Companion website

This book is accompanied by a companion website:

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The website includes:

- Helpful Multiple Choice Questions
- Updates from the author
CLINICAL ARRHYTHMOLOGY

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Foreword

By Dr. Valentin Fuster

When I received the manuscript from Antoni Bayés de Luna and his collaborators to write a foreword for this book, I realized with a glance what a great opportunity this work provides. This has been the rule in books published by Antoni Bayés de Luna; they appear when they are needed most. I still remember his book on electrocardiology, which explained the technique of “Electrocardiography” for beginners in a way that was not only concise but very thorough. This book has been translated into eight languages and remains very successful throughout the world. This also occurred with his book on “Sudden death”, as well as his correlations between electrocardiography and cardiovascular magnetic resonance imaging. But for now I would like to talk about Clinical Arrhythmology, which is what interests us most. The current books on arrhythmias mainly explain the great technological advances being achieved in diagnosis and, in particular, the interventionist treatment of cardiac arrhythmias. However, most of these books fail to examine the clinical aspects closely enough and do not emphasize the crucial role for diagnosis of the surface electrocardiogram, nor do they discuss how the clinical cardiologist or family doctor, or even the emergency medicine doctor, might proceed once this diagnosis is performed, in order to rapidly and efficiently treat the specific arrhythmias in the clinical context in which they appear. The book is full of the experience of Antoni Bayés de Luna teaching electrocardiology and arrhythmias in the style of Paul Puech, Leo Schamroth, and Charles Fisch, with an updated state-of-the-art of the management of arrhythmias.

This book is filled with advice on how to diagnose and effectively treat arrhythmias with classic knowledge that, at the same time, is up-to-date, using many references from 2010. Antoni Bayés de Luna emphasizes the necessity to consult and use the medical guidelines of the scientific societies, while at the same time giving a personal touch derived from his considerable experience. This is especially present in Chapter 1, where he emphasizes the importance that history taking and physical examination still have when diagnosing and treating arrhythmias. He gives a series of recommendations that state the necessity to know heart anatomy and physiology well, in addition to outlining how to approach a case with arrhythmias. I also consider the updated physiopathologic mechanisms of arrhythmias to be of great interest. Later on, in the second part of the book, all the different clinical, electrocardiographic, prognostic, and management aspects of different arrhythmias are clearly commented on. The third part deserves close study because it is where sudden death, being the most important complication of arrhythmia, is examined and discussed in different heart diseases and situations.

I feel that this book demonstrates the great authority of the author, as well as his deep knowledge of clinical arrhythmia and electrocardiography, great didactic capabilities and many years of experience in this field. I am sure it will be extremely useful for doctors who are first faced with cardiac arrhythmias, not only in the diagnosis but also in obtaining a clear idea as to how to focus management of the condition, including the last advances in the treatment through ablation techniques and pacemaker and defibrillator implantation in different types of arrhythmias.

I would like to offer my wholehearted congratulations to Antoni Bayés de Luna for providing all his personal experience in a subject of great clinical importance and based on the crucial value placed on the history taking and especially the surface
Foreword

electrocardiogram in the diagnosis and management of cardiac arrhythmias.

I predict that this book will be a huge success because of its usefulness and timeliness. It will make diagnosis and treatment of different cardiac arrhythmias much easier for students, doctors, and even specialists, without the apprehension often generated in the medical community.

Dr. Valentin Fuster
Director, Mount Sinai Heart Center, New York
Professor of Medicine, Mount Sinai School of Medicine
Past President, American Heart Association
Past President, World Heart Federation
Foreword

By Dr. Pere Brugada i Terradellas

When Professor Antoni Bayés de Luna placed 3 kg of printed material in my hands, I immediately knew what was happening: the “master of masters” had struck again. Undoubtedly, it was a new book. And undoubtedly, it was a book related to electrocardiology, the great love of his life. Knowing him as I have for so many decades, I did not doubt that the manuscript I was now holding had been written to fill a gap in medical knowledge. But what could Antoni have written now that he had not already written? His various books on electrocardiography, published in the most common languages, are known by every admirer of the electrical activity of the heart. No cardiologist has described the electrocardiogram in as much detail as he. His daily work has consisted of the nearly impossible job of dissecting the electrical activity of the heart. And this all without electrocuting himself!

I looked carefully at the title on the first page and those 3 kg soon became lighter: Clinical Arrhythmology. Here was the big secret. Finally, the book that describes the mechanisms, diagnostic clues, and management of cardiac arrhythmias written by the clinical cardiologist for the clinical cardiologist. Thanks to great advances in the study of cardiac electrophysiology, arrhythmia mechanisms are well understood today. However, the general cardiologist, the internist, and the general practitioner must depend continuously on the electrocardiogram to define the swelling mechanism in any cardiac rhythm disorder. Combining clinical and electrophysiologic knowledge with an updated approach of medical management, to produce an integrated textbook of clinical arrhythmology is a challenge few would take on. For this, a clinical and scientific tenacity is required that only a chosen few possess, one of whom is Professor Antoni Bayés de Luna.

These thoughts crossed my mind during the minutes I used to look through the manuscript. Antoni, aware of my love for his work, asked if I would like to write a foreword for this book. Absolutely! I said, I would do it with great pleasure, in order to thank him on behalf of myself and many others for his great efforts in teaching, and for the numerous hours of pleasant reading he has given us. To thank him for the great care he has always taken with his books, including this one, naturally, to offer us clear outlines accompanied by greatly didactic diagrams, which are a pleasure to read and study.

Clinical Arrhythmology is obligatory reading for any physician directly or indirectly related to disorders of cardiac rhythm, including cardiologists, internists, sports medicine doctors, and general practitioners. They will find in this book that combination of clinical experience and great electrocardiographic skills is the best way to approach successfully the diagnosis and treatment of cardiac arrhythmias. It is also a superb resource for paramedics who may be faced with cardiac arrhythmias.

Professor Bayés de Luna must be congratulated on his magnificent effort and the excellent end result of this book.

