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Nota di contenuto	NOVEL THERAPEUTIC TARGETS FOR ANTIARRHYTHMIC DRUGS; CONTENTS; Acknowledgments; Contributors; 1. Introduction; References; 2. Myocardial K(+) Channels: Primary Determinants of Action Potential Repolarization; 2.1 Introduction; 2.2 Action Potential Waveforms and Repolarizing K(+) Currents; 2.3 Functional Diversity of Repolarizing Myocardial K(+) Channels; 2.4 Molecular Diversity of K(+) Channel Subunits; 2.5 Molecular Determinants of Functional Cardiac I (to) Channels; 2.6 Molecular Determinants of Functional Cardiac I(K) Channels; 2.7 Molecular Determinants of Functional Cardiac Kir Channels 2.8 Other Potassium Currents Contributing to Action Potential Repolarization2.8.1 Myocardial K(+) Channel Functioning in Macromolecular Protein Complexes; References; 3. The "Funny" Pacemaker Current; 3.1 Introduction: The Mechanism of Cardiac Pacemaking; 3.2 The "Funny" Current; 3.2.1 Historical Background; 3.2.2 Biophysical Properties of the I(f) Current; 3.2.3 Autonomic Modulation; 3.2.4 Cardiac Distribution of I(f); 3.3 Molecular Determinants of the I(f) Current; 3.3.1 HCN Clones and Pacemaker

Channels; 3.3.2 Identification of Structural Elements Involved in Channel Gating
 3.3.3 Regulation of Pacemaker Channel Activity: "Context" Dependence and Protein-Protein Interactions
 3.3.4 HCN Gene Regulation; 3.4 Blockers of Funny Channels; 3.4.1 Alinidine (ST567); 3.4.2 Falipamil (AQ-A39), Zatebradine (UL-FS 49), and Cilobradine (DK-AH269); 3.4.3 ZD7288; 3.4.4 Ivabradine (S16257); 3.4.5 Effects of the Heart Rate Reducing Agents on HCN Isoforms; 3.5 Genetics of HCN Channels; 3.5.1 HCN-KO Models; 3.5.2 Pathologies Associated with HCN Dysfunctions; 3.6 HCN-Based Biological Pacemakers; References; 4. Arrhythmia Mechanisms in Ischemia and Infarction; 4.1 Introduction
 4.1.1 Modes of Ischemia, Phases of Arrhythmogenesis
 4.1.2 Trigger-Substrate-Modulating Factors; 4.2 Arrhythmogenesis in Acute Myocardial Ischemia; 4.2.1 Phase 1A; 4.2.2 Phase 1B; 4.2.3 Arrhythmogenic Mechanism: Trigger; 4.2.4 Catecholamines; 4.3 Arrhythmogenesis During the First Week Post MI; 4.3.1 Mechanisms; 4.3.2 The Subendocardial Purkinje Cell as a Trigger 24-48 H Post Occlusion; 4.3.3 Five Days Post-Occlusion: Epicardial Border Zone; 4.4 Arrhythmia Mechanisms in Chronic Infarction; 4.4.1 Reentry and Focal Mechanisms; 4.4.2 Heterogeneity of Ion Channel Expression in the Healthy Heart
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 4.4.4 Structural Remodeling; 4.4.5 Role of the Purkinje System; References; 5. Antiarrhythmic Drug Classification; 5.1 Introduction; 5.2 Sodium Channel Blockers; 5.2.1 Mixed Sodium Channel Blockers (Vaughan Williams Class Ia); 5.3 Inhibitors of the Fast Sodium Current with Rapid Kinetics (Vaughan Williams Class Ib); 5.3.1 Lidocaine; 5.3.2 Mexiletine; 5.4 Inhibitors of the Fast Sodium Current with Slow Kinetics (Vaughan Williams Class Ic); 5.4.1 Flecainide; 5.4.2 Propafenone
 5.5 Inhibitors of Repolarizing K(+) Currents (Vaughan Williams Class III)

Sommario/riassunto

Profiles potential treatment approaches for cardiac arrhythmias Cardiac arrhythmias of ventricular origin are responsible for the deaths of nearly half a million Americans each year while atrial fibrillation accounts for about 2.3 million cases per year, a rate that is projected to increase 2.5 fold over the next half century. Effectively managing these cardiac rhythm disorders remains a major challenge for both caregivers and the pharmaceutical industry. Filling a gap in the current literature, Novel Therapeutic Targets for Antiarrhythmic Drugs presents the latest treatments f

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