EDITORIAL COMMENT

Athens, Athletes, and Arrhythmias: The Cardiologist’s Dilemma*

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In 490 BC, the first battle for democracy was fought at the Greek village of Marathon. Though overwhelmingly outnumbered by an invading Persian horde, the citizen-soldiers of Athens were victorious. After running 26 miles to Athens to deliver the stunning news, the Athenian professional messenger Phidippides collapsed and died. Unfortunately, the tragedy of unexpected, sudden death in athletes has been repeated many times; Hank Gathers, Sergei Grinkov, and Florence Joyner are recent victims.

Corrado et al. (1) estimated the incidence of sudden death in athletes from 12 to 35 years of age to be 2.3 per 100,000 athletes/year, compared with 0.9 per 100,000 non-athletes/year in the Veneto region of Italy. Van Camp et al. (2) estimated the prevalence of sudden death in high school and college athletes in the U.S. to be 0.4 per 100,000 athletes per year. The majority of sudden deaths in athletes are associated with structural heart disease. Maron et al. (3) reviewed clinical and autopsy data from 158 young athletes (median age 17 years, range 12 to 40 years) who died suddenly. A cardiovascular cause of death was identified in 134 (85%). Structural heart disease was present in 129 athletes. Hypertrophic cardiomyopathy (36%) and anomalous coronary artery origin (11%) were the most common cardiac abnormalities identified. An additional 10% of victims had an unexplained increase in cardiac mass (possible hypertrophic cardiomyopathy), suggesting that the frequency of hypertrophic cardiomyopathy may be even greater. When structural heart disease is absent at autopsy, an abnormality of cardiac ion channels, such as the long-QT syndrome, or subtle structural abnormality, such as the Wolff-Parkinson-White (WPW) syndrome, are potential causes. The relative importance of causes of sudden death in athletes may vary in different populations. In the Veneto area of Italy, arrhythmogenic right ventricular (RV) cardiomyopathy is found in 22% of athletes who die suddenly; hypertrophic cardiomyopathy is found in 2% (4). The presence of heart disease often escapes detection in the asymptomatic young athlete. Of the 134 victims with cardiovascular cause of sudden death reported by Maron et al. (3), 115 had a pre-participation medical evaluation, only 4 were suspected of having cardiovascular disease, and the cardiovascular abnormality responsible for sudden death was correctly identified in only 1.

Recognition of heart disease, and particularly hypertrophic cardiomyopathy, in athletes can be particularly difficult. As a physiologic adaptation to exercise, the athlete’s heart enlarges, increasing in mass and left ventricular (LV) cavity dimension (5). In a small proportion of trained adult athletes, the magnitude of the wall thickness is comparable to that in mild morphologic expressions of hypertrophic cardiomyopathy. Between 1% and 2% of athletes have an LV wall thickness of 15 to 16 mm. Several studies have evaluated methods to differentiate between hypertrophic cardiomyopathy and physiologic hypertrophy of the athlete’s heart (6,7). A small LV cavity dimension, enlarged left atrial diameter, abnormal diastolic filling pattern, pathologic Q waves, and ST-T abnormalities on the electrocardiogram (ECG) favor hypertrophic cardiomyopathy, whereas a peak oxygen consumption >50 ml/kg/min or >20% suggests a training effect most likely to produce physiologic hypertrophy. Asymmetrical hypertrophy or failure of hypertrophy to diminish with the cessation of regular exercise suggests hypertrophic cardiomyopathy (5). Physiologic hypertrophy in the athlete’s heart is symmetric and reversible with deconditioning. Pelliccia et al. (8) observed that during long-term (1 to 13 years; mean 5.6 ± 3.8 years) deconditioning LV cavity dimension decreased by 7%, maximum wall thickness diminished by 15%, and ventricular mass (normalized to height) decreased by 28%. In all of these athletes, wall thickness returned to the normal, but 22% athletes showed persistent chamber dilation (≥60 mm).

Arrhythmias can be a marker for heart disease and generate appropriate concern when discovered in athletes. In a previous report by Biffi et al. (9), a total of 355 of 15,889 athletes had >3 premature ventricular contractions (PVCs) on 12-lead ECG (n = 337) or a history of palpitations (n = 18) and underwent further evaluation. Overall, only 7% of these athletes harbored structural cardiovascular abnormalities. Structural heart disease was 15 times more frequent in athletes with frequent and complex ventricular tachycardias (VTs) than in those with less severe arrhythmias. The 71 patients with ≥2,000 PVCs/24 h were restricted from participating in competitive sports. Over the eight-year follow-up period, the risk of sudden death proved to be exceedingly low. One patient who had arrhythmogenic RV dysplasia died suddenly while playing hockey against medical advice. No sudden cardiac deaths occurred in the athletes who had frequent and complex arrhythmias but no other cardiovascular abnormalities. Annual mortality was

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0.17%. Although these data suggest that some of these athletes may not require restriction from competitive athletics, whether the outcome would have been as favorable if they continued competitive athletics is not known.

