Invasive Electrophysiology for Beginners

Leon Iden
Martin Borlich
Philipp Sommer
Editors

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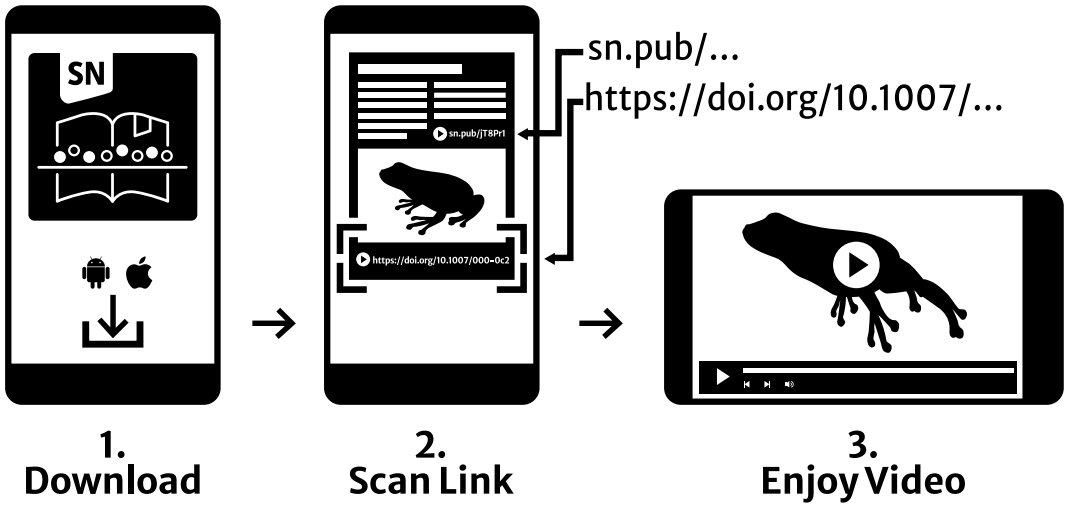

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Foreword

The friendly invitation to write a foreword for a textbook on cardiac arrhythmias elicited two feelings in me: on the one hand, the feeling of aging, because usually only "older semesters" receive such an invitation, and on the other hand—and connected with it—the realization that over the years, I have somewhat lost the proximity to direct application. Essentially, both are correct, albeit somewhat painful. However, I am very happy to fulfill the editors' request to give the book a hopefully appropriate classification and correct perspective with a foreword.

Interventional electrophysiology has undergone an outstanding development over the last (at least) 40 years and, like hardly any other field in medicine, has promoted the most important patient values, a better and/or longer life, in an incredible way. The interventional and thus in many cases also curative treatment of people with highly symptomatic and sometimes even life-threatening cardiac arrhythmias is indeed a great achievement of medicine. The "seniors" among the rhythmologists still have a vivid, rather dreadful, memory of the bleak era of antiarrhythmic drug treatment. With the clinical development and establishment of ablation therapy, the foundation for modern interventional rhythmology was laid. In a breathtaking developmental ride, electrophysiologists managed, step by step and with great consistency in the 1980s and 1990s, to overcome a new hurdle in the interventional treatment of cardiac arrhythmias every 2–3 years. From AV node ablation to the successful ablation of accessory pathways, modulation of the AV node, ablation initially of typical and atypical atrial flutter, and also ventricular arrhythmia, this was, even from today's perspective, an almost incredible success story. With the successful treatment of atrial fibrillation, a treatment option for an arrhythmia that is prevalent to an endemic extent was also successfully developed and established.

The present specialist book on interventional therapy for cardiac arrhythmias excellently summarizes the achievements in a current "Perspective 2022." In addition to the important basics of ablation therapy, the different forms of cardiac arrhythmias are presented and addressed in a practical, understandable, and comprehensible manner. The focus of the individual chapters is on the safe and effective treatment through catheter ablation. In doing so, the current state of knowledge on the pathophysiology of arrhythmias is excellently linked with modern interventional therapy options. At this point, special thanks are due to all those who have driven the successful

development of interventional electrophysiology through knowledge and conviction, but also through courage and determination. Many of the decisive advances on the way to establishing ablation treatment as standard therapy have been achieved by European electrophysiologists, with significant contributions from German electrophysiology as well—the contributions of this book also bear witness to this. I very much hope that the reading will also serve as an incentive to take on further challenges in the interventional management of cardiac arrhythmias and to develop (even) better solution strategies.

