



ASOCIACIÓN NACIONAL  
DE  
MÉDICOS FORENSES

# Spanish Journal of Legal Medicine

## Revista Española de Medicina Legal

[www.elsevier.es/mlegal](http://www.elsevier.es/mlegal)



### REVIEW

## Update on sudden cardiac death: Epidemiology and risk stratification<sup>☆</sup>



Antoni Bayés de Luna<sup>a,\*</sup>, Albert Massó van-Roessel<sup>a</sup>, Luis Alberto Escobar-Robledo<sup>a</sup>, Josep Arimany-Manso<sup>b,c</sup>

<sup>a</sup> Instituto Catalán de Ciencias Cardiovasculares (ICCC), Hospital de Sant Pau, Barcelona, Spain

<sup>b</sup> Unidad de Medicina Legal, Laboral y Toxicología, Departamento de Medicina, Facultad de Medicina, Universidad de Barcelona, Barcelona, Spain

<sup>c</sup> Servicio de Responsabilidad Profesional, Área de Praxis, Colegio Oficial de Médicos de Barcelona, Barcelona, Spain

Received 15 September 2017; accepted 9 October 2017

Available online 13 February 2018

### KEYWORDS

Sudden death;  
Arrhythmias;  
Legal medicine

**Abstract** In this article, a discussion is presented on the following aspects of sudden death, related to legal medicine. The epidemiological aspects and diseases associated with sudden death are discussed first. This is followed by presenting the chain of events leading to the final arrhythmias triggering sudden death, and the legal aspects of this.

This is a comparative study of the final arrhythmias responsible of sudden death in patients with: (1) acute myocardial infarction; (2) with no apparent heart disease; and (3) with heart failure. A comparison is also made between the incidence of ischaemic heart disease, acute coronary thrombosis, and left ventricular hypertrophy.

© 2017 Asociación Nacional de Médicos Forenses. Published by Elsevier España, S.L.U. All rights reserved.

### PALABRAS CLAVE

Muerte súbita;  
Arritmias;  
Medicina legal

### Actualización de la muerte súbita cardiaca: epidemiología y estratificación del riesgo

**Resumen** En este artículo comentamos varios aspectos de la muerte súbita, relacionados con la medicina legal. En primer lugar, se exponen los aspectos epidemiológicos y las enfermedades asociadas a la muerte súbita. Más tarde, se discute la cadena de eventos que llevan a las arritmias finales a desencadenar la muerte súbita y sus aspectos legales.

DOI of original article: <http://dx.doi.org/10.1016/j.reml.2017.10.002>

<sup>☆</sup> Please cite this article as: Bayés de Luna A, van-Roessel AM, Escobar-Robledo LA, Arimany-Manso J. Actualización de la muerte súbita cardiaca: epidemiología y estratificación del riesgo. Rev Esp Med Legal. 2018;44:5–12.

\* Corresponding author.

E-mail address: [abayes@csic-iccc.org](mailto:abayes@csic-iccc.org) (A. Bayés de Luna).

Se hace un estudio comparativo de las arritmias finales responsables de la muerte súbita en pacientes: 1) con infarto agudo; 2) ambulatorios sin cardiopatía evidente y 3) con insuficiencia cardíaca evidente, y también se comparan las diferencias de incidencia de cardiopatía isquémica, trombosis coronaria aguda e hipertrofia ventricular isquémica.

© 2017 Asociación Nacional de Médicos Forenses. Publicado por Elsevier España, S.L.U. Todos los derechos reservados.

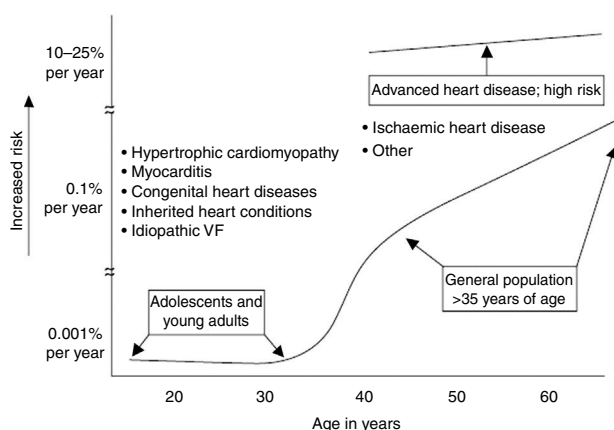
## Epidemiology

Sudden death (SD) probably represents the most significant challenge of modern cardiology due to the large number of cases which exist (in the USA alone there are more than 400,000/year) and due to its resulting significant social impact. However, its incidence is lower in some Mediterranean countries such as Spain.<sup>1–6</sup>

Although SD can even occur in infants, related to repolarisation disorders, autonomic nervous system abnormalities and increased vagal tone, it is in fact rare in the first few decades of life. In young subjects, it often occurs while doing sport.<sup>7</sup> In these cases, it tends to develop in the presence of inherited heart conditions (hypertrophic cardiomyopathy, arrhythmogenic right ventricular dysplasia/cardiomyopathy and channelopathies). The incidence of SD increases gradually, but significantly, from the age of 35 to 40 years, and it is particularly high in the acute phase of myocardial infarction (MI). It is also common in the chronic phase of this disease and in any heart disease, especially in the presence of heart failure (HF)<sup>8</sup> (Fig. 1).

