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SIDE EFFECTS OF ANTIARRHYTHMIC DRUGS AND THE RELEVANCE OF SEARCHING FOR LOW-TOXICITY ANTIARRHYTHMIC AGENTS

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Abstract. Clinical pharmacology for arrhythmias has not much improved over the past few decades, despite recent years of intensive research on antiarrhythmic medications (AAD). It appears that interventional therapy was prioritized over AAD. However, fresh information on the effectiveness and safety of "classical" molecules makes it necessary to create a useful manual to enhance clinical judgment. Even though many guidelines include suggestions for treating AAD, clinical practice may frequently involve overtreatment or undertreatment.1, 2 Furthermore, because of their narrow therapeutic index, complicated drug-drug interactions, and serious cardiac and extracardiac side effects, AADs are not a neutral medication in terms of safety. There is still a gap between the conventional AADs currently used in clinical practice and the "future" AADs, despite significant current research efforts to find target-specific AADs with better safety margins. Compared to other treatment modalities, antiarrhythmic pharmacologic therapy is one of the clinical domains where evidence-based cardiology is less applied. Many doctors rely on the simplistic Singh-Vaughan Williams classification, which has little clinical significance. A position paper on AAD seems warranted after all these years. Our goal is to provide a useful guide for general cardiologists, internal medicine specialists, general practitioners, and cardiology fellows or residents based on current knowledge and specific recommendations rather than a new pharmacology textbook or a review of research viewpoints. PubMed, MEDLINE, and the Cochrane Library (which includes the Cochrane Database of Systematic Reviews and the Cochrane Controlled Trials Registry) were the databases searched for relevant literature. The English-language sources and research with human participants were the main focus. Only where information was crucial to comprehending pathophysiological principles relevant to patient care and similar data from human studies were unavailable were articles pertaining to animal trials cited. When needed, the writers were asked for more details.

Keywords. Mechanisms, pharmacology, drug interactions, arrhythmia, atrial fibrillation, adverse drug responses, anti-arrhythmic medications, anti-arrhythmic drug combinations, Arrhythmias of the ventricles

Introduction. The cornerstone of long-term treatment for patients with cardiac arrhythmias has been and will continue to be antiarrhythmic medications. Antiarrhythmic medication prescriptions tripled between 2004 and 2016, according to a retrospective study conducted in the United States. For antiarrhythmic medications to receive regulatory approval, they must exhibit consistent efficacy and well-defined, controllable hazards. However, careful patient selection, meticulous supervision, and a thorough understanding of these medications' complex and varied pharmacology are necessary for their effective usage in clinical practice. Furthermore, as Levine pointed out, we must have reasonable expectations for success because, like many of our treatments,

antiarrhythmic medications are only meant to alleviate symptoms. Global health is greatly impacted by cardiac arrhythmias. Over the past few decades, efforts have been made to find a definite cure through intrusive procedures [1-5]. However, obstacles like anatomical restrictions, procedural hazards, and complicated arrhythmias still exist despite advancements in invasive therapy by catheter ablation. Furthermore, the generalizability of invasive arrhythmia therapy is limited by the incidence of certain arrhythmic illnesses. For instance, 1-2% of people have atrial fibrillation (AF), the most prevalent persistent arrhythmia. Currently, the capacity of the healthcare system is frequently exceeded by the demand for its intrusive treatment. Due to a lack of staff and resources, only 1% of AF patients in affluent nations currently receive ablation; this number is expected to drop to 10% in the near future. Furthermore, pharmaceutical therapy is still essential for some patients, either as part of peri-procedural care or because of ablation failure. After index ablation, around 50% of patients continue to take anti-arrhythmic medications (AADs), one in six have repeat ablation, and the majority continue to receive concurrent AAD therapy. These results demonstrate that rhythm regulation frequently depends on a combination strategy combining catheter ablation and AADs in contemporary clinical practice. Additionally, AADs are crucial in preventing recurrent arrhythmias in patients with cardiac implantable electronic devices (CIEDs). AADs are also required for conditions like channelopathies, which are frequently inappropriate for ablative therapy [6-11]. The critical significance of these drugs is further highlighted by the acute treatment of arrhythmias in emergency situations. The abbreviation ABC can be used to summarize the existing indications for AADs. AADs are at the forefront as crucial elements in the management of arrhythmias due to the obvious requirement for suitable, backup, and complementary treatments. In order to address this, the European Heart Rhythm Association (EHRA), under the direction of two chairs, brought together specialists from around the world to produce a useful manual on the use of AAD. Each expert was assigned to study the medical literature for a certain segment, and the chairs created the compendium's outline. The final text was subjected to an external review by a separate panel of specialists after the group as a whole considered these reviews. This useful guide methodically traverses the complex terrain of AADs, clarifying their modes of action, effectiveness, and safety profiles in the overall population of arrhythmia patients. Subpopulations with particular arrhythmia mechanisms or traits that could affect the safety and effectiveness of AAD are given extra consideration. The goal of the compendium is to give medical professionals a thorough grasp of these mechanisms so they may make wise choices in the complicated field of cardiac arrhythmias. Additionally, this compilation provides useful guidance and insights regarding the prudent incorporation of these medications into clinical practice [12-19]. It emphasizes how AADs work in conjunction with other therapies such as electrical cardioversion, ablation, cardiovascular medications, and implanted devices. This reveals a synergistic technique that maximizes patient results, guaranteeing a comprehensive and empirically supported approach to rhythm control. There is still a medical need for adjuvant pharmaceutical therapy for individuals following ablation or device placement. Antiarrhythmic medications are frequently administered after AF ablation because it is ineffective in up to one-third of patients. Antiarrhythmic medications are taken by more than half of individuals who have implanted cardioverter defibrillators. Since no medication has been well investigated in this population and no medication has enough safety and effectiveness to have a labeled indication for adjuvant treatment, it is unclear exactly how to provide pharmacologic therapy to these patients [20-26].

