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Arrhythmias and Valvular Heart Disease in Elderly and Senile People

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Introduction

Heart rhythm disorders and valvular heart disease occupy one of the leading places in the structure of cardiovascular morbidity among elderly and senile individuals. Involutive processes developing in the myocardium and the cardiac conduction system, combined with the superimposition of age-related changes onto the consequences of past diseases (rheumatism, coronary artery disease, hypertension), create a complex pathomorphological and pathophysiological background. This significantly modifies the etiology, clinical presentation, and course of arrhythmias and valvular heart defects in geriatric patients. The diagnosis of these conditions is often difficult due to the blunting of symptoms, the nonspecific nature of complaints, and the similarity of manifestations to the "age norm," leading to underdiagnosis. Treatment requires a reconsideration of traditional approaches due to altered pharmacokinetics, a high risk of polypharmacy, and adverse effects. In this regard, an in-depth study of the etiology, clinical features, diagnosis, and treatment principles of arrhythmias and valvular lesions in elderly and senile people is a critically important task of modern geriatric cardiology.

Main Body

Etiology of Arrhythmias

The main cause of heart rhythm disorders in old age is organic heart disease, primarily coronary artery disease (CAD), hypertension, and circulatory insufficiency; less commonly rheumatism, myocarditis, and cardiomyopathies. Other causes include disturbances of acid-base balance (acidosis) and electrolyte balance (hypokalemia, hypomagnesemia); disorders of neurohumoral regulation (pronounced sympathicotonia or vagotonia); intoxications; pathological reflexes from the digestive system (colitis, cholecystitis, diaphragmatic hernia, etc.), respiratory system (chronic pneumonia, pulmonary emphysema, etc.), and spine (cervical and thoracic osteochondrosis). In elderly and senile people, the arrhythmogenic effect of many medications (diuretics, antiarrhythmics, cardiac glycosides, narcotics, psychotropic drugs, glucocorticoids, atropine, etc.) is more common. There is often a combination of several possible causes of arrhythmia, such as CAD + spinal osteochondrosis + hypokalemia [1].

The age-related decline in the functional activity of the sinoatrial node leads to a tendency toward bradycardia characteristic of elderly and senile individuals. In turn, the prolongation of diastole also predisposes to the appearance of heterotopic automaticity. With aging, nervous influences on the heart decrease, while its sensitivity to humoral regulatory factors (catecholamines, acetylcholine, etc.) increases. With age, the number of β-adrenoceptors in the heart decreases, but their sensitivity to mediators increases; the activity of enzymes that inactivate them decreases; the reuptake of mediators by nerve terminals diminishes. Catecholamines and acetylcholine remain in their active sphere for a longer period, provoking various heart rhythm disorders in older people. In old age, the influences of the autonomic nervous system on the heart decrease, with the reduction of parasympathetic influences being more pronounced than that of sympathetic ones. Electrolyte changes in the myocardium, particularly involving potassium ions, also play a significant role in the development of arrhythmias. With age, the content of potassium ions in myocardial cells, erythrocytes, and blood plasma decreases, creating conditions for slow diastolic depolarization in cells and the appearance of heterotopic foci.

Clinical Presentation Diagnosis of Arrhythmias

The analysis of clinical manifestations and diagnosis of arrhythmias in elderly and senile patients is associated with certain difficulties. The reason for seeking medical attention is often not the sensation of an irregular heart rhythm (thumps, interruptions), but general symptoms accompanying arrhythmias weakness, fatigue, anxiety, a feeling of unsteadiness when walking, and other complaints that the patient and their relatives may consider more as manifestations of old age than of cardiovascular pathology. Therefore, the main methods for diagnosing arrhythmias in geriatric patients are direct objective examination (cardiac auscultation, pulse examination) and instrumental examination, including electrocardiography (ECG) in 12 standard leads in the supine position and during various functional tests. The most common of these include physical exertion (Master's test, bicycle ergometry), respiratory tests (Valsalva maneuver, hyperventilation), and orthostatic testing [2]. The use of ECG in combination with these stress tests makes it possible to diagnose the presence of arrhythmia in elderly patients, as well as to clarify the conditions under which it appears or worsens, the localization of the heterotopic automaticity focus, and the quantitative characteristics of the arrhythmia. However, it should be noted that in 20–30% of elderly patients, arrhythmia is detected only with longer-term monitoring of heart rhythm, necessitating 24-hour Holter monitoring. If this