Dr. Pere Brugada i Terradellas
Scientific Director, Centro UZ Brussel Cardiovascular Centre, Brussels, Belgium
Preface

First, I would like to explain why I have written this book. I am a clinical cardiologist who has been especially dedicated to teaching and research in electrocardiography as well as in clinical and non-invasive aspects of arrhythmias and sudden death (SD). Looking back on my life, I have had the opportunity to contemplate how arrhythmology has changed in the last 50 years and become a well defined subspecialty. Currently, arrhythmologists not only need important training as interventionist electrophysiologists, but also a wide knowledge of the epidemiological, clinical, electrocardiographic and genetic aspects of all arrhythmias. My main interest in writing this book is to make available to the clinical cardiologist, or trainee in cardiology, internist or general practitioner interested in this topic, the anatomical and electrophysiological aspects necessary for understanding the mechanisms of arrhythmias and the bases to diagnose and treat them with precision. I do not, however, describe in detail the technical aspects of each diagnostic and therapeutic procedure used today, nor do I discuss the molecular and genetic aspects of cardiac arrhythmias in depth. The reader may find an adequate bibliography for all of this in the text. However, I believe that this book fills a gap. Currently, most arrhythmology books extensively present those aspects related to treatment through invasive methods rather than examine how the diagnosis of an arrhythmia is reached through history taking and careful surface electrocardiography. At the same time, this book also presents a practical, up-to-date focus on prognosis and therapeutic decision making in all types of arrhythmias, including the prevention of SD.

The book is divided into three parts. In the first part, the concept, classification and clinical progress of arrhythmias is presented, with emphasis on its relation to sudden death, as well as the most interesting information still relevant today on the great utility of anamnesis and the physical exam in their diagnosis. Next, the characteristics of each type of cardiac cell are described from an ultrastructural, ionic and electrophysical point of view. Lastly, the most important electrophysiological mechanisms that explain cardiac arrhythmias are discussed.

The second part describes the key elements used to carry out an electrocardiographic diagnosis of the various active and passive arrhythmias, the clinical and prognostic implications and the best method of treatment, explained in a practical way. The current utility of anti-arrhythmic agents and the various techniques (cardioversion and ablation) and implantable devices (pacemakers and defibrillators) available is discussed. They are very useful in the treatment of arrhythmias and the prevention of SD. Finally, in Chapter 7 I describe how to carry out the analytical study and differential diagnosis of different arrhythmias.

The third part deals with the most frequent arrhythmological syndromes, including pre-excitation and channelopathies, as well as other electrocardiographic patterns suggestive of a risk of arrhythmia or sudden death. The most frequent arrhythmias and the markers of SD in different arrhythmias and different situations are also described.

Throughout the book, emphasis is placed on the importance of surface electrocardiography as the basic technique to diagnose arrhythmias at a clinical physician’s level. However, in the Appendix there is a review of other complementary techniques, which at times are very useful in reaching the correct diagnosis or carrying out the most adequate treatment. The reader will find more information about these techniques in the recommended bibliography (see p. xii). Additionally, the Appendix includes an explanation of the basic concepts of sensitivity, specificity and predictive value necessary for the correct evaluation of the different diagnostic electrographic criteria. I also explain the recommendations for both treatment and the application of the various diagnostic tests mentioned in the Scientific Societies guidelines. The physician must of course take these guidelines into considera-
tion but has to bear in mind the individual characteristics of each case, as well as his own personal experience, in order to reach the most accurate diagnosis and offer his patients the best therapeutic option for each arrhythmia. This is the ideal way to proceed, since the guidelines sometimes have not introduced the latest developments and do not contemplate that all the best approaches of management may not be feasible in developing countries. This is what I think has to be considered at all times.

I have tried to present the information here in a coherent and homogeneous way, although at times it may result in repetition in some aspects. I am aware of this, but I believe it to be useful, particularly to the non-expert, in order to reinforce basic knowledge. At the same time, the reader is very often referred for further information either to the cross references related to other sections of the book or sections just before (“see before”) or after (“see after”) the text. I believe that this makes the book more harmonious and allows to the reader to interact better with other parts of the book. Additionally, at the end of each chapter there are self evaluation questions, the answers to which may be found on the pages of the book where the corresponding letter appears in the margin.

In terms of the bibliography, a list of recommended texts for general reference is provided after this preface. In addition, at the end of each chapter there is a bibliography specific to each particular subject. The name of the first author of each article has been inserted in the text in the appropriate place.

I am sure, therefore, that after reading this book the reader will have learned all the basic concepts needed to face the often difficult problem of immediate diagnosis based on an electrocardiographic tracing with an arrhythmia in his future clinical practice. I hope he not only understands the most important clinical, prognostic and therapeutic implications of this diagnosis in every case but also acquires more confidence in this task.

This book is the result of many years of teaching cardiology, especially electrocardiography and arrhythmias. It is a source of pride for me to have V. Fuster and P. Brugada, two of the greatest representatives of cardiology in the world on both sides of the Atlantic, Catalan like me and good friends of mine since the beginning, honor me by writing a glowing prologue for this book. I feel their words not only complement the work but also express its meaning for general cardiologists, cardiology residents and internists.

I would like to express my gratitude to my mentors M. Torner, I. Balaguer, P. Puech and M. Rosenbaum, as well as my collaborators, especially D. Goldwasser, X. Viñolas, M. Fiol, I. Cygankiewicz, J. García Niebla, A. Pérez Riera, P. Iturralde, R. Oter, R. Brugada, W. Zareba, and A. Bayés-Genis for their help in the critical revision of the manuscript, their punctual contributions and their help in selecting references. I am also indebted to J. Riba, J. Guindo, T. Martinez Rubio, M.T. Subirana, R. Elsoua, P. Torner, I. Ramírez, P. Ferres, J. Massó, E. Vallés, X. Gurri, A. Boix, J. Puig, E. Rodríguez, J. Guerra, C. Alonso, A. Carrillo and E. Vallés, among others, who have also been collaborators for many years. Thank you to X. Viñolas along with each and every member of the electrophysiology team at the Hospital de la Santa Creu i Sant Pau and the Hospital Quirón for the excellent images of electrophysiology and ablation they provided. I would like to thank the Fundación Jesús Serra (Catalana Occidente) for their constant support of our research at the Hospital de la Santa Creu i Sant Pau and their decisive help in the creation of Chair for Cardiovascular Research, held by L. Badimon, and also to Laboratorios Dr. Esteve for their continuous support to our ECG postgraduate courses. As always, I would like to thank M. Saurí, who repeatedly typed the manuscript of this book with a smile on her face the entire time. Thank you to Thomas V. Hartman of Wiley-Blackwell Publishers and his magnificent group of collaborators for their excellent work during the publication process of this book. Finally, I would like to dedicate this work to my wife M. Clara and my children and grandchildren, in gratitude for their patience and understanding during its preparation.

