

The prevention and management of sudden cardiac arrest in athletes

Yehia Fanous MD, Paul Dorian MD

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Cardiac arrests that occur during sports competition are dramatic and unexpected, and attract much media interest, as athletes are often young and at the peak of physical fitness. The rate of sudden cardiac arrest during exercise in competitive athletes is about 0.75 per 100 000 athletes per year.^{1,2} Among young athletes, sudden cardiac arrest is usually the first manifestation of underlying cardiac pathology, although one retrospective study found that 29% of athletes had symptoms suggestive of cardiac disease before an arrest.³ Questions arise about the effect of intensive exercise on the heart, the potential value of screening all who would engage in competitive sport for cardiac abnormalities to reduce the incidence of sudden cardiac arrest, and optimal resuscitation protocols to improve survival in those affected. We review evidence related to these questions in light of recommendations in the recent position statement from the Canadian Cardiovascular Society and the Canadian Heart Rhythm Society on pre-participation screening of athletes⁴ (Box 1).

What causes sudden cardiac death in athletes?

Among athletes 35 years of age and younger, the most common underlying conditions (Box 2) identified on clinical examination or at autopsy are “primary electrical disease” with no specific cause found, idiopathic left ventricular hypertrophy, coronary artery anomalies, myocarditis, and heritable disorders such as hypertrophic cardiomyopathy.^{1,3,5-7} Among athletes older than 35 years, coronary artery disease is the most common cause of cardiac death during exercise, and this is by far the most common cause among those older than 45 years.⁸⁻¹⁰

What is the relation between exercise, cardiac disease and sudden cardiac arrest?

When considering the effect of exercise on the heart, it is important to consider the extent to which exercise might either prevent or lead to cardiac disease in a previously healthy heart, and the extent to which it may worsen pathology in a diseased heart.

In the absence of predisposing conditions or symptoms of heart disease, exercise, even at high intensity, poses very little risk to an athlete. The ways in which regular exercise remodels the heart is explained in Box 3. The benefits an individual derives

KEY POINTS

- Sudden cardiac arrest among competitive athletes during exercise is a rare event, usually but not always associated with underlying cardiac pathology.
- Exercise is an effective way to moderate overall cardiac risk in the population, but it is paradoxically both risky and beneficial for people with pre-existing heart disease, though the benefits outweigh the risks.
- Exercise may increase the rate of sudden cardiac arrest in athletes with pre-existing cardiac conditions; however, this is difficult to predict or prevent, which means that screening athletes for underlying cardiac pathology is complex, challenging and of uncertain benefit.
- A recent Canadian position statement recommends a tiered approach to pre-participation screening, advises against the routine use of electrocardiography as a screening tool, and emphasizes the importance of shared decision-making when considering whether to continue or withdraw from competitive sport.
- Establishing effective resuscitation protocols and increasing the availability of automated external defibrillators in settings where competitive sport is undertaken are the most effective strategies in helping reduce the incidence of sudden cardiac death among athletes.

by participating in exercise or sport include a reduced risk of mortality and morbidity, and substantial social and psychological gains.^{18-20,24} One possible exception is the increased risk of developing lone paroxysmal atrial fibrillation in the absence of heart disease, specifically observed in long-term endurance athletes who exercise for 15–30 hours per week for decades or longer.¹⁷ A recent meta-analysis found that such athletes have an increased risk of developing lone paroxysmal atrial fibrillation (odds ratio 1.64, 95% confidence interval [CI] 1.10–2.43) compared with the risk for sedentary people.²⁵ However, the risk of stroke in athletes with atrial fibrillation is lower than that in age-matched non-athletes with atrial fibrillation.²⁶

For most people with chronic conditions such as hypertension, coronary disease with myocardial infarction (MI), and diabetes, engaging in regular exercise reduces the extent of adverse cardiac remodelling.^{27,28} High-intensity interval training in patients with heart failure, hypertension and prior MI has been reported to be safe and to increase exercise capacity.²⁸

In patients with hypertrophic cardiomyopathy, the balance between the risk and benefit is not well understood, with possible risks at very high intensities of exercise.^{29,30} A recent cardiac rehabilitation study highlighted the safety and benefit of moderate-intensity exercise in asymptomatic patients with hypertrophic cardiomyopathy.³¹ An important exception to these reassuring observations is arrhythmogenic right ventricular cardiomyopathy, a heritable cardiomyopathy characterized by fibrofatty infiltration predominantly in the right ventricle. In those with this type of cardiomyopathy, frequent exercise, particularly high-intensity exercise with a resistance component, has been shown in cross-sectional studies to increase the risk of disease progression, severity of the expressed phenotype, and the risk of ventricular arrhythmia.³² This condition is found in only 4%–14% of cases of sudden cardiac arrest in young competitive athletes.^{3,5,32}

