Antonio Pelliccia
Editor

Sports Cardiology Casebook

Foreword by Barry J. Maron

Springer
Sudden death in young athletes engaged in competitive sports is a highly visible, emotionally charged and important public health issue, which has increasingly become part of the consciousness of the general public and practicing sports medicine community. Substantial progress has ensued in this new area of cardiovascular medicine since its inception 25 years ago. Indeed, considerable data are now available from both the U.S. and Europe defining the causes, frequency and demographics of these tragic and counterintuitive events which strike to the core of our sensibilities.

There has been a recent focus (and, in fact, controversy) regarding the most effective and appropriate strategies for mass preparticipation screening for the detection of otherwise unsuspected cardiovascular diseases. Finally, given the identification of a cardiovascular abnormality in a trained athlete, several scientific societies, i.e., American College of Cardiology (ACC), European Society of Cardiology (ESC) and Italian Sports Cardiology Society (SIC Sport) have offered detailed guidelines and consensus recommendations which represent criteria with which clinical decisions regarding management and eligibility/disqualification decisions can be effected.

This multi-authored book, the Sports Cardiology Casebook, edited by Dr. Antonio Pelliccia, a noted international authority on sports medicine, cardiovascular disease in athletes and chair of the ESC guidelines for eligibility/disqualification, is an important contribution to our understanding of this important and growing area of medicine. Dr. Pelliccia has compiled from international experts several case vignettes illustrating some of the dilemmas implicit in reaching the difficult decisions regarding appropriate recommendations in athletes with underlying cardiovascular disease. These are, invariably situations involving individuals greatly invested in sports, often at elite or professional levels with their livelihood dependent on continued eligibility. In drawing the “line in the sand” between eligibility and disqualification, clinicians can rely in large measure on the aforementioned consensus guidelines. . . but, in the end, a substantial measure of individual clinical acumen is often required. Thus, the Pelliccia book represents a particularly useful...
addition to the literature in this field which will facilitate clinical practice related to athletes.

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Introduction

Over recent years, issues related to management of competitive and amateur athletes with cardiovascular disease have (CV) become highly visible and complex medical and public health topics. Particular interest surrounds these issues in consideration that elite and professional athletes represent a special and visible subset of our society, not only for their outstanding physical performances, but also for the substantial economic interests and the intense pressure to which they are exposed by sponsors, sports organizations and media [1–4].

When a cardiovascular disease is found in such athletes, the managing physician is required to solve the compelling issue of appropriate management and recommendations, considering both the impact of sport participation on the course and outcome of cardiac disease and the impact of disqualification on the athlete’s life.

In the last 2 decades participation in a broad spectrum of sport activities (including competitive events) has increased substantially in civilized societies and has become an integral part of the lifestyle of large segments of the population, including very young and senior individuals. In this context, competitive and professional sports have progressively evolved toward globalization, including nowadays not only Western Europe and the U.S., but also large number of East European and African countries, as evident by the changing demographics of elite athletes engaged in professional sports, primarily soccer or basketball.

Due to the unique structure and pressures of competitive sports, athletes with CV disease may not always have adequate knowledge and independent judgment in assessing the risk associated with a competitive sports career. Therefore, the managing physician (and consultant cardiologist) have the ethical, medical and legal
obligation to assess appropriately the overall risk scenario associated with sport lifestyle in an athlete with cardiovascular abnormality and clearly inform the candidate. Indeed, the managing physician is responsible for final recommendations concerning sport participation, with the aim to preserve the innumerable benefits derived from sport (including economic interests in elite/professional athletes) but prevent adverse clinical events and reduce the risk of disease progression.

However, when a disqualification decision is possible, pressure on the managing physician may be intense, and consensus guidelines represent the only legitimate support to the physician’s decision. Under such difficult circumstances, adherence to recommendations released by scientific societies represents the only appropriate defense for the physician, as well as the appropriate manner in which to protect athletes from the unsustainable hazard of sports participation.

Consensus guidelines for eligibility/disqualification decisions in competitive athletes with cardiovascular abnormalities were initially promoted in the U.S. (in 1985 the American College of Cardiology formalized the first Bethesda Conference #16, with subsequent updated #26 and #36 in 1994 and 2005, respectively [5–7]). In Europe, the first consensus document concerning the management and participation in competitive sports of athletes with cardiovascular abnormalities was delivered from the Italian Sports Cardiology Society in 1995 and subsequently updated to 2003 [8], and from the European Society of Cardiology (ESC) in 2005 [9].

