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Impact of Physical Deconditioning on Ventricular Tachyarrhythmias in Trained Athletes

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OBJECTIVES The purpose of this research was to evaluate the impact of athletic training and, in particular, physical deconditioning, on frequent and/or complex ventricular tachyarrhythmias assessed by 24-h ambulatory (Holter) electrocardiogram (ECG).

BACKGROUND Sudden deaths in athletes are usually mediated by ventricular tachyarrhythmias.

METHODS Twenty-four hour ambulatory ECGs were recorded at peak training and after a deconditioning period of 19 ± 6 weeks (range, 12 to 24 weeks) in a population of 70 trained athletes selected on the basis of frequent and/or complex ventricular tachyarrhythmias (i.e., ≥2,000 premature ventricular depolarization [PVD] and/or ≥1 burst of non-sustained ventricular tachycardia [NSVT]/24 h).

RESULTS A significant decrease in the frequency and complexity of ventricular arrhythmias was evident after deconditioning: PVDs/24 h: 10,611 ± 10,078 to 2,165 ± 4,877 (80% reduction; $p < 0.001$) and NSVT/24 h: 6 ± 22 to 0.5 ± 2, (90% reduction; $p = 0.04$). In 50 of the 70 athletes (71%), ventricular arrhythmias decreased substantially after detraining (to <500 PVDs/24 h and no NSVT). Most of these athletes with reduced arrhythmias did not have structural cardiovascular abnormalities (37 of 50; 74%). Over the 8 ± 4-year follow-up period, each of the 70 athletes survived without cardiac symptoms.

CONCLUSIONS Frequent and/or complex ventricular tachyarrhythmias in trained athletes (with and without cardiovascular abnormalities) are sensitive to brief periods of deconditioning. In athletes with heart disease, the resolution of such arrhythmias with detraining may represent a mechanism by which risk for sudden death is reduced. Conversely, in athletes without cardiovascular abnormalities, reduction in frequency of ventricular tachyarrhythmias and the absence of cardiac events in the follow-up support the benign clinical nature of these rhythm disturbances as another expression of athlete's heart. (J Am Coll Cardiol 2004;44:1053–8)

"Athlete's heart" is a complex physiologic and structural cardiac syndrome that develops in response to intensive and chronic athletic training. A growing body of literature has distinguished physiologic left ventricular (LV) remodeling with increased LV chamber size, wall thickness, and mass from the pathologic form of hypertrophy characteristic of cardiomyopathies (1–3). One of the parameters differentiating these two forms of LV hypertrophy is regression in cavity dimension and/or wall thickness, which is confined to "athlete's heart," typically occurring within weeks to months after cessation of training (4,5).

See page 1059

We have reported that frequent and/or complex ventricular arrhythmias occur not uncommonly in trained athletes with physiologic LV hypertrophy (6). However, at present, the potential impact of deconditioning on such ventricular arrhythmias in an athletic population is unresolved. Therefore, the primary aim of this study was to assess the relationship between physical deconditioning and the occurrence of ventricular tachyarrhythmias in our unique population of highly trained athletes.

METHODS

Study population. The case records of the Institute of Sports Science (Rome) from 1984 to 2001 were reviewed. During this time period, 355 athletes had been assessed by 24-h ambulatory (Holter) electrocardiogram (ECG), by virtue of meeting the following criteria: 1) ≥3 premature ventricular depolarizations (PVDs) on resting 12-lead ECG (n = 337), and/or 2) history of palpitations (n = 18). Of the 355 athletes, 71 with particularly frequent and/or complex ventricular arrhythmias (arbitrarily defined as ≥2,000 PVDs and/or ≥1 burst of non-sustained ventricular tachycardia [NSVT]/24 h) were initially considered for inclusion. One athlete (2,100 PVDs and 2 bursts of NSVT on 24-h Holter recording) with arrhythmogenic right ventricular cardiomyopathy (ARVC), who died suddenly while participating in a competitive field hockey game (against medical advice), was

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Abbreviations and Acronyms
ARVC = arrhythmogenic right ventricular cardiomyopathy
LV = left ventricle/ventricular
NSVT = non-sustained ventricular tachycardia
PVD = premature ventricular depolarization

excluded from the study group because he did not undergo detraining, and relevant Holter data were not obtained. Therefore, the final study group comprises 70 athletes with frequent and/or complex ventricular tachyarrhythmias on Holter ECG.