is technically difficult, rhythmography can be of great diagnostic value. In addition to determining the qualitative and quantitative characteristics of arrhythmia, rhythmography allows assessment of the pharmacodynamics of antiarrhythmic drugs, as well as preliminary determination of the patient's baseline autonomic tone, which is important for selecting the optimal antiarrhythmic agent. The use of rhythmography in combination with orthostatic testing and graded exercise on a bicycle ergometer can increase the diagnostic capability of the method to 95–100%.

Treatment of Arrhythmias

Pharmacotherapy for cardiac arrhythmias in elderly and senile patients has specific features; ignoring them significantly reduces treatment efficacy and increases the risk of adverse, often dangerous, effects of antiarrhythmic drugs. These features are related to age-related impairments in absorption, distribution, metabolism, and excretion of drugs, age-related changes in receptors, and changes in patient attitudes toward treatment.

The choice of medication must be guided by the form and characteristics of the arrhythmia, the nature and severity of the underlying disease, the presence of complications and comorbidities, the patient's psycho-emotional status, and information about previous treatment (drugs, doses, duration).

Rapid selection of an effective antiarrhythmic drug, for example for the treatment of extrasystole, can be achieved by conducting a trial drug test. This involves a single dose of the antiarrhythmic drug equal to 1/3 of the daily dose. Treatment is best carried out with one drug, prescribed at an adequate dose. However, it should be taken into account that tolerance to most antiarrhythmic drugs is reduced in elderly patients, so treatment should be started with lower doses than in young or middle-aged patients. Subsequently, if the drug is well tolerated and no dangerous side effects occur, the dose may be increased. Most drugs are characterized by an increased elimination half-life. In addition, for some drugs (isoptin, anaprilin), an increase in elimination half-life has been observed during course treatment. This feature must be considered to determine the optimal regimen for administering single doses. It is important to emphasize that not all side effects should be unequivocally interpreted as a signal to reduce the dose or discontinue the drug. On the other hand, the development of dangerous side effects in patients receiving quinidine, cardiac glycosides, or Novocain amide requires immediate discontinuation of the drug. Treatment with β-blockers should not be stopped abruptly, as this may lead to a deterioration in the patient's condition (development of myocardial infarction, appearance of life-threatening arrhythmias). In such cases, it is advisable to reduce the dose of the β-blocker to a minimum for 1–2 days and only then discontinue the drug [3].

Prevention of polypharmacy remains a pressing problem in the treatment of elderly and senile patients. Rational pharmacotherapy for arrhythmia should, if possible, include a drug that has multiple mechanisms of therapeutic action. Examples of this approach include: β-blockers, isoptin, or cordarone in the treatment of patients with CAD, or the use of cardiac glycosides for extrasystole or permanent atrial fibrillation in the setting of heart failure. A very important condition for successful treatment of heart rhythm disorders is the normalization of homeostasis: correction of acid-base disturbances and electrolyte imbalances, elimination of excessive sympathetic influences on the heart, treatment of heart failure, etc. General supportive therapy (multivitamins, anabolic steroids retabolil, nerobolil; potassium- and magnesium-containing drugs asparkam, panangin) often leads to a reduction or even complete resolution of extrasystolic arrhythmia. Intravenous drip infusion of a polarizing mixture (250 ml of 5–10% glucose solution, 3 g of potassium chloride, 8 IU of insulin or 250 ml of 5–10% glucose solution, 20–30 ml of 10% panangin solution, 8 IU of insulin) is particularly effective and should, whenever possible, be combined with the antiarrhythmic drugs being used. This is because potassium ions, by enhancing the effect of all antiarrhythmic drugs, accelerate the process of normalizing sinus rhythm. Combination antiarrhythmic therapy (two or more antiarrhythmic drugs) is justified only when monotherapy with drugs having different mechanisms of action fails.

