World Bulletin of Public Health (WBPH)
Available Online at: https://www.scholarexpress.net
Volume-57 April, 2026
ISSN: 2749-3644

CLINICAL AND PHARMACOLOGICAL APPROACH TO THE TREATMENT OF CARDIAC ARRHYTHMIA USING ANTIARRHYTHMIC DRUGS

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Article history:
Received: February 12th 2026
Accepted: March 11th 2026

Abstract:
This article provides a comprehensive analysis of cardiac arrhythmias and the clinical and pharmacological characteristics of antiarrhythmic drugs used in their treatment. Arrhythmias arise as a result of disturbances in the heart rhythm and conduction system and manifest with varying degrees of clinical symptoms. In modern medicine, antiarrhythmic agents are considered one of the main tools for normalizing heart rhythm. In particular, the specific features of their use in both children and adults highlight their clinical significance. Therefore, this article discusses the pharmacodynamics, pharmacokinetics, indications for use, adverse effects, and the importance of an individualized approach to therapy. It also substantiates that the patient's general condition, underlying diseases, and the type of arrhythmia are key factors in selecting appropriate treatment in clinical practice.

Keywords: Cardiac arrhythmia, antiarrhythmic drugs, use in children and adults, pharmacokinetics, pharmacodynamics, clinical approach, heart rhythm, drug therapy, adverse effects, cardiology, individualized treatment.

YURAK ARITMIYASI BILAN BOG'LIQ KASALLIKLARNI ANTIARITMIK DORI VOSITALAR ASOSIDA DAVOLASHDA KLINIK-FARMAKOLOGIK YONDASHUV

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Annotatsiya. Ushbu maqolada yurak aritmiyalari va ularni davolashda qo'llaniladigan antiaritmik dori vositalarining klinik-farmakologik xususiyatlari keng tahlil qilingan. Aritmiyalar yurak ritmi va o'tkazuvchanlik tizimining buzilishi natijasida yuzaga kelib, turli darajadagi klinik belgilar bilan namoyon bo'ladi. Zamonaviy tibbiyotda antiaritmik preparatlar yurak ritmini normallashtirishda asosiy vositalardan biri hisoblanadi. Maqolada dori vositalarining farmakodinamikasi, farmakokinetikasi, qo'llash ko'rsatmalari, nojo'ya ta'sirlari hamda individual yondashuvning ahamiyati yoritilgan. Shuningdek, klinik amaliyotda dorilarni tanlashda bemorning umumiy holati, asosiy kasalliklari va aritmiyaning turi muhim omil ekanligi asoslab berilgan.

Kalit so'zlar: Yurak aritmiyasi, antiaritmik preparatlar, farmakokinetika, farmakodinamika, klinik yondashuv, yurak ritmi, dori terapiyasi, nojo'ya ta'sirlar, kardiologiya, individual davolash.

КЛИНИКО-ФАРМАКОЛОГИЧЕСКИЙ ПОДХОД К ЛЕЧЕНИЮ ЗАБОЛЕВАНИЙ, СВЯЗАННЫХ С НАРУШЕНИЯМИ СЕРДЕЧНОГО РИТМА, С ПОМОЩЬЮ АНТИАРИТМИЧЕСКИХ ПРЕПАРАТОВ

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Аннотация. В данной статье проведён всесторонний анализ сердечных аритмий и клинико-фармакологических характеристик антиаритмических препаратов, применяемых для их лечения. Аритмии возникают вследствие

World Bulletin of Public Health (WBPH)
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Volume-57 April, 2026
ISSN: 2749-3644

нарушений сердечного ритма и проводящей системы сердца и проявляются различными клиническими симптомами. В современной медицине антиаритмические препараты являются одним из основных средств нормализации сердечного ритма. Особенно важным является учет особенностей их применения у детей и взрослых, что подчёркивает клиническую значимость этих препаратов. В статье рассмотрены фармакодинамика и фармакокинетика лекарственных средств, показания к применению, возможные побочные эффекты, а также обоснована необходимость индивидуального подхода к терапии. Кроме того, обосновано, что при выборе препаратов в клинической практике ключевыми факторами являются общее состояние пациента, сопутствующие заболевания и тип аритмии.
Ключевые слова: сердечная аритмия, антиаритмические препараты, применение у детей и взрослых, фармакокинетика, фармакодинамика, клинический подход, сердечный ритм, лекарственная терапия, побочные эффекты, кардиология, индивидуальное лечение.

