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Cardiac Arrythmias: Overview, Management, Treatment, and Medical Records Contribution

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Abstract:

Background: Cardiac arrhythmias are significant disorders of heart rhythm and conduction, influenced by genetic, structural, and environmental factors. These conditions range from asymptomatic atrial fibrillation to life-threatening ventricular arrhythmias, necessitating diverse therapeutic approaches. The complexity of arrhythmogenesis stems from electrical remodeling, myocardial ischemia, genetic predispositions, and inflammation.

Aim: This review aims to provide an overview of cardiac arrhythmias, their underlying mechanisms, treatment strategies, and the role of medical records in management.

Methods: A comprehensive search was conducted using PubMed and Google Scholar, focusing on clinical and experimental literature. Keywords such as "cardiac arrhythmias," "ischemic heart disease," and "electrophysiology" guided the literature review. Key findings were synthesized to elucidate pathophysiological mechanisms and therapeutic advancements.

Results: The physiopathology of arrhythmias involves electrical and structural remodeling, ionic disturbances, and genetic and epigenetic factors. Ventricular arrhythmias are acute and severe, while atrial fibrillation progresses chronically, with mechanisms including fibrosis and ectopic activity. Ischemia and inflammation exacerbate arrhythmogenesis through structural damage and cytokine-mediated remodeling. Genetic studies reveal mutations in ion channels and cytoskeletal proteins as critical contributors. Emerging treatments include pharmacological therapies, catheter ablation, and device implantation, supported by robust documentation through medical records.

Conclusion: Advances in understanding cardiac arrhythmias' multifaceted nature have improved diagnostic and therapeutic strategies. Medical records significantly enhance management by documenting progression, treatments, and outcomes, offering insights for personalized care. Future research should focus on integrating genetic, epigenetic, and environmental data to refine preventive and therapeutic approaches.

Keywords: Cardiac arrhythmias, electrophysiology, atrial fibrillation, ventricular arrhythmias, ischemia, inflammation, medical records.

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Introduction:

The original conceptualization of sudden cardiac death in otherwise healthy individuals who frequently experience severe fainting spells is credited to Hippocrates [1]. This phenomenon has since been associated with a range of electrical cardiac disorders, including long-QT syndrome, arrhythmogenic right ventricular dysplasia or cardiomyopathy (Naxos disease), Brugada syndrome, and hypertrophic cardiomyopathy [2]. The 19th-century work of Étienne-Jules Marey marked a significant advancement, as he detailed premature ventricular beats and described various aspects of ventricular contractions. Recent discussions have explored the historical evolution of cardiac electrophysiology in depth [2]. Understanding the physiopathology of cardiac arrhythmias at the subcellular and cellular levels, especially the differentiation between ventricular arrhythmias and atrial fibrillation, remains pivotal for developing innovative pharmacological and non-pharmacological therapies. In clinical settings, atrial fibrillation, characterized as a supraventricular tachyarrhythmia resulting from uncoordinated atrial activation and mechanical dysfunction, represents a prevalent cardiac arrhythmia. This condition arises from structural and electrophysiological alterations within atrial tissues. While atrial fibrillation can occur without underlying cardiac pathology, it is frequently linked to mitral valve disease, heart failure, ischemic heart disease, and hypertension [3,4]. Initial episodes of atrial fibrillation can often be managed pharmacologically, with treatment objectives focused on enhancing survival, minimizing stroke risk, restoring atrial function, reversing ultrastructural remodeling, and alleviating symptoms.

Ventricular arrhythmias, typically catastrophic in nature, demand immediate medical attention, whereas atrial fibrillation is often asymptomatic, undiagnosed, and self-limiting. Over time, atrial fibrillation can progress from a paroxysmal to a chronic form, complicating its management. This progression involves alterations in contributing mechanisms and triggers [5,6]. Key factors driving the progression of atrial fibrillation include pronounced electrical and structural remodeling, changes in conduction and refractoriness, and a predisposition to reentrant circuit formation [7,8]. Additionally, shortened action potentials, reduced refractory periods [9,10,11,12], slowed conduction, and a lower threshold for alternans induction contribute to the condition's pathogenesis [13]. Ultrastructural changes in atrial cardiomyocyte membranes may also play a significant role, though further structural and functional analyses are necessary. These tissue-level alterations profoundly influence conductivity, wave propagation, and reentry potential. Clinical and basic research has facilitated the development of strategies aimed at improving patient quality of life. This review examines experimental and clinical advancements essential for treating cardiac arrhythmias.

General Principles

Cardiac function depends on rhythmic contractions orchestrated by specialized pacemaker cells in mammalian hearts. This coordination is facilitated by a sophisticated electrical conduction system, including the sinoatrial and atrioventricular nodes, the His bundle, right and left bundle branches, fascicles, and Purkinje fibers [14]. These structures interact with cardiomyocytes and other cell types to ensure orderly cardiac function. The cardiac action potential is generated through the highly coordinated activity of plasma membrane ion channel proteins, which enable electrical coupling via gap junctions [15].

In humans, the cardiac action potential comprises five distinct phases (0–4) as described by Larson et al. [16]. Phase 0 involves stimulation by the sinoatrial node, leading to the opening of voltage-activated sodium channels and the generation of a sodium current, which establishes a positive feedback loop. This is followed by rapid repolarization (phase 1), sustained by transient outward potassium currents, and a plateau phase (phase 2) characterized by equilibrium between inward and outward currents. Phase 3 initiates repolarization as calcium currents are inactivated and outward potassium currents dominate. Finally, phase 4 represents the restoration of membrane potential to resting values. Mammalian cardiomyocytes express diverse voltage-gated ion channel subunits that shape action potential waveforms and regulate both automaticity and refractoriness. Interactions among these ionic currents significantly influence ventricular action potential dynamics, as elucidated through molecular genetic studies. In pacemaker cells of the sinoatrial and atrioventricular nodes, as well as the His-Purkinje system, voltage- and calcium-dependent mechanisms play critical roles [18]. The sinoatrial node typically sets the heart rate at 60–100 beats per minute (bpm), with slower intrinsic rates in the atrioventricular node (40–60 bpm) and Purkinje fibers (20–40 bpm). Increases in automaticity may result in sinus tachycardia, driven either physiologically by heightened sympathetic tone or pathologically by hypovolemia, ischemia, or electrolyte imbalances.

Alterations in cellular and electrophysiological properties, such as triggered activity, abnormal automaticity, or reentry, significantly elevate arrhythmia risk. For instance, changes in ionic current expression that decelerate early repolarization and accelerate late repolarization can predispose the myocardium to arrhythmias, including atrial fibrillation [5]. Electrical remodeling, often associated with ion channel gene mutations or myocardial structural disease, further increases arrhythmia susceptibility. In the failing heart, tachycardia-induced remodeling shortens atrial refractoriness, whereas aging or fibrosis slows conduction and prolongs refractoriness [26]. Acute ischemia exacerbates these risks by altering intracellular resistance, shortening action potential duration, and promoting reentrant excitation following reperfusion [27,28]. Recent advances in experimental and computational models have expanded our understanding of cardiac electrophysiology. In silico studies, employing population-based modeling approaches, have enhanced the reliability of arrhythmia risk markers and provided insights into underlying molecular mechanisms [20,32,33]. These developments offer a promising avenue for refining therapeutic interventions.

Genetics

The majority of cardiac arrhythmias are attributed to structural myocardial pathologies; however, they are also influenced by genetic predispositions, environmental risk factors, and epigenetic modifications [39]. These arrhythmias are typically categorized based on their origin, with polymorphic ventricular tachycardia and ventricular fibrillation often linked to primary hereditary arrhythmia syndromes [40,41]. Recent studies, such as the comprehensive review by Wang and Tu [42], have focused on identifying the genetic underpinnings of hereditary arrhythmogenesis. Numerous mutations affecting ion channels critical to cardiac action potentials have been identified. Additionally, mutations have been observed in genes encoding proteins involved in cytoskeletal integrity, calcium handling, sodium transport, and cytokine signaling [43–45]. Structural proteins such as desmin, lamin, titin, and filamin play pivotal roles in maintaining cardiomyocyte integrity and mechanotransduction, with defects in these proteins significantly impacting myocardial stability [46]. Similarly, mutations in genes regulating calcium homeostasis, such as phospholamban, SERCA2, and the ryanodine receptor, have been implicated in arrhythmogenesis. Genome-wide association studies have identified numerous genetic risk variants, predominantly within intergenic or intronic regions, that are associated with atrial fibrillation [47].

