nodes, and fibers, which play a crucial role in the stable functioning of the heart, has been established.

Keywords: *cardiovascular system, structure of the heart, nervous structures of the heart, autonomous regulation, heart failure.*

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Резюме

СОВРЕМЕННЫЙ ВЗГЛЯД НА МОРФОФУНКЦИЮ СЕРДЦА И ВНУТРИСЕРДЕЧНЫХ НЕРВНЫХ СТРУКТУР

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Введение. За последние 10 лет регистрируется высокая смертность от патологий сердечно-сосудистой системы, связанных с вегетативной нервной системой. Смертность от патологии сердца и ее осложнений в основном отмечается среди лиц трудоспособного возраста. При этом взаимосвязь между проводящей системой сердца и

автономной нервной системой и их роль в регулировании устойчивой деятельности сердца занимают большое место в современных научных гипотезах целого ряда ученых. В этой связи актуальны исследования в этом направлении.

Цель исследования. Анализ научной литературы по вопросу морфофункционального обоснования регуляции сердечной деятельности и внутрисердечных нервных структур.

Стратегия поиска. Поиск статей проведен с использованием баз данных поисковых систем PubMed, Google Scholar, Embase, Cochrane Library, а также данных отечественных научных изданий. Глубина поиска 10 лет (2014 - 2024 гг.). Всего было проанализировано 71 научных публикаций.

Результаты. В результате анализа литературы обобщены данные об актуальности морфологического исследования сердечно-сосудистой системы, современных научных выводов по морфо-функции нервных структур сердца.

Заключение. Определено значение морфологических структур и внутрисердечных нервных сплетений, узлов и волокон, занимающих важное место в стабильном функционировании сердца.

Ключевые слова: сердечно-сосудистая система, строение сердца, нервные структуры сердца, автономная регуляция, сердечная недостаточность.

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Түйіндеме

ЖҮРЕК ЖӘНЕ ЖҮРЕКІШІЛІК ЖҮЙКЕ ҚҰРЫЛЫМДАРЫНЫҢ МОРФОФУНКЦИЯСЫ ТУРАЛЫ ЗАМАНАУИ КӨЗҚАРАС

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Кіріспе. Соңғы 10 жылда вегетативті жүйке жүйесімен байланысты жүрек – тамырлар жүйесінің патологияларынан болатын өлім-жітімнің жоғары деңгейі тіркелуде. Жүрек патологияларынан және оның асқынуларынан болатын өлім-жітім негізінен еңбекке қабілетті жастағы тұлғалардың арасында байқалады. Бұл ретте, жүректің өткізгіш жүйесі мен автономды жүйке жүйесі арасындағы өзара қатынас және жүректің тұрақты қызметінің реттелуіндегі рөлі бірқатар ғалымдардың заманауи ғылыми гипотезаларында үлкен орын алады. Осыған байланысты осы бағыттағы зерттеулер өзекті.

Зерттеу мақсаты. Жүрек қызметін реттеу және жүрек ішілік жүйке құрылымдарының морфофункционалдық негізделуі сұрақтары бойынша ғылыми әдебиеттерді талдау.

Іздеу стратегиясы. Мақалаларды іздеу PubMed, Google Scholar, Embase, Cochrane Library мәліметтер қоры бойынша, сонымен қатар отандық ғылыми басылымдар мәліметтерін қолдана отырып жүргізілді. Іздеу жылдықты соңғы 10 жыл (2014 – 2024 жж.). Барлығы 71 ғылыми жарияланымдарға талдау жасалды.

Нәтижелер. Зерттеу нәтижесінде жүрек – тамырлар жүйесін морфологиялық тұрғыдан зерттеудің өзектілігі, жүректің жүйке құрылымдарының морфо-функциясы бойынша заманауи ғылыми тұжырымдар туралы түсініктер берілді.