In the study subjects, 24-h Holter ECGs were initially recorded during periods of peak training, including a conditioning session (an average of 1 h in duration), similar to that usually performed by the athlete; the remaining time was occupied by usual daily activities that may have involved non-competitive and recreational physical activity.

At the time of Holter monitoring during peak training, no athlete was taking antiarrhythmic or other cardioactive medications. However, eight athletes (all with cardiovascular abnormalities) were taking medications at the time of the deconditioning Holter ECG, including beta-blockers (n = 5), propafenone (n = 2), or enalapril (n = 1).

Mean age of the athletes was 25 ± 12 years (range, 15 to 33 years); 51 subjects (72%) were male. These athletes were engaged in a variety of sports disciplines, most commonly soccer (n = 15; 21%), basketball (n = 10; 14%), and volleyball (n = 7; 10%). They also presented a broad spectrum of athletic achievement with 25 (35%) participating at an elite level, including 18 competing in the Olympic Games or World Championships (five finalists or medalists).

Control group. A total of 148 athletes without structural heart disease, of similar age (26 ± 10 years) and gender (78% males) as the study population, with less frequent ventricular arrhythmias (≥100 to <2,000 PVDs/24 h and no episode of NSVT) were assembled as a control group. The 148 controls underwent a second Holter ECG 3 to 6 months after the first study, and at the same level of training (without deconditioning). The time period between these two Holter recordings obtained during training (19 ± 4 weeks; range, 12 to 24 weeks) was the same as between the active and deconditioned phases in the 70 athletes with frequent and/or complex ventricular tachyarrhythmias.

Echocardiography. Echocardiographic studies were performed using Hewlett-Packard 77020 AC or Sonos 5500 (Andover, Massachusetts). Images of the heart were obtained in multiple cross-sectional planes using standard transducer positions. The LV cavity dimensions, anterior ventricular septal and posterior free wall thicknesses, and left atrial dimension were obtained from M-mode echocardiograms in accordance with previous recommendations (7). To enhance the accuracy of LV wall thickness measurements, these dimensions were verified from two-dimensional images (1); LV ejection fraction was measured from end-diastolic and end-systolic volumes in the apical four-chamber view.

Left ventricular mass was calculated by the formula of Devereux (8) and normalized to body surface area. Parameters of LV filling were obtained with pulsed Doppler echocardiography (9).

Diagnostic criteria. ARVC. Echocardiographic criteria used for the clinical diagnosis of ARVC included right ventricular cavity dilation and/or segmental thinning, bulging or aneurysm formation, and wall motion abnormalities, as suggested by the Task Force of the European Society of Cardiology and Federation of Cardiology (10).

Myocarditis. Diagnosis of myocarditis was based on laboratory evidence of an inflammatory condition involving myocardium, associated either with segmental LV wall motion abnormalities and cavity enlargement, and confirmed in selected cases (n = 4) by biopsy showing histopathology in accord with the Dallas criteria (11).

Dilated cardiomyopathy. Dilated cardiomyopathy was diagnosed based on marked LV cavity dilation (end-diastolic dimension ≥60 mm) associated with systolic LV dysfunction (ejection fraction <50%) and/or segmental wall motion abnormalities, and differentiated from physiologic cavity enlargement as previously reported (3,12).

Mitral valve prolapse. Mitral valve prolapse was identified by evidence of elongated, thickened, and redundant leaflets billowing beyond the mitral annulus plane during systole, in the parasternal long-axis echocardiographic view (13).