MicroRNAs are key regulators of electrical and structural remodeling processes in cardiomyocytes, and their dysregulation can precipitate atrial fibrillation [48]. Long non-coding RNAs have also been implicated in modulating fibrosis, ion channel function, and energy metabolism [49–51]. Epigenetic factors, including DNA methylation and histone modification, may provide additional links between genetic predispositions and atrial fibrillation, although further research is required. Emerging evidence suggests

that histone deacetylases, through gene silencing, may influence the post-transcriptional regulation of cytoskeletal and conductive proteins, thereby contributing to arrhythmogenesis [52–54]. Despite advancements, significant knowledge gaps remain concerning the molecular mechanisms underlying cardiac arrhythmias and their pathogenesis, particularly regarding the interplay between genetic and epigenetic factors [48]. Epigenetics, defined as heritable changes in gene expression that do not involve alterations to the DNA sequence [55], investigates the interactions between external risk factors and genetic regulatory mechanisms. Processes such as non-coding RNA expression, DNA methylation, and histone modifications significantly influence gene expression, impacting cellular structure and function [56,57]. These mechanisms, while either inherited or acquired, have an unclear role in atrial fibrillation regulation and warrant further exploration.

Myocardial Ischemia

Acute coronary occlusion leads to significant pathological changes in cardiomyocytes within the ischemic zone due to an abrupt cessation of oxygen supply, halting oxidative phosphorylation and depleting intracellular energy stores. Consequently, myocyte contractile function is impaired, and ischemic heart disease becomes a major risk factor for life-threatening cardiac arrhythmias [58]. Ischemic injury induces both structural and electrical destabilization due to heightened sympathetic activity and diminished parasympathetic tone, predisposing individuals to arrhythmias [29,59]. Changes in membrane potential, depolarization velocity, and refractory periods in ischemic versus non-ischemic regions further exacerbate electrical instability. For instance, during acute myocardial infarction, increased automaticity within the atrioventricular node can result in focal atrial tachycardia [60]. Parasystole, often triggered by ischemia, arises when an ectopic pacemaker competes with the sinus node for dominance, leading to conduction abnormalities [61,62]. Additionally, ischemia-induced fibrosis disrupts myocardial conduction by reducing velocity and causing unidirectional blocks, wave breaks, and reentry circuits [63,64]. Rapid reperfusion of an ischemic artery, though essential for salvaging viable myocardium, may cause reperfusion injury, aggravating cardiomyocyte damage. This process, characterized by oxidative stress, calcium influx, and altered pH levels, contributes to reperfusion-induced arrhythmias [30,65–68]. The severity of these arrhythmias is closely tied to ischemia duration, with intermediate durations associated with higher incidences of malignant arrhythmias [29]. Ischemia and reperfusion lead to profound structural and electrical remodeling, altering ion channel activity, intracellular ion dynamics, and myocardial conduction. Fibrotic tissue exacerbates these changes, promoting conduction mismatches, unidirectional blocks, and reentrant circuits [72]. Fibroblast proliferation and abnormal automaticity are key contributors to arrhythmia progression, highlighting the potential of anti-fibrotic therapies as preventive strategies [73].

Atrial fibrillation, in ischemic contexts, is typically driven by ectopic activity from pulmonary veins, reentrant circuits, or a combination thereof, resulting in irregular atrial rhythms [74–75]. These electrical disturbances are linked to downregulated repolarizing potassium currents and altered calcium handling, reducing the myocardium's repolarization reserve and increasing susceptibility to early afterdepolarizations [24]. The interplay between altered intracellular calcium dynamics, sodium currents, and gap junction protein expression (e.g., connexin 43) significantly influences ventricular conduction during ischemia. Redistribution or loss of connexin 43 exacerbates conduction abnormalities, predisposing affected myocardium to arrhythmias [83–85]. Animal studies suggest that reducing connexin 43 expression or altering its distribution could mitigate atrial fibrillation risk [88,89]. This comprehensive synthesis underscores the intricate molecular, structural, and electrophysiological dynamics contributing to arrhythmogenesis in myocardial ischemia and highlights potential therapeutic avenues.

Inflammation

The role of inflammation as a contributory factor in cardiac arrhythmias has been historically underestimated. However, its significance in the onset of atrial fibrillation is becoming increasingly evident, as demonstrated by elevated levels of inflammatory proteins in affected individuals [90,91,92]. Cardiac or systemic inflammation is typically part of the body's nonspecific response to injury [93]. In a structurally normal heart, acute inflammation can precipitate supraventricular ectopic beats, whereas in cases of febrile

illness, a heightened incidence of malignant arrhythmias has been documented, though without sudden cardiac death [94]. In compromised hearts, electrophysiological remodeling—including alterations in ion channel expression and ionic homeostasis—can lead to prolonged action potential duration, long-QT syndrome, Torsade de Pointes, atrioventricular block, and other repolarization abnormalities that exacerbate myocardial instability [95]. Inflammatory cytokines contribute to arrhythmogenic syndromes through mechanisms such as inflammatory channelopathies, disruptions in ion homeostasis, and ultrastructural remodeling. The anti-inflammatory effects of statins have been shown to reduce atrial end-refractory periods and atrial fibrillation duration in both animal and human studies [96,97]. The emergence of cardiac arrhythmias in patients with COVID-19 has been increasingly reported in recent studies [98,99,100]. These arrhythmias may result from elevated inflammatory cytokines, including TNF, IL-1, and IL-6, which exert direct effects on cardiac function and induce systemic alterations [95]. In severe COVID-19 cases, the use of glucocorticoids or IL-6 receptor antagonists has been associated with reduced cardiovascular mortality [101].

Diet and Metabolic Disorders

A pathological association between dietary imbalances (e.g., dyslipidemia, obesity, Type 2 diabetes, and insulin resistance) and cardiac dysfunction has been proposed. Under normal physiological conditions, the heart relies on free fatty acids as its primary energy source, maintaining a regulated balance between lipid uptake and oxidation. Metabolic disorders disrupt this equilibrium, leading to excessive lipid levels that surpass adipocyte storage capacity, resulting in intracellular lipid droplet accumulation and subsequent cardiac dysfunction. Such myocardial lipid accumulation impairs fatty acid metabolism, induces inflammation, and heightens the risk of sustained or fatal arrhythmias [102,103,104]. Moreover, excessive free fatty acids amplify inflammatory responses [105]. The toxic effects of free fatty acids, coupled with the activation of intracellular signaling pathways, result in ion channel remodeling, which adversely impacts cardiac electrical activity and conduction [106,107,108]. Protective effects of polyunsaturated fatty acids, through mechanisms involving modulation of channel gating, membrane properties, and cardiac connexins, have been highlighted [88,109,110]. These findings underscore the intricate relationship between dietary factors, inflammation, and the development of arrhythmias.

Pharmacotherapy

The Vaughan Williams classification system (referenced in Larson et al. [16]) remains a cornerstone for categorizing anti-arrhythmic drugs based on their interaction with ionic channels and their effects on action potentials, sinus node function, and atrioventricular conduction. Class I drugs primarily target sodium channel blockade, inhibiting the rapid inward sodium current, thereby modulating cardiac depolarization, conduction, and prolonging repolarization through delayed rectifier potassium channel inhibition. These agents also influence action potential and effective refractory period durations, impacting automaticity. Class II medications, or beta-blockers, mitigate sympathetic activity, reducing the rate of initial depolarization and thereby suppressing automaticity and conduction velocity [111]. While beta-blockers are noted to decrease sudden death risk in some studies [112], contradictory findings exist [113,114]. Nonetheless, combining beta-blockers with implantable cardiac defibrillators has demonstrated substantial clinical benefits [115,116,117].

Class III agents, such as potassium channel blockers, primarily prolong repolarization by inhibiting delayed rectifier potassium channels [118,119,120]. Class IV drugs, calcium channel blockers, act predominantly on the atrioventricular node by blocking slow inward calcium currents, extending the effective refractory period without significantly affecting cardiomyocytes or the His–Purkinje system [121,122]. Although their utility in ventricular tachycardia is limited, they play a role in managing catecholaminergic polymorphic ventricular tachycardia (an inherited tachycardia in structurally normal hearts) and idiopathic left ventricular tachycardia [16]. Beyond the Vaughan Williams classification, certain agents demonstrate notable anti-arrhythmic properties. For instance, ranolazine, primarily an antianginal medication, exerts anti-arrhythmic effects through late sodium channel (INa-L) blockade, reducing excitability and prolonging the end-refractory period, which limits atrial activation [124,125,126]. Despite

promising actions, the role of late INa inhibition by ranolazine in managing atrial fibrillation remains unconfirmed, with clinical trials such as RAID, MERLIN-TIMI, and RESTYLE-HCM yielding mixed results [125,127,128]. Ivabradine, another antianginal agent, reduces heart rate through mixed sodium–potassium current blockade without affecting inotropy, though it is associated with an elevated risk of atrial fibrillation due to symptomatic bradycardia [129,130,131,132].