Қорытынды. Жүректің тұрақты қызмет атқаруында маңызды орын алатын морфологиялық құрылымдар және жүрек ішілік жүйке өрімдері, түйіндері мен талшықтардың маңызы анықталды.

Түйінді сөздер: жүрек – тамырлар жүйесі, жүрек құрылысы, жүректің жүйке құрылымдары, автономды реттелу, жүрек жетіспеушілігі.

Дәйексөз үшін:

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Introduction

The regulation of the cardiovascular system, particularly the functioning of the heart as the central organ of the vascular system, its conduction system development, and characteristics, remains a critical area of research in modern morphology. While there is ample literature on the structural features of the heart wall, angiogenesis, and vascular-related changes, these topics continue to occupy a leading position in contemporary research.

In this regard, the interaction between the heart's conduction system and the autonomic nervous system, as well as their role in regulating the heart's steady functioning, is emphasized in numerous modern scientific hypotheses. Despite centuries of studying the anatomical features of the autonomic nervous system, the autonomic nature of the enteral division (related to the digestive system), distinct from its two main divisions, is often overlooked. Similarly, within the heart, alongside extrinsic neural structures, the morphological diversity of intrinsic neural structures, which perform autonomous functions akin to the enteral division, has been highlighted.

Advances in neuroanatomical, neurohistological, and immunohistochemical research methodologies have accelerated investigations into the heart's neural structures. Current studies increasingly focus on the morphology of extracardiac nerve plexuses and fibers in comparison to intramural neural structures, yielding valuable insights into the functional regulation of the heart and its unique features.

Thus, understanding the morphofunctional characteristics and variability of the heart's autonomous (intracardiac neural structures) nervous structures is essential for comprehending cardiac function and associated disorders [4, 6, 11, 13, 15, 17, 28].

Among cardiac pathologies associated with neural structures, sudden cardiac death (SCD) remains a significant concern due to its high incidence rate. This condition poses a severe and unpredictable challenge in the field of general medicine, as it often occurs in individuals with no prior history of cardiac disease or detectable pathology. The absence of any warning signs or preceding symptoms complicates its prediction and prevention. According to several researchers, sudden cardiac death is closely linked to the neural structures of the heart.

However, studies by *Dainius H. Pauza and Dmitrij Kvitko* suggest that the coordinating neural structures within the heart, though substantial, do not constitute a fully autonomous system akin to the enteral division. Instead, these structures are part of the conduction system, which is widely recognized for regulating the heart's rhythmic activity. Despite this knowledge, the detailed morphology of the tens of thousands of intrinsic neurons within the heart and their specific characteristics remains under-researched. This gap in understanding makes it challenging to provide comprehensive information about the heart's neural structures and their functional features. Moreover, changes in intracardiac neural structures are believed to underlie various cardiac arrhythmias, leading to dilated cardiomyopathy and heart failure. These findings underscore the importance of further research into the role of neural structures in cardiac pathophysiology [25, 29, 38, 41, 44, 67].

Thus, scientific studies aimed at investigating the features of cardiac neural structures remain highly relevant today. According to data from the World Health Organization and scientific literature, cardiovascular diseases have the highest mortality rate globally. Among these, pathologies associated with disruptions in the heart's neural structures are particularly challenging to diagnose and are significant contributors to sudden cardiac death. Consequently, studying the morphology of neural structures, particularly intracardiac neural networks, has become one of the priority areas in contemporary research.

Aim: To analyze the scientific literature on the morphofunctional basis of the regulation of cardiac activity and intracardiac nervous structures.

Search Strategy

The literature review for this article was conducted using databases such as PubMed, Google Scholar, Embase, and Cochrane Library, along with references from domestic scientific publications. The review focused on scientific articles published over the past decade (2014–2024). It also included randomized studies, systematic reviews, and meta-analyses related to the topic.

