Sudden cardiac death

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Abstract

Sudden death is a frequent event whose causes may not be anticipated, but often has a cardiac origin. Sudden cardiac death is the final consequence of many pathophysiological mechanisms which have caused acute or chronic arrhythmogenic disease. Single or multifactorial triggering factors may interact with the arrhythmogenic substrate to lead to lethal arrhythmias. Stratifying populations according to risk is feasible, but the immediate priority is prevention of triggering and disease-promoting factors.

ICD therapy is the best treatment for most survivors of sudden cardiac death. Drug therapy, catheter ablation, anti-tachycardia surgery or heart transplant are only first-choice treatments for very few patients. Choosing the best therapy is an individual decision based on the patient’s clinical picture, the type of arrhythmia seen and hospital experience in the various techniques.

Key words: Sudden death, ventricular tachycardia, ventricular fibrillation, bradycardia, heart disease

Epidemiology

According to a study by the WHO, the annual incidence of sudden death in industrialized countries ranges from 19 to 159 cases per 100,000 males and from 35 to 64 per 100,000
females. Thus, it represents between 10 and 32% of all natural deaths, depending on the time that elapses from the onset of symptoms to death, making it the most common form of fatal cardiac disease. About 50% of patients with ischemic heart disease die suddenly [1-8].

Sudden death shows a clear circadian rhythm, occurring most frequently between 7 am and 11 am. This concurs with the higher incidence between these times of various manifestations of ischemic heart disease (infarction, coronary spasm, QTc variability, etc). As regards age, there are two peaks in the incidence of sudden death. The first peak is from birth to 6 months of life (infantile sudden death syndrome); the second peak occurs from 45 to 75 years and is characterized by an increasingly greater frequency of ischemic heart disease after the age of 40 [6]. A multivariant analysis carried out on the Framingham study, including all coronary risk factors, showed that age, systolic pressure, cigarette consumption, and relative body weight are all risk factors that are independently related to the incidence of sudden death. In females, besides age, only cholesterolemia and vital capacity were associated independently with increased risk of sudden death.

Pathophysiology

The pathophysiology of sudden death needs to must be viewed as a multifactorial problem that is inseparable from associated diseases and that includes the precursors of sudden death, the final step responsible for sudden death as well as markers and triggering mechanisms of this event [9-15].

Associated diseases
In about 90% of cases, sudden death occurs in persons with heart disease. Most heart diseases may be associated with sudden death (Table I), as we outline below:

**Ischemic Heart Disease**
Although sudden death occurs in all forms of heart disease, in the west, ischemic heart disease is the most commonly occurring associated disease [1-5]. Several mechanisms can produce potentially fatal arrhythmias in patients with ischemic heart disease and it is often difficult to define the precise mechanism of a given episode [9,10,16-19]. At one extreme is the patient without a prior infarction who has an acute occlusion of a major epicardial coronary artery and develops ventricular fibrillation during the immediate phase of acute infarction [7,20-22]. This patient illustrates the role of acute severe transmural and persistent ischemia. At the other end of the spectrum lies the patient with a history of one or more previous myocardial infarctions, in whom postinfarction scarring has provided the anatomic substrate for a rapid reentrant ventricular tachycardia, triggering ventricular fibrillation, with a resultant hemodynamic collapse and sudden death; new ischemia does not necessarily have to be present [10,17,23-26]. The majority of sudden death victims with ischemic heart disease belong to these two groups. In these patients sudden death is usually produced by a complex interaction between several trigger-types — autonomic nervous system dysfunction, electrolyte imbalance, or drug toxicity among others — and the presence of different interactions between electrical instability, residual ischemia and left ventricular dysfunction (Figure 1) [10,27].

