Premature ventricular beats in athletes: to detrain or not to detrain?

Alessandro Biffi,[1] Stefano Palermi ,[1,2] Flavio D'Ascenzi ,[3] Marco Bonifazi,[4] Alessandro Zorzi ,[5] Domenico Corrado[5]

Long-term physical activity induces structural, functional and electrical changes in the heart, reflecting adaptive physiological responses to the increased cardiac output during exercise.[1] These effects are usually reversible after short detraining periods. Differentiating between exercise-induced cardiac changes and early signs of cardiac pathology often poses a clinical challenge, especially in so-called 'grey zone' (borderline) cases.[1] This editorial presents the rationale and considerations for a period of detraining in the diagnostic evaluation of athletes with premature ventricular beats (PVBs).

ULTRASTRUCTURAL CHANGES RELATED TO PHYSICAL ACTIVITY AND VENTRICULAR ARRHYTHMOGENESIS

Evidence suggests that intense physical exercise and training may increase the propensity to develop arrhythmias.[2] Studies have shown that endurance athletes may exhibit a higher incidence of bradyarrhythmias and atrial fibrillation, while the association between physical training and ventricular arrhythmias remains debated.[2]

The mechanisms linking the athlete's heart to arrhythmias, which range from structural to electrical remodelling (increased vagal activity at rest and increased sympathetic stimulation during exercise) are not fully understood. The continuous stretching of cardiac muscle cells during exercise could trigger arrhythmias in athletes. These cells possess mechanically gated ion channels, some of which function as stretch-activated channels. These stretch-activated channels increase the likelihood of opening and the passage of ions following the deformation of the cell membrane due to muscle stretch. Stretch could affect trans-sarcolemmal Na+/Ca++ exchange, triggering ectopic excitation and inducing atrial and ventricular arrhythmias.[3] Acute modulating factors such as haemodynamic stress, electrolytic changes and body temperature, as well as genetic predisposition and the environment, may also contribute to the arrhythmogenic phenomenon in athletes.

During detraining, a progressive decrease in pressure and volume loads and consequent myocardial tissue remodelling might reduce the mechanical stretch. This could normalise electrophysiological and electroanatomic substrates, thereby reducing arrhythmias in athletes due to stretch-activated channels. Even if the exact mechanism is still unknown, the hypothesis is that some individuals are predisposed to react to the increased mechanical myocardial cell stretch induced by exercise training with an increased intracellular release of calcium, which may explain the development of some arrhythmias. Detraining decreases the mechanical stretch by shortening the action potential duration and normalising ventricular repolarisation.[4]

DETRAINING AND PVBS: A COMPLEX RELATIONSHIP

PVBs are frequently found in competitive athletes without cardiovascular abnormalities during preparticipation screening.[5] More than one PVB on a resting ECG or any PVBs with high-risk features require a first-line clinical workup (including medical history, physical examination, maximal exercise stress testing, Holter ECG monitoring and echocardiography) to rule out the presence of underlying cardiovascular disease.[6 7] However, selected cases of athletes with a 'high-arrhythmic risk' profile (box 1) should undergo further imaging or electrophysiological tests to exclude otherwise concealed pathological substrates.

The role of detraining in the evaluation of athletes with PVBs remains a topic of debate, and the scientific literature exploring this topic is limited.

Box 1 High-risk clinical and ventricular arrhythmia features in competitive athletes

1. Family history of SCD or structural CV disease at a young age.
2. Exercise-induced presyncope/syncope or chest pain.
3. Coexistence of pathological ECG abnormalities.[7]
4. Ventricular arrhythmia/PVB morphology: RBBB pattern with wide QRS; LBBB pattern with intermediate or superior axis; multiple morphologies (multifocal PVBs or polymorphic VT).
5. Ventricular arrhythmia complexity: couplets with short interectopic interval; NSVT or SVT; short coupling interval ('R on T').
6. Ventricular arrhythmia/PVB response to exercise: persistence or increase in the number of PVBs and/or the complexity of the ventricular arrhythmia during exercise.
7. Reproducibility of exercise-induced ventricular arrhythmia/PVBs on serial EST or ECG Holter.

CV, cardiovascular; EST, exercise stress testing; LBBB, left bundle branch block; NSVT, non-sustained ventricular tachycardia (≥3 consecutive beats); PVBs, premature ventricular beats; RBBB, right bundle branch block; SCD, sudden cardiac death; SVT, sustained ventricular tachycardia (duration >30 s).

In athletes with PVBs who underwent detraining, the arrhythmic burden has generally been shown to decrease, and retraining is associated with a new, but more limited, increase in the number of PVBs, suggesting a benign variability of the arrhythmic phenomenon over time.[8] The reduction of PVBs with detraining also appears to be greater in athletes with more frequent and complex PVBs at baseline.[9] This variability in the occurrence of PVBs with training/detraining should be considered a common finding in competitive athletes and could be interpreted as a benign phenomenon.[6]

DETRAINING AND THE DIAGNOSTIC WORKUP IN ATHLETES WITH PVBS

A controversial question is whether the response to detraining will have a role in the clinical evaluation and management of athletes with PVBs.

