High prevalence of right ventricular involvement in endurance athletes with ventricular arrhythmias
Role of an electrophysiologic study in risk stratification

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Background Electrocardiographic abnormalities and premature ventricular contractions are common in athletes and are generally benign. However, the specific outcome of high-level endurance athletes with frequent and complex ventricular arrhythmias is unclear. Also, information on the predictive accuracy of different investigations in this subgroup is unknown.

Results We report on 46 high-level endurance athletes with ventricular arrhythmias (45 male; median age 31 years) followed-up for a median of 4.7 years. Eighty percent were cyclists. Hypertrophic cardiomyopathy or coronary abnormalities were present in ≤5%. Eighty percent of the arrhythmias had a left bundle branch morphology. Right ventricular (RV) arrhythmogenic involvement (based on a combination of multiple criteria) was manifest in 59% of the athletes, and suggestive in another 30%. Eighteen athletes developed a major arrhythmic event (sudden death in nine, all cyclists). They were significantly younger than those without event (median 23 years vs 38 years; \( P = 0.01 \)). Outcome could not be predicted by presenting symptoms, non-invasive arrhythmia evaluation or morphological findings at baseline. Only the induction of sustained ventricular tachycardia (VT) or ventricular fibrillation (VF) during invasive electrophysiological testing was significantly related to outcome (RR 3.4; \( P = 0.02 \)). Focal arrhythmias were associated with a better prognosis than those due to reentry (\( P = 0.02 \)) but the mechanism could be determined in only 22 (48%).

Conclusions Complex ventricular arrhythmias do not necessarily represent a benign finding in endurance athletes. An electrophysiological study is indicated for risk evaluation, both by defining inducibility and identifying the arrhythmogenic mechanism. Endurance athletes with arrhythmias have a high prevalence of right ventricular structural and/or arrhythmic involvement. Endurance sports seems to be related to the development and/or progression of the underlying arrhythmogenic substrate.

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KEYWORDS
Athlete; Sports; Arrhythmia; Sudden death
Introduction

Sudden death in competitive athletes is most often due to underlying structural heart disease. Hypertrophic cardiomyopathy (HCM) and coronary anomalies are the most common findings in athletes younger than 35 years, while ischaemic heart disease is the predominant cause above 35 years of age. Other authors pointed to underlying arrhythmogenen right ventricular dysplasia (ARVD), present in 6–27% of competitive athletes with arrhythmias. Electrocardiographic abnormalities on the resting ECG and premature ventricular contractions (PVC) on Holter recordings are known to be more prevalent in athletes. Recent reports have suggested that they do not have ominous prognostic significance in the absence of cardiovascular abnormalities. However, most studies included different types of athletes, with only a minority of high-level endurance athletes. In particular, data are lacking about the clinical outcome of endurance athletes with more than sporadic isolated PVC (often detected incidentally), but presenting with symptoms and/or with frequent or complex ventricular arrhythmias.

We report on a group of 46 high-level endurance athletes, predominantly cyclists, with complex ventricular arrhythmias (i.e. at least non-sustained ventricular tachycardia). The findings at initial work-up and their follow-up (with a median of 4.7 years) is described.

Methods

Study population

The study comprised 46 endurance athletes seen at three cardiological centres. Only athletes participating regularly in intense endurance sports (i.e. ≥3×2 h/week for ≥5 years) were included. A total of 36 athletes presented with symptoms of light-headedness, fatigue or (pre)syncope that were attributable to ventricular arrhythmias. Nine were asymptomatic at initial presentation but complex ventricular arrhythmias (≥1 runs of ≥3 beats at ≥120 bpm of non-sustained ventricular tachycardia (VT)) were documented on ECG, Holter monitoring and/or on exercise testing during routine screening examination. One athlete was included after aborted sudden death but had an extensive earlier non-invasive cardiological evaluation. The subjects were not followed exclusively at our centres, but their medical outcome had to be available throughout the course of the follow-up. Median follow-up was 4.7 years (0.5–23.7 years).