I wish this excellent textbook wide dissemination and recognition and all readers much joy in reading it. Special thanks go to the editors – also for the kind invitation to this foreword.

Gerhard Hindricks

Preface

Dear friends of invasive electrophysiology!

For over 20 years, ablation has been performed regularly and increasingly frequently in many centers. Often with great intensity, but also often in smaller centers with lower case volumes. Getting an idea of what invasive electrophysiology actually means, how it works, and what the treatment goals are is not so easy – we had to realize that there is hardly any suitable literature in the German-speaking world that can provide a good overview for beginners in this field.

Thus, the idea for our book project "Invasive Electrophysiology for Beginners" was born – many great colleagues were immediately ready to contribute articles on the respective topics, and so we can proudly present a practical guide that will be as helpful as a companion in everyday life as it will be as a work for evening reading.

For one thing is important: that electrophysiology is conducted at an excellent level and that we can provide our patients with an optimal treatment strategy based on current knowledge.

And now have fun reading, learning, and puzzling.



Leon Iden Martin Borlich Philipp Sommer

PS: We deliberately refrain from differentiating between female and male forms in this book—electrophysiologists of all genders should always feel addressed.

Contents

Abbreviations

Abl	Ablation catheter (short form)
ACT	activated clotting time; activated coagulation time
AH	Interval between atrial signal and His
ALARA	"As low as reasonably achievable"; radiation protection principle
AP	Accessory pathway
AP	anterior-posterior; X-ray image, in which the beam path in relation to the body goes from front (anterior) to back (posterior)
ARVC	"Arrhythmogenic right ventricular cardiomyopathy"; arrhythmogenic right ventricular cardiomyopathy; Synonym: ARVD
AVNRT	AV node reentry tachycardia
AVRT	Atrioventricular reentrant tachycardia
CCW	"counterclockwise"; indication of the propagation direction of typical atrial flutter
CF	"contact force"; contact pressure
CHA2DS2-VASc-Score	Scoring system for calculating the thromboembolic risk in AF
CL	"cycle length", cycle length
CPVT	"Catecholaminergic polymorphic ventricular tachycardia"; catecholaminergic polymorphic ventricular tachycardia
CRT	Cardiac resynchronization therapy
CS	"coronary sinus"; coronary venous sinus
CT	Computed tomography
CT	Crista terminalis
CTI	Cavotricuspid isthmus
CW	"clockwise"; indication of the propagation direction of typical atrial flutter
DAD	"Delayed afterdepolarization"; late afterdepolarization
DGK	German Cardiac Society
DOAC	"Direct oral anticoagulants"; direct oral anticoagulants; Synonym: DOAC, NOAC
EAD	"Early afterdepolarization," early afterdepolarization

EAT	Ectopic atrial tachycardia
ECMO	"Extracorporeal membrane oxygenation"; extra-corporeal membrane oxygenation
EF	"Ejection fraction"; left ventricular ejection fraction
EGM	"Electrogram"; Elektrogramm
EKG	Electrocardiogram
EP	Electrophysiology
EPS	Electrophysiological study
FAM	"Fast anatomical mapping"; method for 3D creation of maps in the CARTO system
FAT	Focal atrial tachycardia
FBI	"Fast—broad—irregular" (– tachycardia)
FP	"Fast pathway"; faster conduction pathway
FTI	"Force time integral"; force-time integral
HCM	Hypertrophic cardiomyopathy
HFS	High-frequency current
HPSD	"High power short duration"
HRA	High right atrium
HV	Interval between His and ventricular signal
IABP	Intra-aortic balloon pump
ICD	"Implantable Cardioverter" Defibrillator; implantable cardioverter defibrillator
ICE	"Intracardiac echocardiography"; intracardiac echocardiography
IEGM	"Intracardiac electrogram"; intracardially derived ECG
IVC	Inferior vena cava, V. cava inferior
LAA	"Left atrial appendage"; left atrial appendage
LAMRT	Left atrial macroreentrant tachycardia
LAO	"Left-anterior-oblique" projection direction
LAT	"Local activation time," lokale Aktivierungszeit
LGE	"Late gadolinium enhancement"; methodology in cardiac MRI
LIPV	"Left inferior pulmonary vein"; left inferior pulmonary vein
Long RP	Long interval between R-wave and p-wave
LR-AT	"Localized reentrant atrial tachycardia"; Localized atrial reentry tachycardia
LSI	Lesion Index; Lesionsindex
LSPV	"Left superior pulmonary vein"; left superior pulmonary vein
LV	Left ventricle
LVEDD	Left ventricular end-diastolic diameter
LVOT	"Left ventricular outflow tract"; left ventricular outflow tract
MAZE	Designation of a surgical ablation
MR-AT	"Macroreentrant atrial tachycardia"; atrial macro-reentry tachycardia
MRT	Magnetic resonance imaging