Adenosine acts on sinoatrial and atrioventricular nodes, reducing automaticity and effectively addressing repetitive or paroxysmal ventricular tachycardia [133,134,135]. Similarly, digoxin influences both mechanical and electrophysiological cardiac functions via Na+/K+ ATPase inhibition, raising intracellular calcium and enhancing vagal tone. This results in reduced conduction velocity and extended effective refractory periods at the atrioventricular node [16]. However, digoxin's use is now limited to advanced heart failure and refractory atrial fibrillation due to its association with arrhythmia-related mortality risks [136,137]. Although pharmacological interventions are a mainstay in arrhythmia management, they carry risks, including proarrhythmic effects, particularly in patients with structural heart disease [138]. Clinical trials such as the Cardiac Arrhythmia Suppression Trials (CAST I and II) have provided valuable insights but failed to demonstrate substantial protection in severe arrhythmias, leading to their premature termination [139]. A deeper understanding of arrhythmogenic mechanisms at the molecular, cellular, and tissue levels is imperative to advance targeted pharmacological therapies, potentially in conjunction with non-pharmacologic interventions.

Implantable Devices

Implantable electronic pacemakers have become a standard for managing symptomatic bradycardia caused by atrioventricular or sinus node dysfunction. Despite significant technological advances, limitations such as high costs, risks of infection, and device failure persist. Recent developments focus on battery-less devices and gene-therapy-based approaches like biological pacemakers, which modify cardiomyocytes to generate automaticity. However, these methods require further research on gene expression and cellular coupling mechanisms. Implantable cardioverter defibrillators (ICDs) have demonstrated effectiveness in preventing fatal arrhythmias. While these devices improve survival rates, especially when combined with pharmacotherapy, they cannot prevent arrhythmia recurrence. Issues such as painful shocks and reduced quality of life remain concerns, and further studies are needed to optimize their integration with pharmacological treatments.

Catheter Ablation

Catheter ablation has shown superior efficacy compared to pharmacotherapy for managing recurrent arrhythmias, particularly atrial fibrillation. It targets specific areas of the heart, such as pulmonary veins and ganglionated plexi, using radiofrequency or cryotherapy. While effective, the procedure is associated with risks, including collateral damage (e.g., stroke or pulmonary vein stenosis) and recurrence of arrhythmias due to recovered conduction. Advances in electroanatomic mapping and imaging technologies have improved outcomes, though challenges like mapping resolution persist.

Ischemic Conditioning

Ischemic conditioning, involving repeated arterial occlusion/reperfusion cycles, has emerged as a potential protective measure against ischemia-reperfusion-induced arrhythmias. Experimental studies suggest its benefits in preserving cardiac function and reducing arrhythmias. However, its clinical application is limited due to the unpredictable nature of acute myocardial events and the narrow therapeutic time window. Recent research highlights the promise of post-ischemic conditioning, which may offer significant antiarrhythmic properties and complement existing treatment strategies. These non-pharmacologic interventions provide substantial benefits but require continued innovation and validation to address their limitations and expand their applicability in clinical practice [140].

Role of Medical Secretary and Medical Records in Cardiac Arrythmias:

The management of cardiac arrhythmias involves multidisciplinary teams where medical secretaries and medical records play pivotal roles in ensuring efficient clinical workflows, accurate documentation, and continuity of care. These components support the diagnosis, treatment, and monitoring processes, enhancing patient outcomes and contributing to the effectiveness of healthcare delivery.

Medical Secretary as a Liaison in Care Coordination

Medical secretaries serve as essential intermediaries between patients, physicians, and other healthcare professionals. In the context of cardiac arrhythmias, these professionals facilitate the scheduling of diagnostic tests such as electrocardiograms (ECGs), Holter monitoring, or echocardiograms, ensuring timely appointments and follow-ups. By managing communication channels, they help reduce delays in critical diagnostics, which is particularly important for arrhythmias like atrial fibrillation or ventricular tachycardia, where prompt intervention can significantly impact outcomes. Furthermore, medical secretaries organize multidisciplinary meetings, where cardiologists, electrophysiologists, and allied health professionals discuss complex cases. Their role in maintaining accurate schedules, distributing meeting agendas, and ensuring access to relevant medical records ensures that all team members are informed and prepared. This coordination supports personalized treatment plans and reduces the risk of communication breakdowns.

Medical Records in Documentation and Decision-Making

Medical records are central to the management of cardiac arrhythmias. They serve as comprehensive repositories of patient histories, including prior episodes of arrhythmias, risk factors such as hypertension or diabetes, and medication use. Accurate records ensure that healthcare providers can make informed decisions based on a complete clinical picture. For instance, understanding a patient's history of anticoagulation therapy is critical when managing atrial fibrillation to prevent thromboembolic complications. In cardiac care, medical records also support the documentation of non-pharmacologic interventions such as catheter ablation or implantable devices. Recording procedural details, including the location of ablation and device settings, facilitates continuity of care and enables monitoring of long-term outcomes. Additionally, medical records are vital for evaluating the efficacy of interventions by documenting recurring arrhythmias or adverse events, providing valuable data for both clinicians and researchers.

Ensuring Compliance and Data Security

Compliance with legal and ethical standards in cardiac arrhythmia management is crucial, and medical secretaries and records systems play key roles in achieving this. Medical secretaries ensure that patient data is appropriately shared in compliance with regulations like the Health Insurance Portability and Accountability Act (HIPAA) or other regional data protection laws. They manage consent forms and ensure that sensitive patient information is handled securely, minimizing the risks of breaches. Modern electronic medical records (EMRs) systems enhance compliance through automated alerts, such as reminders for anticoagulation monitoring or follow-up visits after device implantation. These features help mitigate risks associated with treatment lapses, ensuring adherence to clinical guidelines. For example, regular updates in EMRs can help healthcare providers monitor whether patients with implantable cardioverter defibrillators (ICDs) have experienced painful shocks, prompting timely adjustments in therapy.

Supporting Research and Quality Improvement Initiatives

Medical secretaries and records are integral to the research efforts aimed at advancing arrhythmia management. High-quality data derived from accurate medical records enable retrospective studies to identify trends, assess treatment efficacy, and refine therapeutic protocols. For instance, analyzing records of catheter ablation patients can provide insights into recurrence rates and factors influencing success, guiding clinical decision-making and future research. Moreover, these roles support quality improvement initiatives. Medical secretaries help track key performance indicators (KPIs), such as the average time from

diagnosis to intervention or patient satisfaction scores. These metrics allow healthcare facilities to identify areas for improvement and implement strategies that enhance care delivery for arrhythmia patients.

Facilitating Patient Education and Engagement

Another critical function of medical secretaries is their involvement in patient education. They assist in disseminating educational materials about cardiac arrhythmias, treatment options, and lifestyle modifications. By providing clear instructions on follow-up care or medication adherence, medical secretaries empower patients to take an active role in their health. Additionally, medical records contribute to patient engagement by enabling clinicians to use visual aids, such as trend charts of heart rate variability, during consultations. Such tools can improve patients' understanding of their condition and foster collaborative decision-making, ultimately improving adherence to treatment plans. Medical secretaries and medical records form the backbone of effective management strategies for cardiac arrhythmias. Through meticulous coordination, comprehensive documentation, and adherence to regulatory standards, they enhance the quality of care provided to patients. Their roles also extend to supporting research, quality improvement initiatives, and patient education, underscoring their multifaceted contributions to healthcare. As cardiac arrhythmia management continues to evolve with technological advancements, the integration of sophisticated records systems and the professional development of medical secretaries will remain critical to achieving optimal patient outcomes.

Conclusion:

Cardiac arrhythmias remain a major clinical challenge due to their diverse etiology and complex pathophysiology. Atrial fibrillation, a common form of arrhythmia, arises from uncoordinated atrial activation and mechanical dysfunction linked to structural and electrophysiological remodeling. Ventricular arrhythmias, though less common, present acute and severe risks requiring immediate intervention. Recent advancements have elucidated the roles of electrical and structural remodeling, genetic predispositions, and environmental factors such as ischemia and inflammation in arrhythmogenesis. These discoveries have expanded therapeutic options, including pharmacological treatments, catheter ablation, and implantable devices like cardioverter-defibrillators. Medical records play a pivotal role in managing cardiac arrhythmias by providing detailed documentation of patient history, disease progression, and treatment outcomes. This data supports personalized care and facilitates the evaluation of therapeutic efficacy. Additionally, comprehensive records enhance research efforts, enabling the identification of trends and contributing factors in arrhythmogenesis. The integration of genetic and epigenetic insights has further refined the understanding of arrhythmias, revealing mutations in ion channels and structural proteins as key contributors. Epigenetic mechanisms, such as DNA methylation and histone modification, offer promising avenues for future research, particularly in understanding atrial fibrillation's regulation. Despite significant progress, challenges remain. The interplay of inflammation, ischemia, and arrhythmias warrants further investigation to identify preventive strategies and targeted therapies. Emerging evidence linking inflammation to arrhythmogenesis underscores the need for anti-inflammatory approaches to complement existing treatments. In conclusion, a multidisciplinary approach that integrates clinical expertise, advanced diagnostics, and comprehensive medical records is essential for managing cardiac arrhythmias. Future efforts should prioritize personalized medicine, leveraging genetic, epigenetic, and environmental data to improve outcomes and quality of life for affected individuals.