Evaluation

Apart from routine non-invasive testing (ECG, Holter, exercise testing) electrophysiological studies (EP studies) were performed in 40 of the 46 athletes, always off antiarrhythmic drugs (≥5×half-life) and without sedation. A supraventricular arrhythmia was excluded. The ventricular stimulation protocols were standard, consisting of fixed rate pacing (at cycle lengths down to 240 ms) and delivery of up to three extrastimuli at basic cycle lengths of 600 and 400 ms, both in the right ventricular apex and outflow tract (down to the ventricular refractory period but never <180 ms). In all but three athletes a similar stimulation protocol was applied during isoproterenol infusion (1–4 µg/min) if the baseline study was negative. Only the induction of monomorphic VT was considered specific under these conditions.

Efforts were made to distinguish automatic or triggered foci from reentrant circuits as the mechanism of the arrhythmia. A focal mechanism was suspected in the case of frequent spontaneous monomorphic (or multiple monomorphic) premature ventricular contractions (PVCs) or runs of PVCs (on Holter or induced by exercise testing or administration of isoproterenol), with variable coupling intervals and shortening of the cycle length with increasing adrenergic tone, and/or with a morphology typical for ‘idiopathic’ right ventricular outflow tract VT. Reentrant arrhythmias were identified by classical criteria during the EP study, like reproducible inducibility by extrastimuli (with a constant or increasing return cycle with decreasing coupling interval), resetting by extrastimuli, entrainment with (concealed) fusion and/or termination by programmed ventricular stimulation.

Signal-averaged ECG measurements were performed on tracings band-pass filtered between 40 and 250 Hz, and with a final average noise level of ≤0.3 µV. The following criteria were used: (1) a filtered QRS duration ≥118 ms for women and ≥120 ms for men was considered abnormal; (2) the duration of the high-frequency low-amplitude signals (<40 µV) was abnormal if more than 39 ms, and (3) the mean voltage of the last 40 ms needed to be >20 µV for normality. Late potentials (LP) were present if 3/3 criteria were fulfilled, and probable for 2/3 positive criteria.

Statistical analysis

Summary values are given as means±SD or median (for non-normally distributed values). The relation between nominal variables was tested using Fischer’s exact test or Chi-square analysis. Comparisons between groups were made by non-parametric Mann–Whitney tests or logistic regression. All tests were two-tailed. Kaplan–Meier analysis was used to study survival free of sustained ventricular arrhythmias and sudden death, with log-rank testing to compare survival across groups. A P-value <0.05 was considered significant. Relative risk ratios were calculated by a Cox proportional hazard method.

Results

Presentation, electrophysiological and morphological findings

Eighty percent of the endurance athletes (37/46) were cyclists, eight (17%) were long distance runners (three marathon) and one a kayaker. The athletes were high-level: 80% regularly participated in competitive sports while also some of the nine ‘recreational’ athletes participated occasionally in competitive events. Only one of the 46 athletes had a familial history of ventricular arrhythmias (ARVD in a brother, himself a competitive soccer player). The median age was 31 years (16–53 years).

Demographic characteristics, presenting symptoms, electrophysiological and morphological findings are summarized in Tables 1 and 2. Most of those referred because of presyncope or syncope had developed symptoms
during exertion (26 of 30; 87%). There was very little evidence for underlying coronary or left ventricular disease: most athletes had high-normal or slightly enlarged left ventricular cavities (≥52 mm in 54%) and a high-normal wall thickness, which were considered normal for the endurance athlete’s heart. Only one athlete had a septal wall thickness of 17 mm, and it was ≥15 mm in four. Coronary angiography revealed bridging over the left anterior descending artery in one (with a stenosis of 20% and without demonstrable ischaemia during exercise).

The worst arrhythmia documented non-invasively at initial evaluation (12-lead ECG, Holter, exercise test) was sustained VT in 17 athletes (37%), non-sustained VT in 24 (52%), and only ventricular extrasystoles or couplets in five (11%). An invasive EP study induced sustained monomorphic VT in 15 out of 40 athletes, of whom 13 (87%) in the baseline state. In six, two or more morphologies were induced. The average cycle length (CL) of the induced VT was 299±57 ms (200–410 ms). In two athletes, the VT degenerated into VF (CL 280 and 300 ms). Non-sustained monomorphic VT was induced in another 10 athletes (five in the baseline state) (301±71 ms).