The rationale for offering both the American and European expert consensus documents is the widely accepted perception that athletes with clinically silent cardiovascular disease harbor increased risk for disease progression and sudden death by virtue of their commitment to intensive training and competition. Conversely, the removal of athletes from this lifestyle is regarded as mechanism by which the risk may be substantially reduced [10, 11].

Both the ACC Bethesda Conference #36 and the ESC recommendations provide specific advice with respect to different cardiac abnormalities and sports, based on the available scientific data, as well as on personal experience of the panel participants. These documents represent the most updated attempt to combine the (scarce) evidence-based data with personal experience of the experts.

However, presentation of cardiovascular disease in individual athletes may present a variety of clinical forms and not always correspond to the schemes reported in the documents, making management of the candidate-athlete troublesome. In these instances it is challenging to translate the recommendations dictated by the scientific societies into the clinical practice.

It was our aim to address this problem with the present textbook entitled “Sports Cardiology Casebook”. It was our intention to provide the managing physician with appropriate tools for solving this problem, i.e., examples derived from the clinical practice, which illustrate how to proceed in the management and final recommendations of an athlete with cardiovascular abnormality by applying the current consensus guidelines.

The textbook includes a large selection of clinical cases, which are intended to offer a wide perspective of the most common problems arising in the cardiovascular evaluation of competitive athletes, as viewed by expert European cardiologists. The
selected cases are listed according to their clinical presentations, i.e., abnormalities discovered by medical history, by the electrocardiogram, and finally by the usual imaging testing (primarily, echocardiography). This structure makes it easy for the physician to search for a similar and appropriate tutorial case.

The clinical cases here reported represent the selection made by physicians and scientists working in Europe within the Sports Cardiology group, and their assessment closely reflect the ESC consensus guidelines [9], in addition to their personal expertise and science. However, in consideration of the scarcity of scientific investigations concerning the effect of regular sport activities on the pathophysiology and clinical course of several cardiovascular disease, caution is needed in applying the present examples to clinical practice, and efforts should be made to tailor precise advice to each athlete-patient.

Therefore, the examples provided here cannot be assumed to represent the standard of care in all instances, but are examples to be applied in practice in an individualized fashion. Indeed, these cases should not restrict the physician’s independent judgment in evaluating the candidate-athlete and cannot be claimed as impairment to physician liberty to search appropriately the best medical management of the patient-athlete.

References

Chapter 1
A 27-Year-Old Professional Cyclist with Palpitations on Effort

Hein Heidbüchel and Axel J.P. Urhausen

Family and Personal History

The patient was a 27-year-old professional cyclist. An elite athlete, he had participated at several international races, including the Tour de France and the World Cycling Championship.

Prior medical history was uneventful. There were no familial antecedents of sudden cardiac death. His father and younger brother were competitive cyclists too, albeit not at a professional level.

The athlete was referred to our observation because of recurrent exercised-induced palpitations that had first occurred 1 month before.

The athlete reported that during exercise the heart rate monitor showed abrupt increase of heart rate, from an average of 170–180 bpm, to 210 bpm for a few seconds. At that moment the athlete experienced palpitations and a sudden fatigue, but no dizziness. He never had a pre-syncope or syncope. It was remarkable that the team physician had, because of these palpitations, given him flecainide IV on the morning before a competition, although no prior ECG diagnosis had been made. Apparently the treatment had been successful in preventing recurrences.

Physical Examination

The physical examination was normal. Blood pressure was 124/72 mmHg.

12-Lead ECG

The ECG at first evaluation is shown in Fig. 1.1. There was a sinus rate of 42 bpm. The QRS complex duration was slightly prolonged (105 ms) with mild,
non-specific repolarization changes. The corrected QTc interval was markedly prolonged (520 ms).

A prior ECG was searched for (recorded during a cardiologic screening performed elsewhere 2 years before): at that time the ECG showed a QTc interval within normal limits (i.e., 440 ms).

**Diagnostic Testing**

The exercise test showed a superb performance, with maximum workload of 550 W and heart rate of 180 bpm. During the test, the QTc interval returned to normal limits (<440 ms), but there was a progressive widening of the QRS complex, with development of a right bundle branch block morphology and indeterminate axis (Figs. 1.2 and 1.3 at 200 W and 500 W workloads, respectively). During the recovery the QRS width decreased gradually.

In addition, sporadic premature ventricular beats (PVBs) with different morphology (mostly with left bundle branch block) were observed during exercise.

The ECG Holter monitoring documented 870 PVBs/24 h, with different morphology.

