Screening for Cardiovascular Risk in the Masters Athlete

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ABSTRACT

Regular physical activity is associated with health benefits and "successful aging" in Masters athletes (>35 years-old). Paradoxically, exercise can increase the risk of heart attacks, irregular heart rhythms, and sudden cardiac death (SCD) in those with underlying disease. SDC is, of course, a concern for both individuals and the organisers of sports events, particularly those involving Masters athletes. There are currently a number of strategies to reduce exercise-related SCD, including pre-participation screening prior to vigorous activity. In addition to detecting disease, screening creates an awareness of risk factors associated with the development of cardiovascular disease and educates on the nature and significance of warning signs. To date, however, optimal screening methods have not been universally agreed. This article starts with overviews of cardiovascular risk and the effects of aging on cardiovascular function and then examines various screening protocols in detail, including physical activity and fitness assessments, electrocardiogram, exercise treadmill test, and imaging techniques. It concludes with a summary of 12 key points and recommendations.

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Introduction

Widespread media coverage on collapses of seemingly healthy and fit athletes in competition has led to increased pre-participation screening and driven discussion in the sporting and medical communities about the most feasible means to prevent such events. However, these traumatic events continue unabated.

A possible explanation for this is the increased participation of older athletes in sports, particularly endurance competitions prompted by emotional and motivational reasons related to self-image and previous success in sport as a youth. Importantly, research indicates the benefits of physical activity in preventing early onset atherosclerosis, type II diabetes, high blood pressure, heart disease, stroke, cancer and arthritis. Thus, regular exercise and sport participation are seen as non-pharmacological means of health management and Masters athletes (those over 35 years-old) are consid-

ered to be "successful agers", slowing the inevitable aging process by achieving the upper limits of physical capacity[7].

However, although there are certainly benefits to exercise, the risk of myocardial infarction, aortic dissection, arrhythmias, sudden cardiac arrest and/or sudden cardiac death (SCD) is increased during and briefly after exercise[1].

In this article we outline the potential risk and mechanisms of SCD, discuss the effects of ageing and activity on cardiovascular function, and explore pre-participation screening prior to vigorous activity as a means for reducing the risks for Masters athletes.

Who is at Risk and Why?

Sudden cardiac death is defined as an event occurring unexpectedly - generally ≤1 hour from the onset of symptoms[2]. A notable feature of SCD is that it is often due to unidentified, asymptomatic cardiovascular disease (CVD) and is the first clinical expression of CVD[3]. In Masters athletes the primary cause of SCD is atherosclerotic disease, whereas in the under 35-year-old athlete, genetic or congenital cardiovascular abnormalities, with the most common being hypertrophic cardiomyopathy (HCM), are predominately responsible[3]. The commonality between the younger and older athlete is that they are asymptomatic and unaware of having CVD. Therefore, strategies such as pre-participation screening have been suggested to mitigate this risk.

The exercise paradox emerges from evidence that vigorous exercise simultaneously triggers and protects against SCD. Both regular trainers (i.e. competitive athletes) and non-habitual but vigorous exercisers (i.e. weekend warriors) have a two to three fold increase risk of SCD versus non-athletes[4]. In a comprehensive study conducted in France between 2005 and 2010 it was observed that 90% of sports-related deaths amongst the general population occurred during recreational sport[5].

Mechanistic reasons by which intense bouts of exercise precipitates SCD include an increase in arterial wall stress from elevated heart rate and blood pressure, coronary artery spasm in diseased segments, activation of the sympathetic nervous system, increase in circulating catecholamines and a decrease in vagal tone[1]. Such mechanisms predispose the heart muscle to lethal irregular heart rhythms, whereas regular exercise increases vagal tone and the electrical stability of the heart, protecting against irregular heart rhythms. In previously asymptomatic individuals who experienced a cardiovascular event during exercise, plaque rupture in the artery was the most common pathogenic mechanism. At cessation of exercise, a decrease in venous return, cardiac output, and blood pressure causes a transient reduction in perfusion of the coronary arteries and ischemia, potentially creating a substrate for an arrhythmia. A proper cool down at the end of exercise can protect against this.