NCC	"Non-coronary cusp"; non-coronary pocket of the aortic sinus
NCX1	Na^+/Ca^{2+} exchanger
NIKM	Non-ischemic cardiomyopathy
PA	posterior-anterior; X-ray image, in which the beam path in relation to the body proceeds from back (posterior) to front (anterior)
PES	Programmed electrical stimulation
PFO	Persistent foramen ovale
PJRT	"Permanent junctional reciprocating tachycardia"; Permanent junctional reentry tachycardia
PPI	Post-pacing interval; post-stimulation interval
PV	Pulmonary vein
PVI	Pulmonary vein isolation
RAA	"Right atrial appendage", right atrial appendage
RAO	"Right-anterior-oblique" projection direction
RF	Radiofrequency
RIPV	"Right inferior pulmonary vein"; right inferior pulmonary vein
RSPV	"Right superior pulmonary vein"; right superior pulmonary vein
RV	Right ventricle
RVOT	"Right ventricular outflow tract"; right ventricular outflow tract
Short RP	Short interval between R-wave and p-wave
SMA	Superior mitral valve annulus
SOP	Standard Operating Procedure; standard procedure
SP	"Slow pathway," slow conduction pathway
SVC	Superior vena cava, V. cava superior
SVT	Supraventricular tachycardia
TA	Tricuspid valve annulus
TCL	"Tachycardia cycle length", cycle length of the tachycardia
TEE	Transesophageal echocardiography
TIA	Transient ischemic attack
TK	Tricuspid valve
TSP	Transseptal puncture
TTE	Transthoracic echocardiography
TTI	"Time to isolation"; time until isolation (of the pulmonary vein)
VBP	"Ventricular Premature Beat"; ventricular extrasystole; Synonym: VES, PVC
VES	Ventricular extrasystole
VHF	Atrial fibrillation
VKA	Vitamin K antagonist
VOP	"Ventricular Overdrive Pacing"; Ventrikuläre Übersteuerungsstimulation (method for differentiating SVT)
VT	Ventricular tachycardia
WOI	"Window of interest"
WPW	Wolff-Parkinson-White (syndrome)

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- What I love about EP: The perfect synthesis of craftsmanship, intellectual work, and teamwork.
- What I don't like about EP: AF redos with isolated veins.
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- What I would like to be able to do: Play a (rhythm) instrument.
- Favorite arrhythmia: Atypical atrial flutter.
- The book is great because it includes the things I would have liked to know from the beginning.

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Electrophysiological Mechanisms of Cardiac Arrhythmias

1

Martin Borlich

1.1 Introduction

Cardiac arrhythmias arise from disturbances in the formation or conduction of impulses, which can also occur in combination. Bradycardic arrhythmias arise from either sinus node dysfunction or impaired conduction in the cardiac conduction system. The fundamental mechanisms for the development of tachycardic arrhythmias are considered to be increased automaticity, triggered activity, and reentry.

Automaticity refers to the ability of cardiomyocytes to spontaneously depolarize and generate impulses without prior stimulation (pacemaker function). Increased automaticity includes the physiologically increased automaticity of cells with primary pacemaker function (e.g., sinus tachycardia during fever) and the abnormally increased automaticity in disturbances and deviations of the regular excitation process (e.g., supraventricular extrasystoles). Triggered activity is the initiation of impulses in cells caused by depolarizing oscillations of the membrane potential, referred to as afterdepolarizations. They occur as a result of preceding action potentials and can be divided into early (EAD—"early afterdepolarization") and late (DAD—"delayed afterdepolarization") afterdepolarizations. These can manifest, for example, as early-onset ventricular extrasystoles and trigger life-threatening ventricular arrhythmias.

In reentry, an anatomical or functional substrate allows sustained circulating excitation. A spreading depolarization wave does not extinguish after the initial tissue activation but reactivates the site of the original excitation. This mechanism is the most common cause of arrhythmias. AV nodal reentrant tachycardia (AVNRT) or atrial flutter, for example, are based on reentry mechanisms.

