Sudden Death, Sport Activities, and Coronary Anomalies

To the Editor:

We read with interest the editorial written by Dr. Boraita[1] on sudden death and sports. In general, we agree with most of the author´s statements. However, we think that an echocardiographic study especially designed for the diagnosis of anomalies of the coronary arteries should be included in the athletic pre-participation cardiological examination (APCE).

Anomalies of the coronary arteries are the second most frequent cause of death in American athletes and the third most frequent in Italian athletes.[2] In the article, published in the same number of the JOURNAL, on the causes of sudden death associated with athletic activity in Spain, coronary anomalies are responsible for 3.2%. However, the limitations described by the authors should be considered (not all cases of sudden death associated with sports are recorded and the number of competitive or recreational athletes is not known).[2] As Dr. Boraita noted in her discussion of coronary anomalies, it is true that the results of the screening assessment can be discouraging because often no clinical, or physical findings are detected. The athlete may even have a completely normal electrocardiogram and exercise stress test in the presence of a potentially lethal coronary anomaly. How can the presence of a coronary anomaly be safely excluded in high performance athletes using non-invasive techniques? Transthoracic echocardiography allows the outflow ostia of both coronary arteries to be identified, as well as the initial section of both arteries in some cases.[3] Thus, transthoracic echocardiography has been widely used in pediatric patients 4 and even in high performance athletes.[3] Evidently, «poor echocardiographic windows» are the principal limitation of this non-invasive technique. Nevertheless, we must note that this is a study population (young people and athletes) with a low prevalence of «poor windows.»[3,4] In addition, we must consider the important advances in echocardiography in recent years, such as the introduction of devices (e.g., second harmonics) that appreciably improve the images obtained.

The question to be asked when we are confronted with a high competition athlete with a «poor echocardiographic window» that does not allow the presence of a coronary anomaly to be excluded is whether more tests are indicated. We think that they are. We must remember that we are dealing with a population of young people, under the age of 35 years, in which the presence of a coronary anomaly carries an added risk of sudden death.[5] Depending on the experience of the examining team and the preferences of the athlete, we would recommend the performance of a transesophageal echocardiogram[6] (which resolves all of the limitations of transthoracic echocardiograph) or a helical CT with intravenous contrast[7] (a technique available in most hospitals in Spain) or, if possible, vascular magnetic resonance imaging.[8]

In conclusion, we believe that the correct identification of the coronary ostia (by transthoracic echography or other techniques when unclear) should be included in the cardiological examination of high performance athletes. In the future, should this be recommended for all people who practice organized sports and demanding recreational activities?

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Response

To the Editor:

I would like to thank Drs. Barriales Vila, Moris de la Tassa, and Penas Lado for the interest that they have shown in my editorial published in your journal.[1] I am grateful for their interesting comments, although I do not agree with some of their thoughts about the purpose of athletic pre-participation cardiological examinations (APCE).

APCEs should be carried out for the early detection of cardiac pathologies that constitute a risk of sudden death in athletes. Therefore, the susceptible population is very large, since more than 12 million Spaniards practice either federation or recreational sports. This means that the diagnostic tests included in this examination should be sensitive and specific, as well as simple and inexpensive. The APCE meets almost all the necessary requirements for being considered of public interest, but is still not accepted or recognized by health authorities in most countries, or even by most sports federations.[2] In fact, in many countries in the world, as in Spain, a physical examination is require only in certain high-risk sports and activities like boxing, hunting, diving, speed-boating, or motorcycling. This absence of acceptance is due mainly to economic arguments of cost-effectiveness. The cost of such examinations is not accepted by either National Health Services or individual athletes.

On the other hand, the causes of sudden death vary widely with the age of the athlete.[3] In young athletes (<30 years), a group that includes most high performance athletes in Spain according to the study by Suárez-Mier and Aguilera,[4] arrhythmogenic cardiomyopathy of the right ventricle constitutes about one-fourth of all causes. In contrast with other published series, hypertrophic cardiomyopathy and anomalies of the coronary arteries (2 cases) show a low prevalence. As the authors themselves note, this was probably because the organs studied were from judicial autopsies in which forensic pathologists had previously diagnosed these pathologies. Likewise, it should not be overlooked that the low incidence of coronary anomalies could also be due in part to the effectiveness of the APCE in Spain, even if the examination could be much better.