Autopsy and clinical studies differ in their findings of coronary thrombi and the evidence of new ischemia in sudden death patients. Evidence of coronary artery thrombi is reported in between 20% and 50% of sudden death victims. However, only about 25% of patients resuscitated from an out-of-hospital cardiac arrest will develop new Q-wave myocardial infarctions. While there may be persistent repolarization abnormalities and enzymatic evidence of necrosis in another 25% of patients, these changes are not specific for a
new infarction and may be caused by prolonged hipoten-
sion during a primary arrhythmia. There is also evidence
suggesting a contributing role for acute ischemia without
any signs or symptoms of infarction [9,10,21,22,27].

Coronary spasm and other diseases of the coronary arter-
ies not due to atherosclerosis, such as an abnormal left
coronary origin, may give rise to either myocardial infarction
with late ventricular tachycardia or to arrhythmias mediated
by acute intermittent ischemia and sudden death [6,7,27,28].

Cardiomyopathies
Idiopathic dilated cardiomyopathy is the primary cardiac di-
agnosis in 5-10% of resuscitated cardiac arrest victims.
Sudden death accounts for about a half of all deaths in pa-
tients with this diagnosis. Sudden death often occurs rela-
tively late in the course of dilated cardiomyopathy, after he-
modynamic symptoms have been present for some time.
Even with well-documented clinical episodes of ventricular
tachycardia or fibrillation, similar arrhythmias are frequently
not inducible at the time of electrophysiologic study [29,30].
In patients with dilated cardiomyopathy and very advanced
heart failure, bradiarrhythmia, rather than tachyarrhythmia,
is the final event that leads to sudden death.

In hypertrophic cardiomyopathy, sudden death often oc-
curs in young adults who usually have had no prior cardiac
symptoms. Polymorphic ventricular tachycardia or primary
ventricular fibrillation appear to be the initial arrhythmia at
the time of cardiac arrest. Patients with hypertrophic car-
diomyopathy are also at risk from AV block and the severe
hemodynamic compromise that may produce a cardiovas-
cular collapse can occur during any rapid supraventricular
tachycardia in these patients. In young athletes under 35,
sudden death occurs fundamentally in patients with car-
diomyopathy, usually, but not exclusively, of the hyper-
trrophic variety. In contrast, sudden death in athletes over 35
is due to ischemic heart disease in 80% of victims [31,32].

Patients with arrhythmogenic right ventricular dysplasia
may also account for a small number of cases of cardiac ar-
rest and sudden death usually in young adults. The ventricu-
lar tachycardia that these patients typically present exhibits
a left bundle branch block on the ECG, and a QRS pattern
with negative T waves in the right precordial leads is usually
seen aurius sinus rhythm [33,34].

Valvular Heart Diseases
In mitral valve prolapse symptomatic atrial and ventricular
arrhythmias are common but truly life-threatening arrhyth-
mas are extraordinarily rare, except in the presence of cer-
tain complicating conditions such as long QT, electrolyte im-
balance, or drug toxicity.

In young adults with congenital aortic stenoses sudden
death is usually related to exertion probably due to sudden
changes in either ventricular filling or aortic obstruction with
a secondary arrhythmia. In the acquired forms of valvular
heart disease, sudden death is usually a late occurrence
seen in patients with advanced heart failure. Martinez-Rubio
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ease who presents with sustained ventricular tachyarrhyth-
mas or syncope has to be considered as a very high risk pa-
tient because recurrence of sustained ventricular tach-
yarrhythmias or sudden death rate are high despite therapy
[35]. In addition, the data provided by these authors suggest
that such patients should be treated with implantable defib-
rilators while the mechanism of arrhythmias in them is par-
ticularly complex (multifactorial) [35].

Electrophysiological abnormalities
Supraventricular arrhythmias, if associated with very rapid
ventricular rates, may cause hemodynamic collapse and de-
generate to ventricular fibrillation. Atrial fibrillation with rapid
conduction over an accessory pathway in a patient with
Wolff-Parkinson-White syndrome is the supraventricular ar-
rhythmia most frequently associated with sudden death [36].