There was indication of right ventricular arrhythmogenic involvement in a majority of the athletes. Thirty-seven athletes (80%) had ventricular arrhythmias with a left bundle branch morphology, indicating an origin in the RV or the interventricular septum. Also other electrophysiological observations (negative T-waves from V1 to at least V3 on the resting ECG; epsilon-waves, i.e. a delayed component of the QRS complex in V1 or V2, late potentials) and morphological findings (angiography; magnetic resonance imaging, myocardial biopsy) pointed more often to RV than LV disease (Tables 1 and 2).

When the diagnostic criteria for ARVD were applied (Table 4), define RV arrhythmogenic and/or pathological involvement was present in 27 of the athletes (59%) because of the presence of ≥2 major or of one major and ≥2 minor criteria. Moreover, another 14 (30%) had one major and one minor, one major, or two minor criteria. The arrhythmias in these 41 patients had a left bundle branch block morphology in 37 (90%), compatible with a RV origin. From the four patients with an LV septal thickness ≥15 mm, two had definite criteria for RV involvement, and two had very frequent PVCs with left bundle branch block morphology (which also is a major criterion for RV arrhythmogenic involvement). One patient with myocyte fibre disarray on biopsy had no echocardiographic criteria for HCM but instead had two major and two minor criteria for ARVD.

### Table 1 Data at presentation and initial electrophysiological evaluation

<table>
<thead>
<tr>
<th>Baseline variables</th>
<th>No. of pts (%)</th>
<th>Initial evaluation</th>
<th>No. of pts (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>45 (98%)</td>
<td>Holter (n=42)</td>
<td></td>
</tr>
<tr>
<td>Age, years (median &amp; range)</td>
<td>31 (16–53)</td>
<td>Single PVC or couplets</td>
<td>21 (49%)</td>
</tr>
<tr>
<td>Type of sports</td>
<td></td>
<td>Non-sustained VT (120–308 bpm, 3–19 beats)</td>
<td>21 (49%)</td>
</tr>
<tr>
<td>Cycling</td>
<td>37 (80%)</td>
<td>Sustained monomorphic VT</td>
<td>1 (2%)</td>
</tr>
<tr>
<td>Running</td>
<td>8 (17.5%)</td>
<td>Bicycle test (n=44)</td>
<td></td>
</tr>
<tr>
<td>Kayaking</td>
<td>1 (2.5%)</td>
<td>No VT</td>
<td>16 (36%)</td>
</tr>
<tr>
<td>Level of sports activity</td>
<td></td>
<td>Non-sustained VT</td>
<td>15 (34%)</td>
</tr>
<tr>
<td>Professional</td>
<td>17 (37%)</td>
<td>Sustained VT (175–300 bpm)</td>
<td>13 (30%)</td>
</tr>
<tr>
<td>Semi-professional</td>
<td>20 (43%)</td>
<td>Non-invasive evaluation</td>
<td></td>
</tr>
<tr>
<td>Recreational</td>
<td>9 (20%)</td>
<td>Single PVC or couplets</td>
<td>5 (11%)</td>
</tr>
<tr>
<td>Presenting symptoms</td>
<td></td>
<td>No-sustained VT</td>
<td>24 (52%)</td>
</tr>
<tr>
<td>Aborted sudden death</td>
<td>1 (2%)</td>
<td>Sustained monomorphic VT*</td>
<td>17 (37%)</td>
</tr>
<tr>
<td>(Presyncope)</td>
<td>30 (65%)</td>
<td>Electrophysiological study (n=40)</td>
<td></td>
</tr>
<tr>
<td>Palpitations or none</td>
<td>15 (33%)</td>
<td>Negative</td>
<td>10 (25%)</td>
</tr>
<tr>
<td>Baseline 12-lead ECG</td>
<td></td>
<td>Non-sustained VT</td>
<td>15 (38%)</td>
</tr>
<tr>
<td>Normal or LVH</td>
<td>23 (50%)</td>
<td>Monomorphic</td>
<td>8</td>
</tr>
<tr>
<td>Negative T V1–V2</td>
<td>4 (9%)</td>
<td>Polymorphic</td>
<td>5</td>
</tr>
<tr>
<td>Negative T ≥V3</td>
<td>18 (39%)</td>
<td>Both</td>
<td>2</td>
</tr>
<tr>
<td>Pathologic Q waves</td>
<td>1 (2%)</td>
<td>Sustained VT (all monomorphic)</td>
<td>15 (38%)</td>
</tr>
<tr>
<td>Epsilon waves</td>
<td>9 (19.5%)</td>
<td>PVC/VT morphology (n=46)</td>
<td></td>
</tr>
<tr>
<td>Signal averaged ECG (n=38)</td>
<td></td>
<td>LBB</td>
<td>37 (80%)</td>
</tr>
<tr>
<td>≤1 positive criterium</td>
<td>23 (61%)</td>
<td>RBB</td>
<td>3 (7%)</td>
</tr>
<tr>
<td>2 positive criteria</td>
<td>5 (13%)</td>
<td>Polymorph</td>
<td>2 (4%)</td>
</tr>
<tr>
<td>3 positive criteria</td>
<td>10 (26%)</td>
<td>Unknown</td>
<td>4 (9%)</td>
</tr>
<tr>
<td>Filtered QRS duration (40–250 Hz)</td>
<td>124±7 ms</td>
<td>≥120 ms</td>
<td>39 (84%)</td>
</tr>
<tr>
<td>≥120 ms</td>
<td>39 (84%)</td>
<td>Low amplitude signals ≤40 µV</td>
<td>33±13 ms</td>
</tr>
<tr>
<td>≤40 ms</td>
<td>21 (35%)</td>
<td>≥40 µV</td>
<td></td>
</tr>
<tr>
<td>≤20 µV</td>
<td>14 (31%)</td>
<td>RMS last 40 ms</td>
<td></td>
</tr>
<tr>
<td>≤20 µV</td>
<td>14 (31%)</td>
<td>≥20 µV</td>
<td></td>
</tr>
<tr>
<td>≥40 µV</td>
<td>21 (35%)</td>
<td>≤20 µV</td>
<td></td>
</tr>
<tr>
<td>≥120 ms</td>
<td>39 (84%)</td>
<td>≤20 µV</td>
<td></td>
</tr>
<tr>
<td>≤40 µV</td>
<td>21 (35%)</td>
<td>≥20 µV</td>
<td></td>
</tr>
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</table>