INTRODUCTION. Cardiovascular diseases remain one of the most pressing problems in modern medicine. Among them, cardiac arrhythmias occupy a special place. Arrhythmia is a disruption of the normal rhythmic activity of the heart, which can manifest as tachycardia, bradycardia, or irregular heartbeats. Arrhythmias develop due to various causes, including myocardial ischemia, electrolyte imbalances, dysfunction of the autonomic nervous system, endocrine disorders, and improper use of medications. Therefore, their treatment requires a comprehensive approach. Antiarrhythmic drugs act on the electrical activity of the heart, regulating the generation and propagation of impulses. However, along with their effectiveness, these drugs can also produce adverse effects. For this reason, a clinical-pharmacological approach is of critical importance. Furthermore, special caution is needed when using these drugs depending on the patient's age, as antiarrhythmic medications may be employed in both pediatric and geriatric practice.

Treating cardiac arrhythmias in children is one of the most urgent and complex challenges in the practice of both pediatricians and adult cardiologists. Although many treatment methods overlap with those used in adults, the physiological characteristics of a child's developing body, along with the fact that many adult arrhythmia-causing factors are often absent in children, lead to differences in the pathogenesis of arrhythmias and the approaches to their treatment [1].

MATERIALS AND METHODS. In preparing this article, modern scientific literature, clinical guidelines, pharmacology textbooks, and research findings in the field of cardiology were thoroughly studied. During the analysis, the following methods were employed: literature analysis – recent scientific articles and clinical recommendations were reviewed, comparative method – the effects and applications of various antiarrhythmic drugs were compared, clinical observation data analysis – the effectiveness of arrhythmia treatment was assessed, systematic approach – arrhythmias were classified, and criteria for drug selection were summarized. As study material, information on antiarrhythmic drugs belonging to different classes (I, II, III, IV) and their clinical applications was utilized.

ANALYSIS AND DISCUSSION. According to the classical Vaughan Williams classification, antiarrhythmic drugs are divided into five main classes based on their predominant electrophysiological effects [2]: Class I: Sodium channel blockers – subdivided into three groups: IA (moderate block): quinidine, procainamide; IB (weak block): lidocaine, mexiletine; IC (strong block): flecainide, propafenone. These drugs primarily depress phase 0 depolarization, thereby slowing the conduction velocity in the heart. Class II: Beta-adrenergic blockers (e.g., propranolol, metoprolol, esmolol) – reduce sympathetic stimulation of the heart, decrease automaticity, and prolong refractoriness in nodal tissues. As a result, heart rate decreases and arrhythmias are reduced, especially in stress-related arrhythmias. Class III: Potassium channel blockers – these drugs prolong repolarization and help stabilize heart rhythm. Amiodarone is one of the most effective representatives of this group. It is also important to note that antiarrhythmic drugs exhibit significant interindividual variability in absorption, distribution, metabolism, and elimination. Most are highly protein-bound and undergo hepatic biotransformation via cytochrome P450 enzymes. Their pharmacokinetics are strongly influenced by liver function, renal clearance, and interactions with other cardiovascular drugs. For example, amiodarone has highly complex pharmacokinetics. It is highly lipophilic, accumulates in adipose and myocardial tissue, and has a half-life of up to 50 days, resulting in prolonged effects even after discontinuation. It also inhibits multiple CYP enzymes and P-glycoprotein, causing clinically significant interactions with warfarin, digoxin, and statins. In contrast, lidocaine undergoes rapid hepatic metabolism and is used intravenously for acute ventricular arrhythmias due to its short half-life [3]. Class IV: Calcium channel blockers – drugs such as verapamil and diltiazem slow conduction through the AV node and are used in supraventricular arrhythmias.