Similarly, habitual exercise decreases the risk for triggering a myocardial infarction or SCD, while acute exercise in individuals unaccustomed to regular or vigorous exercise are at greatest risk. For instance, the relative risk ratio (RR) of cardiac arrest during exercise was 5 amongst those with the highest level of physical activity (PA) and 56 amongst those with the lowest levels of PA[6].

ALBERT et al. confirmed that habitually active men (exercising at least 5 times per week) have a much lower RR of SCD (RR=10.9) than men who exercise vigorously less than once a week (RR=74.1)[2]. Furthermore, compared to risk during periods of lighter or no exertion, vigorous exertion transiently increased the risk of SCD from a factor of 14 to 45. Interestingly, this risk was quite elevated in even the most active men.

Effect of Ageing and Activity on Cardiovascular Function

Cardiovascular function naturally declines with age in both trained and untrained individu-

als, at approximately 5-12% and 6-8% per decade for the aerobic and anaerobic systems, respectively[8]. The decline in aerobic capacity, measured by maximal oxygen uptake (VO_2max), is mostly attributed to a decline in heart rate max (HRmax) of approximately 0.8bpm/year[8].

Despite the reduction in HRmax, a concomitant reduction in VO_2max is not observed in Masters athletes, suggesting that oxygen pulse increases caused by regular training compensate for the decline in HRmax. For example, in a study comparing Masters long-distance runners and sprinters, both groups had higher levels of aerobic capacity compared to recreationally active individuals, with the endurance runners exceeding both of the other groups[7]. In another study, an Olympic level rower preserved anaerobic exercise capacity to at least the age of 40[10].

In a cross-sectional study examining the anaerobic and aerobic systems collectively, a decline in anaerobic power and capacity in both sprint and road cyclists (aged 35-64) was observed[8]. The same study showed no change in and aerobic capacity, concluding age has a greater negative effect on the anaerobic system[8]. Therefore, the probable effects of a decline in exercise capacity are the effects of sedentary lifestyles and co-morbidities rather than age.

Additionally, regular exercise has been shown to have positive effects on the arterial system, whereas age, a sedentary lifestyle, and an unhealthy diet, have negative effects, causing the arteries to become stiff and consequently increasing the workload of the heart. Regular exercise prevents stiffening by improving endothelial function, releasing vasodilators and enhancing the elasticity of the vessels. In a systematic review examining flow-mediated dilation (a measure of vascular health and a predictor of cardiovascular events) in Masters athletes compared to age-matched healthy controls, it was observed that high levels of exercise training attenuated age-related decline in vascular function[11].

What is the Optimal Dose of Exercise?

Exercising five to 10 minutes a day is associated with a marked reduction in death from all causes and CVD[12], with larger volumes of activity/exercise leading to greater benefits[13]. The Harvard Alumni study, one of the first (initial questionnaires were administered between 1962 and 1977 and then again between 1977 and 1985) and largest (n=10,629 men) studies to analyse the effect of change in risk factors (i.e. level of physical activity, smoking, blood pressure, body weight) on mortality, reported moderately vigorous sport activity (> 4.5 metabolic equivalent hours or METs) resulted in a substantial reduction in mortality from all causes (23%), and from coronary heart disease (CHD) (41%) compared to less vigorous classmates[14].

Furthermore, the most active men (> 3500 kcal per week) decreased the risk of death by half compared to the least active (less than 500 kcal per week) (RR 0.52 vs. RR 1.0, confidence interval (CI) 95%). More recently, CARRICK-RANSON et al. sought to examine lifelong exercise dose on the metabolic and hemodynamic systems, by tracking exercise frequency[15]. They reported four or more weekly sessions (aerobic activity lasting at least 30 minutes) over a lifetime (at least 25 years) was effective in preserving cardiovascular structure and function in seniors (60 years old and older). Specifically, VO_2max increased in a dose-dependent manner across the groups of exercisers being greatest in those committed to exercising (4-5 sessions/week) and Masters athletes (≥ 6 sessions/wk plus regular competitions). Improved ventricular-arterial coupling (relationship between the heart and arterial system, a determinant of cardiovascular health) and heart rate control (indicator of decreased sympathetic drive) during exercise was also greatest in these groups, with oxygen delivery and extraction improving in all trained groups (at least 2 sessions per week).