Are frequent arrhythmias in and of themselves a sufficient reason to restrict physical activity? How can those that are a marker for heart disease and sudden death risk be distinguished from benign arrhythmias? In this issue of the Journal, Biffi et al. (10) extend their previous study. Specifically, they assess the impact of deconditioning on ventricular arrhythmias in their previously identified population of athletes and correlate those findings with presence of heart disease in a manner similar to that suggested to distinguish physiologic from pathologic hypertrophy in athletes. Of the 355 athletes with ≥3 PVCs on 12-lead ECG (n = 337) or history of palpitation (n = 18), 70 had ≥2,000 PVCs/24 h and/or ≥ burst of nonsustained VT on ambulatory ECG recording. The one patient who died suddenly while playing field hockey was excluded. The remaining 70 athletes underwent a deconditioning period of at least 3 months. After deconditioning, a second 24-h recording demonstrated a reduction in arrhythmia for the group overall. Total PVC frequency decreased by 80%, couplets decreased by 80%, and the number of nonsustained VT episodes decreased by 90%. Fifty athletes (71%) showed partial reduction of arrhythmia (PVCs decreased to <500/24 h and absence of nonsustained VT) or complete absence of ventricular arrhythmias.

Reversibility of arrhythmia with deconditioning did not, however, always indicate absence of structural heart disease. Although 74% of patients who had a decrease in ectopy had no cardiovascular abnormalities, heart disease was present in 26%, including mitral valve prolapse (n = 5), myocarditis (n = 3), dilated cardiomyopathy (n = 3), and arrhythmogenic RV cardiomyopathy. In the 29% of the athletes for whom no change of frequency was observed after deconditioning; 13 (65%) had no cardiovascular abnormalities; the remaining 35% had disease including arrhythmogenic RV cardiomyopathy (n = 4), mitral valve prolapse (n = 1), myocarditis (n = 1), or dilated cardiomyopathy (n = 1). The decrease in LV mass after detraining did not differ between athletes who had reversibility of arrhythmias compared to those with no change. Of 24 athletes who underwent electrophysiology study, only one had inducible sustained VT. During the 8 ± 4 years follow up, all 70 athletes survived without cardiac arrests. The 37 athletes with no structural heart disease and partial or complete reduction of ambient arrhythmia after deconditioning resumed competitive sports without restriction, as did six athletes who had cardiovascular abnormalities but who had a reduction in arrhythmias with detraining. No sudden deaths occurred in these individuals during the remaining follow-up time.

There are important caveats. Only two 24-h Holter recordings were obtained. Although the authors report that a control group composed of subjects with less frequent arrhythmia had similar severity of arrhythmia on two recordings, ambient ventricular ectopy often displays substantial day-to-day variability. Fluctuations of >60% in total PVCs are not uncommon (11). Detraining itself would presumably alter the subjects’ activity level during the two recording periods, because the workout during the second recording would presumably be less strenuous than before detraining. A decrease in frequency of ectopy may not, therefore, reflect changes in cardiac electrophysiologic properties. It is possible that those subjects who had a decrease in ectopy at the second study simply had greater spontaneous variability than those with similar severity of ectopy at both studies. The QRS morphology of the arrhythmia was not assessed and could be helpful in assessing presence of a structural abnormality. Idiopathic PVCs often originate from the RV outflow tract and have a characteristic left bundle branch block and inferior-axis QRS morphology. Arrhythmogenic RV cardiomyopathy can also, however, cause this morphology. Thus, it cannot be established with certainty whether the ventricular arrhythmias in athletes without heart disease were specifically related to a training effect, perhaps associated with hypertrophy, or simply benign idiopathic arrhythmia such as RV outflow tract ventricular ectopy (which can be encountered in sedentary individuals).

What is the approach to evaluation of athletes who are found to have ventricular ectopy? First, an underlying cardiac abnormality is relatively common, and careful evaluation is warranted with echocardiography and exercise stress testing. The ECG should be assessed for markers of WPW, Brugada, and long-QT syndromes, although these disorders are not usually associated with frequent ambient ectopy. Although not addressed in the present study, a family history of sudden death should be sought and, if present, would warrant more extensive evaluation for genetic disorders, potentially including electrophysiologic study. Patients with structural heart disease, in particular hypertrophic, dilated, or RV cardiomyopathies, should be restricted from competitive athletics, following current accepted guidelines (12). In the absence of structural heart disease or markers of these other arrhythmogenic syndromes, the risk of sudden death is likely quite low. A decrease in ventricular arrhythmias produced by detraining may further support the safety of allowing a return to athletics. It should be recognized, however, that a favorable decrease in arrhythmia frequency does not exclude the possibility of underlying heart disease and that substantial spontaneous variability in arrhythmia frequency is common. A careful cardiac evaluation and case-by-case assessment is required.

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