The use of antiarrhythmic drugs in elderly patients, especially when administered intravenously, requires mandatory monitoring of the patient's condition pulse, blood pressure, ECG (heart rhythm monitoring).

Paroxysmal heart rhythm disorders in elderly and senile patients deserve special attention and require rapid diagnosis and emergency treatment. Prolonged arrhythmia attacks in this patient population very quickly lead to the development of severe cardiovascular, coronary, and cerebral insufficiency and are potential factors for mortality. Treatment should begin immediately after confirmation of the diagnosis by ECG, using intravenous administration of highly active antiarrhythmic drugs. For ventricular paroxysmal tachycardia, intravenous administration of lidocaine, mexiletine, or cordarone is indicated; for supraventricular arrhythmias, isoptin, cordarone, or cardiac glycosides are indicated. For all paroxysmal tachycardias or tachyarrhythmias, intravenous drip infusion of a polarizing mixture is advisable. The patient's severe condition, rapid progression of circulatory insufficiency and worsening of coronary circulation, as well as the ineffectiveness of drug therapy, are absolute indications for electrical defibrillation of the heart. In patients with acute myocardial infarction and paroxysmal arrhythmia, defibrillation is the primary method of rhythm normalization, since the use of all antiarrhythmic drugs aggravates arterial hypotension and the decrease in contractility of the ischemic myocardium [1].

In cases of conduction disturbances (sinoatrial, atrial, and atrioventricular blocks, bundle branch blocks), treatment measures should first be directed at treating the underlying disease complicated by these conditions improving coronary circulation in CAD, anti-inflammatory therapy for rheumatism, discontinuing drugs that contribute to conduction disturbances (antiarrhythmics, cardiac glycosides, etc.), and prescribing drugs that improve metabolic processes in the myocardium (riboxin, ATP, anabolic steroids, vitamins, etc.).

In cases of severe bradycardia (heart rate less than 40 beats/min), which poses a real threat to the patient's life, parenteral administration of cholinolytics (atropine sulfate), sympathomimetics (isoprenaline, alupent, etc.) is used; if ineffective, rate-increasing endocardial ventricular pacing or transesophageal left atrial pacing is employed.

When Adam-Stokes-Morgagni syndrome occurs, the main efforts should be directed at restoring effective cardiac activity. After diagnosis, immediate measures should be taken, including closed-chest cardiac massage and artificial ventilation, intravenous administration of sympathomimetic drugs, atropine sulfate, and calcium chloride.

In elderly patients with sick sinus syndrome (sinoatrial blocks, sinus and atrial extrasystole, severe bradycardia, and other clinical

manifestations), extreme caution is required when prescribing antiarrhythmic drugs to prevent paroxysmal arrhythmias, as there is a risk of cardiac asystole. In these cases, drugs with an anticholinergic component of action (rhythmilen, belladonna preparations) may be recommended [4].

Most known antiarrhythmic drugs are among the potent medications. Moreover, in some patients, they may cause a paradoxical effect worsening the arrhythmia. Therefore, uncontrolled treatment of heart rhythm disorders with antiarrhythmic drugs in elderly and senile people is unacceptable.

Valvular Heart Disease

In recent decades, the proportion of elderly and senile patients with rheumatic valvular heart disease among those with cardiovascular conditions has increased. This refers to valvular defects acquired at a young age, whose auscultatory signs change with age. This is due to the addition of atherosclerotic processes and changes in several parameters of systemic and intracardiac hemodynamics in older age groups.

Often, the doctor explains auscultatory "findings" as atherosclerotic heart disease, without considering the possibility of past rheumatism.

At the same time, recognizing valvular defects in older age groups becomes fundamentally important, as it sometimes allows the development of heart failure to be linked to the activation of rheumatism and, consequently, to adjust therapy. In this regard, the physician's geriatric awareness often permits correct interpretation of micro-symptoms during cardiac percussion and auscultation, while the use of instrumental research methods helps objectify the picture [5].

