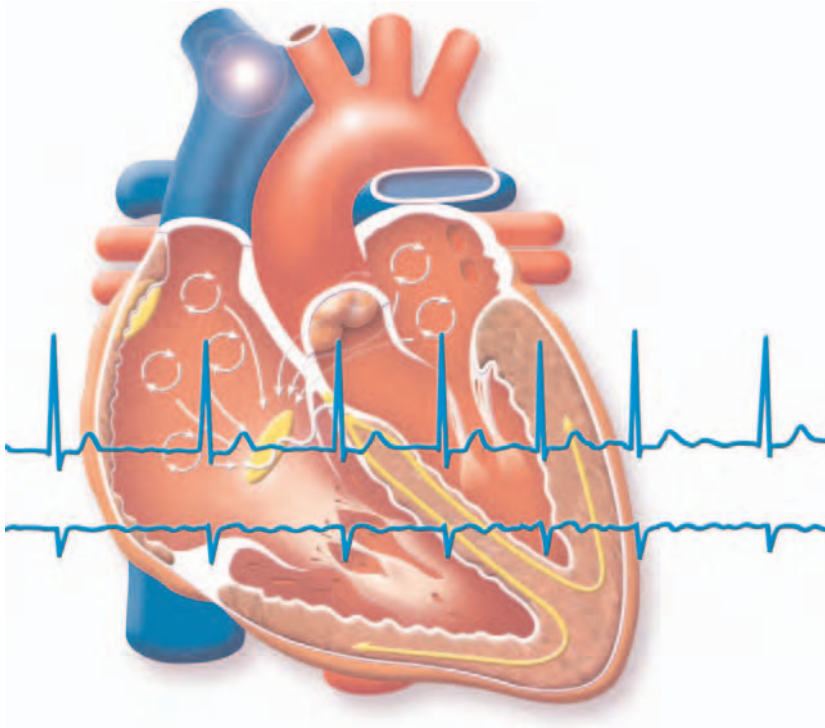


Fast Facts



Fast Facts: Cardiac Arrhythmias

Gerry Kaye, Steve Furniss and Robert Lemery





Fast Facts: Cardiac Arrhythmias



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Declaration of Independence

This book is as balanced and as practical as we can make it.
Ideas for improvement are always welcome: feedback@fastfacts.com



Fast Facts: Cardiac Arrhythmias

First published May 2010

Text © 2010 Gerry Kaye, Steve Furniss, Robert Lemery

© 2010 in this edition Health Press Limited

Health Press Limited, Elizabeth House, Queen Street, Abingdon,
Oxford OX14 3LN, UK

Tel: +44 (0)1235 523233

Fax: +44 (0)1235 523238

Book orders can be placed by telephone or via the website.

For regional distributors or to order via the website, please go to:
www.fastfacts.com

For telephone orders, please call 01752 202301 (UK), +44 1752 202301 (Europe),
1 800 247 6553 (USA, toll free), +1 419 281 1802 (Americas) or
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A CIP record for this title is available from the British Library.

ISBN 978-1-903734-88-9

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Fast Facts: Cardiac Arrhythmias/

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Medical illustrations by Dee McLean, London, UK.

Typesetting and page layout by Zed, Oxford, UK.

Printed by Latimer Trend & Company Ltd, Plymouth, UK.

Text printed with vegetable inks on biodegradable and
recyclable paper manufactured using elemental chlorine
free (ECF) wood pulp from well-managed forests.