References:

1. Mirchandani, S.; Phoon, C.K. Sudden cardiac death: A 2400-year-old diagnosis? *Int. J. Cardiol.* **2003**, *90*, 41–48.
2. Luderitz, B. Historical perspectives of cardiac electrophysiology. *Hell. J. Cardiol.* **2009**, *50*, 3–16.
3. O'Neill, M.; Jaïs, P.; Hocini, M.; Sacher, F.; Klein, G.J.; Clémenty, J.; Haïssaguerre, M. Catheter ablation for atrial fibrillation. *Circulation* **2007**, *116*, 1515–1523.

4. Levy, S. Factors predisposing to the development of atrial fibrillation. *Pacing Clin. Electrophysiol.* **1997**, *20*, 2670–2674.
5. Schotten, U.; Verheule, S.; Kirchhof, P.; Goette, A. Pathophysiological mechanisms of atrial fibrillation: A translational appraisal. *Physiol. Rev.* **2011**, *91*, 265–325.
6. Schotten, U.; Dobrev, D.; Platonov, P.G.; Kottkamp, H.; Hindricks, G. Current controversies in determining the main mechanisms of atrial fibrillation. *J. Intern. Med.* **2016**, *279*, 428–438.
7. Allessie, M.; Ausma, J.; Schotten, U. Electrical, contractile and structural remodeling during atrial fibrillation. *Cardiovasc. Res.* **2002**, *54*, 230–246.
8. Nattel, S.; Harada, M. Atrial remodeling and atrial fibrillation: Recent advances and translational perspectives. *J. Am. Coll. Cardiol.* **2014**, *63*, 2335–2345.
9. Iwasaki, Y.K.; Nishida, K.; Kato, T.; Nattel, S. Atrial fibrillation pathophysiology: Implications for management. *Circulation* **2011**, *124*, 2264–2274. [
10. Lalani, G.G.; Schricker, A.; Gibson, M.; Rostamian, A.; Krummen, D.E.; Narayan, S.M. Atrial conduction slows immediately before the onset of human atrial fibrillation: A bi-atrial contact mapping study of transitions to atrial fibrillation. *J. Am. Coll. Cardiol.* **2012**, *59*, 595–606.
11. Skibsbye, L.; Jespersen, T.; Christ, T.; Maleckar, M.M.; van den Brink, J.; Tavi, P.; Koivumäki, J.T. Refractoriness in human atria: Time and voltage dependence of sodium channel availability. *J. Mol. Cell. Cardiol.* **2016**, *101*, 26–34.
12. Zheng, Y.; Xia, Y.; Carlson, J.; Kongstad, O.; Yuan, S. Atrial average conduction velocity in patients with and without paroxysmal atrial fibrillation. *Clin. Physiol. Funct. Imaging* **2017**, *37*, 596–601.
13. Narayan, S.M.; Franz, M.R.; Clopton, P.; Pruvot, E.J.; Krummen, D.E. Repolarization alternans reveals vulnerability to human atrial fibrillation. *Circulation* **2011**, *123*, 2922–2930.
14. Stătescu, C.; Sascău, R.A.; Georgescu, C.A. Functional Anatomy in Arrhythmias and Vascular Support of the Conduction System. In *Cardiac Arrhythmias: From Basic Mechanism to State-of-the-Art Management*; Kibos, A.S., Knight, B.P., Essebag, V., Fishberger, S.B., Slevin, M., Țintoiu, I.C., Eds.; Springer: London, UK, 2014; pp. 35–42.
15. Kanno, S.; Saffitz, J.E. The role of myocardial gap junctions in electrical conduction and arrhythmogenesis. *Cardiovasc. Pathol.* **2001**, *10*, 169–177.
16. Larson, J.; Rich, L.; Deshmukh, A.; Judge, E.C.; Liang, J.J. Pharmacologic Management for Ventricular Arrhythmias: Overview of Anti-Arrhythmic Drugs. *J. Clin. Med.* **2022**, *11*, 3233.
17. Tse, G. Mechanisms of cardiac arrhythmias. *J. Arrhythmia* **2016**, *32*, 75–81.
18. Lakatta, E.G.; Vinogradova, T.; Lyashkov, A.; Sirenko, S.; Zhu, W.; Ruknudin, A.; Maltsev, V.A. The integration of spontaneous intracellular Ca2+ cycling and surface membrane ion channel activation entrains normal automaticity in cells of the heart's pacemaker. *Ann. N. Y. Acad. Sci.* **2006**, *1080*, 178–206.
19. Jalife, J.; Delmar, M.; Anumonwo, J.; Berenfeld, O.; Kalifa, J. *Basic Cardiac Electrophysiology for the Clinician*, 2nd ed.; Wiley-Blackwell: Hoboken, NJ, USA, 2009.
20. Vagos, M.; van Herck, I.G.M.; Sundnes, J.; Arevalo, H.J.; Edwards, A.G.; Koivumaki, J.T. Computational Modeling of Electrophysiology and Pharmacotherapy of Atrial Fibrillation: Recent Advances and Future Challenges. *Front. Physiol.* **2018**, *9*, 1221.
21. Ravens, U.; Wettwer, E. Ultra-rapid delayed rectifier channels: Molecular basis and therapeutic implications. *Cardiovasc. Res.* **2011**, *89*, 776–785.

22. Skibsbye, L.; Poulet, C.; Diness, J.G.; Bentzen, B.H.; Yuan, L.; Kappert, U.; Matschke, K.; Wettwer, E.; Ravens, U.; Grunnet, M.; et al. Small-conductance calcium-activated potassium (SK) channels contribute to action potential repolarization in human atria. *Cardiovasc. Res.* **2014**, *103*, 156–167.

23. Liang, B.; Nissen, J.D.; Laursen, M.; Wang, X.; Skibsbye, L.; Hearing, M.C.; Andersen, M.N.; Rasmussen, H.B.; Wickman, K.; Grunnet, M.; et al. G-protein-coupled inward rectifier potassium current contributes to ventricular repolarization. *Cardiovasc. Res.* **2014**, *101*, 175–184.

24. Tomaselli, G.F.; Marban, E. Electrophysiological remodeling in hypertrophy and heart failure. *Cardiovasc. Res.* **1999**, *42*, 270–283.

25. Nattel, S. Ionic determinants of atrial fibrillation and Ca^{2+} channel abnormalities: Cause, consequence, or innocent bystander? *Circ. Res.* **1999**, *85*, 473–476.

26. Shah, M.; Akar, F.G.; Tomaselli, G.F. Molecular basis of arrhythmias. *Circulation* **2005**, *112*, 2517–2529.

27. Opie, L.H.; Coetzee, W.A. Role of calcium ions in reperfusion arrhythmias: Relevance to pharmacologic intervention. *Cardiovasc. Drugs Ther.* **1988**, *2*, 623–636.

28. Cascio, W.E.; Yang, H.; Johnson, T.A.; Muller-Borer, B.J.; Lemasters, J.J. Electrical properties and conduction in reperfused papillary muscle. *Circ. Res.* **2001**, *89*, 807–814.

29. Manning, A.S.; Hearse, D.J. Reperfusion-induced arrhythmias: Mechanisms and prevention. *J. Mol. Cell. Cardiol.* **1984**, *16*, 497–518.

30. Tanaka, K.; Hearse, D.J. Reperfusion-induced arrhythmias in the isolated rabbit heart: Characterization of the influence of the duration of regional ischemia and the extracellular potassium concentration. *J. Mol. Cell. Cardiol.* **1988**, *20*, 201–211.

31. Noble, D. A modification of the Hodgkin–Huxley equations applicable to Purkinje fibre action and pace-maker potentials. *J. Physiol.* **1962**, *160*, 317–352.

32. Passini, E.; Britton, O.J.; Lu, H.R.; Rohrbacher, J.; Hermans, A.N.; Gallacher, D.J.; Greig, R.J.H.; Bueno-Orovio, A.; Rodriguez, B. Human In Silico Drug Trials Demonstrate Higher Accuracy than Animal Models in Predicting Clinical Pro-Arrhythmic Cardiotoxicity. *Front. Physiol.* **2017**, *8*, 668.