## Associated diseases

As we have just stated, acute ischaemic heart disease (IHD) is frequently associated with SD in adults. In most cases of



**Figure 1** Correlation between incidence of SD and age. Also note that SD is associated with different diseases throughout life.

Source: Taken from Myerburg RJ. Circulation. 1992; 85 Suppl 1:12.

**Table 1** Diseases associated with SD: autopsy study.

| SD victims (n = 204)                       | No. | %    |
|--------------------------------------------|-----|------|
| <i>Cardiovascular diseases (n = 183)</i>   |     |      |
| <i>Heart diseases (n = 161)</i>            |     |      |
| Coronary heart disease                     | 119 | 58.4 |
| Hypertensive heart disease                 | 20  | 9.9  |
| Valve diseases                             | 5   | 2.4  |
| Idiopathic LVH                             | 4   | 1.9  |
| Dilated cardiomyopathy                     | 4   | 1.9  |
| Hypertrophic cardiomyopathy                | 3   | 1.5  |
| Arrhythmogenic RV cardiomyopathy           | 3   | 1.5  |
| Myocarditis                                | 1   | 0.5  |
| Congenital heart diseases                  | 1   | 0.5  |
| Amyloidosis                                | 1   | 0.5  |
| <i>Vascular disease (n = 22)</i>           |     |      |
| Pulmonary embolism                         | 8   | 3.9  |
| Aortic dissection                          | 9   | 4.4  |
| Brain haemorrhage                          | 5   | 2.4  |
| <i>Non-cardiovascular diseases (n = 7)</i> |     |      |
| Gastrointestinal disorders                 | 3   | 1.5  |
| Pulmonary disorders                        | 4   | 1.9  |
| <i>No findings (n = 14)</i>                | 14  | 6.9  |

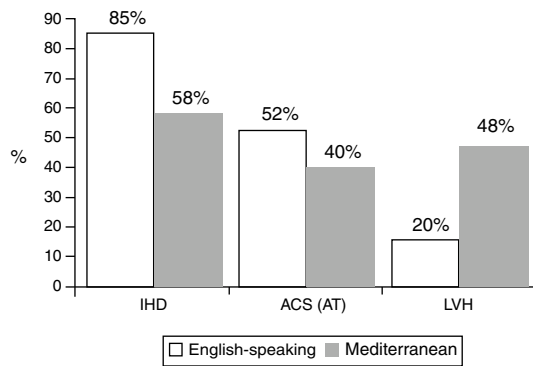
SD, aside from acute IHD and channelopathies, HF or, at least, ventricular dysfunction can be identified. HF can be associated with idiopathic cardiomyopathy, or be present in patients with chronic IHD, hypertension, cardiomyopathies of another aetiology, etc. Inherited heart conditions can cause SD at any age, but their overall impact is small (Fig. 1).

It is worth highlighting, however, that they are responsible for many cases of SD which occur before the age of 35 years. Inherited heart conditions are more prevalent in males and can occur during physical exercise (cardiomyopathies), at rest or during sleep (channelopathies).

Our group conducted a study (EULALIA study) which included 204 cases of SD occurring in a Mediterranean region.<sup>9</sup> This study analysed the epidemiological, anatomical and pathological aspects of diseases associated with SD. Table 1 shows the pathological diagnosis obtained in this group of patients.

It is striking, when compared to similar studies in English-speaking countries,<sup>10</sup> that the incidence of cases of IHD detected in the autopsy is lower ( $\approx 80$ – $90\%$  vs  $58\%$ ) and that, within these cases, the incidence of acute thrombosis and

Results from studies from English-speaking countries versus Mediterranean studies



**Figure 2** Comparative study of the incidence of ischaemic heart disease (IHD), acute coronary syndrome with infarction: acute thrombosis (AT) and left ventricular hypertrophy (LVH). Source: Taken from Subirana et al.<sup>7</sup>

pathological expression of acute MI is also lower (52% vs 40%) (Fig. 2). All of this is consistent with the already known evidence,<sup>1,3,4,11</sup> that the incidence of IHD in Mediterranean countries is lower, which is probably related not only to the diet, but also to the overall way of life and to environmental aspects (Mediterranean culture). On the other hand, we have observed a greater number of SD cases which present left ventricular hypertrophy (48% vs 20%)<sup>9,12</sup> (Fig. 2). From a clinical point of view and compared with the Maastricht study,<sup>13</sup> SD victims from the EULALIA study presented fewer reports of angina pectoris history (20% vs 37%), which is consistent with the lower number of cases of IHD found in the autopsy. In our series, the incidence of possible associated inherited diseases was 3% of cases (hypertrophic and right ventricle arrhythmogenic cardiomyopathies) and no findings were found in the post-mortem in around 7% of cases. This suggests that some of them probably suffered a channelopathy.