A late potential ECG was positive for three criteria (filtered QRS duration 139 ms; HFLA duration 56 ms and RMS40 17 µV; Fig. 1.4).
Fig. 1.2  The ECG recorded at 200 W during exercise testing. There is sinus tachycardia with a rate of 100 bpm. Compared to the baseline ECG, there is a shortening of the QTc interval, but a slight increase of the QRS duration with an incomplete right bundle branch pattern is seen.

Fig. 1.3  The ECG recorded at 500 W during exercise testing. There is a further increase of the QRS duration with development of complete right bundle branch block.
Fig. 1.4  Late potentials were present with three out of three positive criteria: filtered QRS duration 139 ms (normal \( \leq 120 \) ms); duration of the high-frequency low-amplitude signals \( \leq 40 \mu V \) (HFLA40) 56 ms (normal <40 ms), and root mean square voltage of the last 40 ms (RMS40) 17 \( \mu V \) (normal \( >20 \mu V \))

Echocardiography showed dilatation of the left ventricle (diastolic and systolic diameter 58 mm and 38 mm, respectively) with concordant dilatation of the other heart chambers. LV septal thickness was 15 mm and posterior wall thickness 12 mm. The overall morphologic pattern was judged to be expression of the athlete’s heart [1].

Cardiac magnetic resonance imaging excluded hypertrophic cardiomyopathy, but showed a disproportionate (albeit mild) dilatation of the right ventricle (RV) with slightly reduced systolic function. Early diastolic flattening of the septum and a distended inferior vena cava suggested RV overload.

Coronary angiography and ventriculography showed normal coronary arteries, a slightly dilated but normally contractile left ventricle, and a slightly dilated and mildly hypokinetic right ventricle, without significant regional wall motion abnormalities.

An electrophysiological (EP) study was performed. It excluded a concealed accessory pathway and AV nodal re-entrant tachycardia. It could not induce any atrial arrhythmias nor any ventricular arrhythmias (with up to three extra stimuli at two basic cycle lengths of 600 and 400 ms at baseline, and after administration of 1, 2 and 3 \( \mu g/min \) of isoprenaline). During isoprenaline infusion, however, sporadic PVBs and couplets, again with left bundle branch morphology and variable axis, were induced.
In conclusion, the cardiovascular evaluation suggested abnormal ventricular conduction slowing throughout the ventricles, of unknown aetiology, associated with PVBs and positive late potentials. Moreover, there were signs of RV overload (which were likely related to the frequent PVBs with left bundle branch morphology), but these abnormalities did not fulfil the criteria for arrhythmogenic RV cardiomyopathy (ARVC). Together with the late potentials, there were two minor criteria for ARVC, which were not sufficient for definite diagnosis [2].

**Recommendations and Treatment**

The patient was advised to stop competitive sports and was requested to perform only moderate training with a long-term ECG recorder and his rate monitor, with the purpose of recording the spontaneous arrhythmias.

Two weeks later an event recording during training revealed polymorphic ventricular tachycardia which lasted more than 15 s. It reproduced the subjective symptoms and was recorded as an abrupt heart rate acceleration on his heart rate monitor (Fig. 1.5).

![Event recording during training revealing polymorphic ventricular tachycardia which lasted >15 s. It reproduced the subjective symptoms and was documented as an heart rate acceleration on his heart rate monitor](image-url)
Clinical Course

The athlete was advised to stop any competitive and intensive sports activity. A low dose beta-blocker was started (metoprolol 25 mg once daily). Class-1 drugs or amiodarone were considered to be an unsuitable option given the intraventricular conduction delays. It was also suggested to consider prophylactic ICD implantation, which the athlete declined.

Given the impact of the therapeutic decisions, the athlete was advised to seek a second opinion. The importance of showing all the available data for such a second opinion was stressed, and a file with copied documents was assembled for this purpose.

The patient consulted six other cardiology centers, taking the documentation of the original evaluation with him. In four the same advice was given, whereas in two others the recommendation was given to continue competitive sports under beta-blocker treatment, based on the finding that arrhythmias during exercise tests were non inducible under treatment with metoprolol 25 mg/day. The patient, therefore, restarted competition.

Nevertheless, 6 months later and despite continued beta-blocker treatment the athlete showed an exercised-induced non-sustained ventricular tachycardia (10 beats, 220 bpm) during exercise testing performed in one of the latter centers. At that time, also in that center the athlete was finally advised to stop any competitive sports activity.

