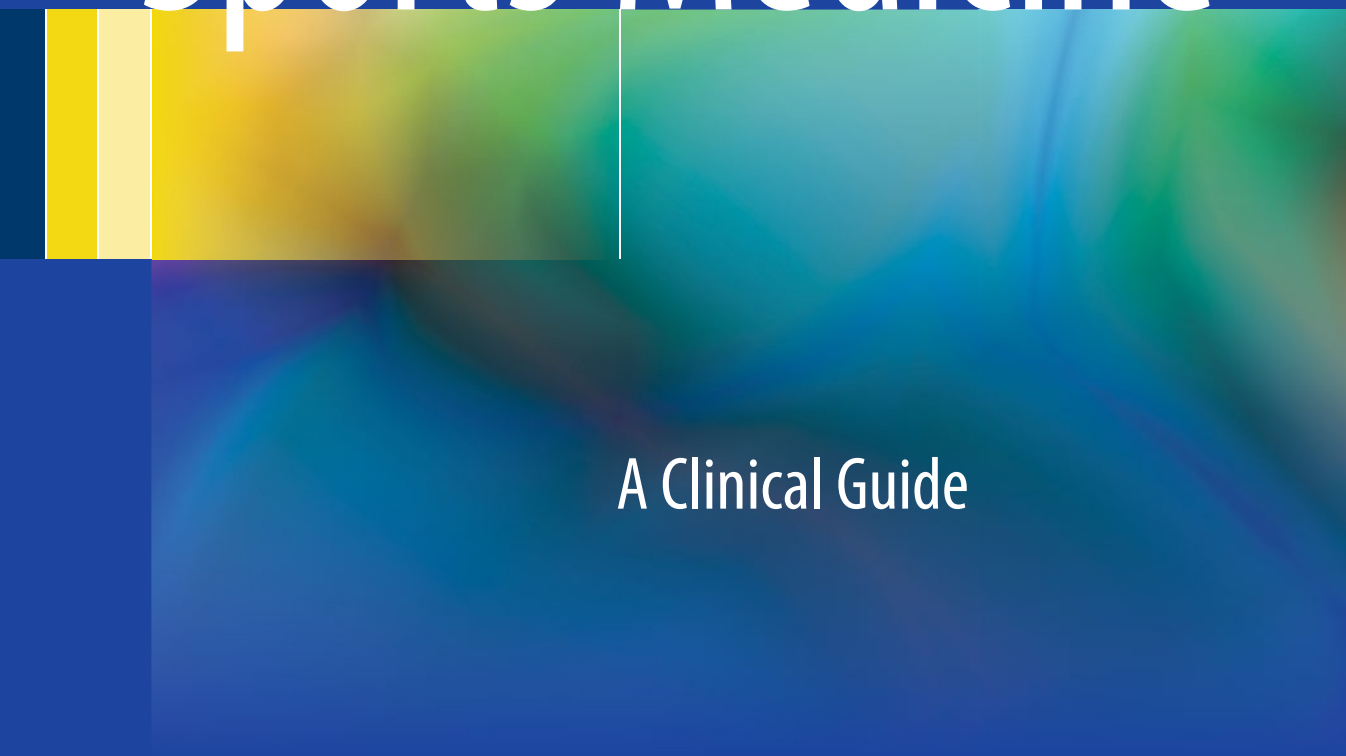
Timothy L. Miller
Editor

Endurance Sports Medicine



A Clinical Guide

Springer

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To my family, in particular my wife Nicole and our children Gavin, Ashton, Avery, and Sydney and my parents Tom and Kathleen, for always believing in me and showing patience and understanding when I take on "yet another project."
To the many endurance athletes who have had their seasons and careers cut short due to overuse injuries, know that your hard work and efforts have not gone in vain and, in fact, were the inspiration for this book.

Preface

Endurance Sports Medicine has been in many ways a labor of love for the authors and the editor. It is the culmination of many years of experience with injuries and conditions as athletes, as researchers, and as sports medicine care providers. This textbook compiles the many concepts, experiences, and techniques required to approach and treat the complexities of conditions that affect endurance sports participants. I truly appreciate the contributions of the authors—many of whom are considered pioneers and leaders in the field of sports medicine—who have provided their invaluable insights and pearls. As a developing field of sports medicine, endurance medicine continues to expand its understanding of overuse injuries as athletes continue to push the limits of running, cycling, swimming, wheelchair, skiing, rowing, cross-fit sports, adventure and obstacle course racing, and many other demanding activities. Traditional strategies for treating overuse conditions such as simply stopping the causative activity or sport are no longer considered an acceptable option for many competitive athletes. Alternative training methods including a holistic approach to the evaluation, treatment, and prevention of activity-related conditions are now the standard of care as is evidenced throughout the 21 chapters of this book. This textbook details strategies for not only treating and preventing injuries and conditions but also for optimizing an athlete's performance. Though it is too early to determine whether we can obviate the need to have athletes completely abstain from their sport of choice in response to an injury or condition, we can decrease the time lost from training and competition and allow for a more safe and predictable return to full activity. It is my hope that this textbook will be a valuable guide for sports medicine physicians, orthopedists, athletic trainers, physical therapists, coaches, officials, and athletes in understanding the needs of the determined individuals who participate in endurance sports.

Gahanna, OH, USA — Timothy L. Miller, MD

Contents

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Part I

Medical Conditions

Cardiovascular Evaluation and Treatment of the Endurance Athlete

1

Jennifer A. Michaud Finch and Aaron L. Baggish

Introduction

Endurance athletes have been the focus of scientific investigation for more than a century. Early investigators, including the Swedish clinician Henschen [1] and Darling [2] of Harvard University, demonstrated increased cardiac dimensions in Nordic skiers and in university rowers, respectively. In the early 1900s, White [3], regarded by some as the father of contemporary cardiology, studied radial contours among Boston Marathon competitors and was the first to report marked resting sinus bradycardia in long-distance runners [4].