When supraventricular paroxysmal tachycardia (SPT) develops, treatment initially begins with vagal

World Bulletin of Public Health (WBPH)
Available Online at: https://www.scholarexpress.net
Volume-57 April, 2026
ISSN: 2749-3644

maneuvers. In children, their effectiveness is highest within the first 20–30 minutes after the onset of an attack. If the attack cannot be stopped during this period, antiarrhythmic drugs (AAD) are administered. In such cases, adenosine (ATP) is the drug of choice. Its clinical effectiveness is due to its rapid onset (up to 10 seconds) and relatively rare, short-lived adverse effects (cough, warmth, flushing, bradycardia). The initial dose is administered intravenously as a 1% solution rapidly (over 3–4 seconds), undiluted, at 0.1 mg/kg. For rapid attack relief, age-adjusted doses can also be used: up to 6 months – 0.5 ml, 6 months – 1 year – up to 0.7 ml, 1–3 years – 0.8 ml, 4–7 years – 1.0 ml, 8–10 years – 1.5 ml, 11–14 years – 2.0 ml. If the heart rhythm is not restored within 1–2 minutes, the dose may be doubled and, if necessary, repeated once more [2]. Key Principles of the Clinical-Pharmacological Approach: identification of Arrhythmia Type – The type of arrhythmia must be accurately determined to select the most appropriate drug for each specific form. Consideration of Individual Patient Characteristics – Age, sex, underlying diseases, and other patient-specific factors must be taken into account when choosing therapy. Pharmacokinetic Considerations – Absorption, distribution, metabolism, and elimination of the drug play a crucial role in its effectiveness and safety. Assessment of Adverse Effects – Antiarrhythmic drugs themselves can sometimes provoke arrhythmias (proarrhythmic effect), so monitoring and risk evaluation are essential. Combination Therapy – In certain cases, multiple drugs may be used together to achieve optimal therapeutic effect.

RESULTS. The analysis showed that the treatment of atrial fibrillation can be carried out using both drug-based and non-drug methods. Non-drug approaches include surgical interventions, minimally invasive electrotherapy methods (such as radiofrequency catheter ablation), and the use of implantable antiarrhythmic devices. Drug therapy is divided into two main directions: emergency management of the most dangerous forms of atrial fibrillation and long-term (chronic) pharmacotherapy. Emergency intervention is especially necessary in cases with a high risk of heart failure, circulatory arrest, or sudden death. Such high-risk arrhythmias primarily include ventricular tachycardia (VT) progressing to fibrillation and bradyarrhythmias. In children over one year old, supraventricular paroxysmal tachycardia (SPT) rarely directly causes circulatory arrest. However, in infants, SPT poses a significantly higher risk and is considered a potential of sudden death.

In patients with Wolff–Parkinson–White (WPW) syndrome who develop SPT, intravenous administration of giluritmal (ajmaline) at a dose of 1 mg/kg (not exceeding 50 mg) has proven effective. Isoptin (verapamil) is also commonly used in practice to terminate SPT attacks. Although this drug may induce bradycardia and arterial hypotension, it is effective in treating polymorphic (polytopic) atrial tachycardia in children. Age-specific doses of isoptin are as follows: up to 1 month – 0.2–0.3 ml, up to 1 year – 0.3–0.4 ml, 1–5 years – 0.4–0.5 ml, 5–10 years – 1.0–1.5 ml, Over 10 years – 1.5–2.0 ml. It is particularly important to emphasize that isoptin is absolutely contraindicated in tachycardia of unknown origin with a wide QRS complex and in WPW syndrome, as accelerated anterograde conduction via accessory pathways can lead to transformation of SPT into ventricular fibrillation. In children, the following drugs are also used as first-line agents to terminate SPT attacks: cordarone (amiodarone): intravenously, in 5% glucose solution, at a dose of 5 mg/kg; digoxin: intravenously, slowly, in saline, at a dose of 0.1–0.3 ml; novocainamide: intravenously, slowly, in saline, at 0.15–0.2 ml/kg, up to a maximum of 17 mg/kg. To prevent arterial hypotension, mesaton 1% solution is administered at 0.1 ml per year of age, not exceeding 1.0 ml. The efficacy of antiarrhythmic drugs in supraventricular tachycardia is enhanced when used in combination with sympatholytic tranquilizers (e.g., relanium, tazepam, radedorm).