Recently, the Copenhagen City Heart Study (2015) found a U-shaped association between

all-causes of mortality and the dose of jogging (n=1,098 joggers and 3,950 healthy non-joggers). Dose was quantified by examining pace, quantity, and frequency of jogging. Compared to sedentary non-joggers, 1 to 2.4 hours of jogging per week was associated with the lowest mortality (Hazard Ratio (HR) 0.29, 95% (CI])). The optimal frequency of jogging was 2-3 times per week (HR 0.32, 95% CI) associated with a slow (HR: 0.51, 95% CI) or average (HR: 0.38, 95% CI) pace. Interestingly, the strenuous joggers did not exhibit a mortality rate statistically different from that of the sedentary group.

Many individuals exercise beyond the evidence-based recommendations, with some endurance athletes accumulating workloads of 200 to 300 MET hours per week, which is five to ten times greater than the standard recommended amount. For example, cyclists exercise for several hours on most days of the week combining aerobic with intense anaerobic dashes, frequently sustaining heart rates around 80% of max for prolonged periods. Similarly, rowers, triathletes, and swimmers also exhibit analogous combinations of endurance and power.

Counter-intuitively, it has been proposed that endurance exercise may be potentially harmful with increasing evidence revealing impairment of right ventricular (RV) function during and after activity[16], RV arrhythmias[17], left atrial dilation[18], atrial fibrillation (AF)[19] and atrial flutter[20] in long-term endurance exercisers. Increased left ventricular (LV) wall thickness is also associated with endurance exercise, but is more commonly seen in strength emphasised activities[21]. However, unlike the RV where there has been reported RV dilation, reduced RV ejection fraction (EF), and RV remodeling, intense endurance exercise does not significantly affect LV volume or function[17].

Possible mechanisms for the increased prevalence of AF and RV dysfunction and associated arrhythmias have been postulated. AF, as reported by a recent meta-analysis, has a significantly higher predominance in athletes (23%) versus non-athletes (12.5%)[19]. The potential pathophysiological mechanisms for AF and atrial flutter include increased atrial ectopic beats, shifts in electrolytes, atrial enlargement with dilation and fibrosis, increased vagal tone and bradycardia, and inflammatory changes[2].

Although an increased prevalence of complex ventricular arrhythmias in endurance-trained athlete has been reported, only limited evidence exists for exercise-induced myocardial fibrosis[23, 24].

Right ventricular myocardial fibrosis, dysfunction and arrhythmias, referred to as 'exercise-induced arrhythmogenic right ventricular cardiomyopathy (ARVC), can exist without underlying genetic abnormalities. It has been suggested that the culprit for ARVC may be long durations and repetitive 'hits' of endurance exercise, whereby repeated bouts of vigorous exercise increases stress on the RV myocardium. This later form of exercise results in apoptosis, and myocardial remodeling, by which the myocardium is replaced with fibrous tissue creating a substrate for lethal arrhythmias[16,17,25]. In turn, another probable cause may be a genetic predisposition, where a high level athlete, may unmask the latent mutation if they are a carrier[17].

Post exercise decrease in RV function has also been linked to increased cardiac biomarkers identifying myocardial damage[16]. Suggested reasons for this increase include longer race durations, higher intensities, older age, and low fitness, however, the data lacks consistency[26]. Adequate rest appears sufficient to allow for complete recovery[27], but further study is required to determine what long-term consequences of such mal-adaptations may be.

Pre-Participation Screening

Maintaining physical fitness has been scientifically proven to decrease cardiac events since a disproportionate number of events occur in individuals unaccustomed to physical activity. However, neither superior athletic abil-

ity nor regular physical training nor the absence of coronary risk factors guarantees against an exercise death[28]. Therefore, the following have been suggested as means to reduce exercise-related SCD: 1) modifying activities in high-risk individuals, 2) reporting and evaluating new symptoms, 3) training fitness personnel for emergencies, 4) making automatic external defibrillators (AED) available in all sporting venues and 5) pre-participation screening prior to vigorous activity. .

Though these steps appear prudent they lack systematic evaluation. In particular, pre-participation screening has received considerable attention in the young competitive athlete, whereas limited data exists on Masters athletes despite the higher risks for coronary artery disease Therefore, screening of the Masters athletes needs to be examined to first gain a better understand of the risks involved but more importantly to establish the appropriate method(s) to prevent cardiac events.