Antoni Bayés de Luna
Plaza Catedral, Vic
October 2010
Recommended General Bibliography


PART I

Anatomical and Electrophysiological Considerations, Clinical Aspects, and Mechanisms of Cardiac Arrhythmias
CHAPTER 1
Clinical Aspects of Arrhythmias

Definition of arrhythmia

Arrhythmias are defined as any cardiac rhythm other than the normal sinus rhythm. Sinus rhythm originates in the sinus node. The electrocardiographic characteristics of normal sinus rhythm are:

- A sinus stimulus originates in a sinus node and subsequently occurs at appropriate rates of conduction transmitted through atria, the atrioventricular (AV) junction, and the intraventricular specific conduction system (ISCS). It initiates a positive P wave in I, II, Vf, V1–V6, and positive or ± in leads III and V1.

- In adults, in the absence of pre-excitation, the PR interval ranges from 0.12 to 0.20s.

- At rest, the sinus node discharge cadence tends to be regular, although it presents generally slight variations, which are not evident by palpation or auscultation. However, under normal conditions, and particularly in children, it may present slight to moderate changes dependent on the phases of respiration, with the heart rate increasing with inspiration.

- In adults at rest, the rate of the normal sinus rhythm ranges from 60 to 80 beats per minute (bpm). Thus, sinus rhythms over 80bmp (sinus tachycardia) and those under 60bmp (sinus bradycardia) may be considered arrhythmias. However, it should be taken into account that sinus rhythm varies throughout a 24-h period, and sinus tachycardia and sinus bradycardia usually are a physiologic response to certain sympathetic (exercise, stress) or vagal (rest, sleep) stimuli. Under such circumstances, the presence of these heart rates should be considered normal.

- As we have already stated, it is normal to observe a certain variation in sinus rhythm in association with the respiratory rate when at rest. Thus, the evidence of a completely fixed heart rate both during the day and at night is suggestive of arrhythmia. In addition, it is important to remember that:

1) The term arrhythmia does not mean rhythm irregularity, as regular arrhythmias can occur, often with absolute stability (flutter, paroxysmal tachycardia, etc.), sometimes presenting heart rates in the normal range, as is the case with the flutter 4×1. On the other hand, some irregular rhythms should not be considered arrhythmias (mild to moderate irregularity in the sinus discharge, particularly when linked to respiration, as already stated).

2) A diagnosis of arrhythmia in itself does not mean evident pathology. In fact, in healthy subjects, the sporadic presence of certain arrhythmias, both active (premature complexes) and passive (escape complexes, certain degree of AV block, evident sinus arrhythmia, etc.) is frequently observed.

Classification

There are different ways to classify cardiac arrhythmias.

- According to the site of origin: arrhythmias are divided into supraventricular (including those having their origin in the sinus node, the atria, and the AV junction), and ventricular arrhythmias.

- According to the underlying mechanism: arrhythmias may be explained by: 1) abnormal formation of impulses, which includes increased heart automaticity (extrasystolic or parasystolic mechanism) and triggered electrical activity, 2) reentry of different types, and 3) decreased automaticity and/or disturbances of conduction (see Chapter 3).
From the clinical point of view: arrhythmias may be paroxysmal, incessant, or permanent. In reference to tachyarrhythmias (an example of an active arrhythmia, see later), paroxysmal tachyarrhythmias occur suddenly and usually disappear spontaneously (i.e. AV junctional reentrant paroxysmal tachycardia); permanent tachyarrhythmias are always present (i.e. chronic atrial fibrillation); and incessant tachyarrhythmias are characterized by short and repetitive runs of supraventricular (Figure 4.21) or ventricular (Figure 5.4) tachycardia. Extrasystoles may also occur in a paroxysmal or incessant way. If they persist they may also be described as incessant or permanent (e.g. permanent atrial tachycardia or bigeminal rhythm) (see Chapter 3, Mechanisms responsible for active cardiac arrhythmias). Some bradyarrhythmias, such as advanced AV block (an example of passive arrhythmia, see later), may also occur in a paroxysmal or permanent form.

Finally, from an electrocardiographic point of view, arrhythmias may be divided into two different types: active and passive (Table 1.1).

- **Active arrhythmias**, due to increased automaticity, reentry, or triggered electrical activity (see Chapter 3 and Table 3.1), generate isolated or repetitive premature complexes on the electrocardiogram (ECG), which occur before the cadence of the regular sinus rhythm. The isolated premature complexes may be originated in a parasystolic or extrasystolic ectopic focus. The extrasystolic mechanism presents a fixed coupling interval, whereas the parasystolic presents a varied coupling interval. Premature complexes of supraventricular origin (p') are generally followed by a narrow QRS complex, although they may be wide if conducted with aberrancy. The ectopic P wave (P') is often not easily seen as it may be hidden in the preceding T wave. In other cases the premature atrial impulse remains blocked in the AV junction, initiating a pause instead of a premature QRS complex (Figures 4.1C and 7.3). The premature complexes of ventricular origin are not preceded by an ectopic P wave, and the QRS complex is always wide (≥0.12 s), unless they originate in the upper part of the intraventricular specific conduction system (see Chapter 5, Electrocardiographic diagnosis).

- **Passive arrhythmias** occur when cardiac stimuli formation and/or conduction are below the range of normality due to a depression of the automatism and/or a stimulus conduction block in the atria, the AV junction, or the specific intraventricular conduction systems (ICS).