Since the work done by these pioneering investigators, advances in our understanding of cardiac adaptations to endurance exercise have largely paralleled advances in diagnostic technology. The development and subsequent widespread availability of chest radiography in the 1950s facilitated studies that demonstrated global cardiac enlargement in trained athletes thereby confirming the earlier physical examination findings of Darling and Henschen by showing global cardiac enlargement in trained athletes [5–7]. In the present era, the electrocardiogram (ECG) and contemporary noninvasive imaging modalities including echocardiography, cardiac computed tomography, and cardiac magnetic resonance imaging have more comprehensively characterize the electrical, structural, and functional cardiac adaptations that accompany long-term endurance training [8–11].

The last 25 years have seen tremendous increase in endurance sport participation among men and women of all ages as evidenced by the fact that there was a record-setting number of running event finishers, nearly 20 million in the United States, in 2013 [12]. At no time in the past has it been more critical for care providers to possess the fundamental skills required for the care of the active endurance athlete. Effective care of this population requires an understanding of the cardiovascular demands of exercise, a familiarity with training-related cardiovascular adaptations, and a structured approach to the athlete with symptoms suggestive of cardiovascular disease. This chapter is written to provide the clinician with a basic foundation of knowledge in these principal areas of patient care with emphasis on the endurance athlete.

Overview of Normal Physiological Adaptations to Endurance Training

Differentiating adaptive physiology from occult pathology is the most important task for the clinician tasked with caring for endurance athletes.

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As detailed below, sustained endurance training lasting for weeks to years often leads to numerous changes in cardiac structure and function. At times, these changes may be challenging to differentiate from those that occur in the disease processes that place athletes at risk for sudden cardiac death. The process of differentiating adaption from disease necessarily begins with an understanding of what cardiac changes are expected in a healthy endurance athlete and what physiology stimulates these adaptations.

All forms of exercise require an increase in skeletal muscle work. There is a direct relationship between exercise intensity (external work) and the body's demand for oxygen. The oxygen demand during exercise is met by increasing pulmonary oxygen uptake (VO_2). In addition, the cardiovascular system is responsible for transporting oxygen-rich blood from the lungs to the skeletal muscles, a process quantified as cardiac output (in liters per minute). Exercise-induced cardiac remodeling enhances the cardiovascular system's ability to meet the demands of exercising skeletal muscle. The Fick equation (cardiac output = $VO_2 \times$ arterial venous $O_2\Delta$) can be used to quantify the relationship between cardiac output and VO_2. As suggested by this equation, there is a direct and inviolate relationship between VO_2 and cardiac output in the healthy human.

Cardiac output (CO), the product of stroke volume and heart rate, may increase five- to six-fold during maximal exercise effort. Coordinated autonomic nervous system function, characterized by rapid and sustained parasympathetic withdrawal coupled with sympathetic activation, is required for this process to occur. Heart rate among healthy athletes may range from fewer than 40 beats per minute at rest to greater than 200 beats per minute in a young, maximally exercising athlete. Heart rate increase is responsible for the majority of cardiac output augmentation during exercise. Peak heart rate is determined by age, gender, and genetics and cannot be increased by exercise training. Stroke volume is defined as the quantity of blood ejected from the heart during each contraction. In contrast to heart rate, stroke volume both at rest and during exercise may increase significantly with prolonged exercise training. Cardiac chamber enlargement and the accompanying ability to generate a large stroke volume are direct results of endurance exercise training and are the cardiovascular hallmarks of the endurance-trained athlete. Stroke volume increases during exercise as a result of increases in ventricular end-diastolic volume and, to a lesser degree, sympathetically mediated reduction in end-systolic volume.

Hemodynamic perturbations that occur during exercise constitute the primary stimulus for exercise-induced cardiac remodeling (EICR). Specifically, changes in cardiac output and peripheral vascular resistance vary widely across sporting disciplines. Although there is considerable overlap and clinically oriented physiology-based sport classification scheme has been developed [13], exercise activity can be classified into two forms with defining hemodynamic differences. Isometric exercise, commonly referred to as strength training, is characterized by short but intense bouts of increased peripheral vascular resistance and normal or only slightly elevated cardiac output. This increase in peripheral vascular resistance causes transient but potentially marked systolic hypertension and left ventricular "pressure" challenge. Strength training physiology is dominant during activities such as weight lifting, track-and-field throwing events, and American-style football. In contrast, isotonic exercise (i.e., endurance exercise) involves sustained elevations in CO, with normal or reduced peripheral vascular resistance. Such activity represents a primary volume challenge for the heart that affects all four chambers. This form of exercise underlies activities such as long-distance running, cycling, rowing, and swimming. It must be emphasized that endurance sports all involve high isotonic stress but that specific disciplines vary considerably as a function of the amount of concomitant isometric stress.

Left Ventricular Adaptations to Endurance Exercise

Enlargement of the left ventricle (LV) is common among endurance-trained athletes. This represents physiologic, eccentric LV hypertrophy (LVH) or eccentric LV remodeling as dictated by

endurance discipline [14]. Eccentric LVH is characterized by an increase in LV chamber size accompanied by proportionate increase in LV wall thickness and is common among endurance sports that involve high levels of isometric physiology. Rowing is an example of a sport that has been shown to promote eccentric LVH due to the inherent physiologic combination of sustained high cardiac output and pulsatile surges in systolic blood pressure that occur during the drive phase of each oar stroke [15]. In contrast, long-distance running is an endurance sport discipline characterized by marked and sustained increases in cardiac output coupled with stable and only modestly elevated arterial blood pressure. This physiologic milieu promotes eccentric LV dilation during which LV chamber size may increase and most often do so proportionally more than accompanying adaptive LV wall thickening.