After 1 year he was admitted to yet another hospital with sustained ventricular tachycardia which had developed during recreational cycling (230/min.). A new EP study was performed, which triggered two sustained ventricular tachycardias, both with left bundle branch block morphology. An ICD was recommended, but rejected again by the patient.

After 6 months he was re-admitted to yet another hospital with sustained ventricular tachycardia (the trigger of which is unknown). Ablation was not performed since it was not considered to be definitive treatment. An ICD was recommended based on the evidence of progressive arrhythmogenic disease in the athlete, albeit without unequivocal diagnosis of ARVC. The patient again rejected the ICD.

After 2 months he collapsed during a solo cycling training, was resuscitated late and died 2 weeks later.

Discussion

This case illustrates the difficult work-up of an athlete presenting with palpitations, and the difficulty in coming to a conclusive diagnosis on the aetiology of ventricular arrhythmias, which may convey a lethal outcome.

It also illustrates the special care needed in evaluating athletes with palpitations induced by exercise, such as in this case, where ventricular conduction disturbances and right ventricular enlargement were seen, but it was unclear whether the palpitations were due to repetitive ventricular premature beats or ventricular tachyarrhythmia or sustained atrial arrhythmia.
Imaging examinations are not always sufficient for diagnosis, as in this case, where the electrophysiological work-up was warranted. Indeed, although the initial QTc interval in our patient was prolonged, there was no further evidence for a congenital long QT syndrome (which was also excluded by the familial screening). This case is also certainly not a typical presentation of catecholaminergic polymorphic VT and there was no familial history of it.

On the other hand, there was conduction abnormality (right bundle branch block; late potentials), increasing during faster heart rates, and of progressive nature.

Some authors have suggested that, in the absence of epsilon-waves on the surface-ECG, signal-averaged ECG recording revealing late potentials may be a sign of delayed (right) ventricular activation and of a pro-arrhythmogenic substrate [3]. Late potentials may be a subtle but definite criterion for ARVC [2]. Unfortunately, genotyping was not available to exclude further a channelopathy or cardiomyopathy in this case. Eventually, not only the absence of a familial history but also the progressive nature of the conduction disturbances (with a normal ECG pattern seen 2 years before presentation) argues against pre-existing and/or familial disease.

Polymorphic ventricular tachycardia occurring during exercise, as documented in this case, always carries a bad prognosis, since it may point to an underlying inherited electrophysiological disorder or to underlying structural disease.

Given the undeniable presence of underlying pathologic substrate (albeit without conclusive diagnosis), this patient was definitely considered ineligible for competitive sports like cycling [4]. Although this advise was debated after the first evaluation, there was clear medical consent in this respect after documentation of repeat arrhythmias, despite beta-blocker treatment.

A sense of concern is also related to the inability for the several consultant cardiologists to implement their advise into practice. The lack of national law obliging athletes to obtain a medical clearance before entering competitive sport (such is the case in Italy) put the cardiologists in the position of not being able to withdraw this patient at high risk from sport activity.

In conclusion, palpitations during exercise, fast rates on a heart rate monitor, symptoms of hemodynamic compromise (like a sudden fatigue during exercise in this case), and even the finding of sporadic VPBs during an exercise test should be taken seriously and should trigger a comprehensive evaluation for ruling out underlying structural heart disease and potentially lethal ventricular tachyarrhythmia (see also Chapters 19 and 21 in this respect).

That goal needs a tailored and individualised work-up in selected cases, and may also exceed the standard routine protocol suggested in the recommendations [4].

References


Chapter 2
Impaired Performance in a Master Long-Distance Runner

François Carré

History

A 58-year-old male long distance runner complained of reduced exertion performance during the last 3 months despite an intensive training schedule. He felt “broken” beyond 12 km/h, and experienced respiratory “blockage” during high speed running, associated with a dramatic decrease of his maximal running speed. During the last 5 years he had short episodes of palpitations during training. Previous cardiovascular testing (12-leads ECG, echocardiography, maximal exercise test, ECG Holter monitoring) demonstrated a few isolated supraventricular and ventricular premature beats. His general physician recently performed a comprehensive haematological check-up, which showed no abnormalities.

Athletic History

This athlete has been active in long distance running for the last 20 years and participated in long-distance events, from half-marathon to 100-km races. The current training schedule was between 80 and 100 km per week (five sessions a week of running and one session of cycling).

Family History

His familial history revealed no known congenital or other cardiovascular diseases and no known cases of premature (< 50 years) sudden cardiac death in close relatives.

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