Exercise has been shown to be both risky and beneficial for people with existing heart disease, creating something of a paradox. The instantaneous risk of a catastrophic event is transiently increased for such people during and immediately after exercise;

Box 1: Evidence used in this review

We consulted recent Canadian and international guidelines on screening athletes to guide this review. We conducted a search of Ovid and the Cochrane Library for articles within the past 5 years using key terms related to “sudden cardiac death” or “arrest” in athletes, “the athlete’s heart,” “pre-participation screening” and “automated external defibrillation in sports.” Most evidence cited is observational in design.

Box 2: Causes of sudden cardiac arrest in athletes*^{1,3,5-10}

Age group ≤ 35 yr

- Primary electrical disease with no specific cause identified
- Idiopathic left ventricular hypertrophy
- Anomalous origin of the coronary arteries
- Heritable cardiomyopathies:
 - Hypertrophic cardiomyopathy
 - Arrhythmogenic right ventricular cardiomyopathy
- Myocarditis
- Electrical disorders:
 - Long QT syndrome, catecholaminergic polymorphic ventricular tachycardia

Age group > 35 yr

- Coronary artery disease
- Idiopathic left ventricular hypertrophy
- Heritable cardiomyopathies:
 - Hypertrophic cardiomyopathy
 - Arrhythmogenic right ventricular cardiomyopathy
- Primary electrical disease with no specific cause identified
- Myocarditis

*In order of decreasing prevalence.

furthermore, this risk increases with increased duration of exertion.³³ The instantaneous acute relative risk of sudden death (compared with at rest) for women is 2.38 (95% CI 1.23–4.60) at an absolute risk of 1 per 36.5 million hours of exertion.³⁴ The increased risk is attributable to increases in myocardial demand and adrenergic output, which can induce an arrhythmogenic state or ischemia, precipitating sudden cardiac arrest.³⁵ However, both the lifetime and acute instantaneous risk of sudden cardiac death are decreased in those who perform habitual exercise; the instantaneous risk is no longer significant in those who participate in 2 or more hours of exercise per week (relative risk 1.49, 95% CI 0.61–3.61).^{33,34} This benefit is a consequence of the positive structural

Box 3: The athlete’s heart

Systematic engagement in regular exercise results in predictable changes to the cardiac architecture, termed “structural” and “functional” remodelling. The physiologic adaptations that occur in response to exercise are often evident on standard 12-lead electrocardiography. Such findings are considered normal in an athlete and typically do not warrant further investigation.¹¹

Exercise-induced remodelling

Exercise-induced cardiac remodelling occurs as an adaptation to permit and enhance the increased cardiac output required during exercise. Such changes are specific to the exercise loads encountered in a particular sport.

Structural remodelling

Structural remodelling results in increases in myocardial wall thickness (concentric hypertrophy) and in chamber size (eccentric hypertrophy).^{12,13} This results in normalization of the left ventricular mass-to-volume ratio with prolonged training.¹³⁻¹⁵ Functional remodelling results in higher vagal tone and slowing of resting heart rate, more rapid heart rate recovery after exercise and improved diastolic function.¹⁴

Does physiologic remodelling from exercise cause harm?

In a small minority of athletes, the degree of structural adaptation resembles structural changes seen in pathologic states, such as hypertension-induced left ventricular hypertrophy, hypertrophic cardiomyopathy and arrhythmogenic right ventricular cardiomyopathy.¹⁶ In middle-aged competitive endurance athletes with decades of high-intensity endurance sport, a greater than expected frequency of myocardial fibrosis and coronary calcification, detected on imaging, has intensified debate on whether “high doses” of exercise can damage the heart and cause maladaptive (“adverse”) remodelling, with possible harmful consequences.¹⁷

It is difficult to reconcile these concerns with the well-established observed reduction in cardiovascular death observed after many years of low-, moderate- and high-intensity exercise.^{18,19}

One unproven hypothesis suggests that very high-intensity exercise over decades, in healthy individuals, can increase the relative risk of cardiac events, compared with moderate exercise.¹⁷ Such a U-shaped curve of mortality as a function of exercise is not seen in older long-distance ski racers, and the fastest (presumably the fittest, with most intensive training) have even larger reductions in mortality than their slower counterparts.^{20,21} It is important to emphasize, however, that the benefits of exercise are near maximal at as little as about 10 metabolic equivalent of task (MET) hr/wk of leisure time activity.²² Recent evidence indicates that ≥4–5 exercise sessions per week of endurance training is optimal for deriving structural and functional cardiovascular benefits in late life.²³

and functional adaptations, which provide protection from the pro-arrhythmic electrophysiologic, hemodynamic, autonomic and coagulation changes associated acutely with exertion and chronically with dysfunctional remodelling.^{34,35}

Is there value in pre-participation screening to prevent sudden cardiac arrest?