The main purpose of the presented manuscript is to provide a brief analysis of the side effects of antiarrhythmic drugs and the relevance of searching for low-toxicity antiarrhythmic agents based on the results of authoritative scientific works.

The meaning and fundamentals of anti-arrhythmic medication methods. Anti-arrhythmic medications are pharmaceuticals that alter the electrical activity of the heart in order to prevent or treat cardiac arrhythmias. This section looks at how they work, including how they affect ion channels, tissue selectivity, and pharmacokinetics (PK). It also looks at how genetics affects their safety and effectiveness. The way anti-arrhythmic medications work. Automatism, triggered focal activity brought on by early afterdepolarizations (EADs) or delayed afterdepolarizations (DADs), and re-entry are the three main ways that arrhythmias appear. Re-entry is the most common of these. Three key factors are essential to the appearance of this later process. To start the re-entrant electrical activity, a trigger is necessary [5-11]. An ectopic beat that originates from a particular area of the heart and isn't always connected to the re-entrant circuit could be this trigger. Second, in order to maintain the irregular beat, a re-entrant circuit—a channel that permits the electrical impulse to circulate inside the heart tissue—is required. Reduced refractoriness, slower conduction (or both), and unidirectional block all encourage re-entry inside the circuit. Finally, the vulnerability to re-entry processes is significantly influenced by the total autonomic condition. Arrhythmic events can be made more or less likely by the interaction of sympathetic and parasympathetic effects on the electrical characteristics of the heart. Understanding these basic mechanisms and how they interact is essential to comprehending the impact of AADs. It serves as the foundation for focused interventions and customized treatment plans meant to address the unique mechanisms behind each patient's arrhythmic appearance. However, a thorough analysis of them is outside the purview of this useful compilation [14-21].

Arrhythmia-based treatment. When selecting an AAD, there are a number of factors to take into account, one of which is whether the medication is meant to prevent or terminate arrhythmias. There are many exceptions to the general rule that medications that effectively stop a particular arrhythmia frequently have preventative qualities. Amiodarone, for example, is quite good at preventing AF but less effective at stopping acute episodes. Beyond these dynamics, the drug's efficacy for preventing vs terminating arrhythmias can be greatly impacted by variables like the mode of administration and its use-dependent or reverse use-dependent effects. Furthermore, different areas of heart tissue are affected differently by AADs. Class I and III medicines mostly affect the working myocardium at both the atrial and ventricular levels, whereas Class II and IV medications mainly decrease conduction and extend refractoriness in the sinus and AV nodes. Because Class I AADs, especially Class Ic, can delay conduction and encourage ventricular re-entry and pro-arrhythmia, their administration is generally discouraged in the presence of ventricular structural alterations or scarring [16-23]. The degree of ventricular myocardial contractility depression and the likelihood of HF aggravation are other important factors to take into account. These factors are more noticeable with Class Ic and Class IV drugs but less so with quinidine or amiodarone. In SHD, polymorphic VT is often a sign of myocardial ischaemia. In addition to normal coronary management, intravenous β-blockers and amiodarone are thought to be the best AADs in instances that are hemodynamically stable. Mexiletine and lidocaine347 might also be useful, and the latter AADs could be used as supplemental treatments. Polymorphic VT/VF, which is caused by triggering from surviving Purkinje fibers, can occasionally occur in post-infarct patients without any signs of cardiac ischemia. They start with a relatively short-coupled PVC (350 ms) and can happen with either a lengthy QT (pseudo-TdP) or a normal QT (PVT with normal QT). These Purkinje-triggered PVTs cause storms that respond well to quinidine but are resistant to amiodarone, lidocaine, mexiletine, β-blockers, and Class Ic medications [11-15].