### Chain of events which leads to the final arrhythmias triggering sudden death

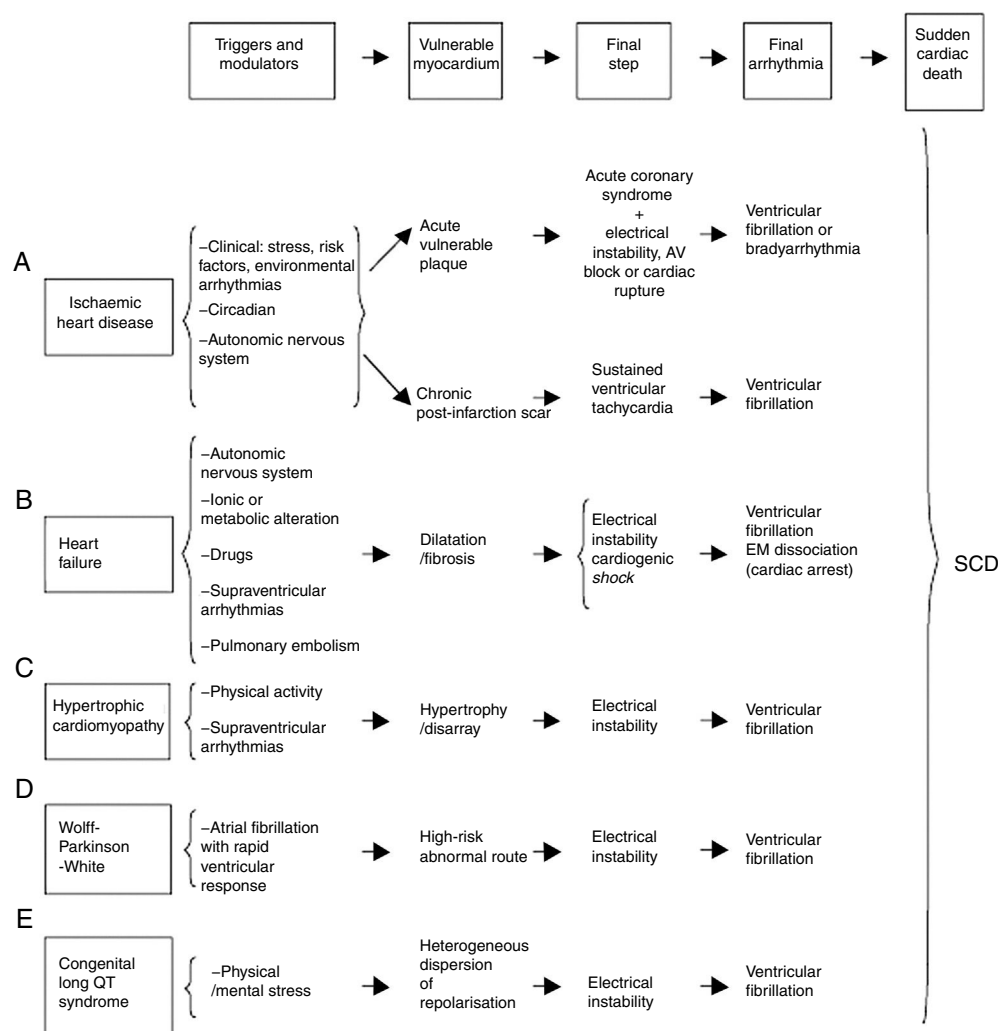
SD is the final stage in a chain of events leading to a cardiac arrest, in general, due to ventricular fibrillation (VF) or, less often, due to an extreme bradyarrhythmia.<sup>14</sup> In all cases, there are a series of modulating or triggering factors which, acting on the vulnerable myocardium, precipitate SD. Fig. 3 shows how this chain of events occurs in different heart diseases. VF tends to be triggered by a premature ventricular contraction (PVC) in the presence of different modulating or triggering factors, among which an appropriate genetic and environmental setting, or the increase in sympathetic activity in relation to physical or mental stress should probably be included. Although this does not have consequences in normal situations, it could trigger SD in the presence of acute ischaemia (Fig. 4). VF is often triggered by sustained ventricular tachycardia (VT) (Fig. 5) or VT in torsades de pointes (Fig. 6). SD presents less frequently as a consequence of a bradyarrhythmia (Fig. 7).