Early cross-sectional data from large heterogeneous cohorts of European athletes demonstrated high prevalence of left ventricular cavity enlargement and increased wall thickness [16, 17]. Markedly dilated LV chambers (defined as >60 mm) were associated with increased body mass and were most common among those participating in endurance sports (cross-country skiing, cycling, etc.). In clinical practice, 55–58 mm is commonly used to define the upper limits of normal for LV end-diastolic dimensions. Thus, approximately 40 % of trained athletes in the above study had LV dimensions that exceeded normal reference points. Within this exclusively white cohort, a small but significant percentage of athletes (1.7 %) had LV wall thicknesses of 13 mm or greater, and all of these individuals had concomitant LV cavity dilation. Sharma and colleagues [18] also reported a low incidence (0.4 %) of LV wall thickness greater than 12 mm in 720 elite junior athletes and confirmed that increased LV wall thickness is associated with increased chamber size in young athletes. More recently, in a study of nearly 500 collegiate athletes, not a single healthy university athlete had LV wall thickness of greater than 14 mm [19]. In summary, LV wall thickness in excess of 13 mm is a rare finding in healthy athletes, although it can be seen in a small number of healthy, highly trained individuals. This finding is more common in athletes with relatively large body size and those of Afro-Caribbean descent [20]. Thickening of the LV, without associated LV chamber dilation, occurs infrequently in trained endurance athletes and should warrant further workup.

Systolic or contractile function of the dilated LV in athletes has also been an area of active investigation. Studies of resting LV systolic function in endurance athletes consistently demonstrate that LV ejection fraction is generally normal in this population [21]. However, a study of 147 cyclists participating in the Tour de France found that 11 % had an LV ejection fraction of 52 % or less [22]. This finding supports our clinical experience in which we often find that healthy endurance athletes demonstrate mildly reduced LV ejection fraction at rest. Exercise testing can be a very useful adjunct to confirm LV augmentation and to document greater than normal exercise capacity in the hearts of trained athletes [23]. Recent advances in functional myocardial imaging, including tissue Doppler and speckle tracking echocardiography, have also suggested that endurance exercise training may lead to changes in regional LV systolic function that are not detected by assessment of a global index, such as LV ejection fraction [24]. Furthermore, changes in LV apical rotation and twist have been identified in a longitudinal study of rowers participating in endurance exercise training [25]. At present, the role of these newer imaging techniques in the clinical assessment of endurance athletes remains uncertain.

LV diastolic function has also been studied in endurance athletes. Endurance exercise training leads to enhanced early diastolic LV filling as assessed by E-wave velocity and mitral annular/LV tissue velocities [26–28]. In an elegant study utilizing invasive hemodynamic measurements, Levine et al. described improved LV chamber compliance in athletes as lesser increases in pulmonary artery capillary wedge pressure for a given increase in end-diastolic volumes [29, 30]. These changes in LV diastolic function are attributable to a combination of enhanced intrinsic myocardial relaxation and training-induced increases in LV preload. Speckle tracking echocardiography has provided further insight into diastolic function, with enhanced peak early diastolic untwisting rate

observed in rowers after 90 days of endurance exercise training [25]. This ability of the LV to relax briskly during early diastole is an essential mechanism of stroke volume preservation during exercise at high heart rates.

Right Ventricular Adaptations to Endurance Exercise

Endurance exercise requires both the LV and the right ventricle (RV) to accept and eject large volumes of blood simultaneously. It is therefore not surprising that both the RV and LV remodel during chronic endurance sport participation. Cardiac imaging studies using both echocardiography and magnetic resonance imaging document a high prevalence of right ventricular dilation among endurance-trained athletes [26, 31, 32]. Similar to the left ventricle, resting RV systolic function may be mildly reduced among trained endurance athletes and likely reflects the substantial contractile reserve afforded by physiologic RV dilation [33]. Due to the increased volume load during endurance training, RV volume, mass, and stroke volume have all been observed to increase in endurance athletes, largely based on measurements using cardiac MRI [30].

RV function may also be affected by endurance exercise. In similar fashion to that observed in the LV, it is common to see trained endurance athletes with low-normal to slightly depressed resting RV systolic function. When encountered clinically, the use of exercise testing coupled with noninvasive imaging may be useful to document normal or enhanced contractile reserve [34]. To what degree RV diastolic function is impacted by endurance exercise training and whether this is of clinical or physiologic relevance remain unknown.

Atrial Adaptation to Endurance Exercise

In addition to remodeling of the left and right ventricle, numerous studies have shown that atrial enlargement is frequent among endurance-trained athletes [30]. In an early sentinel report, Pellicia et al. [35] presented a large data set of atrial measurements in athletes ($n=1777$) and demonstrated that left atrial enlargement (>40 mm in an anterior/posterior transthoracic echocardiographic view) was present in approximately 20 %. Of note, among athletes with left atrial dilation, few athletes had clinical evidence of supraventricular arrhythmias. In general, atrial enlargement is proportional to the enlargement of the ventricles and is affected by the type of training undertaken [11, 23, 36, 37]. Meta-analysis examining left atrial (LA) size in athletes found that both pooled mean LA diameter and LA volume index were greater than sedentary control subjects. In addition, the LA dilation tracked closely with physiological sporting discipline, with endurance athletes demonstrating the largest differences [38]. Cumulative lifetime exercise training hours have subsequently been shown to be an important determinant of left atrial size [39]. The clinical implications of this finding, particularly with respect to the development of atrial fibrillation, warrant further study.

Endurance training appears to have similar effects on the right atrium (RA) as evidenced by data derived from youthful elite athletes [40], with veteran marathon runners [41], despite the existence of right atrial geometric alterations in endurance athletes; no differences in the right atrial function have yet to be demonstrated [42].