The diagnosis of the underlying mechanism of a cardiac arrhythmia is significant for the selection of appropriate pharmacological or interventional therapy. Invasive electrophysiological studies provide clues about the mechanism through the spontaneous behavior of the arrhythmia and the response to standardized electrophysiological stimulation maneuvers. A definitive proof of the underlying mechanism is not always successful, as these can occur simultaneously and transition fluidly into one another.

▶ The fundamental mechanisms for the development of tachycardic arrhythmias include **increased automaticity**, **triggered activity**, and **reentry**.

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1.2 Disturbances of Automaticity

1.2.1 Automaticity and Hierarchy of Pacemaker Function

The cells of the excitation formation and conduction system are hierarchically organized. Their defining characteristic is the ability to depolarize spontaneously, gradually, and diastolically, generating new action potentials upon reaching a threshold. The normal automaticity of the sinoatrials node determines the heart rate under physiological conditions (see Fig. 1.1).

Spontaneous diastolic depolarization, essential for pacemaker function, arises from a net increase in intracellular positive charges during diastole. (Anumonwo and Pandit 2015).

Cells of the sinoatrial node lack specific potassium channels (K^+ inward rectifier I_{K1}), which are involved in stabilizing the resting membrane potential in other cardiomyocytes. Outward potassium currents are carried by delayed rectifier potassium channels (I_K), which are responsible for repolarization. These voltage-dependent potassium channels are inactivated after the maximum diastolic potential, thus enabling early diastolic depolarization. The decline of the outward K^+ current is followed by the activation of inward ion currents. With the hyperpolarization of the cell membrane, specific inward channels, known as "funny channels" (I_f), are opened. They allow an influx of Na^+ and, to a lesser extent, K^+ ions into the cell interior and are primarily responsible for the slow depolarization in this phase 4 of the action potential (DiFrancesco 2010). Additionally, Ca^{2+} channels (I_{CaT}) open and allow an inward current of Ca^{2+} ions. This triggers local calcium releases from the endoplasmic reticulum and accelerates the final part of the diastole. Rising intracellular calcium concentration activates the Na^+/Ca^{2+} exchanger (NCX1), which transports three Na^+ ions into the cell for one Ca^{2+} ion and generates a net influx of positively charged ions into the cell. Upon reaching the threshold potential of approximately −40 mV, a new action potential is generated. This rapid depolarization and thus the beginning of the new action potential in the sinoatrial node is carried by the opening of L-type calcium channels and not by a sodium influx as in the working myocardium. With this depolarization, inward channels (I_f, I_{CaT}, I_{CaL}) close again, and repolarization begins anew (Issa et al. 2018). The interplay of time- and voltage-dependent ion channels, which underlies the pacemaker activity, is referred to as the "membrane clock" and interacts strongly with intracellular Ca^{2+} signaling ("calcium clock") for the joint regulation of the automaticity of pacemaker cells (Carmeliet 2019).

The cells of the sinoatrial node reach the action potential threshold under physiological conditions earlier than subordinate cells in the hierarchy of the specific conduction system. Their slower diastolic depolarization is interrupted by the conducted action potential. This determines the heart rate by the sinoatrial node. Since spontaneous diastolic depolarization is a normal property of these cell groups, it is considered "normal" or physiological automaticity.

The pacing rate of the pacemaker cells of the sinoatrial node is influenced by the maximum diastolic potential, the threshold potential for triggering an action potential, and the rate of diastolic depolarization. The activity of the sympathetic and parasympathetic nervous systems controls this pacing rate. Parasympathetic influences on the sinoatrial node and the AV node slow the rate of impulse generation by increasing K^+ conductance (hyperpolarization). This prolongs the time to reach the threshold potential. The sympathetic nervous system increases the discharge rate of the sinoatrial and AV nodes through β1-adrenergic stimulation by increasing the net influx of ions, resulting in an acceleration of diastolic depolarization. Similarly, medications or electrolyte shifts can influence the heart rate.