Deconditioning. Based on the Italian guidelines for determining eligibility in competitive athletes with cardiovascular abnormalities (14) (which closely resemble those of the 26th Bethesda Conference) (15), each of the 70 athletes with frequent and/or complex ventricular tachyarrhythmias was disqualified from competitive sports.

The 70 athletes underwent a deconditioning period of at least three consecutive months (mean, 19 ± 6 weeks; range, 12 to 24 weeks). This time period was selected because it has been shown to be sufficient to reverse the cardiac remodeling induced by physical training (5). After deconditioning, each athlete had a second cardiovascular assessment, which also included a 24-h Holter ECG performed under the same conditions as at peak training, except for eight athletes who were receiving pharmacologic treatment with propafenone, sotalol, or enalapril at the time of the most recent Holter. This second 24-h Holter ECG also included a conditioning session similar to that usually performed by the athlete (of about 1 h in duration).

Ventricular tachyarrhythmias were regarded as having partial reversibility when PVDs decreased to <500 PVDs/24 h (in the absence of NSVT) on the second Holter ECG. Arrhythmias were considered to show complete reversibility when PVDs and NSVT decreased to 0/24 h.

Electrophysiologic study. Of these 70 athletes, 24 were selected on clinical and Holter indications for electrophysiologic study: ≥10,000 PVDs/24 h (n = 10), ≥10,000

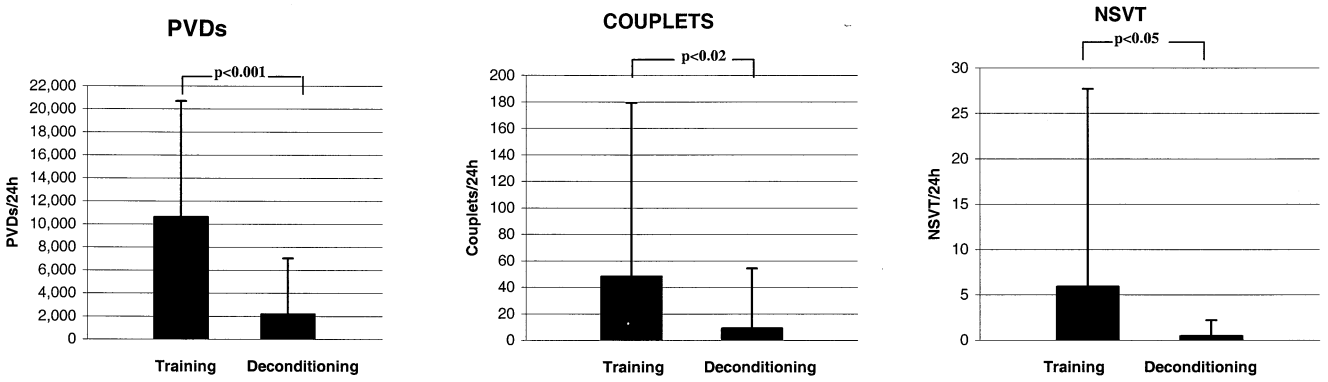

Figure 1. Number of premature ventricular depolarizations (PVD), ventricular couplets, and bursts of non-sustained ventricular tachycardia (NSVT) during 24-h Holter electrocardiogram recording at peak training and after the period of deconditioning in 70 trained athletes.

PVDs and NSVT (n = 9), or ≥2,000 PVDs and NSVT (n = 5). Ventricular stimulation was performed using a programmable stimulator (Medtronic 5328, Medtronic Inc., Minneapolis, Minnesota), delivering rectangular pulses of 2 ms duration at twice the diastolic threshold. Up to three extrastimuli were introduced after eight ventricular paced beats at three drive cycle lengths (600, 500, and 400 ms) and in two right ventricular sites (apex and outflow tract). Programmed stimulation during isoproterenol infusion was subsequently performed in athletes who otherwise were not inducible.