Clinical Presentation Diagnosis of Valvular Heart Disease

In elderly and very old patients, a systolic murmur at the apex and at the Botkin point is most frequently heard, which arises due to the development of atherosclerotic cardiosclerosis. However, it may also be caused by rheumatic involvement of the mitral valve. This necessitates differential diagnosis between mitral regurgitation of atherosclerotic and rheumatic origin. Percussion of the heart is not very informative in this regard, since even in healthy elderly and old people, the left border of relative cardiac dullness is shifted leftward from the midclavicular line by up to 1.5 cm. The apical beat is rarely increased. A more reliable indicator is the widening of the upper border of cardiac dullness, especially if it is combined with a leftward widening of cardiac dullness greater than 1.5 cm.

In mitral regurgitation of rheumatic origin, as a rule, a systolic murmur is heard against a weakened first heart sound, occupying most of systole. It is usually decrescendo, and the first sound is difficult to discern against its background. The presence of equal loudness of the second sound over the aorta and the pulmonary trunk in an elderly patient is equivalent to an accent over the pulmonary trunk, since in elderly individuals the second sound is always accentuated over the aorta.

Phonocardiography (PCG) significantly objectifies auscultation data. Thus, mitral regurgitation associated with rheumatic involvement is often accompanied by splitting of the first sound due to the loss of its middle part the valvular component. The systolic murmur occupies more than 2/3 of systole, although it has low amplitude. As a rule, the murmur is connected to the first sound and is well expressed in the axillary region. In older age groups, the systolic murmur is well conducted to the pulmonary trunk, and sometimes even has a greater amplitude than over the mitral valve. This is explained by the fact that long-standing blood regurgitation into the left atrium leads to its pronounced dilatation and elongation, bringing the pathways of backward blood flow into the atrium closer to the standard microphone placement site over the pulmonary trunk. The presence of equal amplitude of the aortic and pulmonary components of the second heart sound at the apex or the Botkin point is a sign that may indicate mitral regurgitation associated with a rheumatic process. If the pulmonary component of the second sound is higher than the aortic component, then the detected mitral regurgitation should be considered related to rheumatism, while excluding other pathological conditions that could cause pulmonary hypertension (e.g., chronic cor pulmonale). Clearly, when the maximum amplitude of the second sound over the pulmonary trunk in an elderly person is accompanied by other signs indicating mitral regurgitation, the origin of the latter is most likely related to rheumatism. In this case, the position of the heart according to ECG data should be determined, because when the heart rotates around its longitudinal axis clockwise (right ventricle forward), an accentuation of the second sound over the pulmonary trunk may be observed. The rheumatic genesis of mitral regurgitation can also be confirmed by radiographic findings (smoothing of the waist of the heart due to dilation of the pulmonary cone and enlargement of the left atrial appendage, rounding of the left ventricular arch). When evaluating a chest X-ray of an elderly person, unambiguous interpretation of leftward heart enlargement is difficult, as it may be caused by either rheumatic valve disease or atherosclerotic involvement. Identifying a narrowed retrocardiac space due to left atrial enlargement (in the first oblique projection) is important, as this is typically associated with mitral regurgitation of rheumatic origin [6].

In atherosclerotic heart disease, the systolic murmur over the mitral orifice is essentially a murmur of relative insufficiency, but it is often loud because blood regurgitation occurs against the background of sclerosis of the mitral valve leaflets. On auscultation, the first sound is weakened; the second sound is always accentuated over the aorta; its splitting is rarely observed (only in left bundle branch block). As a rule, a systolic murmur is also heard over the aorta, and in many cases, it differs in timbre from the murmur at the mitral orifice. On phonocardiography, a short murmur (no more than 1/3 of systole) is usually recorded, maintaining approximately the same amplitude and shape across all recorded frequencies. This latter sign is most important for confirming the association of the murmur with atherosclerotic heart disease. Over the aorta, a systolic murmur of higher amplitude is usually recorded, sometimes taking the form of a low-amplitude diamond-shaped murmur located in the first half of systole. Radiological examination reveals thickening and dilatation of the ascending aorta, unfolding of its arch, and enlargement of the left ventricle.