*Three patients presented with sustained monomorphic VT, which was not reproduced during exercise testing or Holter. LVH: left ventricular hypertrophy; PVC: premature ventricular contraction; RBB/LBB: right/left bundle branch block morphology; VT: ventricular tachycardia.
Treatment and outcome

All athletes were recommended to stop competitive sports and/or intense physical exercise. Twenty-three received beta-blockers (five in association with a Class I antiarrhythmic drug), and four were treated with amiodarone. Nine received an implantable cardioverter defibrillator (ICD, in some associated with
and six underwent one or two catheter ablation procedures.

Eighteen athletes had a major arrhythmic event during follow-up. Half of them (n=9), all cyclists and not treated with an ICD, died suddenly (after a median of 2 years; range 0.5–9.3 years). Moreover, six of the nine ICD patients received one or more appropriate shocks for VT or VF, while three athletes had recurrences of sustained monomorphic (non-syncopal) VT. All but one episode of sudden death occurred during light or moderate physical activity, none during competitive sports. Eight of those 18 athletes were taking beta-blockers (three in combination with a Class 1 agent and one after a partially successful ablation) and one amiodarone.

Table 3 evaluates the predictive value of foregoing findings for major arrhythmias. No symptoms or arrhythmia findings during non-invasive testing (Holter or exercise test) could predict outcome. Major arrhythmias occurred in 15 athletes with no or only mild symptoms (palpitations) at presentation. Also the recognition of overt structural abnormalities (LV or RV) or their degree (as the count of minor and major ARVD criteria) did not discriminate significantly. None of the four patients with LV wall thickness ≥15 mm developed a major event.

Patients with a major event were younger than those without (median 23 years vs 38 years; P=0.01). The only diagnostic test identifying patients at risk was an invasive EP study (Fig. 1) with a relative risk of 3.4 in inducible patients (P=0.02). Sensitivity, specificity and total predictive accuracy of EP inducibility was 62%, 74% and 70% respectively. However, even 20% of those with a negative EP study developed major arrhythmias. The cycle length of an induced monomorphic arrhythmia was not related to outcome. Although more athletes with major arrhythmias during follow-up had multiple inducible morphologies (5/9, 56% vs. 6/17, 35%), this did not

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**Table 4** Diagnostic criteria for right ventricular dyplasia, as applied in this study (adapted from McKenna et al.)

