EduVision: Journal of Innovations in Pedagogy and Educational Advancements
Volume 2, Issue 4, April 2026
brightmindpublishing.com
ISSN (E): 3061-6972
Licensed under* CC BY 4.0 *a Creative Commons Attribution 4.0 International License.

MAIN CAUSES OF CARDIAC RHYTHM DISORDERS

Urmanbekova Dilfuza Sanatullayevna 1,
Berdiyev Azizbek Ilyos o'g'li 2
Elmurotova Dilnoza Baxtiyorovna 3
Assistant 1, Student 2, Associate Professor, PhD. 3
Tashkent State Medical University1, 2, "Scientific and Technical Center for Radiation and Nuclear Safety" State Institution, Republic of Uzbekistan 3

Abstract
This paper examines the causes of arrhythmia, which are divided into four groups. It is shown that one of the most striking examples of rhythm disturbances is caused by an imbalance between the sympathetic and parasympathetic branches of the autonomic nervous system. Changes in the normal automaticity of the heart lead to the development of sinus arrhythmias.

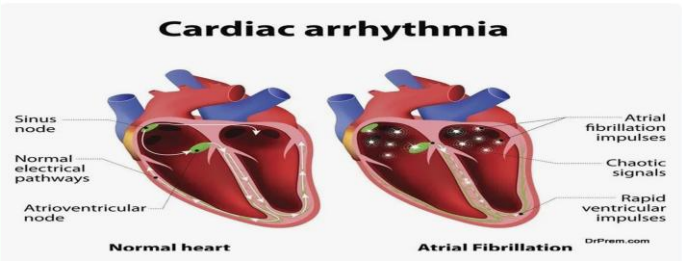
Keywords: Arrhythmia, imbalance, thyrotoxicosis, aldosterone, diuretics, automatism, trigger activity, sinus, frequency.

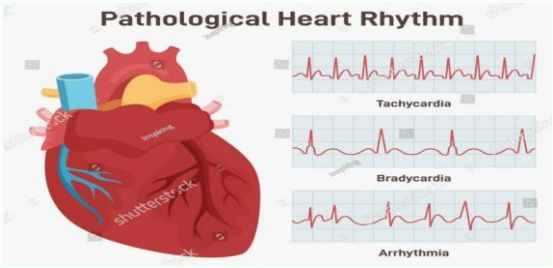
Introduction

Arrhythmia (a—negation of something, Greek rhytmos —flow) is a typical form of cardiac pathology characterized by a disturbance in the frequency and periodicity of excitation impulse generation and/or the excitation sequence of the atria and ventricles. The occurrence of arrhythmias is most often associated with the presence of organic heart disease of various etiologies—ischemia, inflammation, degenerative changes, toxic damage.

However, they are also recorded in individuals with apparently healthy hearts, in whom modern diagnostic methods reveal no pathology. It has been established that the incidence of arrhythmias increases with increasing age. Therefore, in the general population, a correlation has been established between the prevalence of coronary heart disease and the frequency of detection of cardiac arrhythmias. Cardiac arrhythmias are most frequently observed in coronary insufficiency. Thus, in the acute phase of myocardial infarction, arrhythmias are recorded in 95–100% of patients.

EduVision: Journal of Innovations in Pedagogy and Educational Advancements
Volume 2, Issue 4, April 2026
brightmindpublishing.com
ISSN (E): 3061-6972
Licensed under CC BY 4.0 *a Creative Commons Attribution 4.0 International License.*





Arrhythmias are a consequence of a violation of automatism, excitability or conductivity of the myocardium, as well as their combinations.

The causes of arrhythmia can be divided into four groups:

1) disturbances of neurohumoral regulation of electrophysiological processes in the myocardium;

2) organic lesions of the myocardium, its anomalies, congenital or hereditary defects with damage to membranes and cellular structures;

3) a combination of disorders of neurohumoral rhythm regulation and organic heart pathology;

4) arrhythmias caused by the action of toxic substances and drugs (including antiarrhythmic drugs).

One of the main causes of heart rhythm disturbances is a change in the physiological relationship between the tonic activity of the sympathetic and parasympathetic divisions of the autonomic nervous system.