Clinical significance and symptoms

The incidence of the majority of arrhythmias increases with age progressively, and arrhythmias are not frequent in children. Data from the Holter ECG recordings (see Appendix A-3, Holter electrocardiographic monitoring and related techniques) have demonstrated that isolated premature ventricular complexes (PVC) are present in about 10–20% of young people in 24-h recordings, and their presence is nearly a rule in the 80+ age group. Similarly, sustained chronic arrhythmias, such as atrial fibrillation, are exceptional in children, and are present in about 10% of subjects over 80 years.
In this book devoted to providing the basis for the diagnosis, prognosis, and treatment of arrhythmias, we use active and passive classification of arrhythmias (Table 1.1).

- Active cardiac arrhythmias include isolated or repetitive impulses that command heart rhythm, instead of the basic normal sinus rhythm. They are recorded on the ECG tracing as isolated (premature supraventricular or ventricular complexes), repetitive (named runs), or sustained complexes (different types of tachyarrhythmias).
- Many passive cardiac arrhythmias show isolated or repetitive sinus or escape complexes in the ECG tracings with an abnormally slowed heart rate (bradyarrhythmias). This may be due to depression of automaticity or sinoatrial or AV block. However, in some cases the mechanism responsible for the passive cardiac arrhythmia is delayed conduction, which may modify the ECG pattern (first-degree AV block, or atrial or ventricular bundle branch block), but this does not mean that the heart rate has to be slow.

Table 1.1 Classification of arrhythmias according to their electrocardiographic presentation

<table>
<thead>
<tr>
<th>Active arrhythmias</th>
<th>Passive arrhythmias</th>
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<tbody>
<tr>
<td>Supraventricular</td>
<td>Escape complex</td>
</tr>
<tr>
<td>□ Premature complexes</td>
<td>Escape rhythm</td>
</tr>
<tr>
<td>□ Tachyarrhythm</td>
<td>Sinus bradycardia</td>
</tr>
<tr>
<td>▪ Different types of tachycardia</td>
<td>Sinoatrial block</td>
</tr>
<tr>
<td>▪ Atrial fibrillation</td>
<td>Atrial block</td>
</tr>
<tr>
<td>▪ Atrial flutter</td>
<td>Atrioventricular block</td>
</tr>
<tr>
<td>Ventricular</td>
<td>Ventricular block</td>
</tr>
<tr>
<td>□ Premature complexes</td>
<td>Aberrant conduction</td>
</tr>
<tr>
<td>□ Different types of tachycardia</td>
<td>Cardiac arrest</td>
</tr>
<tr>
<td>□ Ventricular flutter</td>
<td></td>
</tr>
<tr>
<td>□ Ventricular fibrillation</td>
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</table>

of age. However, there are arrhythmias that arise particularly in children, such as some paroxysmal and incessant AV junctional reentrant tachycardias (AVJRT), as well as some monomorphic ventricular tachycardias (idiopathic) and polymorphic ventricular tachycardias (catecholaminergic).

The most important clinical significance of arrhythmias is related to an association with sudden cardiac death (Goldstein et al. 1994, p. xii). It is also important to remember that frequently arrhythmias (especially atrial fibrillation), may lead to embolism, including cerebral emboli, often with severe consequences. Also, we have to remember that sometimes fast arrhythmias may trigger or worsen heart failure (HF). We will comment on these aspects.

Arrhythmias and sudden death (SD)

We will now look at some of the most important aspects of SD, a true epidemic of the twenty-first century. However, in other parts of the book (Chapters 8–11) specific aspects of SD in relation to different heart diseases or situations will be discussed in more detail.

Epidemiology

- Sudden death is probably the most challenging issue in modern cardiology, taking into account the remarkably high number of SD cases (the estimated number of SD in the US is approximately 400,000 cases per year, although in Mediterranean countries, such as Spain, the incidence is lower) (Keys and Keys 1975; Masiá et al. 1998; Marrugat et al. 1999; Sans et al. 2005) and the important social impact of SD events.
- Even though SD has been reported in newborns, in whom it has been related to repolarization disorders, alterations of the autonomic nervous system (ANS), and an increase of vagal tone (see Chapter 11, Sudden infant death syndrome), it is indeed very rare in the first decades of life. At this age it often occurs during sports activities (Bayés de Luna et al. 2000) and is often associated with inherited heart disease (hypertrophic cardiomyopathy, arrhythmogenic right ventricular dysplasia/cardiomopathy, and channelopathies). The incidence of SD gradually but significantly increases after 35–40 years of age, and is particularly high during the acute phase of myocardial infarction (MI). It is also frequent during the chronic phase of ischemic heart disease (IHD), as well as in subjects with any heart disease, especially when heart failure (HF) is present (Myerburg et al. 1997) (Figure 1.1).

Associated diseases

- As we have previously discussed, acute IHD is frequently associated with SD in adults. In the majority of cases of SD outside acute IHD or
channelopathies, HF, or at least left ventricular dysfunction, is present. HF may be idiopathic or present in patients with chronic IHD, hypertension, cardiomyopathies, etc. More details on this association will be shown in Chapter 11 (see Chapter 11, Ischemic heart disease, and Heart failure). Inherited heart disease (In.H.D.) can cause SD at any age, but the overall impact is small (Figure 1.1). It should be emphasized, however, that it is responsible for the majority of cases that occur before the age of 35 years. In.H.D. appears more in men and may occur during exercise (cardiomyopathies) or sleep or rest (channelopathies) (see Chapter 9).

- We performed a study (EULALIA trial) that included 204 cases of SD occurring in the Mediterranean area (Subirana et al. 2011). In this study we analyzed the epidemiological and pathological aspects of diseases associated with SD. Table 1.2 shows the diagnosis obtained by the pathologists. When compared with other similar Anglo-Saxon studies (Burke et al. 1997), what caught our attention was that the number of cases presenting with IHD found at autopsy, as well as the incidence of acute thrombosis, as an anatomicopathologic expression of MI, being lower than in previously published Anglo-Saxon studies (80–90% vs. 58%, and 52% vs. 40%, for IHD and acute thrombosis, respectively) (Figure 1.2). Our findings are concordant with previously known evidence (Keys and Keys 1975; de Lorgeril et al. 1999; Marrugat et al. 1999; Sans et al. 2005) that the incidence of IHD in Mediterranean regions is lower, probably related to diet, lifestyle, and environment (Mediterranean culture). In contrast,

![Figure 1.1](image1.png) Relationship between the incidence of sudden death (SD) and age. Note that the sudden death may also be associated with different diseases along the life period (Myerburg et al. 1992).