Prolongued QT syndrome, leading to irregular regulopar-
tiates, sudden death is exceptional. Idiopathic ventricular fib-
rillation represents about 1% of all cases of resuscitated out-
of-hospital cardiac arrest. Many retrospective studies with a
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Precursors of sudden death
Cardiac sudden death occurs as a result of cardiac arrest
due usually to ventricular fibrillation (VF) or more rarely to
malignant bradiarrhythmias [38]. In both cases, a series of
precursors may be identified before sudden death appears
[9,18,24,40-43]. These include triggering mechanisms act-
ing on a vulnerable myocardium to precipitate the appear-
ance of the final step that terminates in either VF or, less fre-
quently, cardiac arrest due to a malignant bradiarrhythmia
though usually secondary to electromechanical dissociation
in patients with severe heart failure.

Final step
Usually the final step leads to ventricular fibrillation. The se-
quence of events occurring in the final step that precipitate
ventricular fibrillation can be called the ‘ventricular fibrillation
cascade’ (Figure 1). These include a final arrhythmia responsible for sudden death and the electrophysiological events that often precede sudden death. Below we describe the arrhythmias and events that constitute the final step in a range of clinical settings:

**Pre-hospital phase of acute myocardial infarction**

In patients whose sudden death is related to an acute myocardial infarction, records taken in mobile coronary units showed that the most frequent final arrhythmia was primary VF unpreceded by ventricular tachycardia (82%) (Figure 2A). In these patients, an R-on-T phenomenon was observed in 70% of cases. Characteristically, an increased heart rate, due to sympathetic overdrive, was seen before the lethal arrhythmia in patients with sudden death related to an acute myocardial infarction.

**Ambulatory patients with out-of-hospital sudden death**

We have published the results of a worldwide survey including 233 cases of patients who have died while wearing a Holter recorder [6,38]. The conclusions of this study are (Figure 2B):

a) Sudden death occurs due to a ventricular tachyarrhythmia in 80% of cases and due a severe bradiarrhythmia in the remaining 20%.

b) VF initiates abruptly in 10% of the individuals, in the rest it is triggered by classical sustained ventricular tachycardia (VT) or less often a ‘torsade de pointe’ ventricular tachycardia. The sustained VT leading to VF was often preceded by sinus tachycardia or a new supraventricular tachyarrhythmia.

On the other hand, in the group of patients with VF, only 12% of patients presented ischemic ST changes prior to the final event. However, the incidence of ischemic ST alterations requires further study using Holter instruments equipped with three leads or more, and should include observations on disturbances in the autonomic nervous system such as heart rate variability.

c) In patients who died suddenly from bradiarrhythmia, the cause was more often sinus depression than atrioventricular block, and the incidence of previous ST changes was surprisingly high (>80%).

**Patients with congestive heart failure**

In patients with end-stage heart failure, Luu et al. [8] have demonstrated that the incidence of ventricular tachyarrhythmias as the final arrhythmia is much lower (40%) than the incidence obtained in ambulatory patients in our series (approximately 80%). Interestingly, all patients who died of VT/VF had a previous myocardial infarction, but previous myocardial infarction was present in fewer than half of the cases of the bradiarrhythmia/electromechanical dissociation group (Figure 2C). This finding, and hyponatremia in the latter group, were the only two parameters that differentiated between the bradiarrhythmia and tachyarrhythmia groups.

**Electrophysiological disorders**

In patients with Wolff-Parkinson-White syndrome who die suddenly it has been shown that the final trigger of VF leading to sudden death is a supraventricular tachyarrhythmia, usually atrial fibrillation with a very rapid ventricular rate [36].
In patients with long QT syndrome, there is evidence that adrenergic hyperactivity produced by physical and/or mental stress may be responsible for triggering malignant ventricular tachycardia of the *torsades de pointes* type [37]. In addition, several drugs might influence the occurrence of this electrophysiological disorder.

**Patients with terminal noncardiac disease**

Terminal cardiac activity has been described in adults who died with no apparent indication of heart disease, generally as a consequence of terminal malignancy present as bradycardia in 87% of patients and ventricular tachyarrhythmia in 17%. Agonal ST segment elevation was often observed.