In elderly and very old patients suffering from CAD, the papillary muscles are often affected, and as a result of their functional insufficiency, mitral valve prolapse develops. In this case, against a weakened first sound, a systolic murmur may be heard, appearing in the middle or end of systole, approaching the accentuated second sound. On phonocardiogram at the Botkin point, a crescendo murmur is most often recorded in the second half of systole, with the second sound having maximum amplitude over the aorta.

When diagnosing combined mitral valve disease in elderly and old people, their "youthful" appearance, the presence of a facial flush, and cyanosis of the lips are striking upon examination. It is important to note the detection of a "thrill" (frémissement cataire)

upon palpation of the precordium, which can be a valuable aid in recognizing mitral stenosis, especially the aphonic form often seen in older age groups. Attention should also be paid to the presence of epigastric pulsation. Usually, in mitral valve disease with predominant stenosis, there is an increase in the area of cardiac dullness to the right and upward. In elderly patients, a marked leftward widening of the border of relative cardiac dullness is generally observed.

As already indicated, combined mitral valve disease in elderly and old people often loses its classic auscultatory picture and becomes aphonic the diastolic murmur disappears, and subsequently the loudness of the systolic murmur decreases. The presence of only a soft systolic murmur is often attributed to atherosclerotic cardiosclerosis, and the valve defect goes unrecognized in the absence of clear anamnestic information. In diagnosing aphonic valve disease, careful study of the medical history, physical examination findings, palpation of the precordium, and determination of the borders of absolute and relative cardiac dullness, as mentioned above, become important. On auscultation, attention should be paid to a snapping first sound, considering that in elderly, even healthy, individuals, the loudness of the first sound at all auscultation points is always less than that of the second sound. Detection of an accentuated and split-second sound over the pulmonary artery indicates the presence of a valve defect. Detection of an opening snap of the mitral valve is an undoubted sign of mitral stenosis.

Phonocardiography significantly expands the diagnostic possibilities for aphonic valve defects. Even in the absence of murmurs, the detection of a high-amplitude, shortened first sound, a split-second sound with an enhanced pulmonary component, an opening snap of the mitral valve, and a prolonged Q–I interval (greater than 0.06 s) allows the diagnosis of mitral stenosis. The presence of a right axis deviation or a non-deviated electrical axis of the heart, “mitral” P waves, and, in atrial fibrillation, coarse fibrillatory waves, can be important aids in recognizing aphonic valve defects. Radiological examination in elderly and old patients does not always reveal typical signs of valve disease, so to detect them, examination of the cardiac shadow in lateral projections with esophageal contrast (enlargement of the left and right atria, right ventricle) is required.

Often in elderly and very old individuals, a loud systolic murmur over the aorta is detected, which may reflect atherosclerotic involvement of the aorta (ejection murmur) or be a sign of aortic stenosis. In differential diagnosis, it should be considered that aortic stenosis is accompanied by a very loud systolic murmur over the aorta, occupying all of systole and radiating to all auscultation points, with the second sound being significantly weakened or inaudible. In atherosclerotic involvement of the aorta, the systolic murmur over it is mainly expressed at the beginning of systole, and the second sound over the aorta is accentuated. On phonocardiography, aortic stenosis shows a high-amplitude diamond-shaped murmur, with the second sound difficult to identify. Electrocardiography shows signs of left ventricular overload; X-ray shows an aortic configuration of the heart [7].

Aortic valve insufficiency is rare in elderly and very old patients. It should be remembered that a protodiastolic murmur over the aorta may appear in old patients with marked dilatation of the ascending aorta and the development of relative valve insufficiency. A distinguishing feature of atherosclerotic aortic valve insufficiency is the good preservation of the second sound over the aorta [5].