![Figure 1.2](image2.png) Comparative study of the incidence of ischemic heart disease (IHD), acute thrombosis (AT), and left ventricular hypertrophy (LVH) in the EULALIA trial (see inner note).

SD victims from the Mediterranean region presented left ventricular hypertrophy more frequently than other studies (48% vs. 20%) (Virmani et al. 2001; Subirana et al. 2011). From a clinical point of view, the victims of SD in the EULALIA trial presented

The majority of SD cases occur in subjects with ischemic heart disease and/or heart failure. It must be emphasized that heart failure is most frequently related to hypertension, chronic ischemic heart disease, cardiomyopathies, and valvular heart disease.

Inherited heart diseases are the main cause of SD in the first decades of life.
anginal episodes less frequently (20% vs. 37%), which was in agreement with the reduced number of cases with IHD found at autopsy, when compared with the Maastricht study (De Vreede-Swagemakers et al. 1997). In our series, the incidence of associated In.H.D. was 3% (hypertrophic cardiomyopathy and arrhythmogenic right ventricular cardiomyopathy). In approximately 7% of cases autopsy did not reveal any changes. Some of these cases might be explained by channelopathies (see Table 1.2).

### Table 1.2 Sudden death victims: pathological abnormalities found in necropsy

<table>
<thead>
<tr>
<th>Sudden death victims (n = 204)</th>
<th>N</th>
<th>%</th>
</tr>
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<tbody>
<tr>
<td><strong>Cardiovascular diseases</strong> (n = 183)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Heart diseases</em> (n = 161)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ischemic heart disease</td>
<td>119</td>
<td>58.4</td>
</tr>
<tr>
<td>Hypertensive heart disease</td>
<td>20</td>
<td>9.9</td>
</tr>
<tr>
<td>Valvular diseases</td>
<td>5</td>
<td>2.4</td>
</tr>
<tr>
<td>Idiopathic left ventricular hypertrophy</td>
<td>4</td>
<td>1.9</td>
</tr>
<tr>
<td>Dilated cardiomyopathy</td>
<td>4</td>
<td>1.9</td>
</tr>
<tr>
<td>Hypertrophic cardiomyopathy</td>
<td>3</td>
<td>1.5</td>
</tr>
<tr>
<td>Arrhythmogenic RV dysplasia/cardiomypathy</td>
<td>3</td>
<td>1.5</td>
</tr>
<tr>
<td>Myocarditis</td>
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<td>0.5</td>
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<tr>
<td>Congenital heart disease</td>
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<td>0.5</td>
</tr>
<tr>
<td>Amyloidosis</td>
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<tr>
<td><strong>Vascular diseases</strong> (n = 22)</td>
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<td></td>
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<tr>
<td>Pulmonary embolism</td>
<td>8</td>
<td>3.9</td>
</tr>
<tr>
<td>Dissection of the aorta</td>
<td>9</td>
<td>4.4</td>
</tr>
<tr>
<td>Cerebral hemorrhage</td>
<td>5</td>
<td>2.4</td>
</tr>
<tr>
<td><strong>Non-vascular diseases</strong> (n = 7)</td>
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<td></td>
</tr>
<tr>
<td>Gastrointestinal disorders</td>
<td>3</td>
<td>1.5</td>
</tr>
<tr>
<td>Pulmonary disorders</td>
<td>4</td>
<td>1.9</td>
</tr>
<tr>
<td><strong>Without findings</strong></td>
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<td>6.9</td>
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Taken from Subirana et al. 2010.

Chain of events leading to final arrhythmias and SD

- SD is the final stage of a chain of events that ends in cardiac arrest, usually due to ventricular fibrillation (VF) or, less frequently, extreme bradyarrhythmia (Bayés-Genis et al. 1995). In all cases there are a number of modulating and/or triggering factors that act on the vulnerable myocardium precipitating SD. Figure 1.3 shows this chain of events in different heart diseases. Ventricular fibrillation (VF) can appear without previous VT, unleashed by a PVC in the presence of other modulating or triggering factors (including genetic and environmental), and/or the sympathetic overdrive secondary to physical or mental stress. Usually under normal circumstances, probably all of these factors would not be of any consequence, but in the presence of acute ischemia they may trigger SD (Figure 1.5). The VF may be secondary to classic monomorphic sustained VT (Figure 1.4) or Torsades de Pointes VT (Figure 1.6). Sudden death is seldom a consequence of bradyarrhythmia (Figure 1.7).

- Therefore, the final arrhythmias that precipitate SD are not always the same (Figures 1.4–1.8). In a study that we performed revising the final causes of SD in 157 ambulatory patients who died suddenly while wearing a Holter recorder (Bayés de Luna et al. 1989), we found that in two-thirds of patients SD was caused by sustained VT that precipitated VF (Figure 1.8, Table 1.3). This was generally accompanied by fast baseline heart rate (sinus tachycardia or rapid atrial fibrillation), which may be considered a sign of sympathetic overdrive (Figure 1.4). VF without previous VT, usually associated with acute IHD, is more frequently seen as a consequence of PVCs with an R/T phenomenon. In our experience with ambulatory patients this pattern was observed in less than 10% of cases (Figure 1.5). Curiously, in 13% of cases, SD was due to Torsades de Pointes VT precipitating VF, generally in patients without severe heart disease but taking antiarrhythmic Class I type drugs because of non-frequent ventricular arrhythmias, sometimes isolated PVCs (pro-arrhythmic effect). We believe that if this study were performed now, the number of cases would be much smaller due to the evidence shown by the CAST study (Echt et al. 1991) demonstrating that class I antiarrhythmic agents are dangerous, especially in patients with heart disease. Thus, currently the prescription of class I antiarrhythmic drugs in post-MI patients is much lower. Finally, cases of SD due to extreme bradyarrhythmia (=15% in our study) (Figure 1.8B) were related more to progressive depression of the sinus node and AV node automatism (Figure 1.7) than to AV block.

