

Push harder to allow pushing harder

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**Editorial on “Ventricular Arrhythmias in Athletes:
Role of a Comprehensive Diagnostic Workup” by A. Dello Russo et al.**

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International sports eligibility recommendations, both in Europe and in North America, state that athletes with ventricular arrhythmias (VA), like premature beats (VPB) or nonsustained VT (nsVT) can participate in all forms of competitive sports if they have a structurally normal heart and no evidence of molecular/genetic or inflammatory disorders.¹⁻³ This statement is clear, but the problem lies in its implementation: when can we decide that there is absence of structural abnormalities, channelopathy, genetic cardiomyopathy or inflammatory disease? Moreover, frequent VPB can be a marker of underlying disease.^{4, 5} Hence, many sports physicians and cardiologists struggle with the recommendation, wondering how extensive an evaluation of an athlete presenting with VPB or other VA should be before a definitive conclusion can be made. It is evident that a more thorough investigation has a higher likelihood to detect any underlying causative mechanism, but this needs to be balanced against the load of investigations for the athlete personally and society at large. This does not only have financial implications, but can also induce increased anxiety and accidental findings that may lead to unnecessary sports termination or therapeutic decisions.

In this issue of the Journal, a group of Italian colleagues describes its eligibility evaluation of a large series of 227 athletes (77.5% of whom were competitive, most athletics and soccer) presenting with VA.⁶ They started with noninvasive evaluation including cardiac MRI (CMR), followed in 188 (83%) by electro-anatomical mapping (EAM) of the right ventricle (and often also of the left ventricle), and even endomyocardial biopsy (EMB) in 42 (15%). Their results make a clear case for these additional investigations: the proportion of athletes with underlying disease increased by 50% with full invasive evaluation, i.e. from 30% to 45%. Interestingly, the former number of 30% is generally cited as the proportion of underlying heart disease in athletes presenting with frequent VPB.⁴ The majority of athletes in this study had at most nsVT, and half of the full cohort was asymptomatic. Whether the high proportion of 45% with underlying disease is related to referral bias, or representative for athletes in general, requires further study. Moreover, in athletes with abnormalities after noninvasive evaluation but without a definitive causative diagnosis, further invasive investigations led to conclusive diagnosis in 87%. Undeniably, the findings indicate that extensive evaluation of athletes presenting with VPBs is required before eligibility can be granted.

The authors have to be congratulated for this large study in a field that is lacking prospective trials that might underpin the international eligibility recommendations. Unfortunately, as is often the case, this study opens more questions than it provides answers. Although the authors stated that they followed a pre-specified protocol, many of the investigative trajectories seem to have been tailored to individual cases by factors that are not always explicit. Some non-definitive noninvasive evaluations were not followed up with invasive testing, and a number of idiopathic VA were further investigated invasively. This does not negate the value of the study, but illustrates the complexity of evaluation, which entails a specialist cardiological and arrhythmological approach.

The study does not give answers on what would be the optimal evaluation pathway for athletes presenting with VA. Unfortunately, genotyping was only considered at the very end of the diagnostic pathway. In many centres now, next generation sequencing is included at an early stage of

evaluation. Results may be available within a couple of weeks, acceptable for athletes before a final decision to continue or modify sports participation is due. Of course, broad genotyping requires well established cardiogenetic expertise, that looks at the evidence for causality of any genetic variation (using a class 1 to 5 categorisation) and its prognostic significance, mandatory for well-founded eligibility decisions. Such approach certainly is not available everywhere yet, but one may wonder whether for important decisions on (dis)continuation of competitive sports in athletes, referral to a regional or national competence centre should not be provided at an earlier stage in the workup. Cardiogenetic workup might even preclude tests like EAM or EMD in a subset of athletes.

Interestingly, 57 athletes considered to have 'idiopathic' VA after noninvasive evaluation were all still considered 'idiopathic' after invasive diagnostic evaluation. The authors do not describe extensively how they defined idiopathic ventricular arrhythmias after initial evaluation, but presumably this was based on the absence of scar on CMR and a typical morphology of idiopathic PVCs.² This illustrates the importance of such noninvasive evaluation which can prevent further invasive tests. The fact that a number of athletes denied invasive evaluation, indicates some reluctance for invasive testing even in this group that benefits from a conclusive diagnosis. One can question whether patients with other definitive diagnoses after initial evaluation (like DCM, MVP, definite ARVC, myocarditis) needed further invasive evaluation. The presence of scar was significantly related to non-eligibility in multivariate analysis in this series, as could be anticipated.⁶ Moreover, the sensitivity of EAM was rather low, as illustrated by the fact that a number of athletes with abnormalities on CMR (functional or fibrosis) had normal EAM. EAM was abnormal in only 24% of athletes.

The authors restricted EMB to athletes presenting with unexplained aborted SCD, with myocarditis, or with presumed ARVC, which seems a rational approach. The findings of EMB illustrate the underestimated prevalence of myocarditis in athletes with VA or SCD. But again, although the authors argue that they diagnosed some ARVC based on EMB (while non-invasive evaluation had suggested myocarditis or indicated non-ischaemic LV scar), I would have preferred to see that diagnosis further corroborated by genetic workup.

The study provides information on the 6-month eligibility outcome of the athletes, but it is not clear whether there was a systematic pathway to guide return to sports. In many athletes, resumption of sports at submaximal levels of intensity is decided in a first stage, including close monitoring with Holter, exercise testing, or even implantable loop recorder. During such follow-up, a progressive relaxation of restrictions is often possible, allowing safe resumption of competitive activities later on. But also sometimes the reverse is true..

Of course, the study also cannot answer the question as to whether the intensive evaluation had prognostic impact: the study has no control arm and no long-term follow-up. That issue remains one of the Achilles' heels of sports cardiological evaluation. Recent evidence indicates that in athletes with genetic conditions (like hypertrophic cardiomyopathy or long-QT syndrome) we may have to evolve into more diversified (and often more relaxed) recommendations.⁷

The field of sports eligibility clearly is a moving target. The good news is that after full evaluation, treatment, and follow-up, 49% of the athletes with VA in Dello Russo' series could return to competitive sports, and 22% more to leisure time sports. The authors should be commended for providing important data in that journey: they pushed hard diagnostically to allow pushing hard by the athletes!

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