Variability of Exercise-Induced Cardiac Remodeling

The magnitude of exercise-induced cardiac remodeling varies considerably across individual athletes. Obvious explanatory factors including sport type, prior exercise exposure, and training intensity/duration do not explain all of this variability. Additional factors including sex, ethnicity, and genetics are contributory [19, 43, 44]. This appears to be true even when cardiac dimensions are corrected for the typically smaller female body size. Definitive explanation for the sex-specific magnitude of exercise-induced remodeling remains elusive. Race is also an important determinant of remodeling, with black

athletes tending to have thicker LV walls than white athletes. A group of white and black athletes have been using echocardiographic imaging and found that nearly 20 % of the black athletes were found to have LV wall thickness of at least 12 mm as compared with 4 % of white athletes. Importantly, 3 % of black athletes in this cohort were found to have wall thickness of greater than 15 mm [45]. Similarly, ethnic-/race-related differences were studied in a group of 440 black and white female athletes using echocardiography. Black female athletes demonstrated significantly greater LV wall thickness and mass compared with the white woman [20].

These numerous adaptations in cardiac structure and function documented above are not exclusive to elite-level athletes. A recent study of middle-aged men who engaged in marathon training also identified biventricular dilation, enhanced left ventricular diastolic function, and favorable changes in non-myocardial determinants of cardiovascular risk after an 18-week recreational running program [46]. The observed structural remodeling in this setting was of sufficient magnitude such that post-training cardiac parameters commonly fell outside the established clinical ranges of normal.

Clinical Implications of Remodeling

Complex cardiovascular demands and adaptations imposed by endurance exercise pose distinct challenges. The overlap between normal, adaptive physiology and pathology and the proper distinction between the two are crucial in the athlete population, where several important causes of athletic sudden cardiac death can be difficult to differentiate from adaptation. The long-term clinical sequela of endurance exercise and the significance of adaptive physiology are still a topic of debate. Although the concept of prolonged endurance exercise participation leading to overuse pathology and premature cardiovascular mortality has been described, there is no definitive evidence to confirm its validity [34, 47–50].

There is a growing body of literature that describes an association between long-term endurance exercise and increased risk of atrial fibrillation [51]. Although routine physical exercise may favorably impact many key determinants of cardiovascular disease (e.g., lipid profiles, blood pressure, and body mass), atrial fibrillation is the exception and has been recognized as a problem for the masters athlete (a group defined as competitors >40 years of age). Numerous underlying mechanisms, including those shared with the general, more sedentary population (e.g., undiagnosed hypertension, excessive alcohol consumption, sleep apnea), and some factors more specific to the aging competitive athlete, including resting vagotonia, chronic inflammation, and left atrial dilation, have been proposed. Greater prevalence of atrial fibrillation among endurance athletes [52] provides an important stimulus for research aimed at better understanding the mechanisms and consequences of endurance exercise. Atrial fibrillation in the endurance athlete will be addressed later in the chapter.

Although cardiac remodeling in response to long-term endurance exercise is often regarded as beneficial, it has been suggested that very extreme exercise bouts could promote permanent structural or functional cardiac changes [34, 48, 53–56]. The RV has been the focus of this debate. We previously reported physiologic RV dilation with focal deterioration of interventricular septal function after a period of intense exercise training [57]. The RV dilation observed may compromise septal function by the way its fibers insert into the LV. Recently, La Gerche and colleagues [48] also demonstrated a relationship between intense endurance exercise and acute reduction in RV function that increased with race duration and correlated with increases in biomarkers of myocardial injury. All immediate postrace right ventricular geometry measurements were increased. According to the Frank–Starling mechanism, the increased volume, area, and dimensions should augment cardiac deformation if contractility is preserved. Therefore, the immediate postrace reductions in the RV may represent a true impairment in RV contractility with acute exercise bouts. In addition, cardiac magnetic resonance imaging with delayed gadolinium enhancement

(DGE) has been used to further characterize RV remodeling.

Septal fibrosis in the locations of RV fiber insertion has been documented in athletes with a longer history of competitive sport, suggesting that repetitive ultra-endurance exercise may lead to more extensive RV change and possible myocardial fibrosis [47, 48]. The cardiac impact of long-term endurance exercise on the RV and the recovery processes may be insufficient to compensate for the extent of injury [34, 48, 58]. This repetitive fatigue may stimulate remodeling and may lead in some to a fibrotic pathologic phenotype. Although the absence of DGE in athletes with modest training histories has been noted, DGE has been reported in 12–50 % of extensively trained veteran athletes [48, 59, 60]. However, the patches of DGE are very small and are focused around the septum and RV insertion points, a region that may indicate local mechanical stresses rather than extensive fibrosis. The relationship between the presence of DGE and long-term outcomes has yet to be defined, and further study is needed.

There has been speculation about whether noninvasive surrogates of cardiac injury may identify athletes at greater risk. Increases in cardiac troponin and beta-type natriuretic peptide after endurance sporting events reflect myocardial injury and have been reported [61]. Significant increases in serological markers, potentially indicating myocardial damage, during and/or directly after a marathon run have been reported [56, 62, 63]. However, the elevated cardiac biomarkers regressed to normal values within a period of 24–48 h and, therefore, may be the result of transient and reversible alterations of the cardiac myocytes without negative clinical consequences [64, 65]. Hanssen et al. [49] combined measurements of cardiac biomarkers with cardiac magnetic resonance, including DGE, demonstrating an absence of detectable myocardial necrosis despite a transient increase in cardiac biomarkers. Further experimental studies are warranted to determine the clinical consequences of these increased cardiac biomarkers.

Electrical Remodeling

In addition to structural and functional myocardial remodeling, electrical remodeling occurs in response to endurance sport training. These adaptations manifest as distinct changes on the 12-lead electrocardiogram (ECG) and are thus important for clinicians to be capable of differentiating ECG patterns which result from training and from those suggestive of the disease which may increase the risk of sudden death during sport participation [30]. The 12-lead ECG provides rapid and relatively inexpensive information about cardiac electrical conduction and myocardial structure. Therefore, the ECG is the initial test of choice in the athletic patient with symptoms suggestive of heart disease and plays an important, though still controversial, role in the pre-participation screening of asymptomatic athletes. Because of the heightened vagal tone that accompanies physical conditioning, trained athletes commonly demonstrate benign arrhythmias and conduction alterations, including sinus bradyarrhythmia, junctional rhythm, first-degree atrioventricular block, and type I second-degree atrioventricular block (i.e., Wenckebach phenomenon) [66].