Pre-participation screening refers to use of a systematic approach to the evaluation of athletes before competition or participation, to identify existing cardiac pathology that may put an athlete at risk of sudden cardiac arrest, and to identify any other important health concerns. The premises of screening are that athletes at high risk of sudden death can be identified (prediction), and that an intervention — usually involving restriction from competitive or high-intensity sport — will substantially reduce the risk of early death in these predisposed people (prevention).

Unfortunately, the first premise has major limitations, and the second is unproven. For “prediction” to be effective, screening needs to be sensitive and allow the detection of conditions most commonly responsible for sudden death. False-positive findings can lead to unnecessary additional testing and the unfortunate restriction of athletes who are actually at low risk. For the promise of “prevention” to be realized, it is necessary to show that sports restriction truly reduces the risk of death.

The only study to suggest reduced risk of death with pre-participation screening of competitive athletes was a retrospective cohort study in which the control (unscreened) population had a sudden death rate much higher than in unscreened populations reported in subsequent studies of sports-related sudden cardiac death.^{2,36} Many guidelines from different organizations have made recommendations regarding the process of pre-participation screening. Medical and family history and a corresponding physical examination are recommended as part of all routine screening programs.^{4,37} However, the use of electrocardiography (ECG) in the screening process remains controversial owing to a paucity of evidence supporting its use.^{4,8,9}

What is the best approach to screening athletes in a primary care setting?

History and physical examination

Since a retrospective study found that 29% of athletes who died suddenly during competition had symptoms suggestive of cardiac disease before cardiac arrest,³ it is important to routinely ask athletes the following questions:³⁷

1. Have you ever felt severely dizzy or faint, unexpectedly short of breath, or had chest pain during or immediately after exercise? (Athletes should also be instructed to report these symptoms if they occur at any time after initial screening.)
2. Do you have any first-degree relatives who died suddenly or had severe cardiac disease under age 60?

According to the recently published joint position statement from the Canadian Cardiovascular Society and Canadian Heart Rhythm Society on the cardiovascular screening of competitive athletes, physical examination should emphasize measurement

and comparison of blood pressure in both arms, auscultation for heart murmurs, and examination for physical features of Marfan syndrome.⁴

12-lead ECG

Most recent studies of cardiac screening in athletes have found a 2%–4% rate of abnormalities requiring further testing,³⁸ with a rate of clinically significant abnormalities in about 0.3% of athletes.^{37,39} According to expert consensus, routine use of ECG as a screening strategy for athletes carries low specificity and requires the use of strict interpretation criteria; many of the disorders that can cause sudden cardiac arrest in athletes, including primary electrical disease, anomalous origin of the coronary arteries, and premature coronary artery disease, will not be detected on standard ECG.¹¹

Among younger patients, the ECG is less sensitive in detecting cardiomyopathies than previously thought, showing typical abnormalities in only 25% of adolescents with arrhythmogenic right ventricular cardiomyopathy and 50%–75% of asymptomatic young patients with hypertrophic cardiomyopathy.^{40,41} A recent longitudinal study of adolescent elite soccer players found that 6 out of 8 sudden cardiac deaths (a sudden cardiac arrest incidence of 6/100 000 per year) occurred in athletes who had a negative screen on history, physical examination and ECG (as well as on echocardiography) and were deemed “healthy.”³⁹ The study highlighted the challenges in preventing sudden cardiac deaths in athletes, even with extensive screening protocols. In addition, interpretation of athlete ECGs requires considerable skill, and interobserver variability remains high, even among experienced interpreters of ECGs.⁴²

How should athletes with notable findings on screening be counselled?