Final arrhythmias which precipitate SD are not always the same (Figs. 4–8). In a study, which we conducted, review-

ing the tapes of 157 outpatients who died from SD while they were wearing a Holter monitor,<sup>15</sup> we found (Fig. 8B and Table 2) that in two-thirds of the cases, the SD was due to a sustained VT which precipitated a VF, in general in the presence of a rapid baseline heart rate (sinus tachycardia or rapid atrial fibrillation), revealing an increase in sympathetic activity (Fig. 5). In contrast, VF not triggered by VT, which is often associated with acute IHD, is often presented as a consequence of a PVC with the R-on-T phenomenon. In our experience, in outpatients this pattern was observed in less than 10% of cases (Fig. 4). Curiously, in 13% of cases, SD was due to a VT in torsades de pointes which precipitated the VF. This generally occurred in patients without severe heart disease but who were taking class I antiarrhythmic drugs due to rare ventricular arrhythmias, often isolated PVCs (proarrhythmic effect). We believe that if this study was repeated now, the number of these cases would be much lower because there is evidence, from the CAST study,<sup>16</sup> that type I antiarrhythmic drugs are dangerous, especially in patients with heart disease. Therefore, the prescription of class I antiarrhythmic drugs in post-acute myocardial infarction (post-AMI) patients is currently much lower. Lastly, when extreme bradyarrhythmia was the cause of SD ( $\approx 15\%$  in our study) (Fig. 8B), it was due more to progressive depression of the sinus automatism and of the atrioventricular (AV) junction than to AV block (Fig. 7).

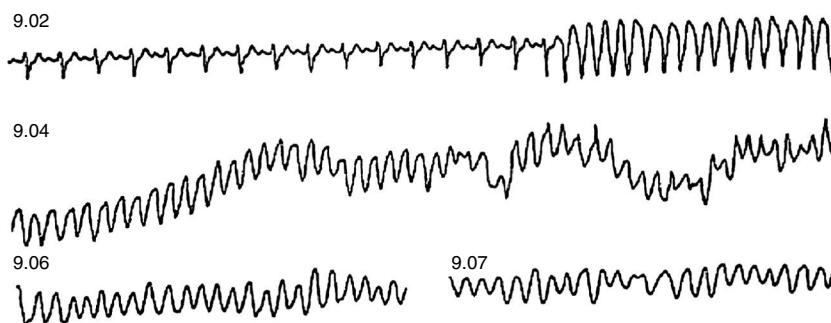
Fig. 8 shows the final arrhythmias which caused SD in patients with different clinical characteristics: (A) on route to the hospital, in a mobile coronary care unit with acute ischaemic symptoms<sup>17</sup>; (B) in outpatients (Holter monitor)<sup>15</sup> and (C) in patients admitted due to severe HF.<sup>16</sup> In the first case, a much higher number of cases of VF without prior VT was observed than in the group of outpatients in our study. This was certainly related to the fact that all patients in group A were in the acute phase of MI. In patients with advanced HF (group C), it was verified that there were many more cases of SD due to severe bradyarrhythmia. This could explain the ineffectiveness of antiarrhythmic drugs in preventing SD in patients with advanced HF. In our series (Fig. 8B), 80% of patients had a reduced ejection fraction, but their functional capacity was, in general, acceptable. These patients were too healthy to die, and many of these cases of SD could have been prevented with adequate treatment, which sometimes consists of not prescribing class I antiarrhythmic agents. We must remember the Hippocratic Oath: *Primum non nocere* (First, do no harm).

Our results were similar to those found in patients with an implantable cardioverter defibrillator with or without cardiac resynchronisation therapy. In this case, rapid VT also developed frequently and was treated with anti-tachycardia stimulation.<sup>18–20</sup> On the other hand, in a small series of post-acute myocardial infarction patients with an ejection fraction below 40%, with an implantable loop recorder, who died suddenly, it was demonstrated that most cases of SD were due to primary VF not triggered by VT. However, the information on the clinical events around the time of the death was unknown. This lack of information and the reduced number of cases makes the comparison of these series with our results difficult. Furthermore, in most patients who died from different types of bradyarrhythmias, death occurred more than 1 h after the onset of symptoms.<sup>21</sup>



**Figure 3** Chain of events which precipitate sudden cardiac death (SCD) and parameters which the different diseases present in the different stages leading to SCD.