Markers and Triggering Mechanisms of Sudden Death

The majority of patients who die suddenly present a vulnerable myocardium. Below we will discuss the different markers of this vulnerable myocardium and also the triggering factors, which acting on this vulnerable myocardium, may precipitate sudden death.

Postinfarction patients are the largest group, so we shall focus initially on them. The risk of sudden death in these patients is related especially to the presence of electrical instability and to its interaction with left ventricular dysfunction and residual ischemia. These three factors form the imaginary triangle of risk of postinfarction complications (Figure 3). In figure 4 we can see various parameters of electrical instability, ischemia and left ventricular dysfunction as markers that should alert us to the danger of a serious complication of myocardial infarction: malignant arrhythmias, new coronary events, and overt heart failure directly or indirectly increase the possibilities of triggering sudden death.

Various morphofunctional parameters (postinfarction scar, left ventricular hypertrophy, reduced ejection fraction), autonomic nervous system parameters (heart rate variability, QT interval, baroreflex sensitivity), and clinical and electrocardiographic findings (previous history, ST depression on surface ECG, number and nature of the ventricular arrhythmias, presence of ventricular late potentials) may be considered as markers of electrical instability and, therefore, of myocardial vulnerability to sudden death [9,18,24,26,42,44-46]. The markers of ischemia and left ventricular dysfunction are also shown in figure 4.

The relationship between ischemia and sudden death depends to a great extent on the duration and severity of ischemia. It is evident that persistent and severe transmural ischemia may induce sudden death in acute infarction. However, in transitory transmural ischemia, such as Prinzmetal’s angina and during PTCA, the relationship between ischemia and malignant ventricular arrhythmias is less certain. During moderate subendocardial ischemia, malignant ventricular arrhythmias are rare.

The five most important triggering factors which act on a vulnerable myocardium are: 1) physical or mental stress; 2) ionic or metabolic disorders; 3) acceleration of sinus rhythm or appearance of a supraventricular arrhythmia or a pause; 4) the arrhythmogenic effect of certain drugs and 5) interaction of electrical instability with ischemia and/or left ventricular dysfunction due to multiple causes [12,21,22,25,27,28,43,47,48].

When sudden death occurs in patients with nonischemic heart disease it is also conditioned by a vulnerable myocardium and certain triggering factors. Heart failure, left ventricular hypertrophy and electrical instability (ventricular arrhythmias, autonomic nervous system dysfunction) are usually the most important markers of vulnerable myocardium, while other factors such as physical or mental stress, ionic or metabolic disturbances, drug administration, etc, may also act as triggers.

**Identification of high risk candidates**

Patients at greatest risk from sudden death (table 2) are those who have previously experienced a malignant ventricular arrhythmia (sustained VT or out-of-hospital cardiac ar-
rest). In patients without previous malignant ventricular arrhythmias, the risk of sudden death is related to the presence of different markers, described earlier and related with the evidence of advanced heart disease with left ventricular dysfunction or even heart failure, and/or evidence of electrical instability and/or residual ischemia when ischemic heart disease is present.

Clinical data are very useful both for risk stratification of patients who have presented severe arrhythmias and for the general population of postinfarction patients. Nonetheless, complementary studies using different technologies need to be undertaken to obtain more information about the triangle of risk (Table 3).

Table 3. Methods to identify a high-risk patient

| Clinical history and physical examination (syncope, angina, etc) |
| Surface ECG (Q-waves, ventricular enlargement, QT interval, arrhythmias, etc.) |
| Exercise testing (ST-segment abnormalities, poor hemodynamic response, arrhythmias, etc.) |
| Ambulatory Holter ECG (arrhythmias, ischemia, autonomic tone, ventricular late potentials, etc.) |
| Electrophysiologic studies (induction of sustained ventricular tachyarrhythmias, anomalous pathways, sinus node dysfunction, etc.) |
| Echocardiography-Doppler (ventricular function, aneurysm, ischemia, valvular dysfunction, etc.) |
| Nuclear studies (ventricular function, aneurysm, ischemia, etc.) |
| Cardiac angiography (coronary artery lesions, ventricular function, aneurysm, biopsies, valvular abnormalities, etc.) |