The ailment, the patient, the medication, and the dosage are all factors to consider while beginning therapy. Following the Cardiac Arrhythmias Suppression Trial (CAST) trials, decision-

making on the initiation of AADs has changed in recent years. The CAST trial, which showed that potent and effective AADs can be potentially hazardous, particularly in the presence of structural heart disease, first highlighted the significance of patient-centered decision-making. Therefore, treating the arrhythmia when it is a sign of the illness differs greatly from treating the illness itself. Pharmacologic treatment for malignant ventricular arrhythmias and sudden cardiac death (SCD) has become less popular as a result of the development of implantable cardioverter-defibrillators (ICD). Currently, rather than suppressing and curing the arrhythmias as in other clinical contexts, AADs are mostly used as an additional therapy to ICD in malignant ventricular arrhythmias to prevent electrical storm and repeated shocks.Atrial fibrillation (AF) is a prevalent arrhythmia that has led to numerous advancements in pharmaceutical therapy and catheter ablation due to its high prevalence and the associated health and financial ramifications. The clinical state of the patient, the structural and functional substrate, and the cellular and molecular mechanisms behind arrhythmias are currently the main areas of focus [5-13]. This method differs significantly from the relatively empirical one proposed by the Singh-Vaughan Williams categorization. Antiarrhythmic medications continue to play a significant role in treating symptoms or preventing the decline of heart function caused by tachycardia, irregular rhythm, or dyssynchrony (such as that caused by frequent ventricular premature beats). Antiarrhythmic medications are an effective way to stop well-tolerated arrhythmias from developing into malignant arrhythmias. Antiarrhythmic medications have a limited therapeutic window and a tiny plasma concentration interval between the initial hazardous dose and the lowest effective dose, or between undertreatment and the toxic or proarrhythmic impact. Additionally, the pharmacological effects of AADs are linked to a high degree of inter-subject variability. In this consensus statement, we also include the rate-controlling medications when discussing AAD due to practical considerations and the numerous concurrent actions. Drug effects are, in fact, influenced by a variety of factors, including race, gender, genetics, temperature, drug-drug interactions, triggering factors, neurohormonal changes, illness severity and condition, disease-induced substrate remodeling, etc [14-24].

Proarrhythmia treatment. Accurately identifying and confirming drug-induced proarrhythmia and quickly stopping the implicated substance are necessary for effective treatment. The identification and modification of risk factors (e.g., female gender, advanced age, renal or liver dysfunction, underlying structural/ischemic heart disease, hypokalaemia, hypomagnesaemia, high drug doses/concentrations, rapid intravenous administration, bradycardia, QT prolongation, QT dispersion, and pre-existing channelopathies) that may be linked to the onset or worsening of arrhythmias is also crucial. The first line of treatment for drug-related proarrhythmia is to stop the offending medication; however, in certain cases, the implantation of an ICD must be taken into consideration based on the patient's unique characteristics and the potential risk of potentially fatal ventricular tachyarrhythmias. The following are used to treat drug-induced TdP, which is frequently associated with Class IA or Class III AAD: intravenous magnesium sulfate administration regardless of serum magnesium levels (e.g., 2 g bolus followed by another 2 g bolus and continuous infusion in case of arrhythmia persistence); raising heart rate using isoproterenol or overdrive pacing at rates greater than 70 beats per minute to reverse bradycardia and avoid pauses that could prolong repolarization and promote TdP [2-10]. Restoring serum potassium to the high-normal range (i.e., 4.5–5.0 mEq/L) in order to rectify hypokalaemia, albeit there is little evidence to support this approach. Atrial flutter with 1:1 AV conduction and persistent sluggish VT are examples of sodium channel blocker-related proarrhythmias, which are often caused by slowing of conduction. In addition to stopping the offending medication, therapy involves controlling the ventricular response with an intravenous beta-blocker or calcium antagonist; on the other hand,

persistent sluggish VT can be reversed by intravenous sodium or sodium bicarbonate. Flecainide-related ventricular arrhythmias have reportedly been successfully treated with beta-blockers [13-17].

What's fresh? The ABC framework's introduction: AAD use is categorized into Appropriate, Backup, and Complementary therapies using a novel conceptual paradigm. This clever conceptualization encourages a practical, tiered approach to rhythm regulation by assisting doctors in placing pharmaceutical decisions in perspective with procedures options like ablation or device therapy. Emphasis on application outside the traditional guidelines: This compendium, in contrast to previous papers, emphasizes the practical, scenario-based use of anti-arrhythmic medications (AADs), including drug initiation protocols, patient-specific modifications, and drug switching methods, particularly in situations when invasive treatments are impractical or ineffective. Expanded drug classifications and updates on emerging agents: It places the document at the forefront of clinical pharmacology by incorporating, in a simplified form, contemporary updates to the Vaughan Williams (VW) classification, such as the Oxford 2018 expansion (e.g., Class 0 for ivabradine) and introducing new and upcoming agents like small-conductance calcium-activated potassium channel inhibitors, etripamil, and budiodarone. Put an emphasis on combining other therapies: Compared to previous publications, the paper offers a more integrated and patient-specific treatment approach by offering detailed guidance on how to coordinate the use of AAD with other therapies, such as ablation, anticoagulation, device therapy, and even electrical cardioversion [3-14].