Treatment of Valvular Heart Disease

As in younger patients, treatment of rheumatism during its active phase is necessary (antimicrobial, anti-inflammatory therapy). Given the low activity of the process in elderly patients, glucocorticosteroids are rarely prescribed, especially since the risk of complications (increased blood pressure, blood glucose levels, development of peptic ulcers of the gastrointestinal tract) increases with age. Nonsteroidal Anti-Inflammatory Drugs (NSAIDs) are prescribed in minimal daily doses, considering age-related changes in the gastrointestinal tract that increase the risk of gastralgia, ulcerative changes, and bleeding. With age-related decline in the detoxifying function of the liver and excretory capacity of the kidneys, the elimination half-life of drugs is prolonged, leading to rapid development of toxicity. The daily dose of acetylsalicylic acid should not exceed 3.0 g. The use of pyrazolone derivatives is limited (fluid retention). Derivatives of phenylpropionic acid ibuprofen at a daily dose of 0.4–0.6 g; naproxen (naprosyn) 0.5 g per day; phenylacetic acid voltaren 0.075 g per day; indoleacetic acid indomethacin (methindol) up to 0.1 g per day are successfully used. The latter can be administered as rectal suppositories of 0.05 g twice daily, thus avoiding gastric irritation [8].

When prescribing cardiac glycosides, a strictly individual approach is necessary; as a guideline, the dose should be approximately half that used in younger patients. Vitamin therapy is indicated. The use of anabolic steroids methandienone (nerobol) 0.005 g per day is possible in the absence of neoplastic processes (prostate adenoma, etc.). When prescribing diuretics, the addition of aldosterone antagonists spironolactone (veroshpiron) 0.075 g per day is mandatory.

Conclusion

Thus, the analysis of the presented data indicates that arrhythmias and valvular heart disease in elderly and senile individuals have significant etiopathogenetic, clinical, and therapeutic features. The main causes of rhythm disorders are organic heart disease (CAD, hypertension), electrolyte disturbances, imbalance of autonomic regulation, and the arrhythmogenic effect of drugs, with a combination of several factors often being observed. Clinical diagnosis of arrhythmias in geriatric patients is challenging due to the nonspecific nature of complaints, which requires the widespread use of instrumental methods (ECG with stress tests, Holter monitoring, rhythmography). Treatment must be individualized, starting with low doses of antiarrhythmics, taking into account their delayed elimination and increased risk of side effects, with priority given to monotherapy and correction of homeostasis.

Regarding valvular lesions, the key problem is the differential diagnosis between rheumatic origin (often aphonic forms of valve defects) and atherosclerotic changes of the heart. A comprehensive assessment of auscultatory, phonocardiography, electrocardiographic, and radiological data plays a decisive role here. Treatment of such patients requires cautious use of anti-inflammatory drugs (due to the risk of gastrointestinal complications) and cardiac glycosides (in reduced doses), with mandatory monitoring of liver and kidney function. Timely recognition and adequate treatment of arrhythmias and valvular pathology in the elderly can improve quality of life, prevent the development of severe heart failure and life-threatening conditions, which underscores the need for geriatric awareness among physicians of all specialties [9-20].