Statistics. Data are expressed as mean ± SD. Differences between means were assessed by unpaired or paired Student *t* test, where appropriate. A two-tailed p value of <0.05 was considered statistically significant.

RESULTS

Ventricular tachyarrhythmias at peak conditioning and after deconditioning. PEAK CONDITIONING. Frequency of PVDs on 24-h Holter ECG performed during the period of peak training and competition ranged from 2,089 to 43,151 (mean, 10,611 ± 10,078), including 21 athletes (34%) with ≥10,000 PVDs. Each of the 70 athletes had ≥1 couplet (mean, 48 ± 131; range, 1 to 280); 37 athletes (53%) also had 1 to 179 bursts of NSVT (mean, 6) consisting of 3 to 28 consecutive beats at heart rates of 130 to 270 beats/min. Of the 70 athletes, 8 (11%) reported frequent or prolonged palpitations (each with >10,000 PVDs or NSVT), but none had episodes of impaired consciousness or other cardiac symptoms.

DECONDITIONING. After the deconditioning period, the overall study group showed significant reduction in PVDs, couplets, and NSVT; PVDs decreased from 10,611 ± 10,078 to 2,165 ± 4,877 (80% reduction; p < 0.001); couplets from 48 ± 131 to 9 ± 45 (80% reduction; p < 0.02); and NSVT from 6 ± 22 to 0.5 ± 2 (90% reduction; p = 0.038) (Fig. 1).

Individual subject analysis showed that after deconditioning, 50 of 70 athletes (71%) showed partial or complete reversibility of ventricular arrhythmias. Partial reversal of ventricular tachyarrhythmias (i.e., to <500 PVDs and absence of NSVT/24 h) was evident in 34 athletes; complete abolition of arrhythmias (no PVDs and NSVT/24 h) occurred in 16 athletes (Figs. 2 and 3). In the remaining 20 athletes (29%), frequency of ventricular arrhythmias showed no significant reduction in frequency after detraining (i.e., persistence of ≥500 PVDs/24 h), including 6 in whom runs of NSVT persisted. No athlete showed an increase of ventricular arrhythmias after deconditioning.

Ventricular tachyarrhythmias in the control group. Control group athletes did not show significant variability in ventricular arrhythmias between the two Holter ECGs obtained during training (19 ± 4 weeks interval training period). For example, mean number of PVDs on the first Holter was 1,211 ± 850 and on the second was 1,050 ± 648 (p = NS) (Table 1).

Relation of change in ventricular tachyarrhythmias with deconditioning to cardiovascular abnormalities. Of the 50 athletes who showed reversibility of ventricular arrhythmias after deconditioning, most (37; 74%) had no cardiovascular abnormalities, and 13 (26%) had cardiac abnormalities, including mitral valve prolapse (n = 5), myocarditis (n = 3), dilated cardiomyopathy (n = 3), and ARVC (n = 2).

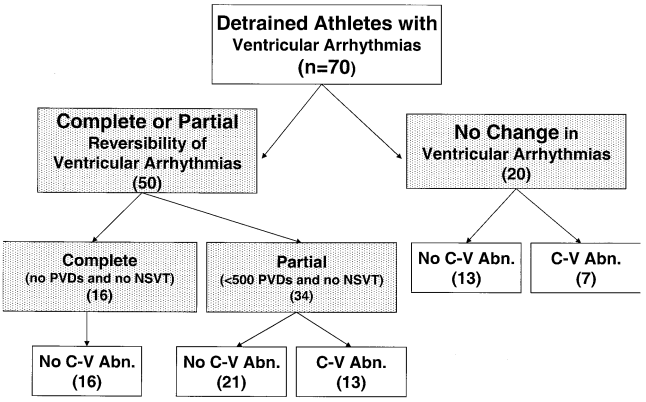

Figure 2. Effect of deconditioning on frequent and/or complex ventricular tachyarrhythmias in 70 trained athletes. C-V abn. = cardiovascular abnormalities; NSVT = non-sustained ventricular tachycardia; PVDs = premature ventricular depolarizations.