Currently pre-participation screening methods are dependent on the age of the athlete, country, and level of sport played. In Italy, mandatory screening has existed since 1982 for all athletes involved in competitive sports. Unfortunately, no European screening recommendations exist for cardiovascular evaluation in apparently healthy, middle-aged or older individuals participating in recreational sporting activities[4,29]. A recent study by the European Association of Cardiovascular Prevention and Rehabilitation (EACPR) aimed to establish the most practical method of cardiovascular evaluation in this population[29]. The consensus was an individual approach, with the level of testing dependent upon the individual risk profile and the intended level of physical activity. Initially, a validated questionnaire, such as the American Heart Association (AHA) or PAR-Q was used to evaluate risk. If the questionnaire results were positive and/or the intended level of activity was moderate to high, a screen by a physician would be performed. The assessment by the physician would include a family and personal symptoms history questionnaire, a physical examination, an evaluation using the SCORE (Systematic Coronary Risk Evaluation) system to assess cardiovascular risk and a resting 12-lead electrocardiogram (ECG). If the physician deemed the patient as high risk, a maximal exercise test would be performed. In the event of a positive exercise test, further evaluation, appropriate treatment and individually prescribed physical activity will be conducted. For those subjects greater than 40 years of age and involved in competitive sport, a periodical cardiovascular evaluation with exercise test to exhaustion is required.

In the United States, the American Heart Association (AHA) recommends selective screening involving a history, physical examination and a maximal exercise treadmill test for all competitive athletes above the age of 40 (men) or 50 years (women) with an additional risk factor according to family history or a blood test[30]. Unlike the EACPR, individuals are not stratified based on habitual activity levels. The American College of Sports Medicine's (ACSM) new recommendations reduce the emphasis on the need for a medical evaluation prior to initiating a progressive exercise regimen in healthy, asymptomatic individuals. The intention is to remove any barriers in order to get people active[31]. However, before engaging in physical activity a PAR-Q or modified AHA/ACSM health fitness pre-participation screening questionnaire is recommended, with a maximal exercise test for high-risk individuals (symptomatic or known cardiovascular, pulmonary, renal, or metabolic disease), and in those that are moderate risk (≥ 2 risk factors) who want to engage in vigorous activity. Further, many major marathons now mandate a medical certificate deeming the participant medically fit to participate.

Cardiovascular risk scores

Cardiovascular risk scores to predict a person's 10-year risk of cardiovascular disease has been widely accepted varying slightly in different countries. In Europe, the SCORE system is used. The calculated risk is based on age, sex, blood pressure, total cholesterol and

smoking history. An individual is considered high risk if he/she has one of the following: 1) the presence of multiple risk factors resulting in a calculated risk score of >5%, 2) markedly raised total blood cholesterol (>8mmol), low density lipoproteins (LDL) (>6mmol) or a blood pressure greater than 180/110 mmHg, 3) presence of diabetes with microalbuminuria, 4) strong family history of premature CVD in first degree relatives < 50 years of age, and a body mass index (BMI) >28[29]. Low risk is viewed as someone with a <5% risk according to the SCORE system, no history of diabetes mellitus, no family history of premature CAD and a BMI of less than 28[29].

In Canada, the Canadian Cardiovascular Society (CCS) recommends the initial risk assessment be completed in 30 to 74 year-olds with unknown CVD at the baseline examination using the Framingham Risk Score (FRS)[32]. It estimates a patient's 10-year risk by categorising risk into three categories - 0-9% (low), 10-19% (intermediate), ≥20% (high) - of developing "total" cardiovascular events. This takes into consideration the individual's age, sex, total cholesterol, high density lipoproteins (HDL), total cholesterol, blood pressure, smoking history, presence of diabetes and whether or not they are being treated for high blood pressure. The Framingham risk score ranks coronary artery disease CAD event risk in a graded fashion, with risk of CAD death or nonfatal myocardial infarction being 14.3 times higher in the high-risk group versus the low risk group. The newly modified FRS also includes family history of premature CVD, doubling an individual's risk when CVD is present in a first-degree relative before 55 years of age for men and 65 years of age for women.