To comprehensively explore the relevance of the topic, a range of keywords and terms were used: cardiac function regulation, myocardial cellular composition, cardiomyocyte types, extracardiac neural structures, intracardiac neural structures, autonomic regulation, cardiac ganglia, cardiac nerve plexuses, adrenergic and cholinergic ganglia, and immunohistochemical studies of neural structures. In total, 71 scientific publications were analyzed during the literature review, providing a detailed characterization of the topic's relevance.

Results and Discussion

A Modern Perspective on the Morphology of Intracardiac Neural Structures

Research on the neural regulation of cardiac function ranks among the most pressing issues in contemporary morphophysiology and practical cardiology. Given the heart's primary role as a pump, its consistent operation ensures stable and efficient hemodynamics. In this context, the maintenance of steady cardiac activity is largely influenced by extracardiac neural regulation, which includes the sympathetic and parasympathetic nervous systems. These systems function as a unified neuroendocrine network, providing stimulatory and inhibitory control mechanisms. However, in addition to external neural structures, the role of intracardiac neural structures is particularly significant in maintaining consistent heart function. These structures are increasingly being recognized as a distinct system. The morphology of intracardiac neural structures and their distribution underscore their unique role in cardiac regulation [39, 60, 68].

Further exploration into the intricate organization and functionality of intracardiac neural elements highlights their importance in both physiological and pathological states of the heart. Understanding their interaction with extracardiac systems is crucial for advancing cardiac research and developing targeted interventions for various cardiac disorders.

Morphological and Functional Features of Intracardiac Neural Structures

Literature data and several research studies have confirmed that sympathetic fibers within the myocardium are located along the coronary arteries. Additionally, afferent and efferent sympathetic fibers are positioned within the superficial layer of the epicardium, extending deeply to reach the endocardium. In contrast, the parasympathetic fibers, particularly those associated with the vagus nerve, are primarily intramural or sub-endocardial and ascend to the sub-epicardial region near the atrioventricular groove.

The parasympathetic division plays a significant role in regulating cardiac function by modulating norepinephrine levels. This regulation occurs through the suppression of cAMP-mediated phosphorylation of cardiac proteins such as phospholamban, thereby altering sympathetic activity. Research also reveals that neuropeptides released from sympathetic nerve endings can suppress the effects of the vagus nerve on the heart. However, it is incorrect to state that the heart and its vascular functions are entirely dependent on the ganglionic (autonomic) nervous system, as the cardiovascular system possesses its intrinsic regulatory mechanisms.

From a morphological perspective, the peripheral autonomic innervation of the heart is highly complex and distinctive. The heart, like other somatic organs, is equipped with segmental–peripheral and cranial-level interactions through the peripheral sympathetic and parasympathetic divisions. These interactions facilitate ganglionic innervation, enabling organs to respond rapidly to changes in the body's vital activities. This system also supports the autonomous regulation of individual organs through specific morphological structures, influencing inter-organ autonomic interactions.

This morphofunctional interdependence highlights the complexity of cardiac regulation and its intricate relationship with other organs. The vegetative provision of the heart's contractile function is closely tied to the peripheral autonomous regulation of pacemaker activity within the sinoatrial node, supported by intracardiac neural structures (ICNS). Despite its integration with the heart's overall physiological role, the sinoatrial node remains the primary pacemaker under both normal and pathological conditions.

To fulfill this role, the sinoatrial node is capable of receiving efferent information at both intra- and extracardiac levels. Hemodynamic participation in the body's adaptive processes to environmental changes is achieved through a complex regulatory system that governs cardiac activity. This regulation occurs not only at the extracardiac level but also through the intrinsic regulatory complex located within the heart's morphologically unique and architecturally complex structures [20, 37, 59, 69].

The self-regulation of cardiac function involves a morphologically complex system facilitated by several structures. According to the scientific literature, these structures include intracellular and intercellular processes, with the most critical being the intracardiac neural structures (ICNS). Some researchers refer to the ICNS as the metasympathetic division in their studies.