In animal experiments, virtually any known form of arrhythmia—from sinus tachycardia to ventricular fibrillation—can be induced by influencing certain parts of the brain: the cortex, limbic structures, and the hypothalamic-pituitary system, which is closely linked to the centers of sympathetic and parasympathetic regulation of cardiac activity located in the reticular formation of the medulla oblongata.

One of the most striking examples of arrhythmia caused by an imbalance between the sympathetic and parasympathetic links of the autonomic nervous system is a decrease in the electrical stability of the heart during psychoemotional stress.

Thyrotoxicosis causes cardiac arrhythmia due to increased cardiac adrenergic reactivity . One common endocrine cause of arrhythmia is excessive mineralocorticoid production in the adrenal cortex.

The mechanism of the arrhythmogenic effect of mineralocorticoids (primarily the most active of them, aldosterone) is associated with an imbalance of Na^+/K^+ in the body.

EduVision: Journal of Innovations in Pedagogy and Educational Advancements
Volume 2, Issue 4, April 2026
brightmindpublishing.com
ISSN (E): 3061-6972
Licensed under CC BY 4.0 *a Creative Commons Attribution 4.0 International License.*

Aldosterone, acting on the renal tubules, stimulates reabsorption Na^+ from primary urine and increased $K^{+\text{ excretion}}$ result in hypokalemia , which contributes to disruption of cardiomyocyte membrane repolarization processes and the development of arrhythmia. Organic myocardial lesions that cause arrhythmias include myocardial infarction, cardiosclerosis, myocarditis, cardiomyopathy, and others.

Diuretics (e.g., furosemide), by increasing $K^{+\text{ excretion}}$, contribute to the development of hypokalemia . Cardiac glycosides (e.g., strophanthin), by inhibiting the Na^+/K^+ -ATPase of cardiomyocyte membranes , reduce the activity of this enzyme, which is accompanied by a decrease in K + content and an increase in the Na + concentration in the sarcoplasm of cardiomyocytes . A decrease in the intracellular concentration of K + leads to a slowing of the repolarization processes of cardiomyocyte membranes , which contributes to the development of arrhythmia.

Impulse formation disorders may be caused by impaired automatism and increased excitability of cardiomyocytes .

Automaticity is the ability of cardiomyocytes to spontaneously generate an action potential. All atypical cardiomyocytes (cells of the cardiac conduction system) possess this ability , but working cardiomyocytes (cells of the contractile myocardium) do not .

Excitability is the ability of cells in excitable tissues to perceive a stimulus and respond to it with an excitatory response. Cardiac muscle excitability is expressed in the ability to generate an action potential in response to stimulation.

Normally, only the sinoatrial node (SA node, Keith- Fleck node) exhibits automaticity; it is a nomotopic (i.e., normally located) pacemaker. The rate of impulse generation by SA node cells at rest in adults is 60–90 per minute. The SA node is called the true pacemaker, or first-order pacemaker .

The remaining structures of the cardiac conduction system (atrioventricular node, bundle of His, branches of the bundle of His, Purkinje fibers) are also capable of spontaneously generating impulses, but The natural frequency of discharges of cells in these sections is low.

It decreases the further the cells are from the first-order pacemaker (automatism gradient). Due to this, under normal conditions, the action potential in these cells of the cardiac conduction system arises as a result of excitation from the more frequently discharging upper sections (cells of the SA node), and their own automaticity "doesn't have time" to manifest.

EduVision: Journal of Innovations in Pedagogy and Educational Advancements
Volume 2, Issue 4, April 2026
brightmindpublishing.com
ISSN (E): 3061-6972
Licensed under CC BY 4.0 *a Creative Commons Attribution 4.0 International License.*

Thus, the underlying structures of the cardiac conduction system exhibit automatism only when the flow of impulses from the SA node is disrupted and are therefore called latent (hidden, potential) pacemakers.

Changes in normal cardiac automaticity lead to the development of sinus arrhythmias. The duration of spontaneous diastolic depolarization and, consequently, the impulse generation rate of SA node cells are influenced by the following mechanisms:

1. The rate of spontaneous diastolic depolarization (the most important). As it increases, the excitation threshold is reached more quickly, leading to an increase in sinus rhythm (tachycardia). A decrease in spontaneous diastolic depolarization leads to a slower sinus rhythm (bradycardia).