Pronostic stratification in the thrombolytic era

There are some indications that the prognosis of patients with or without fibrinolytic therapy following an acute myocardial infarction differs. In a study by Farrell et al. in non-thrombolyzed patients, the presence of ventricular premature beats (VPB) during the Holter recording, decreased heart rate variability (HRV), presence of late ventricular potentials, the results of exercise testing and the value of ejection fraction discriminated those patients at high risk of future arrhythmic events. However, in patients who received thrombolytic therapy, only the diminished HRV discriminated between the two groups. Nevertheless, the incidence of these risk markers (late ventricular potentials, low ejection fraction, positive exercise testing, and decreased HRV) did not differ in the two groups [49].

Other results concerning the incidence of certain markers after thrombolysis are contradictory. Pedretti et al demonstrated in post-myocardial infarction patients that thrombolysis significantly reduced the occurrence of arrhythmic events [46]. On the contrary, the GISSI-2 study showed that frequent premature ventricular beats, recorded by Holter monitoring, remained even in the fibrinolytic era as an independent risk factor of total and sudden death in the first 6 months following an acute myocardial infarction [26].

Prevention

The relatively low incidence of sudden death in Southern Europe is related to the lower incidence of ischemic heart disease, in comparison to Northern Europe and North America. Efforts to reduce risk factors and to treat the acute events as rapidly as possible are the best measures to reduce sudden death. The widespread use of recently developed therapies for acute events (thrombolysis, beat-blockers, aspirin, etc) and the global protection of the patient during follow-up are mandatory [15,50-59]. Undoubtedly, thrombolytic treatment has been crucial in decreasing mortality, though in the acute phase of myocardial infarction it is still relatively high. However, it should not be forgotten that a global cardioprotective approach has been very effective in reducing mortality particularly in the post-discharge phase [11,15,55]. Hämaläinen et al. [11] evaluated effectiveness before the thrombolytic era of a long-term multifactorial intervention program to reduce sudden and cardiac death by medication, cessation of smoking, physical exercise, diet, psychosocial support and optimal medical care. The patients included in the intervention group had an incidence of sudden death of 12.6% at 10 years as opposed to 23% in the group that received no standardized intervention.

We should point out that all patients already presenting a crisis of a malignant ventricular arrhythmia (sustained ventricular tachycardia or ventricular fibrillation) must be treated in a reference centre, because of the actual efficacy of non-pharmacological therapies (implantable cardioverter-defibrillator, ablation, etc.).

Another group of patients to mention are those who have not suffered a malignant arrhythmia, but who present high risk markers. In post-myocardial infarction patients with the following markers: low ejection fraction (≤35%) + spontaneous non-sustained ventricular tachycardia on Holter monitoring + inducible ventricular arrhythmia inducible during the electrophysiologic study and non-suppressible due to drug therapy, the MADIT Trial [45] has demonstrated that the implantable cardioverter-defibrillator (ICD) is superior to conventional drug therapy (including amiodarone) for protecting against sudden death. The ICD may also offer the solution in the group of patients with cardiomyopathies at risk of sudden death (Figure 5). In some other cases, such as patients with WPW syndrome and paroxysmal tachyarrhyth-
mias with electrophysiologic parameters of risk, radiofrequency catheter ablation may provide a definitive solution to the problem. In long QT syndrome a good protection may be achieved by blocking the adrenergic activation with beta-blockers or, even better, with left estelectomy.