<table>
<thead>
<tr>
<th>Category</th>
<th>Major Criteria</th>
<th>Minor Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>I. Global and/or regional dysfunction and structural alterations</td>
<td>Severe segmental dilatation of the RV</td>
<td>Mild global RV dilatation and/or ejection fraction reduction with normal LV</td>
</tr>
<tr>
<td></td>
<td>Minor</td>
<td>Mild segmental dilatation of the RV</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Regional RV hypokinesia</td>
</tr>
<tr>
<td>II. Endomyocardial biopsy findings</td>
<td>Fibrofatty replacement of myocardium</td>
<td></td>
</tr>
<tr>
<td>III. Repolarization abnormalities</td>
<td>Inverted T waves in right precordial leads (V2 and V3) (if more than 12 years and no right bundle branch block)</td>
<td></td>
</tr>
<tr>
<td>IV. Depolarization/conduction abnormalities</td>
<td>Epsilon waves or localized prolongation (&gt;110 ms) of the QRS complex in right precordial leads (V1–V3)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Late potentials on signal averaged ECG</td>
<td></td>
</tr>
<tr>
<td>V. Arrhythmias</td>
<td>Left bundle branch block type sustained or nonsustained VT on ECG, Holter or exercise testing</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Frequent ventricular extrasystoles (more than 1000/24 h)</td>
<td></td>
</tr>
</tbody>
</table>

**Fig. 1** Kaplan-Meier curves showing freedom from a major arrhythmia (sustained VT or sudden death) during follow-up in 40 of the 46 athletes in whom an EP study was performed during initial evaluation. Athletes with inducible sustained VT or VF (n=15) had a significantly higher risk for developing major arrhythmias during follow-up (RR 3.4; P=0.02).
reach statistical significance. Inducible patients were younger (30±8 vs 37±11 years; P=0.03). Therefore, after adjusting for age in a Cox proportional hazard model, inducibility lost its statistical significance (P=0.11; RR 2.62 for inducible patients)

In 22 athletes (48%) the arrhythmia mechanism could be identified as focal or due to reentry, based on the findings during Holter, exercise testing and/or EP study (cf. Methods). If the arrhythmia mechanism was focal, outcome was significantly better than in those with a reentrant mechanism: only one of the 13 patients with a focal mechanism developed sustained VT (which was haemodynamically tolerated), although seven had persistent PVCs or non-sustained VT. In contrast five of nine reentry patients developed major arrhythmias during follow-up (RR 8.6; P=0.02), of whom one sudden death, three with appropriate ICD discharges and one patient without ICD who required emergency cardioversion for sustained VT. Unfortunately, the mechanism of the arrhythmias was unknown or unclear in 12 out of the 18 athletes with a major arrhythmic event; in the remaining, it was reentry in five and focal in one.

Discussion

Prognosis and its prediction

A recent report has shown that structural cardiovascular abnormalities are more prevalent in athletes with frequent or complex ventricular arrhythmias.6 Nevertheless, the risk of sudden death was very low (one out of a series of 355; 8 years mean follow-up) whether or not cardiovascular abnormalities were present and despite series of 355; 8 years mean follow-up) whether or not less, the risk of sudden death was very low (one out of a larger athlete cohorts as discussed. An extended work-up combining criteria like those for the diagnosis of ARVD may however point to more subtle forms of underlying structural cardiovascular disease, and therefore indicate increased risk.7,13 Formal ending of all competitive and semi-competitive sports seems mandatory if there is evidence for underlying structural damage.5,14 Also other authors have pointed to the high fatality rate when ARVD is present in a competitive athlete.2 The value of an EP study has been suggested before.6,13,15 and an EP study was the only test in our series that was statistically related to outcome although its predictive accuracy remained low. This stresses the need for a large, prospective and concerted scientific study with regard to the prognostic power of different investigations. It is clear that this is of paramount importance for all athletes with (semi-)professional sports activity. The association between age and EP inducibility may indicate inclusion bias, i.e. athletes with more progressed disease and/or at higher risk probably present with symptoms at younger age.

RV structural changes can result in reentrant arrhythmias, as expected. Less anticipated was the fact that in others the arrhythmia mechanism was of focal origin. A few athletes had a focal mechanism without detectable structural RV damage, although this may have been too limited to be diagnosed.16 Although the numbers are small and preclude definitive statements, it is interesting to note that a focal mechanism seems to be correlated with better outcome than reentry. It strengthens the importance of the underlying structure for prognosis and of an EP study in risk stratification.