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Cert no. SGS COC 005493
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Glossary of abbreviations and terms

AF: atrial fibrillation

ATP: anti-tachycardia pacing

AV node: atrioventricular node

AVNRT: atrioventricular nodal re-entrant tachycardia (also described as junctional tachycardia)

AVRT: atrioventricular re-entrant tachycardia

bpm: beats per minute

CAD: coronary artery disease

Conduction: the speed at which cells propagate electrical wavefronts; different structures within the heart conduct at different rates (e.g. the His-Purkinje system conducts as rapidly as neural tissue [about 3 m/s], whereas conduction at the compact AV node is slower)

CRT: cardiac resynchronization therapy

ECG: electrocardiogram

EP: electrophysiology

HCM: hypertrophic cardiomyopathy

ICD: implantable cardioverter defibrillator

INR: international normalized ratio

LAA: left atrial appendage

LV: left ventricular (function)

Macro or micro re-entrant arrhythmia: circuit that covers a large or small area of the myocardium, respectively

MI: myocardial infarction

NCT: narrow-(QRS)-complex tachycardia

PVI: pulmonary vein isolation

Refractoriness: as the rate of stimulation of a cardiac muscle cell (myocyte) increases, it will continue to contract until the external stimulus falls when the cell has not recovered its excitability from the previous stimulus; at this point the cell is refractory (i.e. it cannot respond to any stimulus)

RFA: radiofrequency ablation (also described as catheter ablation)

RVOT: right ventricular outflow tract

SA node: sinoatrial node

SVT: supraventricular tachycardia

VF: ventricular fibrillation

VT: ventricular tachycardia

WCT: Wide-(QRS)-complex tachycardia (also described as broad complex tachycardia)

WPW: Wolff–Parkinson–White (syndrome)

Introduction

The management of cardiac rhythm has changed beyond all recognition in recent years, moving from a purely diagnostic field to a therapeutic one. Pharmacological therapy formed the basis of treatment for arrhythmias for many years. However, growing dissatisfaction with pharmacological treatments, unacceptable side effects, palliation rather than cure and, in some cases, a worsening of the arrhythmia itself, led to the desire for non-drug-based treatments.

Electrical therapy in the form of external direct-current countershock showed that reliance on drug therapy need not be absolute. The real breakthrough came with invasive electrophysiological studies. These provided deep insights into the mechanisms of arrhythmias in humans and culminated in the use of radiofrequency energy to safely treat, and indeed cure, many common arrhythmias. This approach has revolutionized the management of cardiac arrhythmias. The success of radiofrequency ablation has been dramatic and its applications are constantly being revised and widened. Arrhythmias that were once thought to be incurable, such as atrial fibrillation, can be modified, if not cured, by this technique, and our understanding of the mechanisms and triggers of many arrhythmias has improved.

For many, cardiac electrophysiology is shrouded in mystery and is viewed as difficult and inaccessible. *Fast Facts: Cardiac Arrhythmias* demystifies this exciting field of modern cardiology and enlightens the reader with regard to the mechanisms of arrhythmias, providing a better understanding of the common arrhythmias and how best to manage them. This comprehensive yet succinct handbook has been written for physicians, nurses and technicians seeking a modern overview of the field of clinical cardiac arrhythmias, and will also be useful to medical students and cardiologists in training.

The emphasis of *Fast Facts: Cardiac Arrhythmias* is on the patient with a rhythm abnormality, how they present and how they can be investigated and managed, a marked divergence from the traditional presentation used in earlier texts on electrophysiology. We have grouped the arrhythmias on the basis of the 12-lead ECG patterns rather than

cellular electrophysiology. We begin with the presentation of patients with arrhythmias and examine how arrhythmias are best investigated. The general features of management are covered, including both drug and non-drug therapies. Specific chapters then cover the different types of arrhythmia – supraventricular arrhythmias, atrial flutter, atrial fibrillation, ventricular arrhythmias, and rare and unusual arrhythmias. The final chapter provides a comprehensive overview of cardiac devices – pacemakers and implantable cardioverter defibrillators (ICDs) – which are now well established as the mainstay of treatment for patients with bradycardias, ventricular tachyarrhythmias and some types of heart failure.

Normal conduction

Cardiac cells have a unique ability to depolarize rhythmically. Normally, depolarization within the heart occurs in one direction from the top downwards. The fibrous ring that supports the mitral and tricuspid valves is an electrical insulator, so depolarization can only travel from the atria to the ventricles via the specialized conducting tissues, unless an abnormal electrically active connection, known as an accessory pathway, is present (see Chapter 2, page 14). The normal conduction pathway within the heart is described below (Figure 1.1).

