Progress in Cardiovascular Diseases 54 (2012) 397–406
www.onlinepcd.com

Evaluation of Suspected Right Ventricular Pathology in the Athlete

Jonathan D.S. Sniderman[a,b], Daniel M. Sado[a], Allan D. Sniderman[c], William J. McKenna[a,*]

[a]*Institute of Cardiovascular Science, University College London, and The Heart Hospital, University College London Hospitals Trust, London, UK*
[b]*Great Ormond Street Hospital for Sick Children, London, UK*
[c]*Mike Rosenbloom Laboratory for Cardiovascular Research, McGill University Health Centre, Montreal, Canada*

Abstract Rigorous training remodels the heart of elite endurance athletes to produce the phenotype of the "athlete's heart." This remodeling, which advantages cardiac performance, creates challenges in the diagnosis of cardiac disorders within this population. This is particularly so for right ventricular pathologies because of the limited number of studies documenting the impact of training on right ventricular remodeling. Although arrhythmogenic right ventricular cardiomyopathy is the focus of this review, several other pathologies that may mimic arrhythmogenic right ventricular cardiomyopathy, including right ventricular outflow tract tachycardia, Wolff-Parkinson-White syndrome, Brugada syndrome, pulmonary embolism, cardiac sarcoidosis, myocarditis, and right ventricular infarction, are also included. In particular, the electrocardiographic findings for each condition are highlighted because this is the most informative and easily accessible diagnostic clinical tool. (Prog Cardiovasc Dis 2012;54:397-406)
© 2012 Elsevier Inc. All rights reserved.

The cardiac remodeling that occurs in an elite endurance athlete may overlap with the changes produced by right ventricular (RV) pathology, making diseases more difficult to recognize in the presence of physiological adaptation. However, it is the athlete who appears to be at most risk for diseases of the right ventricle, and therefore, it is the athlete on whom we will concentrate. In this article, an *athlete* is defined as someone involved in high-level endurance sports such as rowing, canoeing, long-distance running, or cycling. Furthermore, we will address young athletes because, with few exceptions, world-class athletes are younger than 40 years, with most being in their 20s. Accordingly, coronary artery disease will not be emphasized. Rather, arrhythmogenic right ventricular cardiomyopathy (ARVC) will be the major focus because ARVC is one of the leading causes of sudden death in athletes and because phenotypical changes in cardiac remodeling in athletes may overlap sufficiently with ARVC to make diagnosis challenging. Missed diagnoses may lead athletes to expose themselves to a greater risk. Overdiagnosis can injure an individual who has chosen to base his or her life around sport. Error either way may trigger medicolegal actions. Moreover, given the genetic nature of some pathologic conditions, identifying those affected is important not only for their health but also for that of their relatives.

Cardiovascular adaptation in the highly trained athlete

The phenotypic changes associated with endurance training are well established and include the development of cardiac hypertrophy and dilation with bradyarrhythmias and increased vagal tone.[1] These adaptations augment stroke volume and, therefore, the ability to increase cardiac output with exercise above the normal as documented by objective measures of exercise capacity, especially peak oxygen consumption, which increases up to 40% to 50%.[2] The magnitude of this beneficial adaptation depends on the duration, intensity, and type of training, being greatest in elite rowers, runners, and canoeists. The extent of remodeling in these

Statement of Conflict of Interest: see page 405.

* Address reprint requests to William J. McKenna, MD, The Heart Hospital, 16-18 Westmoreland St, London W1G 8PH, UK.
E-mail address: william.mckenna@uclh.org (W.J. McKenna).

0033-0620/$ – see front matter © 2012 Elsevier Inc. All rights reserved.
doi:10.1016/j.pcad.2012.01.005

Abbreviations and Acronyms

ARVC = arrhythmogenic right ventricular cardiomyopathy

CMR = cardiovascular magnetic resonance

ECG = electrocardiographic

RVOT-VT = right ventricular outflow tract ventricular tachycardia

WPW = Wolff-Parkinson-White

athletes has the potential to develop morphologic and electrophysiologic features that overlap with "disease" phenotypes.