Figure 1.8 shows the final arrhythmias that cause SD in patients with different clinical settings: (A) in a mobile coronary care unit on route to hospital due to an acute coronary syndrome (Adgey et al. 1982), (B) in ambulatory patients (Holter recording) (Bayés de Luna et al. 1989), and (C) in patients hospitalized because of severe HF (Luu et al. 1989). In the first situation (A), there are more cases of without previous VT than in our ambulatory cohort (B), most probably...
because patients in group A were in the acute phase of an MI. On the other hand, patients with severe HF (group C) presented extreme bradycardiacs more frequently as a cause of SCD. This could be the reason why antiarrhythmic drugs are not efficient in preventing SD in patients with severe HF. In our series (Figure 1.8B), 80% of patients had a depressed ejection fraction (EF), although their functional class was acceptable. These patients were “too healthy to die”, and many of these cases of SD could have been prevented with adequate therapy that sometimes consists of not prescribing an antiarrhythmic agent. We have to remember the Hippocratic Oath “Primum non nocere. The first is to do no harm”.

Our results were similar to those demonstrated in patients treated with implantable cardioverter defibrillators (ICD) with or without cardiac resynchronization therapy (CRT) (ICD-CR). In these cases, fast VTs also frequently appeared and were treated by antitachycardia pacing (Leitch et al. 1991, Grimm et al. 2006). In contrast, in a small series of post-MI patients with an EF<40%, with
an insertable loop recorded, who died suddenly it was demonstrated that the majority of SD were primary VF not triggered by VT. However, information about clinical events surrounding the time of death was not known. This lack of information and the small number of cases make it difficult to compare this series with our results. In the majority of patients who died due to

Figure 1.4 Ambulatory sudden death due to a ventricular fibrillation (VF) in an ischemic heart disease patient treated with amiodarone for frequent premature ventricular complexes. At 9:02 a.m. he presented a monomorphic sustained ventricular tachycardia (VT), followed by a VF at 9:04 a.m. after an increase in VT rate and width of QRS complex.

Figure 1.5 Ambulatory sudden death due to a primary ventricular fibrillation (VF) triggered by a premature ventricular complex (PVC) with a short coupling interval, after a post PVC pause (1120 ms) longer than the previous one (860 ms). Note that the sequence of events started with an atrial premature complex, which caused the first shorter pause.

Figure 1.6 Start of a Torsades de Pointes ventricular tachycardia (VT) in a woman without ischemic heart disease treated with quinidine for runs of non-sustained VT. The Torsades de Pointes VT triggered a ventricular fibrillation (VF).
different types of bradyarrhythmia, death occurred more than 1 h after the onset of symptoms (Gang et al. 2010).

**How to identify patients at risk**
- We know much more about identifying subjects at risk for SD within the group of high-risk patients (history of cardiac arrest, inherited cardiomyopathies, some postinfarction patients, heart failure, etc.) than in the general population in which SD often represents the first manifestation of the disease (Moss et al. 1979; Théroux et al. 1979; Myerburg et al. 1992, 1997). Figure 1.9 shows that these cases (A and B) represent more than 50% of all SD events. Many of these cases represent patients with first acute MI.

As it is impossible to carefully screen the entire general population, it is very difficult to identify subjects with no previous cardiovascular symptoms and no apparent risk factors who are at risk for SD. Currently, all we can do is to perform the following tasks: 1) a detailed study of relatives of SD patients;
**Clinical Aspects of Arrhythmias**

**Figure 1.8** Sudden death: final arrhythmias. A: in patients with acute ischemic heart disease (Adgey et al. 1982). B: in ambulatory patients wearing a Holter monitor, in whom a depressed ejection fraction was present in 80% of cases (Bayés de Luna et al. 1989). C: in patients with advanced heart failure (Luu et al. 1989).

**Figure 1.9** Left: the percentage of patients with SD is much higher in the high-risk groups (D–F) than in the general population (A, B). The total amount of cases occurring in the general population is greater than the number of all other subgroups of patients at risk (Myerburg et al. 1997).
2) when seeing a patient for whatever reason, ask whether there are any family members who have had IHD, In.H.D., or present evident risk factors; and 3) perform a complete physical examination and blood test (testing for risk factors, especially cholesterol and blood glucose) and take a blood pressure and an ECG recording when an adult is visited for the first time.

Trying to identify the subjects at risk for SD is one of the biggest challenges of modern cardiology. In the Framingham study performed on a general population (Kannel and Schatzkin 1985), two things were demonstrated: 1) the presence of alterations in the ECG, especially bundle branch block, and left ventricular hypertrophy, significantly increases the risk of SD, mainly in men; and 2) in a multivariate analysis, the risk of SD increased especially in men, in relation to the amount of risk factors they had (Figure 1.10). Recently, it has been demonstrated that an increase of N-terminal prohormone brain natriuretic peptide (NT-proBNP) levels suggest, more than other previously described biochemical risk factors (dyslipidemia), an increased risk of sudden death in women (Korngold et al. 2009).

Risk stratification by an estimated calcium “score” by multislice computed tomography (CT) scan may help to identify patients with asymptomatic IHD. Nevertheless, this method is not currently recommended as a routine test (Greenland et al. 2000), although it might be useful in patients with multiple risk factors (ATP III Guidelines 2001). The latest ACC and Associated Societies statement does not support the application of this technique in the general population and raises doubts as to whether this test should even be performed in medium- and high-risk patients (Hendel et al. 2006). Also, its indiscriminate use is not recommended by other authors (Bonow 2009). In our opinion, in middle-aged patients with significant family history and/or multiple risk factors, it may be useful to perform the calcium “score”, and, if positive, use a non-invasive coronaryography by means of the latest generation multislice CT scan, which currently emits less radiation. We hope that in the near future magnetic resonance imaging (MRI) will offer more information on heart anatomy and function in a single test, and be safer.