Endurance sports and arrhythmogenic structural RV damage

Our series only included a small minority of athletes with preexisting left ventricular or coronary disease. This contrasts with previously published series of athletes with (aborted) sudden death, that included primarily basketball and American football players (68%) but was virtually free of cyclists.1 The low prevalence of HCM may be attributable to prior screening visits with ECG and echocardiography more easily detecting LV than RV disease. Italian series of athletes with arrhythmias have reported a ‘high’ incidence of ARVD although it only accounted for 6–27% of competitive athletes with arrhythmic manifestations.2,3 while its incidence was
even lower in non-Italian series. A combined analysis of the arrhythmic and morphological findings in our series pointed definitely and probably to a RV origin in 59% plus 30% of the athletes. Exercise may act as the trigger for arrhythmias in athletes with pre-existing ARVD. Given the high prevalence of ARVD in their region, certainly 30% of the athletes. Exercise may act as the trigger for arrhythmias in athletes with pre-existing ARVD. Given

On the other hand, there is some evidence that endurance sports by itself may have contributed to the RV structural and electrical modifications. Arguments for such an hypothesis are: (1) ARVD, and familial ARVD in particular, is a rare condition in our countries, as it is in North-America. Familial ARVD was only evident in one athlete. (2) Immediately after a competitive triathlon event, paradoxical RV dilatation (contrasting with the LV) has been documented echocardiographically. It was attributed to the greater increase in RV work: endurance sports disproportionately lead to greater strain on the RV. (3) Jordaens et al. reported that cyclists with or without documented arrhythmias had more pronounced late potentials (a major ARVD criterion) than basketball players. (4) In most Italian patients with ARVD MRI revealed clear fat infiltration of the wall, which was absent in our series (present in only two out of 28 MRI studies). The ARVD described by these authors may be of a different aetiology and nature (familial, genetic) than the structural RV damage we observed. (5) The incidence of atrial fibrillation is higher in athletes. Atrial dilatation and remodelling has been suggested as a factor explaining this association. Therefore, we speculate that endurance sports by itself may also lead to RV structural damage that might not have developed without the activity. Long-lasting volume overload could be the mechanism leading to or contributing to the development of RV structural changes.

Doping agents are known to increase the risk of cardiac death. Chronic use of performance-enhancing drugs may be more prevalent due to the intense demands during endurance sports. However, its prevalence and the type of drugs can never reliably be assessed. At the other hand, direct cardiac effects of these drugs would be expected to be more obvious in the left ventricle (having the largest mass), contrasting with our observations that the majority of arrhythmias is of right ventricular origin. Indirectly, these drugs may promote volume/pressure overload and arrhythmogenic changes by allowing longer and heavier training and competition. Also, it is well recognized that different types of sports have a different influence on the cardiovascular system. Cycling is a particular type of strain, even when compared with other endurance athletes like runners: on top of the isotonic and aerobic work of the leg musculature, it also includes an important part of isometric work, particularly with the upper part of the body. Moreover, cyclists regularly engage in anaerobic dashes in between long episodes of aerobic work. Practice and competition are performed during much more time than is the case in many other sports.

The type of sports may therefore be an important factor in the prognosis of athletes with arrhythmias. Our data suggest that intense endurance athletes (and cyclist in particular) may have a clinical evolution different from other athletes. Further research is mandatory to assess the importance of the type of sports in the risk stratification of athletes with arrhythmias, and of the different factors involved. If the hypothesis that long-lasting volume overload promotes arrhythmias would prove to be correct, it raises concern about the ever increasing workload presented to competitive endurance athletes.

Conclusions

Complex ventricular arrhythmias in high-level endurance athletes and in cyclists in particular may be associated with a higher risk for developing major arrhythmias than previously expected. Prognostic stratification represents a difficult issue, and may warrant an EP study, also by defining the arrhythmogenic mechanism (focal vs reentry). The pathophysiology of arrhythmias in endurance athletes is poorly understood. Long-lasting volume overload and a higher tendency to resort to performance-enhancing drugs may play a role, revealing an underlying (genetic sub-) substrate and/or promoting its development.

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