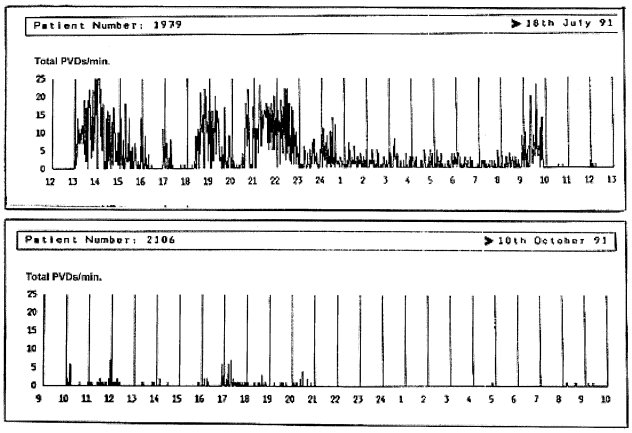

Figure 3. Arrhythmia trends during 24-h Holter electrocardiogram (ECG) recordings showing marked reversibility of premature ventricular depolarizations (PVDs) after three months of deconditioning in a 32-year-old elite bobsledder. **(Top panel)** The 24-h ECG performed during peak training shows 3,288 PVDs, distributed relatively homogeneously, with a slight reduction evident during the evening hours. **(Bottom panel)** Marked reduction in ventricular arrhythmias (to 73 PVDs) is evident after three months of physical deconditioning. The reduction of ventricular arrhythmia after detraining has occurred throughout the 24-h recording period.

In particular, each of the 16 athletes who showed complete reversibility of ventricular arrhythmias after detraining had no cardiovascular abnormalities.

In the remaining 20 athletes for whom ventricular arrhythmias remained substantially unchanged after detraining, 13 had no cardiovascular abnormalities (65%), and 7 (35%) had either ARVC (n = 4), mitral valve prolapse (n = 1), myocarditis (n = 1), or dilated cardiomyopathy (n = 1) (Fig. 2). Therefore, the absence of structural heart disease was similar in athletes with or without reversibility of ventricular arrhythmias after deconditioning (37/50; 74% vs. 13/20; 65%; p = NS).

Relation of change in ventricular tachyarrhythmias with deconditioning to LV mass. During peak training, LV mass index was 115 ± 24 g/m^2 (range, 77 to 170 g/m^2) and after deconditioning decreased to 93 ± 20 g/m^2 (range, 53 to 140 g/m^2). The decrease in LV mass after detraining did not differ between athletes who experienced partial or complete reversibility of ventricular arrhythmias with deconditioning and athletes with no change in arrhythmias (116 ± 22 g/m^2 to 94 ± 18 g/m^2; 19% reduction vs. 114 ± 22 g/m^2 to 93 ± 26 g/m^2; 18% reduction; p = NS).

Table 1. 24-H Holter Monitoring Electrocardiographic Recordings in 148 Control Group Athletes

	First 24-H Holter (Training)	Second 24-H Holter (Training)	P Value
PVDs (range)	100–1,890	88–1,750	NS
PVDs (mean)	1,211 ± 850	1,050 ± 648	NS
No. of athletes with couplet	8 (5%)	6 (4%)	NS
No. of athletes with NSVT	0	0	NS

NSVT = non-sustained ventricular tachycardia; PVD = premature ventricular depolarization.

Relation of change in ventricular tachyarrhythmias to electrophysiologic findings. Of the 24 athletes who underwent electrophysiologic study, 23 (10 with and 13 without cardiovascular abnormalities) showed no ventricular arrhythmias or only non-sustained runs of ventricular tachycardia during programmed ventricular stimulation; most of these 23 athletes (17; 74%) had shown reduced arrhythmia frequency after deconditioning (i.e., 4,376 ± 754 to 336 ± 112 PVDs/24 h).