The difficulties in identifying subjects at risk for cardiac arrest are increased by the fact that the general population is more willing to perform medical examination to detect certain malignancies (colon, breast in women, and prostate in men). Adequate examinations such as a multislice CT (in patients with a positive family history, and/or positive exercise test without symptoms or other risk factors), or genetic tests in special situations (young patients with a family history of SD, suspicion of channelopathies, etc.), to determine apparently silent patients at risk for SD, have to be performed more frequently.

- The third part of this book will deal with different aspects related to SD in different heart diseases and situations. We will explain the mechanisms that trigger fatal arrhythmias and the characteristics of an anatomic or electrophysiologic substrate, if known, that make the myocardium vulnerable to VF/SD.
How to prevent SD

- Obviously, the best way to prevent SD is to identify subjects at risk (see above). In the group with the highest risk (Figure 1.9D–F) it may be necessary, even compulsory, to implant an ICD. (see Chapters 5, 8, and 9, and Appendix A-4). This alone does not prevent SD, but may help to avoid it when the final arrhythmia appears.

- Thus, it is known how to prevent sudden death in patients at high risk (Myerburg et al. 1992). It is much more difficult, however, to prevent SD events in the general population. The true prevention of SD is fighting the associated diseases, such as IHD, HF, and In.H.D. Prevention of IHD should start in childhood, with an adequate health education promoting a healthy lifestyle and an adequate diet to avoid overweight and obesity, preventing the development of risk factors. Of course it is crucial to fight and treat high cholesterol, hypertension, diabetes, and other risk factors when they are present because they are, at least in part, responsible for the presence of atherosclerotic plaques. Also it is important, if possible, to avoid and treat HF adequately from the moment it starts, and it is necessary to diagnose and manage In.H.D. This includes detailed personal and family history (antecedents of syncope/SD), as well as the knowledge that a simple ECG pattern can identify a patient at risk for SD (see Chapter 9). When we look to the future, what we need to reduce the burden of SD is an arteriosclerotic plaque stabilizer, preferably a chemical additive to food or water, that may be given to the global population, stabilizing the fibrous cap of the plaque and reducing the probability of plaque erosion/rupture and SD (Moss and Goldenberg 2010).

The management of a patient resuscitated (survivors) from a cardiac arrest

- Patients resuscitated from an out-of-hospital cardiac arrest should be referred to a reference center for a detailed evaluation, in order to identify the cause of the cardiac arrest, using an exhaustive examination that includes, if necessary, invasive and non-invasive tests. This is the routine approach for patients who suffered a cardiac arrest with and without evident heart disease. A recent report (Krahn et al. 2009) recommends genetic testing only when a genetic disease appears to be responsible for cardiac arrest in the clinical test results. In our opinion, genetic studies need to be more accessible and cheaper. This would encourage their use and would be of great benefit regardless of their current limitations, in addition to studying the relatives of patients with In.H.D. (see Chapter 9).

- Ventricular fibrillation is the cause of cardiac arrest in many cases (see Figure 1.8). Therefore, it is necessary to prevent the first episode, and in any case to organize an appropriate prevention of future episodes. If VF appears to be associated with ischemia, the possibility of revascularization has to be considered (see Chapter 11, Chronic ischemic heart disease). Other possible mechanisms involved in the triggering of VF have to be ruled out, i.e. in rapid AF in patients with Wolff–Parkinson–White syndrome (WPW) the ablation of accessory pathway is compulsory.

In many other cases of cardiac arrest due to VT/ VF, if no trigger is found, it is usually necessary to implant an ICD with a resynchronization pacemaker (CRT), if needed (see Chapters 9–11).

- Obviously, in cardiac arrest due to passive arrhythmias an urgent pacemaker implantation is required (see Chapter 6).

- For more details about ICD indications/implantation see Chapters 5 (Ventricular tachycardias, and Ventricular fibrillation), 9, and 11 (Ischemic heart disease and Heart failure), as well as Appendix A-4, Automatic implantable cardioverter defibrillator (ICD)), and guidelines of scientific societies (p. xii).

Arrhythmias and severe clinical complications

- Regardless of the real risk of SD, arrhythmias may induce severe consequences on the patient’s clinical and hemodynamic status, which may appear as:
  - A crisis of left ventricular failure, and also a congestive HF (tachycardiomyopathy).
  - A crisis of angina (hemodynamic angina).
  - A low cardiac output with dyspnea and weakness.
  - Hypotension that may be significant, even resulting in cardiogenic shock.
  - Embolism, more often systemic, frequently cerebral, sometimes with severe consequences.
  - Dizziness, pre-syncope, syncope. Syncope may be benign or may be a marker of life-threatening arrhythmia (see later).

- More severe symptoms due to tachyarrhythmias are left ventricular failure, hemodynamic angina,
those derived from embolism, dizziness, especially when standing up, and even syncope in some very fast supraventricular tachyarrhythmias, such as atrial flutter 1:1, or VT (fast VT), which may lead to SD.
- The most frequent symptoms related to bradyarrhythmia are a low cardiac output, dizziness, syncope, and even SD.
- These symptoms are especially evident in relation to:
  - The presence or absence of heart disease. An association with either acute ischemia or HF is of special importance.
  - Duration of arrhythmia; the longer it is, the greater the risk of not being well tolerated.
  - Heart rate (fast tachycardia or severe bradycardia) during the arrhythmia.
  - The presence or absence of AV dissociation.
- Consequently, an episode of short duration, with a not very fast heart rate in subjects without heart disease, does not affect the cardiac output, and it will not result in significant hemodynamic impairment. On the other hand, very rapid episodes, especially in patients with heart disease and poor ventricular function, may cause evident hemodynamic impairment resulting in dyspnea, hypotension (Figure 1.11), angina, syncope, and even shock and congestive HF if the arrhythmia is sustained.
- Now we are going to look at some aspects of syncope, the most alarming symptom related with arrhythmias, which may be of practical interest to the reader.

**Syncope: serious or innocent symptom**

**Concept.**
Syncope is the sudden and transient loss of consciousness caused by an important reduction in cerebral perfusion. It is accompanied by loss of muscular tone and a total spontaneous recovery in a short period of time. At times there is only a feeling of dizziness or unsteadiness (pre-syncope) (Garcia Civera et al. 1989).