Atrial depolarization. Conduction originates with self-excitation of the sinoatrial (SA) node, which lies at the junction of the superior vena cava with the upper part of the right atrium. The SA node acts as the heart's pacemaker. A depolarization wavefront spreads down from the SA node to the base of the right atrium and simultaneously spreads to the left atrium over specialized conductive tissue known as Bachmann's bundle. The complete depolarization of the atria gives rise to the P wave on the surface electrocardiogram (ECG; see Figure 1.1). A normal P wave is less than 200 ms wide and smaller than 1 mV in amplitude. It has a low amplitude because the mass of the atria is considerably smaller than that of the ventricles. The P wave is wide because most of the depolarization of the atria occurs by relatively slow cell-to-cell conduction.

Atrioventricular node depolarization. The depolarization wavefront is then directed to the compact atrioventricular (AV) node, which bridges the atria and ventricles near the center of the heart. Conduction through the AV node is slowed in the upper part of the node. This delay allows mechanical contraction of the atria, which is much slower than the electrical activation, to complete before the ventricles contract, and gives rise to the PR interval on the surface ECG (less than 200 ms).

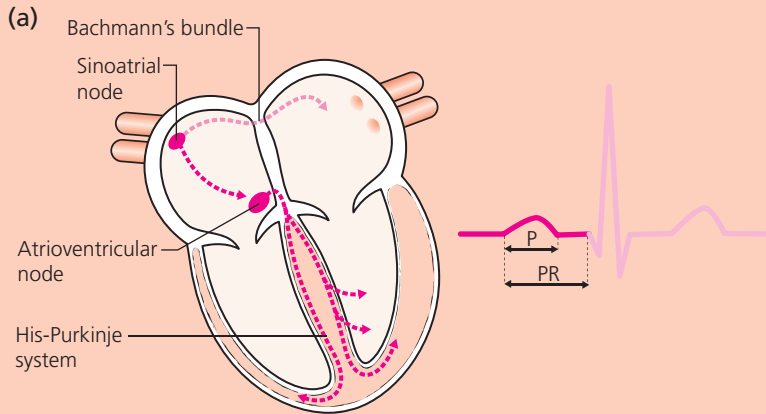


Figure 1.1 Normal conduction pathway within the heart, with corresponding surface ECG. (a) The cardiac impulse arises from the sinoatrial node and depolarizes the atria by cell-to-cell conduction and via Bachmann's bundle, giving rise to the P wave on the ECG. The impulse is slowed through the atrioventricular node, which corresponds to the PR interval on the ECG. The impulse is then conducted rapidly to the His-Purkinje system and bundle branches. (b) The ventricles are depolarized via the His-Purkinje system, giving rise to the narrow QRS complex on the ECG. (c) Electrical recovery of the ventricles corresponds to the T wave. The QT interval is measured from the onset of the QRS complex to a point where the down stroke of the T wave crosses the baseline; the faster the heart beat the shorter the QT interval.

Septal depolarization. Once through the AV node, the wavefront travels through the septum and reaches the specialized His-Purkinje system. The His tissue conducts rapidly and, after splitting to follow the left and right bundle branches, the wavefront depolarizes the ventricles.

Ventricular depolarization and systole. The mass of ventricular tissue far outweighs that of the atria. Hence, the amplitude of electrical depolarization of the ventricles (represented by the surface QRS

Ventricular tachycardia

The most common type of ventricular tachycardia (VT) is related to ischemic heart disease or cardiomyopathic processes. Less commonly, and usually in younger patients, VT arises in a structurally normal heart.

During normal conduction the depolarizing wavefront arrives at a constant speed within the ventricular myocardium. Recovery occurs at a constant speed in the opposite direction to depolarization (see Chapter 1). During a myocardial infarction, although there may be areas of tissue death that are unable to depolarize, there are also coexisting areas of ischemia. Conduction and recovery are variably slow in these ischemic areas (the substrate) and if an impulse is slowed sufficiently it may re-enter a nearby area that has recovered conduction and that may now allow a re-entrant circuit to occur (Figure 2.9). Ventricular ectopics are often the trigger.