Source: Adapted from Bayés-Genís et al.<sup>14</sup>



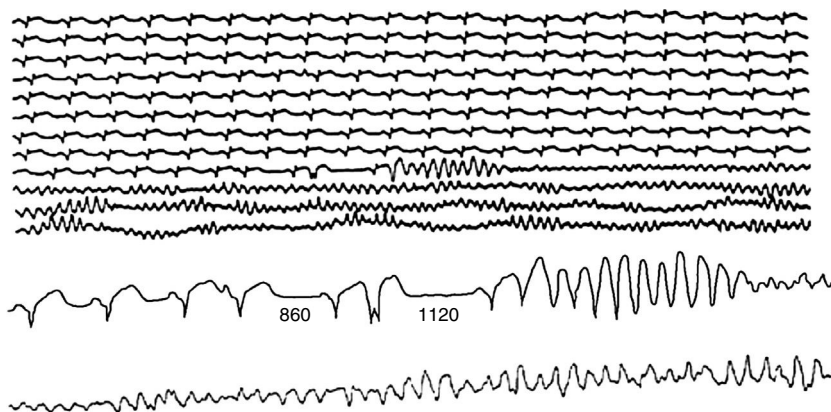
**Figure 4** Outpatient sudden death from VF, of a coronary patient treated with amiodarone due to presenting frequent premature ventricular contractions. At 9.02 a.m., the patient suffered a sustained monomorphic VT, followed by a VF at 9.04 a.m. after an increased rhythm of the VT and a widening of the QRS complex.

## Final considerations

Traditionally, forensic and legal medicine has undertaken the study of sudden cardiac death (SCD), along with other medical specialities. In recent years, several studies<sup>9</sup> have

underlined the importance of complementarity between different medical specialities, and the need for a multidisciplinary approach in order to tackle the problem correctly.<sup>22</sup>

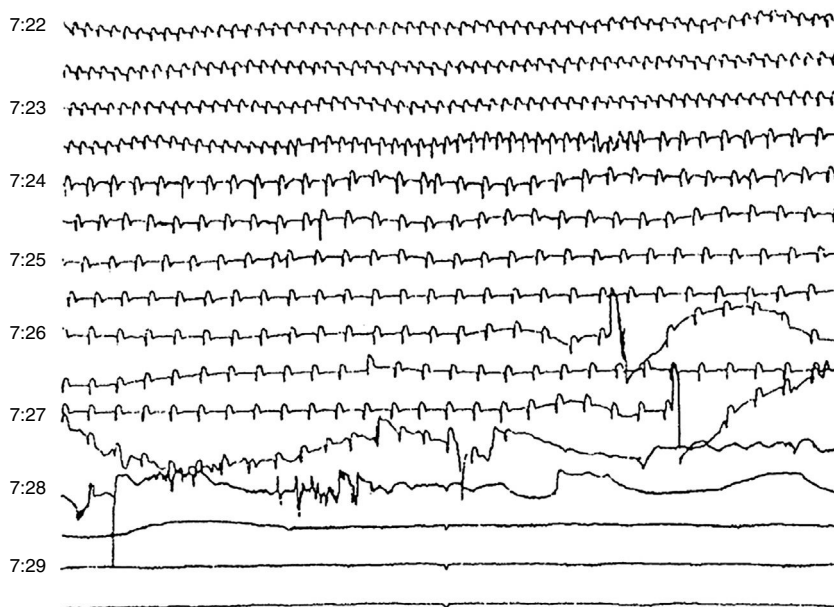
Furthermore, the modernisation of forensic medicine in Spain currently allows for a study of SCD which offers



**Figure 5** Outpatient sudden death from primary VF caused by ventricular extrasystole with short coupling interval, after post-extrasystolic pause (1.120 ms) greater than the previous one, which had caused an isolated ventricular extrasystole. Note that the sequence of events started with an atrial extrasystole which caused the first shorter pause (860 ms).