33. Colatsky, T.; Fermini, B.; Gintant, G.; Pierson, J.B.; Sager, P.; Sekino, Y.; Strauss, D.G.; Stockbridge, N. The Comprehensive in Vitro Proarrhythmia Assay (CiPA) initiative—Update on progress. *J. Pharmacol. Toxicol. Method.* **2016**, *81*, 15–20.

34. Miragoli, M.; Salvarani, N.; Rohr, S. Myofibroblasts induce ectopic activity in cardiac tissue. *Circ. Res.* **2007**, *101*, 755–758.

35. Pastore, J.M.; Rosenbaum, D.S. Role of structural barriers in the mechanism of alternans-induced reentry. *Circ. Res.* **2000**, *87*, 1157–1163.

36. Gaeta, S.A.; Bub, G.; Abbott, G.W.; Christini, D.J. Dynamical mechanism for subcellular alternans in cardiac myocytes. *Circ. Res.* **2009**, *105*, 335–342.

37. Gaeta, S.A.; Krogh-Madsen, T.; Christini, D.J. Feedback-control induced pattern formation in cardiac myocytes: A mathematical modeling study. *J. Theor. Biol.* **2010**, *266*, 408–418

38. Gaeta, S.A.; Christini, D.J. Non-linear dynamics of cardiac alternans: Subcellular to tissue-level mechanisms of arrhythmia. *Front. Physiol.* **2012**, *3*, 157.

39. Ortmans, S.; Daval, C.; Aguilar, M.; Compagno, P.; Cadrin-Tourigny, J.; Dyrda, K.; Rivard, L.; Tadros, R. Pharmacotherapy in inherited and acquired ventricular arrhythmia in structurally normal adult hearts. *Expert. Opin. Pharmacother.* **2019**, *20*, 2101–2114.

40. Prystowsky, E.N.; Padanilam, B.J.; Joshi, S.; Fogel, R.I. Ventricular arrhythmias in the absence of structural heart disease. *J. Am. Coll. Cardiol.* **2012**, *59*, 1733–1744.

41. Killu, A.M.; Stevenson, W.G. Ventricular tachycardia in the absence of structural heart disease. *Heart* **2019**, *105*, 645–656.

42. Wang, M.; Tu, X. The Genetics and Epigenetics of Ventricular Arrhythmias in Patients without Structural Heart Disease. *Front. Cardiovasc. Med.* **2022**, *9*, 891399

43. Bermúdez-Jiménez, F.J.; Carriel, V.; Brodehl, A.; Alaminos, M.; Campos, A.; Schirmer, I.; Milting, H.; Abril, B.; Álvarez, M.; López-Fernández, S.; et al. Novel Desmin Mutation p.Glu401Asp Impairs Filament Formation, Disrupts Cell Membrane Integrity, and Causes Severe Arrhythmogenic Left Ventricular Cardiomyopathy/Dysplasia. *Circulation* **2018**, *137*, 1595–1610.

44. Van der Heijden, J.F.; Hassink, R.J. The phospholamban p.Arg14del founder mutation in Dutch patients with arrhythmogenic cardiomyopathy. *Neth. Hear. J.* **2013**, *21*, 284–285.

45. Forleo, C.; Carmosino, M.; Resta, N.; Rampazzo, A.; Valecce, R.; Sorrentino, S.; Iacoviello, M.; Pisani, F.; Procino, G.; Gerbino, A.; et al. Clinical and functional characterization of a novel mutation in lamin a/c gene in a multigenerational family with arrhythmogenic cardiac laminopathy. *PLoS ONE* **2015**, *10*, e0121723.

46. Austin, K.M.; Trembley, M.A.; Chandler, S.F.; Sanders, S.P.; Saffitz, J.E.; Abrams, D.J.; Pu, W.T. Molecular mechanisms of arrhythmogenic cardiomyopathy. *Nat. Rev. Cardiol.* **2019**, *16*, 519–537.

47. Roselli, C.; Chaffin, M.D.; Weng, L.-C.; Aeschbacher, S.; Ahlberg, G.; Albert, C.M.; Almgren, P.; Alonso, A.; Anderson, C.D.; Aragam, K.G.; et al. Multi-ethnic genome-wide association study for atrial fibrillation. *Nat. Genet.* **2018**, *50*, 1225–1233.

48. Lozano-Velasco, E.; Franco, D.; Aranega, A.; Daimi, H. Genetics and Epigenetics of Atrial Fibrillation. *Int. J. Mol. Sci.* **2020**, *21*, 5717.

49. Cao, F.; Li, Z.; Ding, W.M.; Yan, L.; Zhao, Q.Y. LncRNA PVT1 regulates atrial fibrosis via miR-128-3p-SP1-TGF-beta1-Smad axis in atrial fibrillation. *Mol. Med.* **2019**, *25*, 7.

50. Zhao, J.B.; Zhu, N.; Lei, Y.H.; Zhang, C.J.; Li, Y.H. Modulative effects of lncRNA TCONS_00202959 on autonomic neural function and myocardial functions in atrial fibrillation rat model. *Eur. Rev. Med. Pharmacol. Sci.* **2018**, *22*, 8891–8897.

51. Yao, L.; Zhou, B.; You, L.; Hu, H.; Xie, R. LncRNA MIAT/miR-133a-3p axis regulates atrial fibrillation and atrial fibrillation-induced myocardial fibrosis. *Mol. Biol. Rep.* **2020**, *47*, 2605–2617.

52. Shen, K.; Tu, T.; Yuan, Z.; Yi, J.; Zhou, Y.; Liao, X.; Liu, Q.; Zhou, X. DNA methylation dysregulations in valvular atrial fibrillation. *Clin. Cardiol.* **2017**, *40*, 686–691.

53. Zhang, D.; Hu, X.; Li, J.; Hoogstra-Berends, F.; Zhuang, Q.; Esteban, M.A.; de Groot, N.; Henning, R.H.; Brundel, B.J. Converse role of class I and class IIa HDACs in the progression of atrial fibrillation. *J. Mol. Cell. Cardiol.* **2018**, *125*, 39–49.

54. Zhang, D.; Wu, C.T.; Qi, X.; Meijering, R.A.; Hoogstra-Berends, F.; Tadevosyan, A.; Deniz, G.C.; Durdu, S.; Akar, A.R.; Sibon, O.C.; et al. Activation of histone deacetylase-6 induces contractile dysfunction through derailment of alpha-tubulin proteostasis in experimental and human atrial fibrillation. *Circulation* **2014**, *129*, 346–358.

55. Egger, G.; Liang, G.; Aparicio, A.; Jones, P.A. Epigenetics in human disease and prospects for epigenetic therapy. *Nature* **2004**, *429*, 457–463.

56. Cavalli, G.; Heard, E. Advances in epigenetics link genetics to the environment and disease. *Nature* **2019**, *571*, 489–499.

57. Cao, J. The functional role of long non-coding RNAs and epigenetics. *Biol. Proced. Online* **2014**, *16*, 11. [

58. Priori, S.G.; Blomström-Lundqvist, C.; Mazzanti, A.; Blom, N.; Borggrefe, M.; Camm, J.; Elliott, P.M.; Fitzsimons, D.; Hatala, R.; Hindricks, G.; et al. 2015 ESC Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death: The Task Force for the Management

of Patients with Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death of the European Society of Cardiology (ESC). Endorsed by: Association for European Paediatric and Congenital Cardiology (AEPC). *Eur. Heart J.* **2015**, *36*, 2793–2867.