About 0.3% (3/1000) of screened athletes will be identified by a screening protocol as “possibly at risk.”^{37,39} These athletes may wonder, “What is my risk of sudden death if I continue to compete and exercise?” and “By how much is my risk reduced if I stop competitive sport?” The answers to these questions are challenging and specific to the sport, the type of disorder and the anticipated intensity, frequency and duration of training involved. For every 100 000 athletes screened, about 300 will be identified as possibly at risk and fewer than 1 per year will die suddenly. Among athletes who screen positive, fewer than 0.33% (3.3/1000) will die suddenly (0.001% of screened athletes).^{1,37,39} Importantly, not all athletes identified as “at risk” during the screening process have a relevant diagnosis, that is, a diagnosis for which there is an established increased risk of sudden cardiac arrest.³⁹

These observations underscore the potential harms of a screening program, which in most cases will identify asymptomatic abnormalities not destined to cause harm. Harms may include heightened anxiety for the patient and family, further unnecessary testing, negative changes to lifestyle and adverse economic consequences (such as difficulty with obtaining insurance).⁴³

Based on the best available evidence — observational studies — the recent Canadian position statement recommends a tiered

approach to pre-participation screening⁴ rather than the routine use of ECG screening in all athletes. A focused history and physical examination are augmented, when clinically appropriate, by ECG investigation. Primary care physicians involved in the care of athletes should favour a focused history and physical examination to screen for cardiac abnormalities that increase risk of sudden cardiac death, with additional ECG or other investigations only as indicated.⁴ If an abnormality is found or suspected, consultation with a cardiologist with expertise in the care of such athletes should guide further testing and treatment.

Importantly, the position statement emphasized shared decision-making, in which the values and preferences of an informed athlete are a crucial component of a decision about further testing and sport participation, taking into account the best estimates of the likelihood of a life-threatening event with or without continued competitive sport.⁴ Should athletes at risk be prohibited from participating in sports, despite our lack of ability to predict these events, even among compromised athletes? Even if these events were accurately predictable, would athletes decide to disengage from their sport? In the shared decision-making model, the physician helps the athlete to deliberate and explore the options of treatment and exercise restriction, allowing an informed choice regarding the risks associated with participation in sports.⁴⁴

Framing is important in human decision-making. Athletes should be made aware of the risks of death in other common recreational activities to put the risk of sudden cardiac death in sports in context. Among recreational skiers and snowboarders, the risk of death is as high as 0.25/100 000 days of exposure (days-at-risk),⁴⁵ compared with a risk of sudden cardiac death of 0.7/100 000 person-years (36.5 million days-at-risk) in competitive sport.^{1,4} The risk of death for scuba divers is 3/100 000 participants; for mountain hikers, 6/100 000; for parachutists, 18/100 000; and for mountaineers, 600/100 000.⁴⁵

What can optimize survival following sudden cardiac arrest in athletes?

Despite screening, sudden cardiac arrest may be difficult to predict and therefore to prevent among athletes. Yet, evidence points to excellent survival rates after arrest in this group when appropriate resuscitation is promptly started. A prospective study using 2 years of data from the US National Registry for AED [automatic external defibrillator] Use in Sports showed 89% survival following sudden cardiac arrest among high school athletes.⁴⁶ A recent study found a 93% survival rate following sudden cardiac arrest in Italian sports facilities with an AED on site.⁴⁷ Another recent observational study showed a 100% survival rate among 28 Japanese athletes with witnessed sudden cardiac arrest during a marathon after prompt resuscitation.⁴⁸ Even in the absence of a specific program, survival rates after sport-related cardiac arrest in Toronto, Canada, were 43.8% in competitive athletes and 44.8% in recreational athletes,¹ yet the gap between this rate and those seen in settings in which optimal resuscitation was available highlights the opportunity to reduce death rates from cardiac arrest in sport settings.

Box 4: Unanswered questions

- What is the cause of “unexplained sudden death” (with a normal autopsy) in athletes?
- What are the triggers to cardiac arrest, apart from physical activity per se? Why is it that sudden death often occurs at rest or during practice, and is often not related to maximal exertion during competition?
- If standard 12-lead electrocardiography is not optimal as a screening tool, should efforts exist to create devices or tests that augment current recommended screening protocols?

Adequately trained personnel, education of trainers and bystanders, and access to AEDs at a sports venue are crucial in reducing the incidence of sudden cardiac death after an arrest, as shown by the high survival rates seen in settings with systematic emergency response protocols and rapid deployment of AEDs.^{47,48}

Conclusion

Given the rarity of sudden cardiac arrest in athletes and the lack of a clear relation between screening and reduced cardiac death, several questions remain (Box 4). In the absence of answers to these questions, shared decision-making between athlete and physician is the best approach when athletes who are found to have an underlying cardiac abnormality are considering whether to continue or withdraw from competition.

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Affiliations: Faculty of Medicine (Fanous, Dorian), University of Toronto; Division of Cardiology (Dorian), Department of Medicine, St. Michael's Hospital, Toronto, Ont.

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Correspondence to: Paul Dorian, dorianp@smh.ca