Clinical criteria for differentiating adaptive/training-related ECG patterns from those suggestive of true pathologic heart disease have undergone considerable evolution over the past two decades. In 1998, Corrado et al. presented the results of a 27-year screening initiative (1979–1996) designed to protect young competitive athletes from sudden cardiac death. This landmark paper represented the first publication in which a comprehensive set of 12-lead ECG criteria was proposed for use in asymptomatic athletes [67]. The European Society of Cardiology (ESC) followed in 2005 with the first consensus document presenting quantitative ECG criteria for use in athletes [68]. Although the use of the 2005 ESC criteria was shown to significantly increase the likelihood of detecting underlying cardiomyopathy in athletes, its use was associated with a prohibitive rate of false-positive testing [8]. Specifically, utilizing transthoracic echocardiography as the gold standard for the presence or

absence of structural and valvular heart disease, 12-lead ECG-inclusive screening using the 2005 ESC criteria was associated with a false-positive rate of 16.9 %. The majority of false-positive testing included several common ECG patterns (isolated precordial and limb lead QRS voltage, isolated left atrial enlargement, and incomplete right bundle branch block) and were found almost exclusively among athletes who were found by echocardiography to have evidence of physiologic cardiac remodeling.

The ESC updated their criteria in 2010 [69]. In this update, the writing group divided ECG patterns into two specific groups. "Group 1" was a list of patterns designated as "common and training related" that should not prompt further evaluation for underlying pathology. Key components of the "Group 1" list included sinus bradycardia, first-degree AV block, incomplete right bundle branch block, "early repolarization," and isolated QRS voltage criteria for left ventricular hypertrophy. In parallel, a second group of ECG patterns was designated as those that are "uncommon and training unrelated" among athletes. "Group 2" included many well-established markers of occult structural and electrical diseases including T-wave inversions, ST segment depression, pathologic Q waves, conduction abnormalities including complete bundle branch and left bundle fascicular blocks, gender-specific abnormalities of QT interval duration, and preexcitation.

Most recently, the "Seattle Criteria" [70] and subsequent "revised criteria" [71] have been developed. In sum, these criteria continue to improve the specificity of ECG interpretation in athletes by assigning a benign nature to several common ECG patterns including equivocal QTc intervals, T-wave inversions isolated to leads V1 and V2, and either isolated right axis deviation or right ventricular hypertrophy voltage criteria [72, 73].

Evaluation of the Symptomatic Endurance Athlete

Although participation in sport and regular exercise promotes good health, athletes are not immune to cardiovascular symptoms and disease. Sudden death of an athlete is a tragic event. These deaths often assume a high public profile because of the youth of the victims and the generally held perception that trained athletes constitute the healthiest segment of society. Sudden death during sport is most commonly caused by occult cardiovascular disease [74], and many of the key cardiovascular diseases responsible for sudden death first manifest as symptoms during exercise. The cardiovascular causes of sudden death in athletes have been well documented, and pathology is often age related [66, 74–77]. In the United States, among people <35 years old, genetic heart diseases predominate, with hypertrophic cardiomyopathy being the most common, accounting for at least one-third of the mortality in autopsy-based athlete study populations [74, 75, 77]. Congenital coronary anomalies (usually those of wrong sinus origin) are second in frequency, occurring in 15–20 % of cases. For older athletes (>35 years of age), atherosclerotic coronary artery disease is the predominant cause of sudden death [74]. Symptoms can first manifest during exercise when there are increased cardiovascular demands. Symptoms may be suggestive of underlying pathology and have long-term prognostic implications. The possibility of coronary or other life-threatening cardiac diseases in "physically fit" endurance athletes who have specific cardiac symptoms must be considered. Symptoms sufficiently severe to interfere with an athlete's performance should be exhaustively investigated, and a cardiac cause must be presumed until disproven. The following sections address the most common cardiovascular issues encountered in the clinical care of the endurance athlete.

Chest Pain

Chest pain is a common complaint seen in athletes across the entire age spectrum, and the underlying etiologies of chest pain are both myriad and referable to many organ systems [78] (Table 1.1). The term "chest pain" encompasses vague sensations that carry a low likelihood of cardiac etiology to typical angina that is commonly

Table 1.1 Common cardiac and noncardiac causes of chest pain among endurance athletes

Common cardiac causes	Typical presenting symptoms
Obstructive CAD	Chest pressure or heaviness; pain radiates to the neck, jaw, shoulder, and/or left arm
Coronary vasospasm	Chest pressure or heaviness; pain radiates to the neck, jaw, shoulder, and/or left arm
Aortic or coronary artery dissection	"Tearing" pain, sudden and excruciating pain in the anterior chest radiating to the back or scapula
Anomalous coronary artery	Exertional chest discomfort at high levels of exertion. Exertional shortness of breath often mislabeled as "asthma." Can manifest as syncope or cardiac arrest without premonitory symptoms
Valvular disease	Exertional chest discomfort at high levels of exertion. Exertional shortness of breath often mislabeled as "asthma." Can manifest as syncope or cardiac arrest without premonitory symptoms
Myo- or pericarditis	Often manifests within days to weeks of a clear noncardiac viral infection. Symptoms variable. Positional chest pain that is most severe when lying supine and relieved at least in part by sitting forward; dyspnea and exercise intolerance
Tachyarrhythmias	Palpitations may be associated with chest pressure or heaviness, shortness of breath, and syncope
Noncardiac causes	
Respiratory	Pulmonary embolism
	Pneumonia/pleurisy
	Pneumothorax
Gastrointestinal	Gastroesophageal reflux disease
	Peptic ulcer disease
	Cholecystitis
	Pancreatitis
Musculoskeletal	Costochondritis
	Trauma
	Strain
Other	Anxiety, depression
	Anemia
	Thyrotoxicosis
	Herpes zoster

associated with pathologic underlying cardiovascular disease.