The complexity of cardiac function is directly related to the morpho-functional properties of the myocardium. The myocardium's primary mass comprises working cardiomyocytes (contractile), followed by sinoatrial (pacemaker), transitional, conductive, and secretory cardiomyocytes. The self-regulation of cardiac activity is significantly influenced by metabolic processes within these cardiomyocytes and the transfer of excitation between cells through transitional intercellular processes. This interaction ensures the myocardium's complex and uninterrupted contractions [33, 35, 43].

Among the key contributors to cardiac self-regulation are the intramural neural formations located within the heart's layers (ICNS). These structures include autonomic neurons, preganglionic, and postganglionic fibers found within the neural ganglia of the heart. While these structures are part of the peripheral division of the autonomic nervous system and function as autonomous structural-functional components within the heart, their precise roles remain a subject for further research.

The lack of consensus regarding the morphofunctional characteristics of these structures complicates understanding the development and function of the cardiac neural network, ganglionic apparatus, and neuromuscular connections. These characteristics also depend on the unique features of each organism. Morphologically, the ICNS begins with sensory receptor structures located in the heart tissue and its segments. These receptors are diverse and situated intramurally, receiving signals about the heart's condition and activity, converting them, and transmitting them via afferent pathways.

Research into purinergic regulation during early postnatal ontogenesis reveals that P2X receptor subtypes located within the heart have heterochronic effects on cardiac contractile and chronotropic functions, varying across different heart regions. Intramural neurons are interconnected through synapses and interact via associative fibers, enabling effector responses through axon reflex mechanisms.

The ICNS possesses an extensive mediator system that influences impulse transmission and modulation, affecting conduction speed and response variability. Studies show that the number of these neurons varies among mammals. For instance, in humans (including children), approximately 94,000 neurons are found in epicardial plexuses, with additional neurons grouped or isolated. Subepicardial ganglia can number up to 1,500. This extensive neural network supports viewing the ICNS as an independent system.

Neurons within the ICNS are categorized as motor adrenergic, motor cholinergic, sensory, and interneurons. Adrenergic and cholinergic neurons, in particular, respond to various stimuli differently. Mechanoreceptors influence this response; for example, reduced blood pressure in the left ventricle triggers enhanced myocardial contraction through adrenergic ICNS neurons. Conversely, cholinergic neurons exhibit lower excitability and require stronger stimuli to activate, which in turn suppress adrenergic neuron activity [18, 40, 47, 48, 70].

The ICNS serves as a delicate integrative mechanism that not only evaluates blood circulation within the heart chambers but also monitors changes in the overall arterial and venous systems. In response to these changes, the ICNS ensures adaptive adjustments in cardiac function through autoregulatory mechanisms.

This integrative role highlights the ICNS's essential contribution to maintaining hemodynamic stability by dynamically responding to physiological and pathological changes within the cardiovascular system.

Aspects of Research Focused on Heart Pathologies

When analyzing the genesis and consequences of cardiovascular system pathologies, it has become evident that diseases with a neurogenic origin are a current and relevant issue, requiring an expansion of research. Scientific studies focused on the features of heart nerve structures in various pathologies have resulted in differing opinions among scientists, providing insight into the complex morphofunctional relationship between the heart's conduction system and the ganglionic nervous system, and shedding light on their variability.

Disruption of heart rhythm can occur when the autonomic nervous system fails to fully or partially ensure the functional conduction system. In such cases, functional disorders occur within the nerve cells, fibers, and the networks of ganglia, including the sinoatrial and atrioventricular nodes.