1.2.2 Increased Automaticity

Cells that do not belong to the specific excitation formation and conduction system do not spontaneously depolarize under physiological

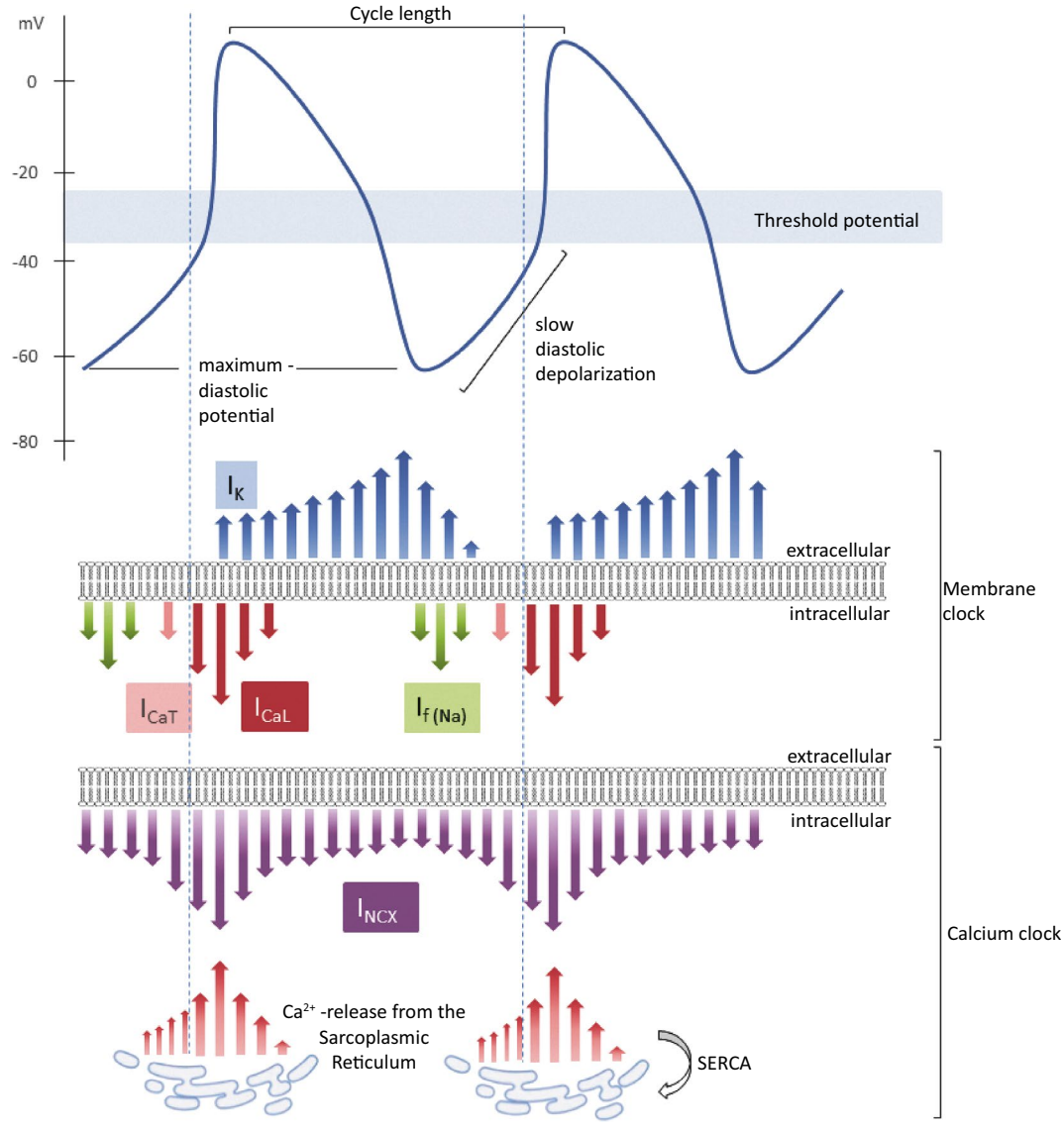

Fig. 1.1 Principle of automaticity in sinus node cells. The course of the membrane potential in sinus node cells (*top*) and the involved ion currents (*middle*) and components of the "calcium clock" are shown. The diastolic depolarization is characteristic of pacemaker cells. The opening of voltage-dependent If channels ("funny channel") and ICaT channels results in slow depolarization through a net influx of positively charged ions. The increase in intracellular Ca^{2+} concentration during diastolic depolarization leads to a local release of Ca^{2+} from the sarcoplasmic reticulum (Ca^{2+}-induced Ca^{2+} release). The sodium-calcium exchanger (NCX1) is also activated, generating a net influx of positively charged ions. Upon reaching the threshold potential, rapid depolarization begins through the opening of L-type calcium channels (ICaL) (no Na^+ influx as in working myocardium). Repolarization occurs through increased conductivity of potassium channels (Ik). The Ca^{2+} pump SERCA transports calcium ions back into the sarcoplasmic reticulum, thereby lowering the intracellular Ca^{2+} concentration (adapted from Murphy C, Lazzara R. Current concepts of anatomy and electrophysiology of the sinus node. J Interv Card Electrophysiol. 2016 Jun;46(1):9-18).

