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Editorial

Sudden cardiac death during sports practice. Is one man's meat another man's poison?

Muerte súbita en el deporte, ¿lo que cura a uno mata a otro?

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When people die suddenly during exercise, their death usually has major social repercussions. If they were competing, the tragedy will probably be rapidly publicized via newspapers, radio, television, and social media. The consequence of this dissemination is that many people who take exercise are immediately considered possible victims of the same misfortune, and skeptics of the benefits of exercise instantly have reasons to justify their sedentary behavior.

There are 2 scientifically proven reasons to dismiss the emotional biases caused by these dramatic deaths. The first is that physical exercise significantly improves the cardiovascular health of all people, both young and old and whether healthy or unwell.[1] The second is that, because the incidence of sport-related sudden death is extraordinarily low, exercise cannot be considered to represent a threat to life, even when intense or strenuous and only in very specific cases.

The overall incidence of sport-related sudden cardiac death is estimated to be about 1/100 000 people per year.[2] This figure varies according to the subgroup analyzed. The incidence is lower in the general population and slightly higher among competitive athletes.

A critical question is why sudden cardiac death occurs. We do not know or, better said, we know very little. It is difficult to obtain this information due to the circumstances typically surrounding these deaths. For a diagnosis to be made, it is vital to know if the person had symptoms (eg, palpitations, dizziness, chest pain). This information is usually very difficult to obtain, even if the person survives the cardiac arrest, because retrograde amnesia is often caused by concussion.[3] It is also, of course, crucial to visualize the cardiac rhythm with an electrocardiogram at the time of the event, which has thus far rarely been possible.

Despite these difficulties, the overwhelming majority of these deaths have a cardiac origin and the most frequently seen arrhythmia is ventricular fibrillation. This arrhythmia, the least well-known of all in terms of the underlying mechanisms, causes a drastic drop in cardiac output. The immediate cessation of cerebral perfusion leads to loss of consciousness within seconds and culminates in asystole within minutes.

Given the absence of clinical and electrocardiographic data, the information on cause of death has to be obtained from an autopsy. This has major limitations. Unless the autopsies are obligatory and performed under the same criteria, the results might represent only a selected population. In addition, cardiac studies are particularly difficult and require specialized knowledge and sophisticated technology. It is thus essential for the autopsy to be protocol-based to obtain maximum clinical information on family history, pathology, toxicology, and genetics.

Accordingly, the study by Morentin et al.,[4] recently published in *Revista Española de Cardiología*, is very interesting. In this work, a multidisciplinary team analyzed the forensic findings of 288 people. All had unexpectedly died during sporting activity in the absence of trauma or violence. The study also included individuals who died up to 1 hour after cessation of the physical activity.[4] The researchers performed a retrospective analysis of all deaths occurring during an 8-year period (2010-2017) in a large national territory. The authors report epidemiological, clinical, pathological, and toxicological data, obtained via a highly protocol-based, homogeneous, and rigorous methodology. All macroscopic and histological studies were performed by forensic pathologists with cardiovascular experience according to international recommendations.

The work represents the largest case series of the general population published in Spain. Its main conclusions are in line with those of the few similar international series published. The incidence of sport-related sudden death was 0.38 cases/100 000 population per year and it mainly affected men (99% of cases). The highest incidence was seen in adults aged between 35 and 54 years (mean, 44 years); 56% were practicing outdoor sports and 44% were practicing indoor sports. Only 4% (11 people, 7 younger than 30 years old) were practicing competitive sport at the time of death. The remaining deaths (96%) were associated with recreational sport.

The cause of death was attributable to the heart in 98% of cases and the underlying disease and the type of sport being practiced were related to the age of the victim. For those older than 35 years, the most common finding was ischemic heart disease (coronary thrombosis, acute infarction, > 75% stenosis, or chronic infarction). Above this age, running and particularly cycling were the most frequent activities. Below this age, the most frequent activity was football and the most common findings were myocardial disease and sudden arrhythmic death syndrome. The latter group includes channelopathies.

In 10% of cases, no underlying disease that could have caused death was identified. In addition, 13% had previous symptoms

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(chest pain, palpitations, or syncope) and 20% had a relevant family history.

One of the interesting findings of the study is that cardiotoxic substances (cannabis, alcohol, QT-prolonging drugs, or cocaine) were detected in 7% of the victims and a combination of them in 3 cases. Of the 10 who tested positive for cannabis, acute ischemic heart disease was the cause of death in 8.

Regarding the type of sporting activity, cycling predominated, even after the data were adjusted for the total population practicing this sport. As the authors note, this does not mean that cycling is the most dangerous sport, given that the population practicing it appears to have more cardiovascular risk factors (including overweight). Nonetheless, this topic needs to be investigated.

In summary, the authors report a set of original and essential data to effectively shed light on sport-related sudden death. Extrapolating, they calculate that it causes 167 deaths annually in Spain (165 men and 2 women). This male predominance, found in almost all sudden cardiac death series, is one of the enigmas still being studied. It is currently considered to be due more to the coexistence of other causes (eg, more cardiovascular risk factors, different autonomic tone, different hormonal environment, higher sport intensity) than to intrinsic differences in the electrophysiological properties of the heart between men and women.[5]

Then, with such a large amount of data, why do we say that we know so little? The answer is very simple. Although the most frequent cardiac finding is ischemic heart disease (63% in the present article), we do not know why some people have ventricular fibrillation and others do not.[6] Massive infarctions often do not cause it, whereas an ischemia that affects just a small myocardial territory can trigger it. In chronic infarction, the fibrotic scar creates, in its interior, channels of healthy myocardium that favor the existence of circuits. In these circuits, electrical activation of the heart can lead to reentrant arrhythmias and very rapid ventricular tachycardias that deteriorate into ventricular fibrillation and cause death. In the cases reported in this article, 26% of the victims were diagnosed with acute ischemic heart disease and 37% with chronic ischemic heart disease. We also cannot rule out the possibility that coronary spasm may sometimes be involved, even in the absence of significant coronary lesions. Accordingly, although the cause of the ventricular fibrillation is not known, prevention of ischemic heart disease should continue to be the main objective to prevent sudden cardiac death.