This is the most worrying symptom of patients with arrhythmias. It can be either the expression of an innocent process or a marker of evident risk of SD. From clinical and prognostic points of view, there are three types of syncope: a) neuromediated via vagal reflex and those due to orthostatic hypotension that are usually benign, b) related to heart diseases, such as severe tachy- or bradyarrhythmia,
Mechanisms

- **A. Neuromediated reflex syncope.** A high percentage of syncopal episodes (>50%) are neuromediated via a vasovagal reflex, often with triggering factors related to increased vagal tone such as cough, micturition, venous puncture, orthostatism, etc. Recent studies have shown that some polymorphism of protein G is associated with family history and may explain the susceptibility to vasovagal syncpe (Márquez et al. 2007; Lelonek et al. 2009).

- **Hypotensive orthostatic syncope** is frequent (10–20%). It is caused by the dysautonomy reflex during orthostatism, which produces a loss of vasconstrictive reflexes in the vessels of the lower extremities, reducing baroreceptor sensitivity and producing reactive hypotension to such a degree that it decreases cerebral flow and induces syncope. **Hypersensitivity of the carotid sinus** (a pause >3 s, or a decrease in blood pressure >30 mm, that occurs after carotid sinus massage for 10 s with or without syncope) may also induce a neuromediated syncope via a vagal stimulus from a sick carotid sinus, and may be the cause of an unexplained fall, especially in the elderly. This syncope is often related to some accidental pressure on the carotid sinus (while shaving, for instance). It may also predict its occurrence during a tilt test.

- **B. Syncopes of cardiac origin** include all syncopes related to arrhythmias (5–10%) and encompass bradyarrhythmias (sick sinus syndrome and advanced AV block), and tachyarrhythmias (very fast supraventricular arrhythmias, i.e. atrial fibrillation with WPW, sustained VT and polymorphic VT of all types). These arrhythmias, which may trigger syncope and VF/SD, are seen especially in IHD, HF and In.H.D. (cardiomyopathies and channelopathies), as well as in other heart diseases and clinical situations (see Chapters 8–11). Also, syncopes of cardiac origin may be related to obstruction of flow (2–3%), as in aortic stenosis, hypertrophic cardiomyopathy (CM), mixoma, etc.

- **C. Finally, syncopes related to neurological disorders** are infrequent, but include the neurological disorders that may induce a brusque reduction in cerebral perfusion, which may happen in some transient ischemic attacks with loss of consciousness (subclavian steal syndrome or bilateral severe carotid artery stenosis). Usually they are accompanied by transient neurological problems (difficulties in speech, movement, etc.).

**Diagnosis and management of patients with syncope**

- First, we have to be certain that the patient has experienced a syncopal episode. This means that all aspects of the definition of syncope must be present: abrupt and transient loss of consciousness, caused by a brusque reduction in cerebral flow, accompanied by a loss of muscular tone, followed by a total spontaneous recovery.

- Next, we must always rule out factors that may provoke prolonged unconsciousness, such as hysterical attacks, hypoglycemia, intoxication including alcohol (heavy drinkers), or dizziness and vertigo (more than true syncope) as happens with otolaryngological disorders among others. We must also exclude epileptic episodes that sometimes produce problems of differential diagnosis, although history taking is very useful in general. During epileptic attacks there is no reduction in cerebral perfusion; however, some epileptic patients may present arrhythmias with syncopal episodes and even SD following an epileptic convulsion (Rugg-Gunn et al. 2004; Tomson et al. 2008).

- **Syncopes may be accompanied by typical seizures** (pallor, no pulse, rapid recovery with facial flush – Stokes–Adams crisis) and may occur at rest, or typically during exercise (see Chapter 9). We want to emphasize that it is extremely important to differentiate neuromediated vasovagal syncopes including orthostatic syncope, which are generally benign, from syncope due to cardiac origin that often present an unfavorable prognosis. The latter includes (see before) syncope due to very slow or very rapid arrhythmias, or an obstruction to flow (aortic stenosis, hypertrophic cardiomyopathy, myxoma, etc.).

- To recognize the origin of syncope we will proceed to the basal study that includes history taking, physical examination, and an ECG (see Figure 1.12). Depending on the results we may have to perform other complementary tests.
It is important to obtain a comprehensive history (Colman et al. 2009), including: 1) family antecedents of syncope or SD, very important in attempting to rule out In.H.D., 2) antecedents of previous heart disease (MI, history of valve heart disease, etc.), 3) the number of prior syncopes, and in adults whether they also occurred during childhood, and 4) the evaluation of: a) the prodromal symptoms or circumstance of appearance (exercise, movements, digestion, emotions, etc.); b) the onset (abrupt or slightly gradual); c) the position in which they occur, such as standing (orthostatic hypotension) or sitting; d) what the patient was doing before (cough, defecation, micturition, carotid sinus massage, neck movements, venous puncture, etc.); e) the recovery of consciousness (rapid or gradual); f) associated events (tongue biting); and g) appearance (pallor), etc.

With all these arguments we will be able, in many cases, to have already an impression of the etiology of syncope. Obviously, it is very important to proceed to physical examination (auscultation of the heart and arteries of the neck, and palpation of the heart and vessels).

Figure 1.12 Algorithm for the management of patients with syncope.

* It will be convenient to know about: 1) any assessment of the patients’ relatives who have died from SD; 2) the presence of anatomic or electrocardiographic structural lesions; 3) if the syncopal episode occurred unexpectedly at rest or during exercise; 4) if there is high suspicion of the neurally mediated syncope or it is still not well defined, and 5) if the syncope was related to neurologic disturbances. Other complementary tests will be necessary if the history taking, physical examination, and the ECG consider that the basal study is abnormal (see abnormal).

** If necessary with: echocardiography (murmur, abnormal ECG, etc), Holter (palpitations), exercise test (exercise arrhythmias), and sometimes electrophysiologic studies, magnetic resonance imaging, coronarography and/or genetic studies.

*** Especially necessary in cases of doubtful or repeated syncopes.