Currently, physical activity levels and lifestyle factors (i.e. dietary and alcohol intake, psychological stress) are not included in current risk scores but should be considered in risk factor management.

It is highly advocated by consensus groups that cardiovascular risk is assessed routinely, with the frequency depending on the presence and severity of risk markers. Furthermore, studies show the greatest positive effects on cardiovascular risk occurs when risk profiles are discussed and given to the patient, motivating individuals to reduce modifiable risk factors (i.e. smoking, blood pressure, cholesterol).

Physical activity and fitness assessment

Neither the level of physical activity nor physical fitness is incorporated into the FRS, but studies have shown that less fit or less active individuals may be at higher risk similar to other risk factors included in the FRS[33,34]. The volume and type of habitual physical activity provides insight into the extent of cardiovascular strain and physiological adaptations and, therefore, should be taken into account when risk stratifying. The absolute intensity of activities can be classified into three groups: low intensity (1.8-2.9 METs), moderate intensity (3-6 METs) and high intensity (> 6 METs)[3]. This latter group includes Master's events such as long-distance cycling, city marathons, long-distance cross country skiing and triathlons. In other words, these activities are typically classified as vigorous.

Lifestyle contributors in risk assessment

Diet (daily consumption of fruits and vegetables and red meat), smoking, and alcohol consumption are risk factors that can be modified decreasing the risk of premature CAD. For example, daily intake of fruits and/or vegetables, consumption of less than two servings of red meat a week, and consumption of less than seven servings of alcohol per week are deemed protective[35]. Conversely, heavy drinking (men ≥21 drinks /week; women ≥14 drinks/week) has an increased risk of all-cause mortality, possibly due to cirrhosis, cancer and violence[36]. Currently smoking is attributed as the strongest risk factor, with risk increasing at every increment and former smoking associated with a smaller risk ratio.

A compelling amount of evidence has associated psychological stress with development of premature CAD. The INTERHEART study found that psychological stress was the third highest risk factor for an acute myocardial in-

farction; only smoking and lipid concentrations (LDL and very low density lipoproteins (VLDL)) being greater[35]. Psychological stress may contribute to CVD risk by activating the sympathetic nervous system (SNS)[37]. Increased SNS activity can promote catecholamine release, general platelet aggregation, endothelial dysfunction, a negative inflammatory response, myocardial contractility, and vascular resistance, resulting in greater arterial pressure and turbulent blood flow, potentially damaging the endothelial lining[38]. Recently, XUE et al. observed that an acute stressful experience has a delayed, negative impact on the function of the endothelium[39]. In addition, repeated exposure to short-term stress might lead to permanent injury of the vasculature. Therefore, assessment of the patients' exposure to both repeated acute and chronic mental stress might be useful in determining their risk of CVD development.

Resting 12-lead electrocardiogram

Screening which includes the resting 12-lead electrocardiogram (ECG) has a high sensitivity to detect potentially lethal cardiovascular disease compared to only the history and physical examination[40]. It has the ability to identify subclinical cardiac disease (i.e. asymptomatic prior myocardial infarction, left ventricular hypertrophy, and fibrosis), arrhythmias, ion channelopathies, coronary artery disease (CAD), arrhythmogenic right ventricular hypertrophy, and HCM. Reasons against use of including the ECG are the potential of presenting false-negatives and -positives. False negatives are of concern in the Masters athlete since the main cause of SCD is CAD, which in most cases cannot be detected with the ECG alone. In lieu of this, it has been suggested that the exercise treadmill test be included during screening since it has a higher sensitivity in detecting flow-limiting CAD. In response to false-positives, an athlete specific criterion has been developed. Previously, the European Society of Cardiology (ESC) criteria was used, however recently, the 'Seattle Criteria' was developed, reducing the false-positive rate from 17% to 4.2% while still identifying the 0.3% of athletes with a cardiac abnormality[41].