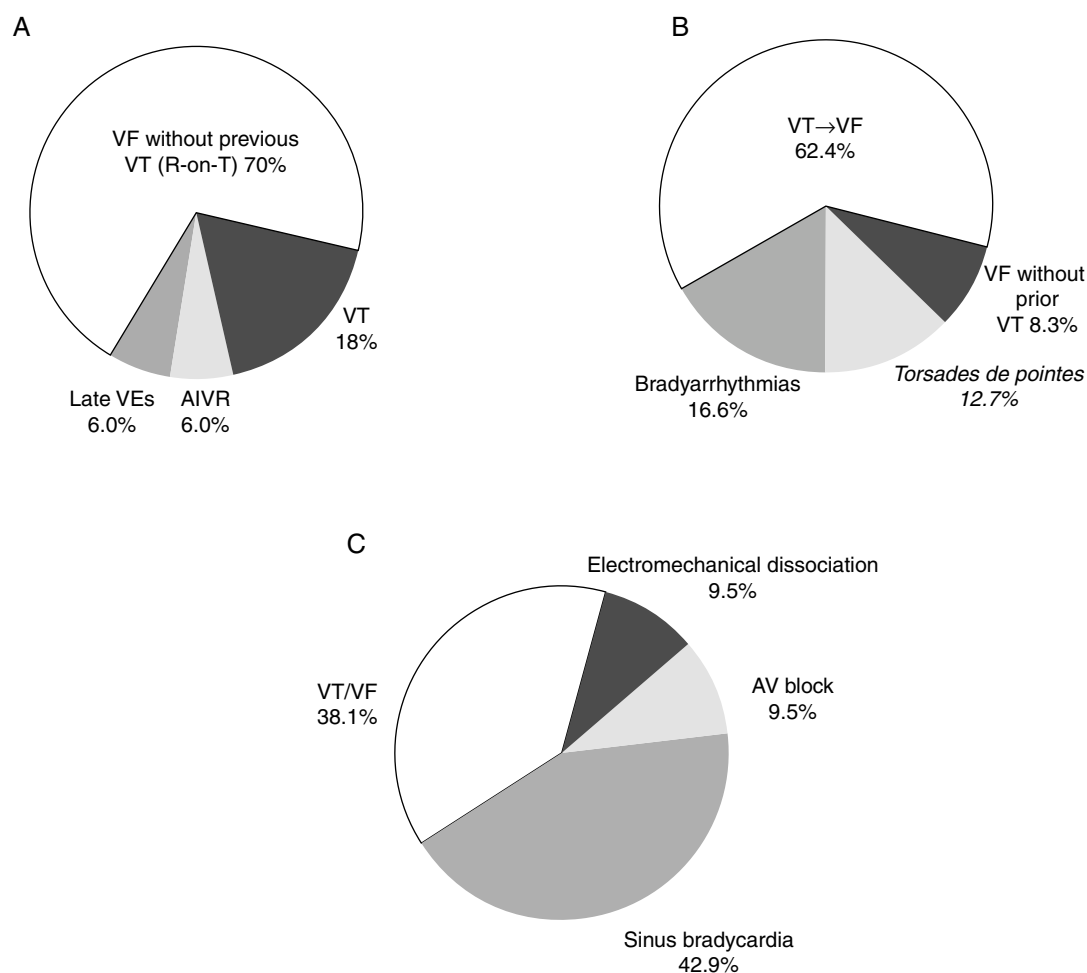
**Figure 6** Onset of a VT in torsades de pointes, in a woman treated with quinidine due to presenting bursts of non-sustained VT without coronary heart disease. In this case, VT in torsades de pointes triggered VF.



**Figure 7** Sudden death due to progressive bradycardia in a patient with acute infarction and probable electromechanical dissociation.

homogeneous results. These results can contribute to an analysis of the clinical setting, and lead to forensic medicine and healthcare medicine interacting with each other, with an undeniable interest for public health.<sup>23</sup> Therefore, an early diagnosis of the anatomical pathology

results of deceased individuals, and the molecular analysis, are crucial to diagnose possible familial cardiomyopathies or channelopathies (e.g. Brugada syndrome) in order for correct prevention to be carried out in affected family members.<sup>24</sup> There are multiple experiences of collaboration



**Figure 8** Sudden death: final arrhythmias (A) in patients with acute ischaemic heart disease.<sup>15</sup> (B) in outpatients in 80% of cases with reduced ejection fraction.<sup>13</sup> (C) In patients with advanced heart failure.<sup>16</sup>

**Table 2** Clinical characteristics of patients who died suddenly while wearing a Holter monitor.

|                                  | Group I primary VF and VT → VF n = 108 | Group II VT in torsades de pointes n = 23 | Group III bradyarrhythmias n = 26 |
|----------------------------------|----------------------------------------|-------------------------------------------|-----------------------------------|
| Age                              | 58                                     | 60                                        | 70                                |
| Gender (males) %                 | 76                                     | 40                                        | 39                                |
| Coronary patients %              | 90                                     | 50                                        | 70                                |
| With no apparent heart disease % | 4                                      | 22                                        | 10                                |
| Antiarrhythmic treatment %       | 51                                     | 70                                        | –                                 |

between medical examiners and cardiology specialists—such as the case of collaboration with physicians responsible for patients wearing an implantable cardioverter defibrillator—which can contribute to improving knowledge about these patients' conditions, to detecting possible malfunctions in the devices and to optimising therapies.<sup>25</sup>