Athletic patients presenting with chest pain most often experience their symptoms during physical exertion. In patients less than 35 years of age, congenital valvular heart disease; genetic cardiomyopathies, such as hypertrophic cardiomyopathy (HCM); and coronary anomalies/malformations are the leading causes of cardiac chest pain. In contrast, among patients over 35 years of age, obstructive coronary artery disease due to atherosclerosis is the most common cause of exertional chest pain [75, 76]. Chest pain can be divided into two categories: chest pain resulting from myocardial ischemia and chest pain from other causes. Myocardial ischemia results from an imbalance between myocardial oxygen demand and coronary blood flow. The most common causes of ischemic chest pain in athletes include atherosclerotic coronary artery disease (typically in older patients), anomalous coronary arteries (typically in younger patients), and genetic cardiomyopathies, most often HCM. Ischemic chest pain can also be caused by less frequent entities including coronary vasospasm, coronary artery dissection, and valvular heart disease, including aortic stenosis (both congenital and acquired), severe anemia, and thyrotoxicosis. Congenital coronary artery anomalies originating from the contralateral sinus of Valsalva and following a course between the aorta and pulmonary artery are a common cause of exertional chest

pain and exercise-related sudden death in athletes younger than 35 years and the literature pretaining to the general population has relevance. Two studies examining atypical chest pain leading to hospital admission found that the causes fell into five categories: musculoskeletal, cardiac, gastrointestinal, respiratory, and miscellaneous. Musculoskeletal causes were the most common (27 %) [79], and in our experience, this finding also applies to athletes with chest pain. A careful physical examination of the chest wall can be relied on to differentiate musculoskeletal chest pain from other causes.

There are several important cardiac conditions that can cause chest pain without myocardial ischemia. The most common are pericarditis, myocarditis, and aortic dissection. Pericarditis and myocarditis are inflammatory diseases of the heart lining and muscle, respectively, which are most often caused by infectious agents (most commonly viruses). Both conditions often follow clinically appreciable infectious syndromes and are typically characterized by positional chest discomfort, fatigue, and in some cases palpitations. Aortic dissection, acute tearing of the aortic wall, is a surgical emergency that classically presents with "tearing" chest or back pain, often localized to the mid-scapular region. Among athletes, aortic dissection most commonly occurs in individuals with underlying aortopathy and is usually precipitated by intense isometric actions (i.e., lifting heavy weight) or direct blunt chest wall trauma. Pre-participation screening, whether confined to standard medical history and physical examination or inclusive of a resting or exercise 12-lead ECG, has limited capacity to identify coronary artery anomalies. Therefore, anomalous coronary lesions should be suspected in the athlete who presents with chest discomfort during or immediately after exertion, even if the athlete has been previously screened with a pre-participation exam. When this diagnosis is suspected, direct, noninvasive imaging of the coronary anatomy is required. Transthoracic echocardiographic imaging is capable of accurately defining coronary anatomy in 90 % of athletes [80]. Given the absence of radiation exposure and the widespread accessibility of this technique, this is the initial diagnostic imaging test in this setting. If transthoracic echocardiography does not yield definitive images of the coronary origins and proximal course, magnetic resonance imaging [79, 81, 82] or computed tomography [83] should be considered, based on institutional preferences and insurance reimbursement patterns.

Syncope

Syncope, transient loss of consciousness followed by complete and spontaneous return to baseline mental status, is common among athletes. In a large series of young Italian athletes, approximately 6 % experienced some form of syncope during their athletic careers [84]. Etiologies of syncope in the endurance athlete range from the benign neurally mediated collapse to life-threatening pathologic conditions, including structural heart disease and primary arrhythmias.

Most syncopes in athletes are attributable to neurally mediated mechanisms involving predominant and often excessive vagal tone related to chronic exercise training. Syncope frequently occurs in athletes outside of sports participation. Typical triggers include anxiety, sudden postural changes, and painful stimuli. While often recurrent, non-exertional, neurally mediated syncope in the athlete is usually a benign condition. A closely related and common fainting syndrome is "post-exertional" syncope, often informally referred to as exercise-associated collapse. Post-exertional syncope occurs following abrupt cessation of exercise, most commonly moderate- to high-intensity endurance exercise, and is caused by a rapid reduction in central venous return. This reduction in venous return, caused by both the cessation of skeletal muscle contraction and altered sympathetic/parasympathetic balance, causes transient cerebral hypoperfusion. The affected athlete typically reports prodromic feelings of warmth, light-headedness, or diaphoresis, which begin within seconds of exercise termination and rapidly culminate in a loss of consciousness lasting from several seconds to a minute. Athletes predisposed to this condition should be

Salt, Sips, Stop, Socks, Sex, Sleep	
	Dietary salt augmentation in the form of liberalized table salt use and consumption of salty snacks prior to training and competition
	Adequate hydration with an emphasis on the ingestion of 18-24 oz. of electrolyte containing fluid prior to training and competition
	Avoid sudden termination of exercise. Emphasize the importance of active cool down sessions after training and competition
	Use of commercially available compression stockings
	Adequate sleep and recovery particularly during times of high intensity / high volume training
	More common in female athletes, particularly those initiating or discontinuing oral contraceptive therapy for birth control or regulation of menses

Fig. 1.1 Conservative steps for the clinical management of neurally mediated syncope among endurance athletes

counseled as this problem has a high rate or recurrence in the absence of avoidance-based therapy. In our practice, we employ the "6-S" algorithm (salt, sips, stop, socks, sex, sleep) as shown in Fig. 1.1.