59. Sridharan, A.; Bradfield, J.S.; Shivkumar, K.; Ajijola, O.A. Autonomic nervous system and arrhythmias in structural heart disease. *Auton. Neurosci.* **2022**, *243*, 103037.
60. De Azevodo, I.M.; Watanabe, Y.; Dreifus, L.S. Atrioventricular junctional rhythm: Classification and clinical significance. *Chest* **1973**, *64*, 732–740.
61. Jalife, J.; Antzelevitch, C.; Moe, G.K. The case for modulated parasystole. *Pacing Clin. Electrophysiol.* **1982**, *5*, 911–926.
62. Gussak, I.; Antzelevitch, C.; Hammill, S.C.; Shen, W.K.; Bjerregaard, P. *Cardiac Repolarization: Bridging Basic and Clinical Science (Contemporary Cardiology)*; Humana Press: Totowa, NJ, USA, 2003.
63. Wu, T.-J.; Ong, J.J.; Hwang, C.; Lee, J.J.; Fishbein, M.C.; Czer, L.; Trento, A.; Blanche, C.; Kass, R.M.; Mandel, W.J.; et al. Characteristics of wave fronts during ventricular fibrillation in human hearts with dilated cardiomyopathy: Role of increased fibrosis in the generation of reentry. *J. Am. Coll. Cardiol.* **1998**, *32*, 187–196.
64. Kawara, T.; Derksen, R.; de Groot, J.R.; Coronel, R.; Tasseron, S.; Linnenbank, A.C.; Hauer, R.N.; Kirkels, H.; Janse, M.J.; de Bakker, J.M. Activation delay after premature stimulation in chronically diseased human myocardium relates to the architecture of interstitial fibrosis. *Circulation* **2001**, *104*, 3069–3075.
65. Braunwald, E.; Kloner, R.A. Myocardial reperfusion: A double-edged sword? *J. Clin. Investig.* **1985**, *76*, 1713–1719.
66. Yellon, D.M.; Hausenloy, D.J. Myocardial reperfusion injury. *N. Engl. J. Med.* **2007**, *357*, 1121–1135.
67. Ferrari, R.; Balla, C.; Malagù, M.; Guardigli, G.; Morciano, G.; Bertini, M.; Biscaglia, S.; Campo, G. Reperfusion Damage- A Story of Success, Failure, and Hope. *Circ. J.* **2017**, *81*, 131–141.
68. Hausenloy, D.J.; Garcia-Dorado, D.; Bøtker, H.E.; Davidson, S.M.; Downey, J.; Engel, F.B.; Jennings, R.; Lecour, S.; Leor, J.; Madonna, R.; et al. Novel targets and future strategies for acute cardioprotection: Position Paper of the European Society of Cardiology Working Group on Cellular Biology of the Heart. *Cardiovasc. Res.* **2017**, *113*, 564–585.
69. Spannbauer, A.; Traxler, D.; Lukovic, D.; Zlabinger, K.; Winkler, J.; Gugerell, A.; Ferdinandy, P.; Hausenloy, D.J.; Pavo, N.; Emmert, M.Y.; et al. Effect of Ischemic Preconditioning and Postconditioning on Exosome-Rich Fraction microRNA Levels, in Relation with Electrophysiological Parameters and Ventricular Arrhythmia in Experimental Closed-Chest Reperfused Myocardial Infarction. *Int. J. Mol. Sci.* **2019**, *20*, 2140.
70. Wang, Y.; Hill, J.A. Electrophysiological remodeling in heart failure. *J. Mol. Cell. Cardiol.* **2010**, *48*, 619–632.
71. Inoue, H.; Zipes, D.P. Conduction over an isthmus of atrial myocardium in vivo: A possible model of Wolff-Parkinson-White syndrome. *Circulation* **1987**, *76*, 637–647.
72. Tomaselli, G.F.; Zipes, D.P. What causes sudden death in heart failure? *Circ. Res.* **2004**, *95*, 754–763.
73. Yue, L.; Xie, J.; Nattel, S. Molecular determinants of cardiac fibroblast electrical function and therapeutic implications for atrial fibrillation. *Cardiovasc. Res.* **2011**, *89*, 744–753.
74. Wijffels, M.C.; Kirchhof, C.J.; Dorland, R.; Allessie, M.A. Atrial fibrillation begets atrial fibrillation. A study in awake chronically instrumented goats. *Circulation* **1995**, *92*, 1954–1968.
75. Moe, G.K.; Rheinboldt, W.C.; Abildskov, J.A. A Computer Model of Atrial Fibrillation. *Am. Heart J.* **1964**, *67*, 200–220.
76. Roden, D.M. Repolarization reserve: A moving target. *Circulation* **2008**, *118*, 981–982.

77. Varro, A.; Baczko, I. Cardiac ventricular repolarization reserve: A principle for understanding drug-related proarrhythmic risk. *Br. J. Pharmacol.* **2011**, *164*, 14–36.

78. Pu, J.; Boyden, P.A. Alterations of Na+ currents in myocytes from epicardial border zone of the infarcted heart. A possible ionic mechanism for reduced excitability and postrepolarization refractoriness. *Circ. Res.* **1997**, *81*, 110–119.

79. Ursell, P.C.; Gardner, P.I.; Albala, A.; Fenoglio, J.J., Jr.; Wit, A.L. Structural and electrophysiological changes in the epicardial border zone of canine myocardial infarcts during infarct healing. *Circ. Res.* **1985**, *56*, 436–451.

80. Maltsev, V.A.; Sabbab, H.N.; Undrovinas, A.I. Down-regulation of sodium current in chronic heart failure: Effect of long-term therapy with carvedilol. *Cell. Mol. Life Sci.* **2002**, *59*, 1561–1568.

81. Sato, T.; Ohkusa, T.; Honjo, H.; Suzuki, S.; Yoshida, M.-A.; Ishiguro, Y.S.; Nakagawa, H.; Yamazaki, M.; Yano, M.; Kodama, I.; et al. Altered expression of connexin43 contributes to the arrhythmogenic substrate during the development of heart failure in cardiomyopathic hamster. *Am. J. Physiol. Heart Circ. Physiol.* **2008**, *294*, H1164–H1173.

82. Rodriguez-Sinovas, A. Cx43 phosphorylation and cardioprotection. *Cardiovasc. Res.* **2009**, *83*, 613–614.

83. Peters, N.S.; Green, C.R.; Poole-Wilson, P.A.; Severs, N.J. Reduced content of Connexin43 gap junctions in ventricular myocardium from hypertrophied and ischemic human hearts. *Circulation* **1993**, *88*, 864–875.

84. Akar, F.G.; Rosenbaum, D.S. Transmural electrophysiological heterogeneities underlying arrhythmogenesis in heart failure. *Circ. Res.* **2003**, *93*, 638–645.

85. Yan, G.-X.; Rials, S.J.; Wu, Y.; Liu, T.; Xu, X.; Marinchak, R.A.; Kowey, P.R. Ventricular hypertrophy amplifies transmural repolarization dispersion and induces early afterdepolarization. *Am. J. Physiol. Heart Circ. Physiol.* **2001**, *281*, H1968–H1975.

86. Wetzel, U.; Boldt, A.; Lauschke, J.; Weigl, J.; Schirdewahn, P.; Dorszewski, A.; Doll, N.; Hindricks, G.; Dhein, S.; Kottkamp, H. Expression of connexins 40 and 43 in human left atrium in atrial fibrillation of different aetiologies. *Heart* **2005**, *91*, 166–170.

87. Polontchouk, L.; Haefliger, J.-A.; Ebelt, B.; Schaefer, T.; Stuhlmann, D.; Mehlhorn, U.; Kuhn-Regnier, F.; De Vivie, E.; Dhein, S. Effects of chronic atrial fibrillation on gap junction distribution in human and rat atria. *J. Am. Coll. Cardiol.* **2001**, *38*, 883–891.

88. Sarrazin, J.-F.; Comeau, G.; Daleau, P.; Kingma, J.; Plante, I.; Fournier, D.; Molin, F. Reduced incidence of vagally induced atrial fibrillation and expression levels of connexins by n-3 polyunsaturated fatty acids in dogs. *J. Am. Coll. Cardiol.* **2007**, *50*, 1505–1512.

89. Daleau, P.; Boudriau, S.; Michaud, M.; Jolicoeur, C.; Kingma, J.G., Jr. Preconditioning in the absence or presence of sustained ischemia modulates myocardial Cx43 protein levels and gap junction distribution. *Can. J. Physiol. Pharmacol.* **2001**, *79*, 371–378.

90. Chung, M.K.; Martin, D.O.; Sprecher, D.; Wazni, O.; Kanderian, A.; Carnes, C.A.; Bauer, J.A.; Tchou, P.J.; Niebauer, M.J.; Natale, A.; et al. C-reactive protein elevation in patients with atrial arrhythmias: Inflammatory mechanisms and persistence of atrial fibrillation. *Circulation* **2001**, *104*, 2886–2891.

91. Psychari, S.N.; Apostolou, T.S.; Sinos, L.; Hamodraka, E.; Liakos, G.; Kremastinos, D.T. Relation of elevated C-reactive protein and interleukin-6 levels to left atrial size and duration of episodes in patients with atrial fibrillation. *Am. J. Cardiol.* **2005**, *95*, 764–767.

92. Issac, T.T.; Dokainish, H.; Lakkis, N.M. Role of inflammation in initiation and perpetuation of atrial fibrillation: A systematic review of the published data. *J. Am. Coll. Cardiol.* **2007**, *50*, 2021–2028.

93. Pohl, D.; Benseler, S. Systemic inflammatory and autoimmune disorders. *Handb. Clin. Neurol.* **2013**, *112*, 1243–1252.

94. Pasquié, J.L.; Sanders, P.; Hocini, M.; Hsu, L.F.; Scavée, C.; Jais, P.; Takahashi, Y.; Rotter, M.; Sacher, F.; Victor, J.; et al. Fever as a precipitant of idiopathic ventricular fibrillation in patients with normal hearts. *J. Cardiovasc. Electrophysiol.* **2004**, *15*, 1271–1276.