Exercise treadmill test

The ECG exercise treadmill test (ETT) is a diagnostic tool for identifying arrhythmias and flow-limiting CAD. It has been advocated as a prognostic tool in risk stratifying those at danger for SCD. Multiple consensus guidelines recommend ETT in asymptomatic patients with diabetes or males >45 years-old or females >55 years-old prior to embarking on a vigorous exercise programme[31,32]. Its use in pre-participation screening to prevent cardiovascular events during exercise is debatable. One criticism is its poor sensitivity and specificity in detecting CAD in asymptomatic patients with low likelihood of the disease (i.e. endurance athletes)[42]. A "truly" positive ETT requires the presence of a flow-limiting coronary lesion (i.e. greater than 70% narrowing), whereas most acute coronary events evolve from vulnerable plaque rupture at mild to moderate stenosis and are less likely to be detected with such a test[1]. However, it has been shown that the predictive value of the ETT improves for patients with cardiovascular risk factors: the age-adjusted RR of an abnormal exercise test for CAD death was 21 in those with no risk factors, 27 in those with one risk factor, 54 in those with two risk factors, and 80 in those with more than three risk factors[43].

The ETT measures exercise capacity. It has the ability to predict cardiovascular risk, facilitating exercise prescription by taking into account further cardiovascular, metabolic and mechanical demands. This evaluation of exercise capacity in a general healthy population may provide information beyond markers of a positive test (i.e. ST depression). Indeed, a positive test is highly suggestive of flow-limiting CAD, but additional risk stratification is possible by considering the degree of ST-segment depression (marker for ischemic coronary artery disease), hypotensive blood pressure response (blood pressure doesn't increase proportionally to the intensity of the exercise), complex ventricular ectopy (extra beats that occur in succession originating from the larger chambers of the heart), slow heart rate recovery, altered heart rate response during activity and reduced exercise capacity, with the latter being a strong predictor of SCD in patients

with disease as well as in healthy patients[30,40]. An exercise capacity of less than six METs is a prominent risk factor and an exercise capacity greater than eight to 10 METs is associated with fewer cardiovascular events and greater longevity in men and women.

Measurement of maximal oxygen consumption by ventilatory gas analysis (i.e. cardiopulmonary test) is a useful indicator for aerobic capacity as well as for training evaluations in athletic populations. Nonetheless, it has not been shown to add relevant clinical information over that of the standard ETT for predicting CAD and is more costly[4].

ECG and ETT

Only a few studies concerning Masters athletes exist in support of the ECG and ETT during the screening process. AAGAARD et al. examined the effectiveness of the ECG in middle-aged, novice runners (n=153) by performing a pre-participation evaluation that included: a medical history, physical examination, SCORE risk evaluation, ECG, echocardiograph (echo), and blood tests[45]. Of the 153 individuals, 14 (9%) required further investigations. The physical examination and ECG identified 12 of the 14 runners requiring further diagnostic evaluation, and the echo discovered the remaining two. Seven of the 14 athletes were diagnosed with CVD, of which three (2%) were discouraged from racing. This demonstrated the importance of using both the physical examination and ECG as first line tests for pre-participation evaluation. It identified individuals in need of a further workup before vigorous exercise, whereas the echo did not substantially add to the diagnostic yield.

A second study performed in Switzerland, examined 785 middle-aged athletes engaged in high-intensity sports, following the EACPR protocol (described above)[46]. Cardiovascular abnormalities were discovered in 22 (2.8%) and 32 (4%) had multiple cardiovascular risk factors, putting them at risk for developing CVD. Three of the participants with a new cardiovascular diagnosis were ineligible for sport for the following discoveries: apical HCM, an old asymptomatic myocardial infarction (MI), and an ascending aorta aneurysm. Importantly, all were discovered through an abnormal ECG. White-Parkinson-White (WPW) (n=1) was also discovered with the ECG, but this athlete was deemed still eligible for sport. The physical examination discovered these remaining cardiovascular abnormalities: systemic hypertension (n=8), mitral valve prolapse (n=5), bicuspid aortic valve (n=3), and mild pulmonary stenosis (n=1), with personal history accounting for the discovery of a vaso-vagal syncope abnormality. This study was in agreement with AAGARD et al's findings, that the ECG plays a pivotal role as a tool in detecting serious cardiac disease.