Syncope that occurs during intense exercise, either with or without prodromal symptoms, should raise suspicion for underlying cardiac disease. Traumatic injury secondary to syncope should further increase the index of suspicion for underlying disease. Current American College of Cardiology/American Heart Association (ACC/AHA) guidelines recommend that the approach to the athlete with exertional syncope begin with a meticulous history, physical examination, resting 12-lead electrocardiogram, and echocardiogram [85]. The medical history should characterize potential triggers, the timing and duration of the event, and the risks associated with future episodes of loss of consciousness. Physical examination should be directed toward signs, most often pathologic murmurs, of occult heart muscle and valve diseases. The resting 12-lead ECG should be inspected for abnormalities of conduction (QT prolongation, preexcitation, pathologic right bundle branch block with early precordial ST elevations suggestive of Brugada syndrome) and findings suggestive of structural heart disease (left bundle branch block, LVH with repolarization abnormalities, diffuse

T-wave inversion). Transthoracic echocardiography is recommended to exclude structural and valvular heart disease in individuals with syncope, especially if any abnormality is detected during medical history, physical examination, or ECG interpretation.

The comprehensive assessment of true exertional syncope often extends far beyond these basic measures and must be tailored to exclude underlying structural and electrical heart disease. We recommend such evaluations be conducted under the supervision of a cardiovascular specialist with expertise in the care of athletic patients. Provocative and customized exercise testing is often high yield and should be designed to approximate the exercise conditions in which the syncope occurred. Careful attention should be given to the exercise ECG for the detection of explanatory arrhythmias. When lab-based exercise testing is inconclusive, ambulatory rhythm monitoring may be necessary. There are numerous ambulatory rhythm monitoring devices available, and the choice should be dictated by the frequency and duration of syncope on an individual patient basis. The use of tilt-table testing in athletes, due to high rates of false-positive testing, is of limited value. In very select cases, most often in the setting of a documented arrhythmia syndrome, invasive electrophysiologic study (EPS) may be both diagnostic and potentially therapeutic [86].

Management of the athlete with syncope is dictated by cause. Individuals with significant structural or valvular heart disease should be managed with appropriate sport restriction, medication, EPS with or without ablation, implantable defibrillator placement, or surgery on the basis of specific pathology [83]. Neurally mediated post-exertional syncope can often be avoided by mandating an active cooldown period after exertion and by paying attention to hydration and supplemental salt intake. In athletes with recurrent neurally mediated syncope despite these first-line treatments, postural training or pharmacologic therapy may be reasonable; however, these should be prescribed only after careful consideration of rules delineating banned substances in athletes.

Palpitations

Palpitations are a frequent complaint in athletes. The heightened vagal tone that accompanies routine exercise training commonly results in marked sinus bradycardia, and the relatively lengthy time period between sinus beats may facilitate an increase in ectopic atrial and ventricular beats. Slow resting heart rate coupled with the keen body awareness that is typical among athletic patients often leads to sensing of these spontaneous depolarizations. In the majority of cases, resting palpitations that are suppressed with exercise, particularly among athletes with no findings during medical history, physical examination, and 12-lead ECG, are a benign phenomenon that requires no further evaluation [87].

The athlete presenting either with palpitations that are exacerbated by exercise or with other findings suggestive of occult cardiac disease requires a comprehensive evaluation by a cardiovascular specialist with expertise in the care of athletic patients. A broad differential diagnosis must be considered. Noninvasive cardiac imaging, customized exercise testing designed to stimulate the demands of training and competition, and ambulatory rhythm monitoring play a key role in the evaluation. The choice of a specific ambulatory rhythm monitoring device is crucial and requires individualized decision making. For symptoms that predictably recur within a 24-h period, simple Holter monitoring may be adequate. If the athlete experiences less frequent, more intermittent symptoms, they are best evaluated with a continuous loop recorder or event (patient-triggered) monitor. For those with very infrequent or elusive symptoms (>1 month between symptoms), an implantable loop recorder may be required.

Palpitations due to benign atrial and/or ventricular ectopy that occur at rest are suppressed by exercise, are not accompanied by primary

structural or electrical diseases, and are best managed conservatively. Often counseling geared toward patient reassurance and avoidance of potential triggers including alcohol, caffeine, and stimulants are sufficient. Among athletic patients with palpitations caused by pathology, disease-specific therapies designed to reduce the burden of arrhythmia (i.e., pharmacologic suppression, intracardiac ablation) and to reduce the risk of sudden cardiac death (i.e., sports restriction, implantable defibrillator placement) are often indicated.

Atrial Arrhythmias

Atrial fibrillation (AF) is the most common arrhythmia in the general population [88]. A rapidly growing body of literature suggests that long-standing participation in endurance exercise may increase the prevalence of atrial fibrillation in athletes [89]. Mechanistic underpinning of this observation remains unknown but is likely multifactorial with contributions from age, years of training, structural remodeling (atrial stretch), scarring, atrial inflammation, changes in the autonomic nervous system (increased vagal tone), hypovolemia, genetic predisposition, and illicit drug use.

A meta-analysis of six studies that involved 655 athletes engaging in diverse sporting disciplines reported a fivefold risk of AF [90]. A recent large study of 52,000 long-distance cross-country skiers demonstrated that atrial fibrillation was the most common arrhythmia (1.3 %) and was related to the number of races competed and faster finishing times, surrogates of endurance training volume and duration, respectively [91]. This has been supported by others who documented lone atrial fibrillation with endurance athletes (mean age 44 years) who report a lifetime practice of sport exceeding 1500 h [92–94].

In an effort to identify as potential modulators and triggers of arrhythmias, Wilhelm studied a cohort of 70 randomly selected athletes entering a ten-mile running race and demonstrated an increase in left atrial volume and P-wave prolongation as evidence of altered atrial substrate and increased vagal tone and atrial ectopy, respectively [39]. The alterations seen in the atria with chronic endurance training may also adversely affect the sinoatrial and atrioventricular nodes.