It is even more difficult to determine the mechanism underlying sudden cardiac death in people with myocardial disease (21% of the cases in this series). Recent data have linked the presence of fibrosis on magnetic resonance imaging to arrhythmic events.[7] Electrophysiological studies can also help to explain if a heart is more or less vulnerable to arrhythmias but their significance greatly depends on the substrate. In chronic ischemic heart disease, the presence of scar tissue facilitates the induction of ventricular tachycardia in programmed pacing and this has been shown to have prognostic implications. In contrast, in cardiomyopathies (except maybe arrhythmogenic dysplasias), the induction of sustained ventricular arrhythmias is not easy or reproducible and has unknown prognostic significance.[8] The same occurs in channelopathies (about 5% in this series), because the inducibility of "malignant" arrhythmias has been linked to arrhythmia development during clinical follow-up in only some cases.

This failure in our ability to predict who will develop ventricular fibrillation is probably largely due to our inability to reproduce the specific circumstances triggering the arrhythmia. Sport not only increases myocardial oxygen demand, but also alters the autonomic tone, catecholamine concentration, pH, and electrolyte levels. To further complicate the problem, an exhaustive study was unable to clarify the reason for ventricular fibrillation or to find a cause of its development.[9] It may be that these people do not experience arrhythmia recurrence.[10]

Accordingly, when we talk about sudden cardiac death, and particularly when we talk about sport-related sudden death, it is important to recognize the deep uncertainty and lack of knowledge. Fortunately, some people experience a witnessed cardiac arrest and are treated in time. They survive thanks to the performance of resuscitation maneuvers and the use of a defibrillator. In some cases, a trigger can be found and treated. If not, a defibrillator may be implanted as "life insurance".

To improve survival from sudden cardiac death, we must invest in the training of bystanders who are able to immediately recognize cardiac arrest and are ready to perform resuscitation maneuvers. This requires implementation of action plans that incorporate the use of semiautomatic external defibrillators in places where sport is practiced and people gather. Technological advances help, by providing relevant information from different monitoring systems, such as watches, phones, and other devices that can recognize heart signals and enable the immediate localization of people requiring help. Applications such as Ariadna, promoted by the Spanish Society of Cardiology, are advances in this regard.[11]

Despite the existence of guidelines and recommendations to prevent sudden cardiac death, the sporting activity of people with predisposing heart diseases (ischemic heart disease, cardiomyopathies, and channelopathies) should be supervised by cardiologists familiar with the treatment of this type of condition.[12] When scientific evidence is lacking, the value of clinical experience and common sense increases even more, if possible.[12] This experience would facilitate the evaluation of the specific circumstances surrounding each heart disease and help these people to live a life as close as possible to their expectations.

Also important is screening prior to sporting participation, although no universally accepted model has been developed to balance its cost and effectiveness. Although the performance of 12-lead electrocardiography, added to clinical history and physical examination, increases the detection sensitivity of heart disease and reduces sudden cardiac death, this model is still being questioned.[13] The detection of false positives may lead to unnecessary tests and disqualify some people for sporting activity without solid scientific evidence.[12] Some have recently proposed that we go a step beyond echocardiography and even systematically apply magnetic resonance imaging to specific populations to rule out heart diseases.[14]

However, before we drastically limit a person's sporting activity and substantially alter their quality of life, we must remember that the incidence of sport-related sudden death is very low, despite its dramatic and emotionally devastating impact. A young person may have a higher chance of death due to mobile phone use while driving than that due to physical exercise, regardless of the intensity of the activity. In addition, raising fears surrounding the practice of sporting activity and physical exercise will deprive society of their extraordinary health benefits.

Personally, I fully support examinations before sporting participation, but more to educate people than to prevent sudden cardiac death (with such a low incidence, an accurate prediction is almost impossible). To prevent sudden cardiac death, we must promote healthy lifestyles, avoid the use of drugs, rapidly identify and treat cardiac arrest, and stress the value of immediately seeking medical care in the presence of certain symptoms.

I believe that the researchers[4] should be congratulated for collecting such a large amount of data from a series of autopsies of people who died of sport-related sudden death. This is probably the fruit of years of work, marked by the successive contributions of Drs Lucena and Suárez-Mier to the understanding of this problem.[15] As they themselves mention, the Spanish

Sports-related Sudden Death Study was launched in 2010, with the participation of the Spanish Society of Cardiology, the Spanish Sports Council, and the Spanish Society of Forensic Pathology.[16] Unfortunately, the then-pioneering project was discontinued but, hopefully, with the magnificent work being carried out by the Sports Cardiology Working Group of the Spanish Society of Cardiology and with the new information technologies, it will soon be possible to automate the data collection and thus create registries that enable much better understanding, among other things, of the incidence and causes of these deaths.

This excellent article by Morentin et al. also shows the extraordinary yield of network-based multidisciplinary work. It will hopefully stimulate prospective clinical research, essential in this field to root our decisions in clinical evidence. Fortunately, the day may be near that ventricular fibrillation—that accursed arrhythmia—can be treated with a simple drug.[17]

CONFLICTS OF INTEREST

None declared.

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