In this clear context of needing a multidisciplinary approach, we consider the coordination of healthcare services (hospital and primary care) with institutions of the Ministry of Justice, Justice departments of the autonomous communities and legal medicine and forensic science

institutes, along with the responsible public health departments, to be necessary. This interrelationship would be very useful to determine the real incidence and to design SD prevention strategies, with regard to diagnostic methods, screening and immediate healthcare procedures in the event of an emergency situation occurring, as various authors have previously underlined.<sup>26,27</sup> Likewise, it would also be desirable for the advisory bodies of both ministries (Health and Justice) to draft a comprehensive action plan which ensures the quality of post-mortem testing and the subsequent familial clinical and genetic testing.<sup>28</sup>

There are experiences of a multidisciplinary approach in areas such as Tarragona (Catalonia), which study the incidence and aetiology of SCD, integrating outpatient emergency departments and forensic and clinical sources (ReCapTa Study).<sup>29</sup>

Lastly, in the face of the common challenge of SD, we believe that it is necessary to create a national registry for cases of SD, funded by the health administration, in coordination with the Ministry of Justice and the autonomous communities competent in matters of Justice.<sup>30</sup>

Not forgetting the information programmes for citizens and the CPR training plans, it should be highlighted, within the prevention plans, that the use of semi-automatic defibrillators in public places is an important breakthrough as a treatment measure in cases of cardiac arrest. There is unanimity among most medical scientific societies that early defibrillation is the "key to survival". The installation of defibrillator devices has become mainstream in non-professional sports venues and also in public places in Spain. However, Spain remains far behind other European countries such as Germany and France.<sup>31</sup>

Finally, it is important to emphasise again that, in the face of the most significant challenge in modern cardiology, the comprehensive collaboration of all individuals involved in the broad field of intervention in SD, starting with those who deal with the preventive side and ending with those who are in charge of the post-mortem diagnosis, is fundamental.

## Conflicts of interest

The authors declare that they have no conflicts of interest.