conditions. However, in, for example, ischemic regions of the heart, more positive resting membrane potentials can occur, which can trigger spontaneous depolarization (depolarization-induced automaticity). The difference between physiological and abnormal (mostly

increased) automaticity is thus that the excitation process of the cells exhibiting increased activity deviates from their normal course. Not only can a lower resting membrane potential lead to abnormal automaticity, but also altered potassium conductivity or abnormal release of Ca^{2+} from the sarcoplasmic reticulum. This results in a change in the excitation process of the cells, resulting in bradycardia or tachycardia (see Fig. 1.2).

Increased automaticity is not very susceptible to suppression by overstimulation. It can be well treated with medication such as beta-blockers or antiarrhythmics, or specifically terminated by ablation (Zipes et al. 2017). Cardiac arrhythmias based on increased automaticity include, for example, inappropriate sinus tachycardia, focal atrial tachycardia, (supra-)ventricular extrasystoles, or certain (often idiopathic) ventricular tachycardias. Increased automaticity as a cause of arrhythmias is not as common as triggered activity or the reentry mechanism.

▶ Clinical arrhythmias based on the mechanism of increased automaticity include focal atrial tachycardia, inappropriate sinus tachycardia, (supra-)ventricular extrasystoles, and certain forms (often idiopathic) of ventricular tachycardia.

1.3 Triggered Activity

Triggered activity refers to the initiation of impulses in heart muscle cells that occurs as a result of afterdepolarizations following previous action potentials. They occur either early during the repolarization phase of the preceding action potential (early afterdepolarizations = EAD) or late after the completion of repolarization (delayed afterdepolarizations = DAD). If these depolarizing oscillations of the membrane potential are large enough to reach the threshold potential for triggering a new action potential, this is considered triggered (see Fig. 1.3). Single extrasystoles or sustained tachycardias can be the result.

1.3.1 Early Afterdepolarizations (EAD)

Early afterdepolarizations (EAD) are oscillations in the membrane potential that occur

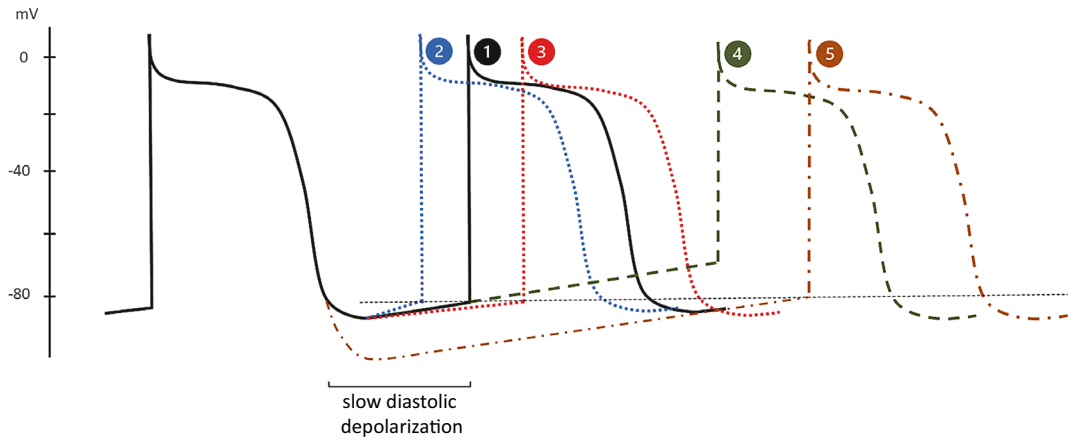

Fig. 1.2 Influence of regular automaticity using the example of a His-Purkinje cell. Two action potentials under physiological conditions are shown (*1*). His-Purkinje cells exhibit spontaneous diastolic depolarization. The cell's impulse rate is increased by accelerating diastolic depolarization (*2*) and is slowed by delaying diastolic depolarization (*3*), increasing the threshold potential (*4*), and starting diastolic depolarization from a more negative resting membrane potential (*5*)