2. The magnitude of the resting potential of the SA node cells.

If the resting potential value becomes more negative (for example, as a result of membrane hyperpolarization under the action of acetylcholine), then it takes longer to reach the excitation threshold (provided that the rate of spontaneous diastolic depolarization remains unchanged) - bradycardia occurs.

If the resting potential of the SA node cells becomes less negative, then, accordingly, less time is required to reach the excitation threshold - tachycardia develops.

3. Change in the excitation threshold.

A more negative value of the excitation threshold of the SA node cells promotes an increase in the sinus rhythm, and a less negative value promotes a decrease in the sinus rhythm .

Various combinations of the three main electrophysiological mechanisms regulating the automatism of the SA node are also possible. Abnormal automatism. Normally, the rhythm of heart contractions is determined by the cells of the SA node; all other cells of the cardiac conduction system discharge, like the working myocardium, under the influence of spreading excitation. The action potential in them arises under the influence of currents from excited areas of the myocardium before their membrane potential reaches the excitation threshold as a result of their own slow spontaneous diastolic depolarization.

Abnormal automaticity is the appearance of pacemaker activity in cardiac cells that are not normally pacemakers (i.e., they become pacemakers instead of SA node cells). If, for one reason or another, excitation of the SA node does not occur or cannot pass to the atrium due to conduction disturbances, the atrioventricular node (AV node,

EduVision: Journal of Innovations in Pedagogy and Educational Advancements
Volume 2, Issue 4, April 2026
brightmindpublishing.com
ISSN (E): 3061-6972
Licensed under CC BY 4.0 *a Creative Commons Attribution 4.0 International License.*

Aschoff - Tawara node) takes over the role of pacemaker - a second-order pacemaker (frequency of the heart pulse rate of 40–60 per minute).

If the conduction of excitation from the atria to the ventricles is completely impaired, the ventricles contract in the rhythm of a third-order pacemaker (pulse generation rate less than 40 per minute), located in the ventricular conduction system. The SA node is called a nomotopic (normally located) pacemaker, and the excitation foci in the remaining parts of the cardiac conduction system are called heterotopic (abnormally located).

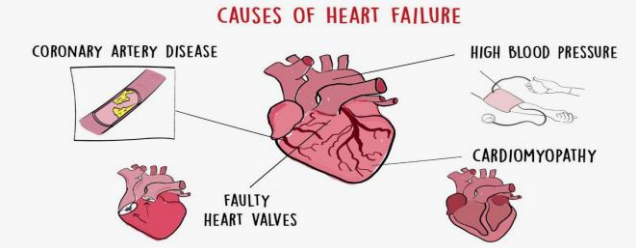
An ectopic pacemaker is a source of cardiac impulses located anywhere in the myocardium other than the SA node. A cardiac contraction caused by an impulse from a heterotopic excitation focus is called an escape rhythm, and the sequence of contractions is called an escape rhythm (e.g., an AV rhythm).

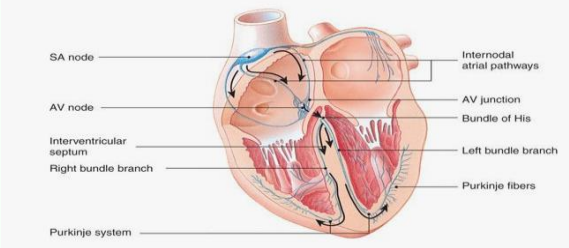
Such rhythms perform a protective function, maintaining a relatively high heart rate (HR) with a marked slowdown in impulses from the SA node.

Heterotopic foci of excitation can determine the heart rate if their impulse generation rate (and therefore frequency) is greater than that of the SA node.

For example, against the background of a high concentration of catecholamines, the automatism of cells in the underlying parts of the cardiac conduction system may increase, and if the frequency of their depolarization is greater than the frequency of impulse generation by cells of the SA node, this leads to the appearance of an ectopic rhythm (for example, extrasystole).