## References

- Marrugat J, Elosua R, Gil M. Epidemiology of sudden cardiac death in Spain. *Rev Esp Cardiol*. 1999;52:717–25.
- Masiá R, Pena A, Marrugat J, Sala J, Vila J, Pavesi M, et al. High prevalence of cardiovascular risk factors in Gerona, Spain, a province with low myocardial infarction incidence. REGICOR Investigators. *J Epidemiol Commun Health*. 1998;52:707–15.
- Keys A, Keys M. How to eat well and stay well. The Mediterranean Way. New York: Doubleday, Garden City; 1975.
- Sans S, Puigdefàbregas A, Paluzie G, Monerde D, Balaguer-Vintró I. Increasing trends of acute myocardial infarction in Spain: the MONICA-Catalonia Study. *Eur Heart J*. 2005;26:505–15.
- Morentin B, Audicana C. Estudio poblacional de la muerte súbita cardiovascular extrahospitalaria: incidencia y causas de muerte en adultos de edad mediana. *Rev Esp Cardiol*. 2011;64:28–34.
- Barbería E, Gotsens M, Xifró A, Castellà J, Rodríguez-Sanz M, Medallo J, et al. Estudio retrospectivo de la muerte súbita cardiovascular extrahospitalaria de adultos de mediana edad en la ciudad de Barcelona. *Rev Esp Cardiol (Engl Ed)*. 2013;66:226–7.
- Bayés de Luna A, Furlanello F, Maron BJ, Zipes DP. Arrhythmias and sudden death in athletes. Dordrecht: Kluwer Academic Publishers; 2000.
- Myerburg RJ, Interian A Jr, Mitrani RM, Kessler KM, Castellanos A. Frequency of sudden cardiac death and profiles of risk. *Am J Cardiol*. 1997;80(5B):10F–9F.
- Subirana MT, Juan-Babot JO, Puig T, Lucena J, Rico A, Salguero M, et al. Specific characteristics of sudden death in Mediterranean Spanish population. *Am J Cardiol*. 2011;107:622–7.
- Burke AP, Farb A, Malcom GT, Liang YH, Smialek J, Virmani R. Coronary risk factors and plaque morphology in men with coronary disease who died suddenly. *N Engl J Med*. 1997;336:1276–82.
- De Longeril M, Salen P, Martin JL, Monjaud I, Delaye J, Mamelle N. Mediterranean diet, traditional risk factors, and the rate of cardiovascular complications after myocardial infarction: final report of the Lyon Diet Heart Study. *Circulation*. 1999;99:779–85.
- Virmani R, Burke AP, Farb A. Sudden cardiac death. *Cardiovasc Pathol*. 2001;10:275–82.
- De Vreede-Swagemakers JJ, Gorgels AP, Dubois-Arbouw WI, van Ree JW, Daemen MJ, Houben LG, et al. Out-of-hospital cardiac arrest in the 1990's: a population-based study in the Maastricht area on incidence, characteristics and survival. *J Am Coll Cardiol*. 1997;30:1500–5.
- Bayés-Genís A, Viñolas X, Guindo J, Fiol M, Bayés de Luna A. Electrocardiographic and clinical precursors of ventricular fibrillation: chain of events. *J Cardiovasc Electrophysiol*. 1995;6:410–7.
- Bayés de Luna A, Coumel P, Leclercq JF. Ambulatory sudden cardiac death: mechanisms of production of fatal arrhythmia on the basis of data from 157 cases. *Am Heart J*. 1989;117:151–9.
- Echt DS, Liebson PR, Mitchell LB, Peters RW, Obias-Manno D, Barker AH, et al. Mortality and morbidity in patients receiving encainide, flecainide, or placebo: the Cardiac Arrhythmia Suppression Trial. *N Engl J Med*. 1991;324:781–8.
- Adgey AA, Devlin JE, Webb SW, Mulholland HC. Initiation of ventricular fibrillation outside hospital in patients with acute ischaemic heart disease. *Br Heart J*. 1982;47:55–61.
- Luu M, Stevenson WG, Stevenson LW, Baron K, Walden J. Diverse mechanisms of unexpected cardiac arrest in advanced heart failure. *Circulation*. 1989;80:1675–80.
- Leitch JW, Gillis AM, Wyse DG, Yee R, Klein GJ, Guiraudon G, et al. Reduction in defibrillator shocks with an implantable device combining antitachycardia pacing and shock therapy. *J Am Coll Cardiol*. 1991;18:145–51.
- Grimm W, Plachta E, Maisch B. Antitachycardia pacing for spontaneous rapid ventricular tachycardia in patients with prophylactic cardioverter-defibrillator therapy. *Pacing Clin Electrophysiol*. 2006;29:759–64.
- Gang UJ, Jons C, Jorgensen RM, Abildstrom SZ, Haarbo J, Messier MD, et al. CARISMA investigators Heart rhythm at the time of death documented by and implantable loop recorder. *Europace*. 2010;12:254–60.
- Basso C, Aguilera B, Banner J, Cohle S, d'Amati G, Henriques de Gouveia R, et al. Guidelines for autopsy investigation of sudden cardiac death: 2017 update from the Association for European Cardiovascular Pathology. *Virchows Arch*. 2017. <http://dx.doi.org/10.1007/s00428-017-2221-0> [Epub ahead of print].
- Xifró-Collamata A, Suelves-Joanich JM. Patología forense y salud pública. *Rev Esp Salud Publica*. 2011;85:419–20.
- Maron BJ. Medical progress: sudden death in young athletes. *N Engl J Med*. 2003;349:1064–75.
- Villuendas Sabaté R, Alcalde Rodríguez O. Retirada autopsica de un desfibrilador automático implantable. *Rev Esp Med Legal*. 2011;37:173–6.
- Puigdefàbregas Serra A, Freitas Ramirez A, Gispert Magarolas R, Castella Garcia J, Vidal Gutierrez C, Medallo Muñoz J, et al. Las muertes con intervención judicial y medicolegal y su impacto en la estadística de causas de muerte en Cataluña. *Rev Esp Med Legal*. 2017;43:13–9.
- Barbería-Marcailain E, Xifró A, Arimany-Manso J. Impacto benéfico de la incorporación de las fuentes forenses a las estadísticas de mortalidad. *Rev Esp Med Legal*. 2017;43:1–4.
- Molina Aguilar P, Giner Blasco J, Izquierdo Macian I, Martínez-Dolz L, Barriales Villa R, Zorio Grima E, et al. Unidades multidisciplinarias en el estudio y prevención de la muerte súbita por cardiopatías familiares. *Rev Esp Med Legal*. 2018;44:46–52.

29. Azeli Y, Barbería E, Jiménez-Herrera M, Bonet G, Valero-Mora E, López-Gomariz A, et al. The ReCaPTa study: a prospective out of hospital cardiac arrest registry including multiple sources of surveillance for the study of sudden cardiac death in the Mediterranean area. *Scand J Trauma Resusc Emerg Med*. 2016;24:127.
30. Arimany-Manso J, Bayés de Luna A. Muerte súbita en el deporte. *Rev Esp Med Legal*. 2011;37:131–3.
31. Desfibrilación semiautomática en España. Informe. Ministerio de Sanidad y Política Social; 2007. Available from: [http://www.msc.es/organizacion/sns/planCalidadSNS/docs/Informe\\_uso\\_Desfibriladores\\_sep\\_07.pdf](http://www.msc.es/organizacion/sns/planCalidadSNS/docs/Informe_uso_Desfibriladores_sep_07.pdf) [accessed 12.09.17].