According to the research of Fedulova M.V., Pigolkin Y.I., and Toklu H.Z., the mechanism of arrhythmia is very complex, with multiple causes. On one hand, it is based on centers located in the brain, specifically in the nuclei of the medulla oblongata. On the other hand, it is based on the damage to the autonomic nerve ganglia and their peripheral parts, as well as the associated morphological structures. Therefore, it is well known that changes in the heart's nervous structures can lead to sudden death. For infants under one year of age, forensic examinations have revealed that in over 92% of cases, morphological changes are observed in the nerve ganglia, alongside changes in the layers of the heart wall. These changes are associated with hypertrophy of cardiomyocytes, which, in turn, are predicted to lead to disruptions in the heart's contraction function. This hypothesis is supported by morphological criteria such as changes in the heart's conduction system, apoptosis in neurons, fragmentation of nerve fibers, and axonal degeneration. Furthermore, the primary mechanism behind arrhythmia is the death of atypical cardiomyocytes. These conclusions were drawn from complex studies that identified these morphological changes through scientific research. [19, 26, 27, 52, 64].

Scientific conclusions regarding the role of ganglionic regulation in the genesis of heart arrhythmias have led to the formation of two opposing views. In particular, during ischemic heart disease, increased sympathetic activity is known to contribute to the development of arrhythmias, while activation of the parasympathetic component has the opposite, protective effect. However, in cases of sudden death with autonomic dysfunction, morphological studies of the myocardium reveal a sharp decrease in catecholamines in adrenergic fibers and changes in the vegetative nerve ganglia. Furthermore, ischemia of the ventricular walls leads to activation of the parasympathetic component in some areas, while ischemia in other areas increases the tone of sympathetic afferent nerves. This suggests that during heart pathologies, the intramural neural structures and autonomic nervous system exert various effects, and also that changes in myocardial contraction due to remodeling during ischemic heart disease lead to structural alterations in the neural components. These morphological alterations cause electrical instability, which can result in fatal arrhythmias.

The mechanism of neurovascular changes associated with heart pathologies cannot be considered in isolation. Depending on the stages of ontogenesis, including age-related changes, these alterations may appear as a manifestation on their own or may be exhibited simultaneously with other pathologies, potentially supporting one another. Scientific studies have shown that early involution of the sympathetic component appears around the age of 35-40. Forensic examinations in cases of death unrelated to heart disease show that structural changes in vascular and vegetative nerve fibers are indicative of disruptions in nerve trophism. With aging, involutive changes in sympathetic innervation increase, and reactive changes in adrenergic neurons can lead to further destructive changes. Additionally, powerful stimuli like myocardial stretch receptors and the stimulation of cholinergic neurons with low excitability in vagus nerve fibers result in heart rate slowing, which in turn inhibits adrenergic neurons. These phenomena are further exacerbated by the presence of atherosclerotic changes, leading to the development of additional pathologies [1, 5, 8, 10, 21].

According to the studies by Pooja Bhadoria and R. Ambiga, today, left ventricular hypertrophy (myocardium) is considered one of the manifestations of heart remodeling caused by arterial hypertension. Changes in the left ventricular myocardium are multifactorial and often occur in stages. Therefore, even mild left ventricular hypertrophy is a significant factor contributing to the onset of cardiovascular diseases, lowering the prognosis of patients with arterial hypertension, increasing the risk of myocardial infarction and sudden cardiac death, and leading to arrhythmias and heart failure. Cardiovascular diseases are the leading cause of death in many developed countries, accounting for 40% of all deaths. In this context, left ventricular myocardial hypertrophy is frequently observed in many such diseases.

During the development of cardiovascular disease, the dysfunction of the left ventricle, manifesting as a decrease in systolic (contractile) and/or diastolic (active relaxation) function, is common. This dysfunction is associated with an increase in the heart's pumping function and minute volume through compensatory mechanisms. Among these mechanisms, the primary role is played by the hypertrophy of the left ventricle with an increase in muscle mass [32, 57, 63].

The remodeling of heart structures, influenced by external environmental factors and age-related changes, involves a process of adaptation. Additionally, changes in the endothelial cells of blood vessels and subsequent structural remodeling are well documented. Research in this area has focused on the connection between left ventricular myocardial hypertrophy and the heart's neural structures, leading to investigations of gene polymorphism. These studies show that complex morphological changes in the sympathetic-adrenal system occur, indicating that the sympathetic system's effect on adrenergic receptors influences cardiac neural structures through the presynaptic inhibition of norepinephrine release, thus affecting autonomic regulation.