95. Lazzerini, P.E.; Capecchi, P.L.; Laghi-Pasini, F. Systemic inflammation and arrhythmic risk: Lessons from rheumatoid arthritis. *Eur. Heart J.* **2017**, *38*, 1717–1727.

96. Kumagai, K.; Nakashima, H.; Saku, K. The HMG-CoA reductase inhibitor atorvastatin prevents atrial fibrillation by inhibiting inflammation in a canine sterile pericarditis model. *Cardiovasc. Res.* **2004**, *62*, 105–111.

97. Tveit, A.; Grundtvig, M.; Gundersen, T.; Vanberg, P.; Semb, A.G.; Holt, E.; Gullestad, L. Analysis of pravastatin to prevent recurrence of atrial fibrillation after electrical cardioversion. *Am. J. Cardiol.* **2004**, *93*, 780–782.

98. Lazzerini, P.E.; Laghi-Pasini, F.; Boutjdir, M.; Capecchi, P.L. Inflammatory cytokines and cardiac arrhythmias: The lesson from COVID-19. *Nat. Rev. Immunol.* **2022**, *22*, 270–272.

99. Coromilas, E.J.; Kochav, S.; Goldenthal, I.; Biviano, A.; Garan, H.; Goldbarg, S.; Kim, J.-H.; Yeo, I.; Tracy, C.; Ayanian, S.; et al. Worldwide Survey of COVID-19-Associated Arrhythmias. *Circ. Arrhythmia Electrophysiol.* **2021**, *14*, e009458.

100. Wang, Y.; Zheng, Y.; Tong, Q.; Wang, L.; Lv, G.; Xi, Z.; Liu, W. Cardiac Injury and Clinical Course of Patients with Coronavirus Disease 2019. *Front. Cardiovasc. Med.* **2020**, *7*, 147.

101. Group WHOREAfC-TW; Shankar-Hari, M.; Vale, C.L.; Godolphin, P.J.; Fisher, D.; Higgins, J.P.T.; Spiga, F.; Savović, J.; Tierney, J.; Baron, G.; et al. Association between Administration of IL-6 Antagonists and Mortality among Patients Hospitalized for COVID-19: A Meta-analysis. *JAMA* **2021**, *326*, 499–518.

102. Sharma, S.; Adrogue, J.V.; Golfman, L.; Uray, I.; Lemm, J.; Youker, K.; Noon, G.P.; Frazier, O.H.; Taegtmeyer, H. Intramyocardial lipid accumulation in the failing human heart resembles the lipotoxic rat heart. *FASEB J.* **2004**, *18*, 1692–1700.

103. Aromolaran, A.S.; Colecraft, H.M.; Boutjdir, M. High-fat diet-dependent modulation of the delayed rectifier K(+) current in adult guinea pig atrial myocytes. *Biochem. Biophys. Res. Commun.* **2016**, *474*, 554–559.

104. Aromolaran, A.S.; Boutjdir, M. Cardiac Ion Channel Regulation in Obesity and the Metabolic Syndrome: Relevance to Long QT Syndrome and Atrial Fibrillation. *Front. Physiol.* **2017**, *8*, 431.

105. Lundman, P.; Boquist, S.; Samnegård, A.; Bennermo, M.; Held, C.; Ericsson, C.-G.; Silveira, A.; Hamsten, A.; Tornvall, P. A high-fat meal is accompanied by increased plasma interleukin-6 concentrations. *Nutr. Metab. Cardiovasc. Dis.* **2007**, *17*, 195–202.

106. Rennison, J.H.; Van Wagoner, D.R. Impact of dietary fatty acids on cardiac arrhythmogenesis. *Circ. Arrhythmia Electrophysiol.* **2009**, *2*, 460–469.

107. Park, T.S.; Goldberg, I.J. Sphingolipids, lipotoxic cardiomyopathy, and cardiac failure. *Heart Fail. Clin.* **2012**, *8*, 633–641.

108. Ali, A.; Boutjdir, M.; Aromolaran, A.S. Cardiolipotoxicity, Inflammation, and Arrhythmias: Role for Interleukin-6 Molecular Mechanisms. *Front. Physiol.* **2018**, *9*, 1866.

109. Billman, G.E.; Kang, J.X.; Leaf, A. Prevention of sudden cardiac death by dietary pure omega-3 polyunsaturated fatty acids in dogs. *Circulation* **1999**, *99*, 2452–2457

110. Leaf, A.; Xiao, Y.F.; Kang, J.X.; Billman, G.E. Prevention of sudden cardiac death by n-3 polyunsaturated fatty acids. *Pharmacol. Ther.* **2003**, *98*, 355–377.

111. Grandi, E.; Ripplinger, C.M. Antiarrhythmic mechanisms of beta blocker therapy. *Pharmacol. Res.* **2019**, *146*, 104274.

112. Al-Gobari, M.; El Khatib, C.; Pillon, F.; Gueyffier, F. beta-Blockers for the prevention of sudden cardiac death in heart failure patients: A meta-analysis of randomized controlled trials. *BMC Cardiovasc. Disord.* **2013**, *13*, 52.

113. McMurray, J.; Køber, L.; Robertson, M.; Dargie, H.; Colucci, W.; Lopez-Sendon, J.; Remme, W.; Sharpe, D.N.; Ford, I. Antiarrhythmic effect of carvedilol after acute myocardial infarction: Results of the Carvedilol Post-Infarct Survival Control in Left Ventricular Dysfunction (CAPRICORN) trial. *J. Am. Coll. Cardiol.* **2005**, *45*, 525–530.

114. Exner, D.V.; Reiffel, J.A.; Epstein, A.E.; Ledingham, R.; Reiter, M.J.; Yao, Q.; Duff, H.; Follmann, D.; Schron, E.; Greene, H.; et al. Beta-blocker use and survival in patients with ventricular fibrillation or symptomatic ventricular tachycardia: The Antiarrhythmics Versus Implantable Defibrillators (AVID) trial. *J. Am. Coll. Cardiol.* **1999**, *34*, 325–333.

115. Puljevic, M.; Velagic, V.; Puljevic, D.; Milicic, D. Propranolol efficiency in prevention of sustained ventricular tachycardia in patients with implanted cardioverter-defibrillator: A case series. *Croat. Med. J.* **2014**, *55*, 75–76.

116. Connolly, S.J.; Dorian, P.; Roberts, R.S.; Gent, M.; Bailin, S.; Fain, E.S.; Thorpe, K.; Champagne, J.; Talajic, M.; Coutu, B.; et al. Comparison of beta-blockers, amiodarone plus beta-blockers, or sotalol for prevention of shocks from implantable cardioverter defibrillators: The OPTIC Study: A randomized trial. *JAMA* **2006**, *295*, 165–171.

117. Boutitie, F.; Boissel, J.-P.; Connolly, S.J.; Camm, A.J.; Cairns, J.A.; Julian, D.G.; Gent, M.; Janse, M.J.; Dorian, P.; Frangin, G. Amiodarone interaction with beta-blockers: Analysis of the merged EMIAT (European Myocardial Infarct Amiodarone Trial) and CAMIAT (Canadian Amiodarone Myocardial Infarction Trial) databases. The EMIAT and CAMIAT Investigators. *Circulation* **1999**, *99*, 2268–2275.

118. Bashir, Y.; Thomsen, P.-E.B.; Kingma, J.; Møller, M.; Wong, C.; Cobbe, S.M.; Jordaens, L.; Campbell, R.W.; Rasmussen, H.S.; Camm, A. Electrophysiologic profile and efficacy of intravenous dofetilide (UK-68,798), a new class III antiarrhythmic drug, in patients with sustained monomorphic ventricular tachycardia. Dofetilide Arrhythmia Study Group. *Am. J. Cardiol.* **1995**, *76*, 1040–1044.

119. Mounsey, J.P.; DiMarco, J.P. Cardiovascular drugs. Dofetilide. *Circulation* **2000**, *102*, 2665–2670.

120. Baquero, G.A.; Banchs, J.E.; De Palma, S.; Young, S.K.; Penny-Peterson, E.D.; Samii, S.M.; Wolbrette, D.L.; Naccarelli, G.V.; Gonzalez, M.D. Dofetilide reduces the frequency of ventricular arrhythmias and implantable cardioverter defibrillator therapies. *J. Cardiovasc. Electrophysiol.* **2012**, *23*, 296–301.

121. Singh, B.N.; Nademanee, K. Use of calcium antagonists for cardiac arrhythmias. *Am. J. Cardiol.* **1987**, *59*, 153B–162B.

122. Singh, B.N. Comparative mechanisms of action of antiarrhythmic agents: Significance of lengthening repolarization. In *Control of Cardiac Arrhythmias by Lengthening Repolarization*; Singh, B.N., Ed.; Futura Publishing Company: Mount Kisco, NY, USA, 1988; pp. 53–127.