During an attack, if a wide QRS complex tachycardia is observed on ECG, it is not always possible to clearly distinguish whether it is ventricular tachycardia (VT) or supraventricular tachycardia with conduction abnormalities. If the type of arrhythmia cannot be precisely determined, treatment is conducted as for VT. In all such cases, calcium channel antagonists are contraindicated. Termination of Ventricular Tachycardia and Management of Bradyarrhythmias in Children: The first-line drug for terminating ventricular tachycardia (VT) is lidocaine. It is administered intravenously, slowly, in 5% glucose solution, at an initial dose of 1 mg/kg (as a 1–2% solution). If the heart rhythm is not restored, the drug may be repeated every 5–10 minutes at half the initial dose, ensuring that the total dose does not exceed 3 mg/kg. For VT, second-line agents include novocainamide, giluritmal (ajmaline), cordarone (amiodarone), or β-blockers, using previously indicated dosages. In patients with prolonged QT interval experiencing "torsade de pointes" (pirouette-type) tachycardia, the drug of choice is magnesium sulfate. It is administered as a 10% solution, 25–50 mg/kg (maximum 2 g) over 1–2 minutes. If ineffective, the

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Volume-57 April, 2026
ISSN: 2749-3644

dose can be repeated after 5–10 minutes. Among bradyarrhythmias, symptomatic bradycardia, asystole, or electromechanical dissociation (sinus bradycardia on ECG without a pulse) requires urgent treatment. In children, the main interventions for asystole are adrenaline and atropine administration. Adrenaline: If small intravenous doses (0.01 mg/kg) are ineffective and cardiac arrest occurs, higher doses of 0.1–0.2 mg/kg IV are administered, repeated every 3–5 minutes as needed. Atropine: Typically used after adrenaline for bradyarrhythmias. Intravenous dose: 0.02 mg/kg (maximum single dose: 0.5 mg in young children, 1.0 mg in adolescents), repeatable every 5 minutes (total maximum dose: 1.0 mg for young children, 2.0 mg for adolescents). Long-term Pharmacotherapy of Arrhythmias in Children Long-term pharmacotherapy of arrhythmias in children is aimed at correcting both intracardiac (within the heart) and extracardiac (outside the heart) mechanisms that contribute to their development [3]. When antiarrhythmic drugs are appropriately selected, they demonstrate high efficacy, and an individualized approach significantly improves treatment outcomes. Adverse effects are often associated with incorrect dosing or inappropriate drug selection. Drugs like amiodarone, despite their broad spectrum of action, require cautious long-term use due to potential systemic effects. Beta-blockers are considered among the safest options for arrhythmias associated with cardiovascular diseases. This highlights the importance of a personalized, clinically informed approach when planning chronic antiarrhythmic therapy in pediatric patients, balancing efficacy with safety.

CONCLUSION. Treatment of cardiac arrhythmias is a complex, multi-stage process that requires a deep clinical-pharmacological approach. The clinical specificity of therapy is based on balancing efficacy and risk, meaning it is essential to choose a drug that stabilizes heart rhythm while producing the least systemic toxicity [4]. Proper selection of antiarrhythmic drugs plays a critical role in improving patients' quality of life and preventing complications. Additionally, an individualized treatment plan is necessary for each patient. The effectiveness and safety of arrhythmia therapy can be significantly enhanced through a chronotherapeutic approach, which involves determining the daily pattern of arrhythmia using Holter monitoring and administering two-thirds of the daily dose of antiarrhythmic drugs (AAD) so that the maximum effect occurs just before the expected time of arrhythmia intensification or onset. Although modern cardiology increasingly employs invasive methods, pharmacotherapy remains one of the primary treatment strategies for managing arrhythmias.

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