Similar changes in the heart's neural structures have been identified under physical stress. Morphologically, these include changes in nerve cells of the nodal apparatus, functional load in conductive cardiomyocytes, hypertrophy, and acute swelling, while limiting physical load is associated with reparative changes. In comparative studies of alcoholic cardiomyopathy and ischemic heart diseases, changes in vascular and cellular membrane permeability have been shown to disrupt electrical impulse conduction and generation, leading to ventricular fibrillation.

These changes represent the remodeling of the conduction system of cardiomyocytes and the heart's neural structures (GITQ). Hypertrophic cardiomyopathy, typically seen in secondary (arterial hypertension) hypertrophy, often presents as asymmetric concentric hypertrophy of the left ventricle, despite the variety of myocardial remodeling patterns. Modern perspectives on hypertrophic cardiomyopathy suggest that without a clear assessment of remodeling, numerous variations in the structural changes of the left ventricle exist depending on the exact location of hypertrophy. In contemporary cardiovascular research, the concept of "remodeling" is not universally defined. Remodeling refers to a complex process of structural and functional disruption of the heart, resulting from excessive load or loss of part of the viable myocardium. It is crucial to understand the evolutionary responses that allow damaged tissue to quickly adapt to new conditions in response to pathological agents affecting the heart [9, 30, 34, 46, 53, 55, 58].

Thus, the study of the characteristics of intracardiac neural structures in various cardiovascular pathologies shows that these structures are characterized by a range of features, from simple morphological changes to alterations at the cellular level. Age-related changes in the intracardiac neural structures, along with general hemodynamic patterns, are capable of receiving information from the heart wall and intracardiac morphofunctional indicators, which, through integration with the extrinsic nervous system, can alter the heart's contractile activity. It can be predicted that the autonomous system of the intracardiac neural structures plays a key role in maintaining the heart's stable rhythm and that these structures undergo changes in the form of remodeling during pathological conditions.

Conclusion

Recent scientific research over the past decade has established a link between the autonomic nervous system and cardiovascular system pathologies in relation to mortality. This highlights the significant relevance of contemporary scientific studies focusing on cardiovascular issues. A key feature of these studies is the high mortality rate from heart-related complications, particularly among individuals of working age. As outlined in the literature, sudden cardiac death and secondary manifestations of certain changes lead to disruptions in the heart's contractile function, primarily due to alterations in the heart's neural structures.

Given that coronary circulation is primarily regulated by the autonomic nervous system, autonomic-reflex changes, such as hemodynamic, metabolic, and energy disturbances, are considered prognostic indicators of functional changes. Heart rhythm, as an indicator of these changes, is influenced by the dynamic interactions of the morphological divisions of the ganglionic nervous system, affecting the heart's conductive system and intracardiac neural structures. In maintaining heart function, the role of the extrinsic neural structures (the peripheral part of the autonomic nervous system), along with the cellular elements (neurons, contractile cardiomyocytes), and particularly intracardiac neural structures (intracardiac nervous system and heart's autonomic nervous system), is pivotal [2, 14, 22, 23, 31, 36, 42, 51, 54, 65].

Efferent neurons within the intracardiac neural structures not only serve as a means of transmitting impulses from preganglionic fibers to postganglionic fibers but also play a critical role in regulating other vital heart functions. The most important factor influencing the heart's rhythmic contraction and its changes is the interaction between impulses from preganglionic fibers and those participating in the autonomous regulation process. As a result, the heart's regular and rhythmic contraction is controlled through the complex mechanisms of intense impulse transmission. Thus, studying the architecture of intracardiac neural structures, their morphological components, and changes in the context of cardiovascular pathologies remains a crucial area of scientific research [3, 12, 16, 24, 45, 49, 50, 56, 61, 62, 66].

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