References

1. Matsumoto Y, Mori Y, Kageyama S, Sato H (2026) Long-term changes in QT interval in hemodialysis patients. Renal Failure 48: 2641970.
2. Zhao N, Li W, Chen Q, Zhang Q, Yu Q, et al. (2026) In-hospital and short-term outcomes in patients with atrial fibrillation undergoing chimeric antigen receptor-T cell therapy. Hematology 31: 2662692.
3. Cataldo-Miranda P, Koh HJW, Stub D, Zoungas S, Kaye DM, et al. (2026) Beta-blocker therapy and all-cause mortality after a coronary event: A matched nested case-control study using the Victorian Cardiac Outcomes Registry linked data. Cardiology 51: 103340.
4. Ismaili D, Virtanen P, Im J, Brunnbauer P, Saleem U, et al. (2026) Higher sensitivity to ouabain-induced toxicity in human induced pluripotent stem cell-derived cardiomyocytes than human adult heart tissue despite similar Na+/K+-ATPase pump current amplitudes. BJP 183: 3093-3110.
5. Liang Z, Bai M, Guo H, Gao Y, Feng W, et al. (2026) Stagger-folding leaflet design for improved durability in transcatheter heart valves. J Mech Behav Biomed Mater 179: 107447.
6. Xiong W, Tan GH, Chen H, Chen MH, Wu RH, et al. (2026) Toxicarioside H induces cytoprotective autophagy by hindering the progression of necroptosis in triple-negative breast cancer cells. Transl Oncol 68: 102779.
7. Takeji Y, Taniguchi T, Morimoto T, Shirai S, Kitai T, et al. (2026) Cardiac Damage Stages and Clinical Outcomes in Patients with Severe Aortic Stenosis. JACC 5: 101729.
8. Kundi H, Leon M, Cohen DJ, Popma A, Kobayashi Y, et al. (2026) Early Postrranscatheter Aortic Valve Replacement Heart Failure Hospitalization and Outcomes in Older Patients. Journal of American Heart Association 15: 1-12.
9. Zhang Y, Carr J, Hancox JC, Harmer SC, Dempsey CE (2026) Quantitative analysis of trafficking defects induced by heterozygous expression of hERG voltage sensor domain variants.channels 20: 2672228.
10. Wu X, Meng J, Yang Y, Sheng D, Liu X, et al. (2026) Comparative efficacy and safety of apixaban and rivaroxaban versus warfarin in atrial fibrillation patients with end-stage renal disease: a systematic review and meta-analysis.Meta-Analysis 48: 2666454.
11. Kang SH, Park SY, Lim YJ, Kim BY, Do JY (2026) Association between ultrafiltration variability and clinical outcomes in patients undergoing hemodialysis.Ren Fail 48: 2620166.
12. Paraskevaidis I, Tsougos E, Kourek C (2026) Quadruple therapy in heart failure: Why, when, and in whom. Curr Probl Cardiol 51: 103352.
13. Fujita E, Hadano J, Hashimoto J, Matsumoto-Miyai K (2026) Co-administration of digoxin and trans-2-decenoic acid ethyl ester improves motor learning performance in mice. Behav Brain Res 25: 116225.
14. Yan Y, Zhou X, Gu M, Ding Y, Zhao X, et al. (2026) Arenobufagin suppresses lung cancer cell growth by disrupting mitochondrial function and inducing relocalization of ATP synthase.Toxico 15: 109067.
15. Myagmardorj R, Fortuni F, Mantegazza V, van Wijngaarden AL, Wu HW, et al. (2026) Extent and Progression of Cardiac Damage in Patients with Primary Mitral Regurgitation Undergoing Surgical Repair. European Journal of Cardio-Thoracic Surgery 68: 1-11.
16. Ho D, Li T, Leow R, Sia CH (2026) Rheumatic heart disease and mitral stenosis. Future Cardiology 22: 541-555.
17. Pereira T, Fernandes RM, Mata E, Azevedo O, Bento D, et al. (2024) Transthyretin amyloid cardiomyopathy in severe aortic stenosis submitted to valve replacement: a multicenter study. Multicenter Study 20: 419-430.
18. Lyon J, Nash PS, Ahmed N, Aram L, Balogun M, et al. (2026) Early Versus Delayed Anticoagulation in Acute Ischemic Stroke According to Atrial Fibrillation Subtype and Time of Diagnosis: Subgroup Analysis of the OPTIMAS Randomized Controlled Trial. Randomized Contolled Trial 57: 1513-1525.
19. Wańha W, Kuźma Ł, Kowalewski M, Iwańczyk S, Grygier J, et al. (2026) Primary Results of the SALAMANDER Registry: A Multicenter Observational Cohort Study. Observational Study 19: e046625.
20. Zhou J, Zhang Z, Lan Y, Yang M, Qiu T, et al. (2026) Long-Term Outcomes of Oral Propranolol for Facial Infantile Hemangioma. Plast Reconstr Surg 157: 142-149.