A large number of heart disease patients—currently more than half of post-infarction patients have a low risk of sudden death (without significant markers of electrical instability, ventricular dysfunction and/or ischemia). In these cases, the usual postinfarction treatment consists of acetilsalycilic acid, beta-blockers and control of risk factors (tobacco, high cholesterol levels, high blood pressure, etc). Recently, several studies [56-67] have firmly established the value of secondary (e.g. 4S Trial) and primary (e.g. WSO Trial) coronary prevention in reducing mortality by using inhibitors of the HMG CoA reductase. The inhibitors of the angiotensin converting enzyme have proven especially useful in patients with myocardial infarction and left ventricular dysfunction but are also recommended in all post-infarction patients after the ISIS-4 and GISSI-3 Trials. Amiodarone does not give significant advantages against placebo (EMIAT Trial) or has only been effective in some subsets of patients (CAMIAT Trial). Other antiarrhythmic agents have shown a significant increase in mortality (Class I agents —CAST Trial—; d Sotalol —SWORD Trial—).

Thus, although prognosis has improved, sudden death remains a frequent clinical occurrences and on unresolved multifactorial problem which needs further research.

References


About the authors

Professor Antonio Bayés de Luna was born at Vic (Barcelona) in 1936. He is a Cardiology specialist at the School of Cardiology at the University of Barcelona and at the Institute of Cardiology and Hammersmith Hospital in London. He is married to M.Clara Genís Piqueras and has five children. In 1971 he became Associate Lecturer at the Autonomous University of Barcelona and Head of the Unit of Electrocardiography and Arrhythmias at the Sant Pau Hospital. At present he is Professor of Cardiology at the Autonomous University of Barcelona and was Director of the Department of Cardiology and Cardiac Surgery at the Sant Pau Hospital from 1992 to 1998. He used this post to encourage essential research at the Sant Pau Hospital. At present he is Director of the Catalan Institute of Cardiology at the University of Barcelona and Head of the Department of Cardiology and Cardiac Surgery at the Sant Pau Hospital from 1992 to 1998. He used this post to encourage essential research at the Sant Pau Hospital. At present he is Director of the Catalan Institute of Cardiology at the Sant Pau Hospital. Between 1973 and 1974 he was President of the Catalan Cardiology Association; 1983-1984, President of the Spanish Cardiology Association; 1983-1985, President of the Catalan Association of Aid to Cardiology (ACARD); and between 1983 and 1985, Vice-president of the Hispanic Foundation for Cardiology. He has been member of the Myocardioathy Council of the International Society and Federation of Cardiology (ISFC) since 1991, and has been active in various ISFC heart disease campaigns. In September 1994 he was elected President of this Society, and ended his term as Executive President in January 1999. He has been deeply involved in activities of the European Society of Cardiology (ESC) as member of: nucleus of the working-group on Arrhythmias (1986-1993); Scientific Executive Committee (1986-1992); and the Nominations Committee of the ESC Council (1985-1991). In 1992 he was President of the XIV European Congress of Cardiology. He has represented the ESC on the Executive Committee of the European Board of Specialists in Cardiology, and at present is member of its Library and History Committee. He is also a member of the nucleus of the International Council of Electrocardiology, and of the Founder Nucleus of the International Society for Holter Monitoring, of which he is also Vice-president. He has received various prizes and honours, most notably «Catalan Doctor of the Year» (1991) and Spain’s «Doctor of the Year» (1991 and 1994). He is Honorary Member or Correspondent of 19 National Cardiology associations and was made Doctor Honoris Causa by the University of Lisbon in 1997. In 1998 he was awarded the «Health Sciences» prize of the Uriach Foundation. In addition, he is on the editorial board of over 12 prestigious Spanish and international journals. He was also founder and editor of the journal Revista Latina de Cardiología. He has organised annual postgraduate courses and major meetings and symposia. He was President of the European Congress of Cardiology in 1992 and the Sixth International Congress on Ambulatory Monitoring in 1994, both held in Barcelona. He has been invited to lecture at major congresses and meetings in 39 countries round the world. His extensive scientific work has concentrated mainly on Electrocardiography, arrhythmias, sudden death, Holter technology, risk stratification and silent ischaemia. The fruit of this work is over 100 «peer review» original articles, many of them published in the most prestigious Cardiology journals (Circulation, American Journal of Cardiology, American Heart Journal, Chest, European Heart Journal, Archives de Maladies du Coeur, Revista Española de Cardiología, along with basic research studies already done in our country etc.). His impact on the Department of Cardiology of the Sant Pau Hospital, which he directs, led to around 200 articles in the last 4 years. He is also author of five books first published in English or Spanish. His book on Clinical Electrocardiography, first published in Catalan.
and Castilian Spanish, was later translated into French, English, Russian, Polish and Turkish. It received excellent reviews. Copies are attached of the review in the New England Journal of Medicine and of the foreword by Prof. Eugene Braunwald to the next edition in English. He has edited 15 books and published over 190 chapters in other books and monographs.