123. Van der Werf, C.; Zwinderman, A.H.; Wilde, A.A. Therapeutic approach for patients with catecholaminergic polymorphic ventricular tachycardia: State of the art and future developments. *Europace* **2012**, *14*, 175–183.

124. Antzelevitch, C.; Belardinelli, L.; Zygmunt, A.C.; Burashnikov, A.; Di Diego, J.M.; Fish, J.M.; Cordeiro, J.; Thomas, G. Electrophysiological effects of ranolazine, a novel antianginal agent with antiarrhythmic properties. *Circulation* **2004**, *110*, 904–910.

125. Olivotto, I.; Camici, P.G.; Merlini, P.A.; Rapezzi, C.; Patten, M.; Climent, V.; Sinagra, G.; Tomberli, B.; Marin, F.; Ehlermann, P.; et al. Efficacy of Ranolazine in Patients with Symptomatic Hypertrophic Cardiomyopathy: The RESTYLE-HCM Randomized, Double-Blind, Placebo-Controlled Study. *Circ. Heart Fail.* **2018**, *11*, e004124.

126. Antzelevitch, C.; Burashnikov, A.; Sicouri, S.; Belardinelli, L. Electrophysiologic basis for the antiarrhythmic actions of ranolazine. *Heart Rhythm.* **2011**, *8*, 1281–1290.

127. Morrow, D.A.; Scirica, B.M.; Karwatowska-Prokopczuk, E.; Murphy, S.A.; Budaj, A.; Varshavsky, S.; Wolff, A.A.; Skene, A.; McCabe, C.H.; Braunwald, E.; et al. Effects of ranolazine on recurrent cardiovascular events in patients with non-ST-elevation acute coronary syndromes: The MERLIN-TIMI 36 randomized trial. *JAMA* **2007**, *297*, 1775–1783.

128. Zareba, W.; Daubert, J.P.; Beck, C.A.; Huang, D.T.; Alexis, J.D.; Brown, M.W.; Pyykkonen, K.; McNitt, S.; Oakes, D.; Feng, C.; et al. Ranolazine in High-Risk Patients with Implanted Cardioverter-Defibrillators: The RAID Trial. *J. Am. Coll. Cardiol.* **2018**, *72*, 636–645.

129. Fox, K.; Ford, I.; Steg, P.G.; Tardif, J.C.; Tendera, M.; Ferrari, R. Ivabradine in stable coronary artery disease without clinical heart failure. *N. Engl. J. Med.* **2014**, *371*, 1091–1099.

130. Fox, K.; Ford, I.; Steg, P.G.; Tendera, M.; Ferrari, R.; BEAUTIFUL Investigators. Ivabradine for patients with stable coronary artery disease and left-ventricular systolic dysfunction (BEAUTIFUL): A randomised, double-blind, placebo-controlled trial. *Lancet* **2008**, *372*, 807–816.

131. Swedberg, K.; Komajda, M.; Böhm, M.; Borer, J.S.; Ford, I.; Dubost-Brama, A.; Lerebours, G.; Tavazzi, L.; on behalf of the SHIFT Investigators. Ivabradine and outcomes in chronic heart failure (SHIFT): A randomised placebo-controlled study. *Lancet* **2010**, *376*, 875–885.

132. Cocco, G.; Jerie, P. Torsades de pointes induced by the concomitant use of ivabradine and azithromycin: An unexpected dangerous interaction. *Cardiovasc. Toxicol.* **2015**, *15*, 104–106.

133. Lerman, B.B.; Ip, J.E.; Shah, B.K.; Thomas, G.; Liu, C.F.; Ciaccio, E.J.; Wit, A.L.; Cheung, J.W.; Markowitz, S.M. Mechanism-specific effects of adenosine on ventricular tachycardia. *J. Cardiovasc. Electrophysiol.* **2014**, *25*, 1350–1358.

134. Lerman, B.B.; Stein, K.M.; Markowitz, S.M.; Mittal, S.; Slotwiner, D.J. Right ventricular outflow tract tachycardia: An update. *Card. Electrophysiol. Rev.* **2002**, *6*, 68–71.

135. Markowitz, S.M.; Litvak, B.L.; Ramirez de Arellano, E.A.; Markisz, J.A.; Stein, K.M.; Lerman, B.B. Adenosine-sensitive ventricular tachycardia: Right ventricular abnormalities delineated by magnetic resonance imaging. *Circulation* **1997**, *96*, 1192–1200.

136. Ziff, O.J.; A Lane, D.; Samra, M.; Griffith, M.; Kirchhof, P.; Lip, G.Y.; Steeds, R.P.; Townend, J.; Kotecha, D. Safety and efficacy of digoxin: Systematic review and meta-analysis of observational and controlled trial data. *BMJ* **2015**, *351*, h4451.

137. Vamos, M.; Erath, J.W.; Hohnloser, S.H. Digoxin-associated mortality: A systematic review and meta-analysis of the literature. *Eur. Heart J.* **2015**, *36*, 1831–1838.

138. Williams, E.S.; Viswanathan, M.N. Current and emerging antiarrhythmic drug therapy for ventricular tachycardia. *Cardiol. Ther.* **2013**, *2*, 27–46.

139. Greene, H.L.; Roden, D.M.; Katz, R.J.; Woosley, R.L.; Salerno, D.M.; Henthorn, R.W. The Cardiac Arrhythmia Suppression Trial: First CAST... then CAST-II. *J. Am. Coll. Cardiol.* **1992**, *19*, 894–898.

140. Kingma, J., Simard, C., & Drolet, B. (2023). Overview of cardiac arrhythmias and treatment strategies. *Pharmaceuticals*, *16*(6), 844.

اضطرابات نظم القلب: نظرة عامة، الإدارة، العلاج، ودور السجلات الطبية

الملخص:

الخلفية :اضطرابات نظم القلب هي حالات مرضية مهمة تتعلق بإيقاع القلب ونظام التوصيل الكهربائي، وتتأثر بعوامل وراثية وبنيوية وبيئية. تتراوح هذه الحالات من الرجفان الأذيني غير المصحوب بأعراض إلى اضطرابات البطين القاتلة، مما يستلزم اتباع نهج علاجي متنوع. يعود تعقيد اضطرابات نظم القلب إلى إعادة التشكيل الكهربائي، ونقص التروية القلبية، والعوامل الوراثية، والالتهاب.

الهدف :تهدف هذه المراجعة إلى تقديم نظرة عامة على اضطرابات نظم القلب وآلياتها الأساسية واستراتيجيات علاجها، بالإضافة إلى دور السجلات الطبية في إدارتها.

الطرق :تم إجراء بحث شامل باستخدام قواعد البيانات *PubMed* و*Google Scholar*، مع التركيز على الأدبيات السريرية والتجريبية. تم استخدام كلمات مفتاحية مثل "اضطرابات نظم القلب"، "مرض القلب الإقفاري"، و"الكهروفسيولوجيا" لتوجيه مراجعة الأدبيات. وتم تجميع النتائج الرئيسية لتوضيح الآليات الفيزيولوجية المرضية والتطورات العلاجية.

النتائج :تشمل الفيزيولوجيا المرضية لاضطرابات نظم القلب إعادة التشكيل الكهربائي والبنيوي، واضطرابات الأيونات، والعوامل الوراثية والبيئية. تُعد اضطرابات البطين حادة وشديدة، بينما يتطور الرجفان الأذيني بشكل تدريجي مع آليات تشمل التليف والنشاط الشاذ. يؤدي نقص التروية والالتهاب إلى تفاقم اضطرابات النظم من خلال الأضرار البنيوية وإعادة التشكيل بوساطة السيتوكينات. تكشف الدراسات الوراثية عن طفرات في قنوات الأيونات والبروتينات الهيكلية كعوامل أساسية. تشمل العلاجات الحديثة الأدوية، واستئصال القسطرة، وزرع الأجهزة، مع دعم كبير من التوثيق في السجلات الطبية.

الخاتمة :أدت التطورات في فهم الطبيعة متعددة الجوانب لاضطرابات نظم القلب إلى تحسين استراتيجيات التشخيص والعلاج. تسهم السجلات الطبية بشكل كبير في الإدارة من خلال توثيق تقدم الحالة والعلاجات والنتائج، مما يوفر رؤى للرعاية الشخصية. يجب أن تركز الأبحاث المستقبلية على دمج البيانات الوراثية والبيئية لتطوير استراتيجيات وقائية وعلاجية محسنة.

الكلمات المفتاحية :اضطرابات نظم القلب، الكهروفسيولوجيا، الرجفان الأذيني، اضطرابات البطين، نقص التروية، الالتهاب، السجلات الطبية.