Such ectopic rhythms can occur during myocardial ischemia, hypoxia, electrolyte imbalances, etc. When the myocardium is damaged (e.g., by ischemia, etc.), working cardiomyocytes can acquire a pathological capacity for automaticity. If the rate of spontaneous diastolic depolarization of such cardiomyocytes exceeds the impulse generation rate of the SA node cells, these cells establish their own heart rate, becoming an ectopic pacemaker.





EduVision: Journal of Innovations in Pedagogy and Educational Advancements
Volume 2, Issue 4, April 2026
brightmindpublishing.com
ISSN (E): 3061-6972
Licensed under CC BY 4.0 *a Creative Commons Attribution 4.0 International License.*

Trigger activity. Increased excitability of cardiomyocytes most often causes arrhythmias through the mechanism of trigger activity (induced, starting, from the English " trigger ") .

The electrophysiological basis of trigger activity (trigger automatism) is early and late postdepolarizations .

Early afterdepolarization is a premature depolarization of cardiomyocytes that occurs during the repolarization phase of the action potential, when the membrane potential has not yet reached the resting potential.

The conditions for the occurrence of early afterdepolarizations are a prolongation of the repolarization phase of the action potential and bradycardia. If repolarization slows and, consequently, the overall duration of the action potential increases, premature spontaneous depolarization may occur at a time when the repolarization process is not yet complete.

With a decrease in the frequency of the main heart rhythm (bradycardia), there is a gradual increase in the amplitude of early suprathreshold oscillations of the membrane potential, which, upon reaching the excitation threshold, can cause the formation of a new action potential even before the completion of the initial one.

This premature action potential is considered triggered (induced) because it owes its origin to the early afterdepolarization emanating from the main action potential.

In turn, the second (induced) action potential, due to its early post-depolarization, can cause a third, also triggered action potential, etc.

If the source of trigger activity is in the ventricles, then ventricular extrasystole or polymorphic (paroxysmal) ventricular tachycardia (ventricular tachycardia of the "pirouette" type - torsade) may develop. de pointes).

The occurrence of early post-depolarizations is facilitated by: hyperka Techolaminemia , hypokalemia , acidosis, ischemia. Late postdepolarization is a premature depolarization of cardiomyocytes that occurs immediately after the completion of the repolarization phase , i.e., when the electrical charge of the membrane corresponds to the resting potential.

Subthreshold oscillations of membrane potential, which may normally be present but never manifest themselves, in pathological conditions accompanied by an increase in the intracellular concentration of Ca2+ in cardiomyocytes , can increase in amplitude, reaching the excitation threshold.

EduVision: Journal of Innovations in Pedagogy and Educational Advancements
Volume 2, Issue 4, April 2026
brightmindpublishing.com
ISSN (E): 3061-6972
Licensed under CC BY 4.0 *a Creative Commons Attribution 4.0 International License.*

$^{+}$ concentration activates nonselective ion channels, allowing increased influx of cations (primarily Na +) from the extracellular environment into the cardiomyocyte . As a result, the negative charge of the inner membrane surface decreases, reaching the excitation threshold, and a series of premature action potentials occurs. Late postdepolarizations often occur during myocardial infarction and can be caused by cardiac glycosides and catecholamines.