Most research in cardiac remodeling in athletes has centered on the left ventricle. Numerous studies have demonstrated that strength training is associated with concentric thickening of the left ventricle with associated electrocardiographic (ECG) changes of left ventricular hypertrophy.[1] By contrast, endurance athletes have an eccentric increase in the size of the left ventricle; that is, the increase in cavity size is more pronounced than the increase in wall thickness.[3] The ECG variants associated with exercise training include early repolarization changes, such as ST elevation in V_3 to V_6, with an elevated J point and peaked upright T waves (Fig 1).[4] There are important racial differences in both the appearance of the ECGs in an athlete and the frequency of these abnormalities. Biphasic and inverted T waves in anterior chest leads (V_1-V_3) are rare in white athletes but may be seen in up to 25% of elite Afro-Caribbean athletes. These findings warrant additional investigation, although, often, no other abnormalities are detected after a complete workup, including familial investigation (Fig 2).[5,6] The increased vagal tone associated with endurance training results in sinus bradycardia, lengthened PR interval, and Wenckebach phenomena.[4]

The effects of athletic training on the right ventricle are not as clearly described. Whether concentric RV remodeling occurs is unclear; most reports describe eccentric RV remodeling with training.[7-11] Indeed, some studies have suggested that eccentric remodeling may be more pronounced in the right ventricle than in the left because of proportionately higher increases in wall stress during exercise.[11] An increase in RV volume has been associated with the presence or development of incomplete or complete right bundle-branch block.[12] Nevertheless, the increase in RV dimensions with endurance training only uncommonly reaches the threshold for pathologic dilation used in ARVC echocardiography measurements, whereas wall motion abnormalities have not been described with physiologic dilatation. These are key points. Moreover, RV remodeling due to training demands a high degree of training and is found only in individuals involved in demanding endurance training.

Arrhythmogenic RV cardiomyopathy

Arrhythmogenic right ventricular cardiomyopathy is a desmosomal disease linked to mutations of multiple genes implicated in maintaining the structure of the intercalated discs.[13-15] It is believed that instability of the intercalated

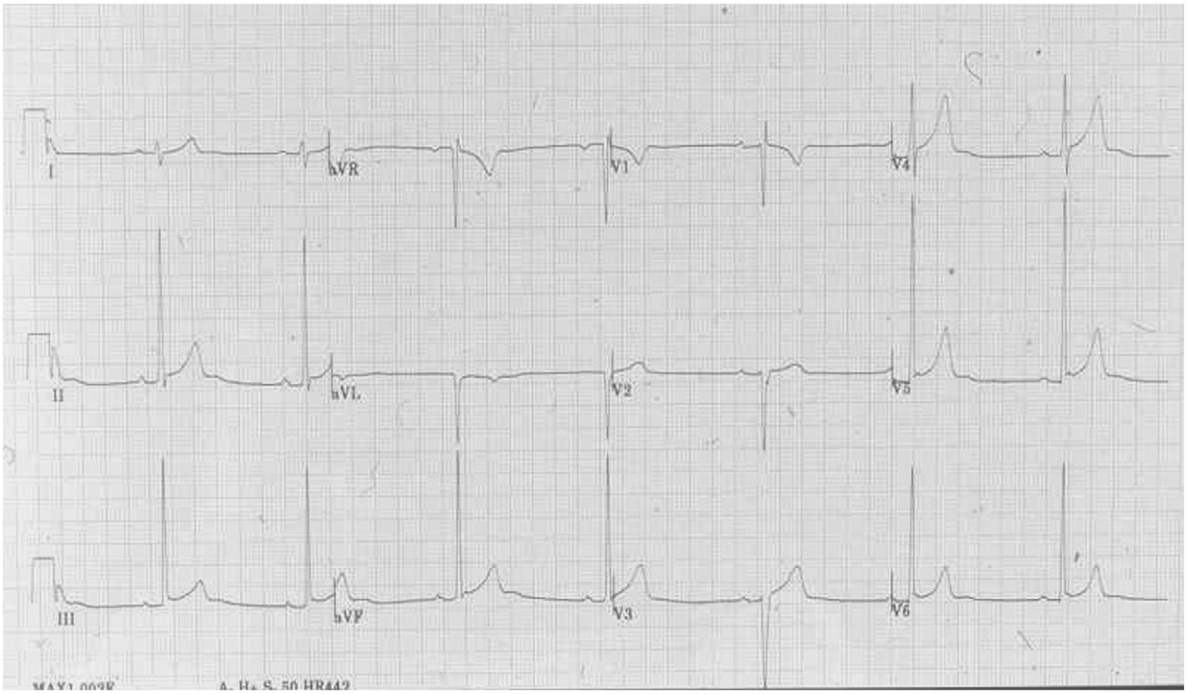

Fig 1. Electrocardiogram of a 17-year-old elite rower with a rightward axis, prominent QRS voltages, tall peaked T waves, and inferolateral J-point elevation. On 2-dimensional echocardiography, the LV wall thickness is normal and the LV end-diastolic dimension is 60 mm.

Download English Version:

https://daneshyari.com/en/article/3006914

Download Persian Version:

https://daneshyari.com/article/3006914

Daneshyari.com