For these reasons, the Working Group on Sports Cardiology proposes a simple screening protocol suitable for use in the entire population and capable of identifying or indicating the possible presence of a high risk cardiac pathology.[5] In fact, the advanced cardiological examination of young competitive athletes includes, in addition to a maximum exercise stress test, transthoracic Doppler echocardiogram for the identification of possible anomalies in the origin of the coronary arteries and hypertrophic cardiomyopathies that have not been diagnosed because they course with an atypical or even a normal ECG. Unfortunately, arrhythmogenic cardiomyopathy may remain undetected even by experts with these examinations. In my personal experience of cardiological examinations of the Spanish population of high performance athletes, including more than 5000 athletes throughout four olympic cycles, the ostia and initial sections of both coronary arteries were clearly identified in all of them. On the other hand, no coronary anomaly was suspected in any of the athletes excluded. In contrast, in the case of hypertrophic or arrhythmogenic cardiomyopathy examinations had to be complemented by other studies, including invasive studies, when the results were dubious. In fact, in the only two cases of sudden death that occurred in this population, the causes of death were exercise-induced anaphylaxis and biventricular diffuse, patchy subepicardial arrhythmogenic cardiomyopathy.

One might think that this population is highly selected and that it is not very likely that an athlete with a coronary anomaly would reach highly competitive circles since these sports are extremely demanding and coronary malformations would limit athletic performance. However, I would like to emphasize that, while this is true, there are athletes involved in sports with a moderate or high cardiovascular demand, which include 34 athletic specialties. Some of these specialties have a low static and dynamic component and, therefore, a low cardiovascular demand, which is why an athlete with these coronary anomalies could reach the highest circles of competition.

Undoubtedly, in the case of athlete in which there is a well-founded suspicion of the presence of a coronary anomaly, other diagnostic tests should be made before authorizing participation in competitive sports. Such tests might include transesophageal echocardiography, helical computed tomography with intravenous contrast, or vascular magnetic resonance imaging. The situation changes when considering whether these techniques should be included in the cardiological examination of high performance athletes or in the APCE screening for sports or demanding recreational activities.

I hope that in the not too distant future, thanks to the work of authors like Suárez-Mier and Aguilera and the efforts of the Spanish Society of Cardiology and the Spanish Federation of Sports Medicine, advanced APCE including an echocardiogram will not be limited to high performance athletes, but extended to the entire population of young people who practice organized sports or demanding recreational activities.

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Factor XIII Val34Leu Polymorphism and Premature Myocardial Infarction

To the Editor:

We read with real interest the exhaustive review by Professor Navarro of genetic polymorphisms and coronary artery disease.[1] Nonetheless, we would like to further discuss the influence on myocardial infarction of the Val34Leu polymorphism of coagulation factor XIII (FXIII). This polymorphism conditions a change of valine for leucine only 3 amino acids from the site of activation by thrombin. It has been confirmed that it facilitates the activation of FXIII, increasing and accelerating the stabilization of fibrin.[2] Nevertheless, paradoxically, it is has been observed that this polymorphism could play a protective role against both venous and arterial thrombosis,[3] as Professor Navarro indicates in his review.[1]

We studied the Val34Leu genotype of FXIII in 180 patients who suffered myocardial infarction before the age of 45 years and in 585 controls.[4] We found that the frequency of the Leu/Leu genotype was greater in patients (7.5% vs 3.8%; *P*=.05). After adjustment for other cardiovascular risk factors, this genotype was found to be an independent risk factor for the development of premature infarction (*P*=.019; odds ratio [OR], 3.54; 95% confidence interval [CI], 1.22-10.22).

Recently we analyzed the possible influence of this polymorphism on response to fibrinolytic treatment in premature myocardial infarction.[5] We observed that patients with the Val/Val genotype more frequently met criteria for non-invasive reperfusion (*P*=.006; OR, 5.11; 95% CI, 1.28-21.12). Therefore, the results of our study demonstrate the existence of clinical manifestations of the type suggested by the results of experimental studies.[6] These findings enhance the interest of this polymorphism in myocardial infarction and should be examined in prospective multicenter studies. It is even more important to know the influence this polymorphism has on fibrinolytic treatment in populations where it has been shown to be a mutation that protects against the development of thrombotic phenomena.

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