References

1. А.Н. Глебов, Ф.И. Висмонт. Патофизиология нарушений ритма сердца : учеб.-метод. пособие – Минск : БГМУ, 2014. – 39 с.
2. Е.Б. Букреева, В.В. Боярко, Н.С. Ямкина. Нарушения сердечного ритма: учебное пособие – Томск, 2017. – 88 с.
3. Elmurotova D.B., Ro‘zimatova Sh. Sh., Umarova F.S. Insonning estetik tafakkuri // Лучшие интеллектуальные исследования, ISSN:3030-3680, Ч.45, Т.1, С.130-135, май-2025, Россия. https://scientific-jl.com/luch/article/view/13889
4. Elmurotova D.B., Farmonova Sh.Sh., Umarova F.S. Borliq va bo‘shliq: mavjudlik chegaralari haqida tafakkur // Лучшие интеллектуальные исследования, ISSN:3030-3680, Ч.44, Т.5, С.411-416, май-2025, Россия. https://scientific-jl.com/luch/article/view/13407
5. Elmurotova D.B., Jo’rayeva R.A, Umarova F.S. Bilimning chegarasi va rad etilishi”: eskeptitsizm va bilimga bo‘lgan ishonchsizlik muammosi // Лучшие интеллектуальные исследования, ISSN:3030-3680, Ч.44, Т.5, С.417-423, май-2025, Россия. https://scientific-jl.com/luch/article/view/13408
6. Elmurotova D.B., Kattaxodjayeva D.U., Komiljonov J.O. Blood and its functional structure // Texas Journal of Medical Science, Peer Reviewed International Journal, V.45, ISSN NO: 2770-2936, June 2025, P.40-44. https://zienjournals.com/index.php/tjms/article/view/6318
7. Elmurotova D.B., Odilova N.J., Odilova E.U. Invasive Radiation Diagnostic Methods // American Journal of Bioscience and Clinical Integrity, ISSN: 2997-7347, V.2., N.10, 2025. P.21-27.Amerika, https://biojournals.us/index.php/AJBCI/article/view/1555/1308
8. Elmurotova D.B., Kattaxodjayeva D.U., Samatov A.X., G’ulomov F.O. Classification of bone joints // Miasto Prysztosci, IF-9.9, ISSN-2544-980X, V.67,

EduVision: Journal of Innovations in Pedagogy and Educational Advancements
Volume 2, Issue 4, April 2026
brightmindpublishing.com
ISSN (E): 3061-6972
Licensed under CC BY 4.0 *a Creative Commons Attribution 4.0 International License.*

2025, P.126-130. Poland. https://miastoprzyszlosci.com.pl/index.php/mp/article/view/7255/6825

9. Elmurotova D.B., Yusupov N.N., Sattarov Y.K., G'oyibnazarov R.B. Remature hearing loss// Miasto Prysztosci, IF-9.9, ISSN-2544-980X, V.66, 2025, P.206-210. Poland. https://miastoprzyszlosci.com.pl/index.php/mp/article/view/7156
10. Elmurotova D.B., Kattaxodjayeva D.U., Komiljonov J.O. Structure of the brain section of the head // Miasto Prysztosci, IF-9.9, ISSN-2544-980X, V.65, 2025, P.313-317. Poland. https://miastoprzyszlosci.com.pl/index.php/mp/article/view/7033
11. Elmurotova D.B., Kattaxodjayeva D.U., Tanirbergenova N.O. The Main Tasks of Biophysics and the Physical Foundations of Biological Structures// American Journal of Bioscience and Clinical Integrity, ISSN: 2997-7347, V.2, N.10, 2025, October P.71-77. https:///biojournals.us/index.php/AJBCI/article/view/1596/1345
12. Elmurotova D.B., Imamova L.N., Orifqulova M.F. Indications for holter monitoring // Miasto Prysztosci, IF-9.9, ISSN-L:2544-980X, V.62, 2025, P.155-160. Poland. https://miastoprzyszlosci.com.pl/index.php/mp/article/view/6828
13. Elmurotova D.B., Qo'ziyev S.R. Retinitis pigmentosa: clinical picture and diagnostic insights // Western European Journal of Linguistics and Education, ISSN (E): 2942-190X, V.3, Issue 10, October 2025, P.113-120. Germany, https://westerneuropeanstudies.com/index.php/2/article/view/2901/20091
14. Elmurotova D.B., Qo'ziyev S.R. Current Perspectives on the Etiology and Epidemiology of Retinitis Pigmentosa // American Journal of Bioscience and Clinical Integrity, ISSN: 2997-7347, V.2, N.10, 2025, October P.119-124. America. https://biojournals.us/index.php/AJBCI/article/view/1641/1380
15. Elmurotova D.B., Urmanbekova D.S., Bozorov M.B. Main risk factors of myocardial infarction // American Journal of Bioscience and Clinical Integrity, ISSN: 2997-7347, V.2, N.12, 2025, Dekabr P.6-9. https://biojournals.us/index.php/AJBCI/article/view/1763/1476