Treatment and prognostication of endurance athletes with atrial fibrillation is difficult due to the paucity of large-scale prospective, randomized clinical trials and guidelines focusing directly on this population. In the absence of such resources, current guidelines for the management of atrial fibrillation in the general population must be considered when caring for endurance athletes. Therapeutic options for athletes with AF must be individualized and may include strategies including restriction of endurance exercise dose, rhythm maintenance pharmacotherapy, and intracardiac ablation. We routinely assess the risk–benefit balance of anticoagulation pharmacotherapy for all athletes with AF and develop an individualized strategy that emerges from a shared decision-making strategy.

Long-Term Effect of Chronic Endurance Training

Regular exercise is highly effective for prevention and treatment of many chronic diseases and improves cardiovascular (CV) health and longevity. The American Heart Association recommends at least 30 min of moderate-intensity aerobic activity at least 5 days per week for a total of 150 min for overall cardiovascular health. Moderate-intensity exercise is considered to be any activity causing a raised heart rate and increased breathing but being able to speak comfortably and includes a brisk walk at 4 mph or cycling at 10–12 mph. The intensity of physical exercise is usually expressed in terms of energy expenditure or metabolic equivalents (METs). One MET represents an individual's energy expenditure while sitting quietly for 1 min (equivalent to about 1.2 kcal/min for a person weighing 72 kg). Moderate-intensity exercise is equivalent to 3–6 METs, whereas athletes typically

perform in excess of 15 METs [95]. The "dose" of exercise consists of a number of factors, including intensity (how hard), duration (how long), and frequency (how often).

People who exercise regularly have markedly lower rates of disability and a mean life expectancy that is 7 years longer than that of their physically inactive contemporaries [96–98]. It is clear that mild to moderate exercise is better from a health outcomes perspective than no exercise; it remains unknown and actively debated whether an upper limit of exercise dose exists above which adverse effects outweigh benefits [50]. Preliminary data suggests that there may be a U-shaped relationship between exercise dose and adverse cardiovascular events [99, 100]. Among 1000 joggers prospectively studied, those who ran for 30–45 min, three times a week, had a lower mortality than sedentary non-joggers, while more strenuous joggers had a mortality rate that was not statistically different from this study's sedentary group. Similarly, a 15-year observational study of 52,000 adults found that runners had a 19 % lower risk of all-cause mortality compared with non-runners, with U-shaped mortality curves for distance, speed, and frequency. Running distances of about 1–20 miles per week, speeds of 6–7 miles per hour, and frequencies of 2–5 days per week were associated with lower all-cause mortality, whereas higher mileage, faster paces, and more frequent runs were not associated with better survival [101]. In our opinion, these data generate important hypotheses that will require further work to confirm or refute.

Sudden Death During Endurance Competition

Sudden cardiac death (SCD) during endurance sporting events is a rare phenomenon. Collectively, this topic has been most thoroughly examined in the context of marathon running as summarized in Table 1.2. The largest database documenting sudden death during marathon running was comprised by the RACER study group. Kim et al. [62] investigated the incidence and outcome of cardiac arrests during marathon and half-marathon races in the United States during the decade spanning from 2000 to 2010. In the 10.9 million runners examined, 59 cases of cardiac arrest occurred in association with either marathon or half-marathon races. Men were significantly more likely to experience cardiac arrest and SCD than women. The event rates were significantly correlated with the age of the runners and significantly more frequent in "full" marathon runs (42.195 km) than in "half"-marathon runs. Notably, more than half the cases of sudden cardiac death occurred in the final mile.

The RACER authors calculated that the incidence rates of cardiac arrest and sudden death during long-distance running races were 1 per 184,000 and 1 per 259,000 participants, respectively. Thus, event rates among marathon and half-marathon runners are relatively low, as compared with other athletic populations, including collegiate athletes (1 death per 43,770 participants per year) [102], triathlon participants (1 death per 52,630 participants) [103], and previously healthy middle-aged joggers (1 death per 7620 participants) [104], suggesting that the risk associated with long-distance running events is no higher and may be lower when compared with other vigorous physical activities. These rates compare favorably with those presented by other investigators.

Conclusion

In summary, since the days of Henschen and Darling, understanding of sport-specific changes in cardiac structure and exercise-induced cardiac remodeling has advanced significantly. In order to define the natural history of endurance-induced cardiac remodeling, longer-duration longitudinal study is required. Examination of prolonged exposures to exercise training will be necessary to determine normative values across the age and training spectrums of athletic patients.

Table 1.2 Major reports

Studies	Race	# of runners	Deaths *N* (rate)	Cardiac arrest *N* (rate)	Mean age (years)	Male (%)	Etiology
RACE Paris registry (2006–2012) [105]	Marathon, half-marathon, 20 K	511,880	2 (0.4/100,000)	9 (1.8/100,000)	42.8	100	Eight MI, three arrhythmias, one ARVD, five heat stroke
RACER (2000–2010) [62]	Marathon, half-marathon	10,900,000	42 (0.4/100,000)	59 (0.5/100,000)	42	86	14 HCM +/− other, one heat stroke, one ARVC, two hyponatremias, seven presumed dysrhythmias, seven MI, three unknown
Webner (1976–2009) [106]	Marathon	1,710,052	10 (1/171,005)	30 (1/57,002)	49.7	93	Seven CAD, one ACA, one unknown, one no autopsy
Redelmeier and Greenwald (1975–2004) [107]	Marathon	3,292,268	26 (0.8/100,000)	N/A	41	81	21 CAD, four electrolyte abnormalities, two ACA, one heat stroke
Maron and Roberts (1976–1994) [108]	Marathon	215,413	4 (0.5/100,000)	0.9/100,000	39	75	Three CAD, one ACA
Maron and Roberts (1995–2004) [109]	Marathon	220,606	1 (0.45/100,000)	3 (1.4/100,000)	47	100	Three CAD, one mitochondrial myopathy

HCM hypertrophic cardiomyopathy, *ARVC* arrhythmogenic right ventricular cardiomyopathy, *CAD* coronary artery disease, *ACA* anomalous coronary artery, *MI* myocardial infarction

This information, coupled with the impact of detraining, will help to more clearly distinguish the boundary between physiology and pathology in athletic patients.

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