Sometimes the VT circuit covers a relatively large area and may have complex re-entry circuits. These are often deep within the myocardium and occur over a relatively large area, which is why ablation is often difficult and less successful than if the abnormal substrate area is microscopic (as in SVTs).

Ventricular fibrillation

Ventricular fibrillation (VF) is now thought to have a re-entrant basis. It most commonly occurs during (or after) myocardial infarction or ischemia; less often it is related to electrolyte disturbance or drug therapy. VF has been described in 'normal' hearts and is often related to an early-cycle ventricular ectopic falling within the vulnerable period of ventricular recovery. Ablation of these ectopics has been shown to prevent recurrent fibrillation. It is thought that these ectopics allow localized re-entry.

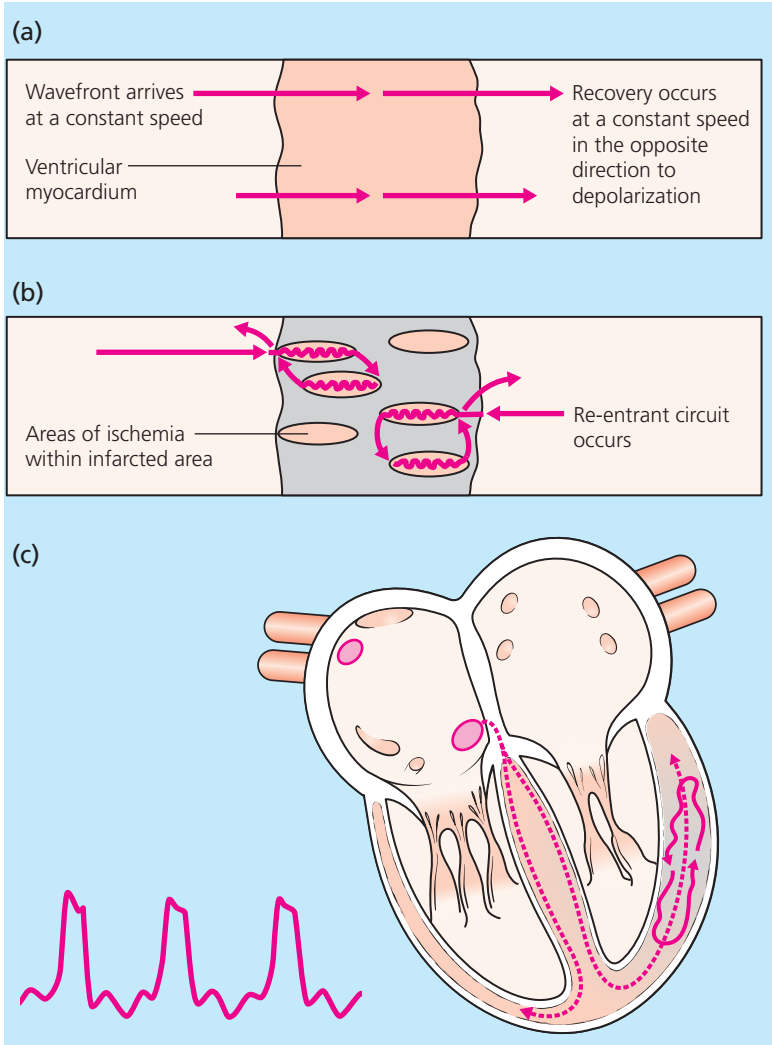


Figure 2.9 (a) In normal ventricular myocardium, depolarization and electrical recovery are uniform. (b) Following a myocardial infarction or ischemia, areas of damaged but viable tissue depolarize and recover more slowly than surrounding tissues. These areas allow impulses to re-enter locally thereby establishing a tachycardia, which may arise within a relatively small area of the ventricle but (c) spreads out to involve the remainder of the ventricular myocardium, giving rise to ventricular tachycardia. The surface ECG shows very wide (broad) QRS complexes.