Discussion. For many years, the tools available to medical professionals treating patients with cardiac arrhythmias were extremely limited. Digoxin, which has long been used to treat dropsy, has also been shown to prevent other supraventricular arrhythmias by slowing the ventricular response to atrial fibrillation (AF) by vagally mediated suppression of AV nodal conduction. Later in the 20th century, beta-adrenergic and L-type calcium channel blockers joined it for comparable purposes. Quinidine, procainamide, and lidocaine were the primary options for directly suppressing atrial or ventricular arrhythmia until a number of potential medications were introduced in the late 1970s. New sodium channel-blocking medications, such as disopyramide, encainide, flecainide, ethmozine, and propafenone, were approved after a series of clinical trials. These medications were restricted due to the possibility of heart failure or ventricular proarrhythmia. A small number of them are still in use today, but they are limited to specific patient subgroups [1,4,5]. For instance, we discovered from the CAST (Cardiac Arrhythmia Suppression Trial) that patients with ischemic heart disease, a recent myocardial infarction, and frequent premature ventricular complexes were more likely to die when given medications with strong effects on the fast sodium current. Flecainide and propafenone are commonly used in the post-CAST era to control paroxysmal AF in people without structural heart disease, and new research indicates that they may also be utilized in patients with little coronary artery disease.3 Mexiletine, a congener of lidocaine, has continued to be used in therapeutic settings for certain ventricular reasons, such as treating torsade de pointes in individuals who are not responding to intravenous magnesium. Due to the infrequent occurrence of neutropenia, tocainide, another lidocaine congener, had limited use in the suppression of ventricular arrhythmias. The European Heart Rhythm Association Practical Compendium of Anti-arrhythmic Drugs (AADs) provides guidance on these medications, emphasizing their clinical application and the worldwide consequences of cardiac arrhythmias [6-12]. The purpose of this publication is to give clinicians useful guidance on managing arrhythmias using pharmacological techniques. Clinical limitations, procedural hazards, and the intricacy of some arrhythmias are just a few of the ongoing difficulties in treating arrhythmias that are highlighted in the compendium. Atrial fibrillation is particularly common, and the need for invasive treatment frequently exceeds the

capabilities of current healthcare systems. Pharmacological treatment is therefore still crucial. This is especially important for individuals who have channelopathies or cardiac implantable electronic devices, for which ablation is frequently not an appropriate course of action. In these situations, anti-arrhythmic medications are essential. The ABC framework for AAD therapy is introduced in the compendium: A (Appropriate therapy) for patients for whom AADs are the most effective treatment option; B (Backup therapy) as an adjuvant to invasive procedures like catheter ablation; and C (Complementary therapy) in conjunction with other therapies. Each class of AADs' methods of action, efficacy, safety profiles, and medication interactions are all covered in length in this publication. With a focus on pro-arrhythmia risk mitigation, the compendium also addresses practical issues such as initiation, combination methods, monitoring, follow-up, particular populations, and adverse impact management. In order to maximize patient outcomes, it also investigates the combination of AADs with other therapy methods. In conclusion, this compilation is a vital tool for medical professionals, providing useful guidance and evidence-based perspectives to successfully negotiate the challenges of managing arrhythmias [21-29].

Conclusions. The continually high prevalence of cardiac arrhythmias, the synergistic effects of AADs in conjunction with other treatments, and their indispensability in treating acute episodes all highlight the enduring significance of AADs in the face of diminishing attention owing to the emergence of alternative therapies. When it comes to managing cardiac arrhythmias, AADs continue to fulfill the ABC strategy, which stands for appropriate therapy, backup therapy, and complementary therapy.

The continued development of novel and promising AADs, in spite of the strict regulations that lead to a lengthy, complex, and expensive development process, emphasizes this importance even more. New or infrequently utilized therapeutic techniques, like individual self-administration for arrhythmia termination, may be prompted by these advancements. Proper selection requires an understanding of how these medications function, but it is also necessary to take into account any potentially dangerous interactions with other medications or patient circumstances.

This excellent guide provides a thorough overview of the information required to prescribe these drugs, which are powerful and helpful but also have the potential for serious side effects. For medical experts looking to maximize the treatment of cardiac arrhythmias, striking this equilibrium is crucial.

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