Given the potential negative impacts that detraining may have on athlete performance, compromising their training

[1]Med-Ex, Medicine and Exercise srl, Medical Partner Scuderia Ferrari, Rome, Italy
[2]Public Health Department, University of Naples Federico II, Sulmona, Italy
[3]Department of Medical Biotechnologies, University of Siena, Siena, Italy
[4]Department of Medicine, Surgery, and NeuroScience, University of Siena, Siena, Italy
[5]Cardiac, Thoracic and Vascular Sciences, University of Padova, Padova, Italy

Correspondence to Dr Alessandro Biffi, Med-Ex, Medicine & Exercise, Medical Partner Scuderia Ferrari, Rome, Italy; a.biffi@libero.it

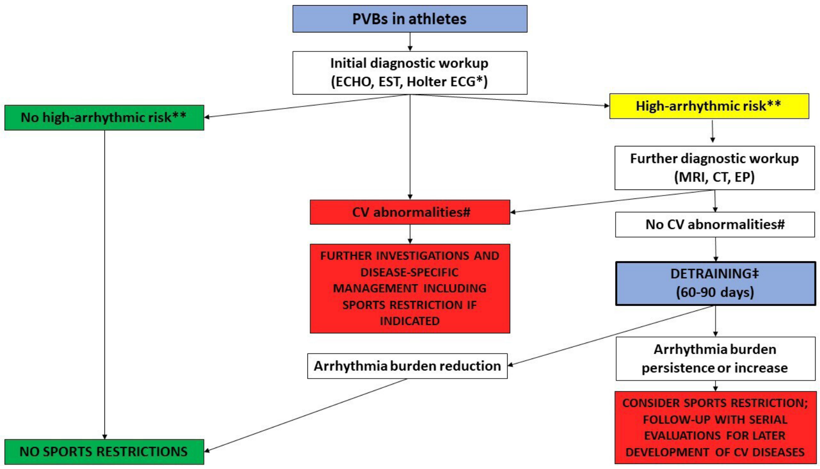

Figure 1 Clinical workup of athletes with premature ventricular beats: potential role of detraining‡. *24-hour 12-lead Holter ECG monitoring, including at least one exercise training session. **'High-arrhythmic risk' is defined based on the clinical and ventricular arrhythmia features detailed in box 1. ‡Detraining is defined as the complete cessation of all kinds of athletic activities. #CV abnormalities are defined as morphofunctional and/or structural heart abnormalities; coronary artery disease; inducibility of ventricular tachycardia or fibrillation. CV, cardiovascular; EP, electrophysiological study; ECHO, echocardiography; EST, exercise stress testing; PPS, preparticipation screening; PVBs, premature ventricular beats.

programme and jeopardising selection or participation with a team, and considering that detraining is not the gold standard in the management of athletes with PVBs, it is essential to be cautious when recommending this approach. The decision should weigh the potential additional diagnostic value of deconditioning against its detrimental consequences for the athlete, particularly for elite and professional athletes, especially in the era of advanced imaging techniques which can help distinguish a 'normal' heart from underlying structural heart disease.[1]

We postulate that detraining can have an important role in the cardiac evaluation of selected athletes with a 'high-arrhythmic risk' profile when clinical evaluations do not show cardiovascular abnormalities (figure 1). Demonstration of a reduction of the ventricular arrhythmia/PVB burden after detraining suggests the potential arrhythmogenic role of neuroautonomic remodelling in the athlete's heart that characteristically reverses with deconditioning, and thus indicates a benign nature of the arrhythmia/PVB and supports the safe return to competitive sports. If detraining does not lead to a reduction in the ventricular arrhythmia/PVB burden, more cautious decision-making for competitive sports eligibility should be considered, and repeat evaluation is indicated to exclude that ventricular arrhythmias/PVBs may herald the later development of an identifiable cardiovascular disease.

CONCLUSIONS

Physical deconditioning and detraining can modulate ventricular arrhythmogenicity of the athlete's heart. This effect is the expression of multiple mechanisms including the level of training and myocardial remodelling (through ion channel regulation), the imbalance of autonomic nervous system changes, and, rarely, the early manifestations of structural or genetic heart disease. We propose that the prescription of detraining in competitive athletes with PVBs may be useful in the diagnostic workup of selected cases with 'high-arrhythmic risk' in the absence of an identified cardiovascular disease.

Twitter Stefano Palermi @stefanopalermi

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ORCID iDs
Stefano Palermi http://orcid.org/0000-0003-1558-4857
Flavio D'Ascenzi http://orcid.org/0000-0002-0947-6836
Alessandro Zorzi http://orcid.org/0000-0002-3578-0583

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