Antoni Martínez Rubio was born in Sabadell in 1961. After taking his degree in Medicine and Surgery at the Autonomous University of Barcelona (1985), he worked at the German universities of Düsseldorf and Münster, specialising in Internal Medicine and Cardiology. His basic research area was for many years the electrophysiology of the heart. He received a magna cum laude for his PhD. thesis (University of Münster) on: «Electrophysiological parameters that determine the induction of ventricular tachycardia versus ventricular fibrillation during programmed ventricular stimulation». Since the end of 1996 he has worked at the Sta. Creu and St. Pau Hospital in Barcelona as a clinical cardiologist and electrophysiologist. He has undertaken new lines of research in Arrhythmology and Ischaemic Cardiopathy, and received the Pfizer research grant from the Spanish Cardiology Association in 1999. He is author or co-author of over 250 papers to international congresses, chapters in books and articles in peer-review journals (Circulation, Journal of the American College of Cardiology, European Heart Journal, European Journal of Clinical Investigation, American Journal of Cardiology, Pace, Zeitschrift für Kardiologie etc.). He has received invitations to lecture in several countries and is a reviewer for several international journals. He is a member of, and participant in, various organisations: German Society of Cardiology, Spanish Cardiology Association, Working Group on Arrhythmias of the European Society of Cardiology, and the Scientific Committee of the European Society of Cardiology. He is assistant editor of Timely Topics in Medicine – Cardiovascular, Fellow of the European Society of Cardiology (F.E.S.C.), Fellow of the American College of Cardiology (F.A.C.C.), and lecturer at the School of Nursing of the Autonomous University of Barcelona. He is also a member of the committee of experts of the European Commission on: Evaluation of therapies through multinational large scale studies/trials taking into account advances in modern technology and optimised use of databases, registries, reagents and sample banks in «Chronic and degenerative diseases, cancer, diabetes, cardiovascular diseases and rare diseases»; «Quality of life and Management of Living Resources» of the Fifth Framework Programme for RTD and Demonstration Activities (European Commission, Brussels).

Antoni Bayés Genis (Vic 1968). He graduated from the Autonomous University of Barcelona in 1992 with a degree in Medicine and Surgery. He did his Cardiology Residency from 1993 to 1997 at the Sant Pau Hospital in Barcelona. During his Cardiology training he undertook clinical research in the team led by Dr. Bayes de Luna, which focussed on the study of the causes triggering sudden death and on the use of an old medicine, Colchicine, to treat refractory Pericarditis. In 1998 he was awarded a Ph.D cum laude for his thesis on «Haemostatic markers in acute coronary syndromes». He published two articles in international journals and a book chapter based on the thesis, which had been financed by a CICYT scholarship. In the same year, 1998, he started cardiovascular research work, using porcine models to study coronary disease, at the laboratory of Dr. L. Badimon. Since 1999 he has been at the Mayo Clinic on a grant for further study from La Caixa. Here he is a research fellow at the «Center for Applied Vascular Biology» with Dr. R.S. Schwartz. The main aims of this research are to deepen our understanding of the causes triggering coronary restenosis after angioplasty and to develop further new intra-coronary stents to maintain the vascular lumen more open for a longer period.