Only one athlete, a 32-year-old cyclist with ARVC and 12,000 PVDs and 5 NSVT bursts on Holter ECG, had induced sustained ventricular tachycardia (by two extrastimuli); oral administration of sotalol reduced ventricular arrhythmias (to <500 PVDs and absence of NSVT).

Follow-up. The 70 study athletes and the 148 controls were periodically examined at our institute over 8 ± 4 years after identification of ventricular tachyarrhythmias. Over this follow-up period, each of the 70 athletes and each of the 148 controls survived without experiencing cardiac symptoms or events. The 37 of the 70 athletes with partial or complete reversibility of the ventricular tachyarrhythmias after deconditioning (and without cardiovascular abnormalities) resumed competitive sports without restriction. In addition, six athletes with cardiovascular abnormalities (three with mitral valve prolapse and without significant regurgitation, and three with healed myocarditis), who had shown partial and substantial reduction of ventricular arrhythmias after deconditioning, were also allowed to resume competitive activity.

The remaining 27 of the 70 athletes were permanently disqualified because of structural cardiovascular diseases such as dilated cardiomyopathy or ARVC, and/or frequent, and/or complex ventricular arrhythmias, which were not reversible after deconditioning (either in the presence or absence of cardiovascular abnormalities).

Pharmacologic treatment. Pharmacologic treatment, with beta-blocker or propafenone at the time of the first Holter ECG, did not influence reversibility of ventricular tachyarrhythmias with deconditioning. The proportion of athletes with partial or complete reversal of ventricular arrhythmias was similar in those taking cardioactive drugs (5 of 8; 63%) as in those without medications (42 of 62; 70%; p = NS).

DISCUSSION

We have previously shown that intense athletic conditioning may be associated with the occurrence of frequent and/or complex ventricular tachyarrhythmias on ambulatory (Holter) ECG (6). These observations are extended in the present study where we demonstrate that detraining can reverse this process, whether or not structural cardiovascular abnormalities are present. Indeed, frequent and/or complex ventricular tachyarrhythmias in 70 highly trained and elite athletes were particularly sensitive to short periods of deconditioning (19 weeks on average), including complete

reversibility in about one-fourth and partial reversibility in almost one-half.

The mechanisms that explain reduction in ventricular tachyarrhythmias with deconditioning are probably complex, but likely are related to autonomic nervous system changes associated with high-intensity training and detraining. Intensive endurance training has been shown to shift autonomic modulation from parasympathetic to sympathetic predominance (16), which may predispose to an electrical instability of the ventricles and eventually trigger ventricular tachyarrhythmias (17). Alternatively, sinus bradycardia, characteristic of athlete's heart (with lengthening of the R-R interval) could facilitate the emergence of PVDs (18,19). The increase in cardiac mass induced by training is an unlikely explanation for the ventricular tachyarrhythmias in our athletes, given the mild degree of cardiac remodeling present in our study group, and the observation that decreased cardiac dimensions after deconditioning was similar in athletes with and without reversible ventricular arrhythmias. The possibility of significant spontaneous variability of ventricular arrhythmias in our study group is unlikely, given the lack of arrhythmia variation documented in the control group of trained athletes.

The removal of athletes with cardiovascular disease (such as hypertrophic cardiomyopathy) from intense training and competition has been promoted as a strategy to reduce the risk for sudden death (14,15,20,21). Recently, Corrado et al. (22) showed that the risk for sudden death in young competitive athletes with cardiovascular disease was 2.5-fold greater than in non-athletes. These data suggest that sports activity itself may act as a trigger for life-threatening ventricular tachyarrhythmias during intense physical exertion in susceptible individuals with silent cardiovascular diseases, thereby predisposing to cardiac arrest. Based on our present data, in which no athlete (with or without cardiovascular abnormalities) experienced a clinical event or sudden death during follow-up after deconditioning, we propose that such a favorable outcome may be related to the reduction in ventricular arrhythmias associated with detraining. Therefore, the deconditioning effect on arrhythmias is a potential mechanism by which disqualification from intense competitive sports may reduce the risk for sudden cardiac death in those athletes with structural heart disease and frequent and/or complex ventricular arrhythmias. Our data support, therefore, the restriction from competitive sports in athletes with frequent and/or complex ventricular tachyarrhythmias and structural heart disease, as suggested by the present recommendations (14,15,20). Conversely, this study also identified a subset of athletes without cardiovascular abnormalities, and with reversibility of ventricular arrhythmias, who resumed training and competition and experienced a benign course. Therefore, resolution of ventricular tachyarrhythmias with deconditioning may justify resumption of competition without risk in these athletes without heart disease.

However, some caution is suggested in interpreting our data in the context of certain clinical circumstances. For example, we cannot exclude the possibility that in an occasional athlete the observed reduction in ventricular arrhythmias was due to the resolution of previously unrecognized structural heart disease (such as myocarditis), rather than to a deconditioning effect. Nevertheless, given the large number of trained athletes with ventricular tachyarrhythmias in our cohort (and the rarity of cardiovascular abnormalities in a young athletic population) (23), this explanation seems very unlikely for the vast majority of athletes in the present analysis.

Recommendations for the eligibility of athletes without cardiovascular disease or abnormalities, but with frequent and/or complex ventricular tachyarrhythmias on ambulatory Holter ECG, are presently unresolved and have not yet been definitively addressed in formal expert consensus panels such as the Bethesda Conference (15) or the Italian guidelines (14). However, it is our current practice to initially withdraw such athletes from all training and competition for three to six months and then reevaluate with ambulatory Holter ECG monitoring for the presence of these arrhythmias. If ventricular tachyarrhythmias are greatly reduced in frequency or abolished at the end of the detraining period, then competitive sports participation can be resumed. Close follow-up of athletes with frequent and/or complex ventricular arrhythmias is recommended for the assessment of new symptoms, and/or to detect worsening of arrhythmias, or the possible expression of a previously undiagnosed cardiovascular abnormality with late clinical onset (24). On the other hand, no limitation in sports activity is recommended for athletes without cardiovascular abnormalities and less frequent arrhythmias (such as in the 148 control group athletes), due to their favorable prognosis and the observation that sports training and competition were not associated with an increase in arrhythmia frequency.

In those athletes with underlying cardiovascular disease or abnormalities, and with frequent and/or complex ventricular tachyarrhythmias, permanent (or temporary in selected cases) disqualification from most competitive sports is indicated. These guidelines are based primarily on recommendations for the athlete's particular structural heart disease (i.e., hypertrophic cardiomyopathy, ARVC, dilated cardiomyopathy, or myocarditis) (23). With particular regard to mitral valve prolapse, exertion-related sudden cardiac death is a known, albeit uncommon, consequence, particularly in those athletes with frequent and/or complex ventricular arrhythmias (23). Therefore, it is prudent to disqualify from competitive sports athletes with mitral valve prolapse associated with frequent and/or complex ventricular arrhythmias (15,23).

In conclusion, frequent and/or complex ventricular tachyarrhythmias in highly trained athletes are sensitive to short periods of deconditioning. This reversibility of arrhythmias after deconditioning was observed both in athletes with and without cardiovascular abnormalities. In athletes with heart disease, resolution of arrhythmias after detraining could

explain the mechanism by which the restriction of these athletes from intense sports competition may reduce their risk for sudden death. Conversely, in athletes without cardiovascular abnormalities, the reversibility of ventricular tachyarrhythmias (and the absence of cardiac events in the follow-up period) support the benign clinical nature of these arrhythmias as another expression of athlete's heart.

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