An Italian study sought to evaluate the usefulness of both a resting and exercise ECG in a pre-participation screen using a five-year cross-sectional study design, examining 30,065 participants with a mean age of 30.4 (range 5-92)[47]. An abnormal resting 12-lead ECG was observed in 6% with 80% considered training-related changes and 1.2% were distinctly abnormal. The ETT detected 4.9% abnormalities, which led to 159 restrictions from sport after further examinations. In the initial screen, personal history, physical examination, or both suggested problems in six (3.7%) of those disallowed to participate. Almost all those who were advised not to participate in exercise exhibited signs of cardiac abnormalities on the exercise ECG. Of note, 56 of those with a potentially fatal cardiac disease had a normal resting ECG, demonstrating that the ETT does add value. The causes for exclusion were valve diseases (24%), hypertension (19%), arrhythmias (18%), CAD (9%), conduction disorders (7%), and cardiomyopathies (5%). The most prevalent cardiac abnormalities observed during stress testing, comprised of findings suggestive of CAD and arrhythmias, which is in line with the current consensus on causes of SCD in this population. Based on these findings, the ECG and ETT can identify those with cardiac abnormalities in the middle-aged and older athletes, therefore, it seems important that they be included in the initial screening process.

Autonomic nervous system assessment

The ECG can also provide information on the state of the autonomic nervous system (ANS). This is useful in detecting autonomic imbalance, a term used to indicate a relative or absolute decrease in vagal activity (i.e. parasympathetic nervous system) or an increase in sympathetic activity (i.e. fight or flight response). This imbalance is known to predict SCD risk. As a result, tools monitoring heart rate variability have gained attention, however, at this time an ANS based risk assessment has a predictive value that is likely too low to reliably screen for SCD[48].

Cardiovascular imaging

The addition of cardiovascular imaging as a first line tool in screening is controversial due to concerns with cost-effectiveness. However, improvements in cardiovascular imaging technology coupled with therapeutic options for cardiovascular disease have led to an increased consideration of using imaging as a viable option for pre-participation screening[49]. Imaging tests that have received the most attention are echocardiography, coronary artery calcium scoring, cardiac computed tomography and cardiac magnetic resonance imaging.

Currently, international sporting bodies such as the International Federation of Association Football (FIFA), the International Cycling Union (UCI) and the National Basketball Association (NBA) mandate echocardiography (echo) as part of the first line screening process, whereas, neither the European Society of Cardiology (ESC) nor AHA does.

The rationale for including the echo is that it is relatively inexpensive, accessible, and free of any direct adverse effects. For screening purposes, it has the capability of detecting CVD that is not always evident on an ECG (i.e. coronary anomalies, valvular disease, and some cardiomyopathies), which could potentially decrease false-positives and follow-up testing. It has also been useful for detecting an old myocardial infarction where damage has occurred. However, the current consensus suggests it doesn't substantially add to the prognostic detection of flow-limiting CAD and is not economically viable.

Cardiac computed tomography (CCT) and/or coronary artery calcium scoring (CACS) and cardiac magnetic resonance imaging (CMR) are highly sensitive tests in diagnosing CVD. Recent AHA guidelines support CCT use in asymptomatic individuals with intermediate cardiovascular risk or low risk with positive family history for premature CAD [50]. Its use is deemed inappropriate for those of low cardiovascular risk and in high-risk patients[50].

In addition to diagnostic capabilities, the CMR may play a prognostic role in detecting subclinical myocardial damage, clearly defining an athlete's heart from one that is pathological. A recent study investigating the potential prognostic significance of the CMR concluded that its usefulness in detecting asymptomatic athletes remains unclear and cannot be recommended as a routine examination in the care of athletes at this time[51].

Currently, the limitations of the CCT, CACS and CMR are high financial costs, inaccessibility, a potential harm due to contrast reactions, radiation exposure and whether or not the Masters athlete would benefit beyond the current risk factor management strategies (i.e. FRS). Therefore, these concerns far outweigh the benefits for inclusion in screening. However, they prove crucial and beneficial as secondary screening tests where the initial evaluation suspects a cardiomyopathy, coronary anomaly or myocarditis[49].

Additional roles of the pre-participation screen

Though the pre-participation screen is not perfectly sensitive in detecting all CVD, it has the potential to decrease mortality apart from detecting CVD, as well as the capacity to detect disorders related to SCD. It creates an awareness of risk factors, decreasing co-morbidities and ultimately slowing the atherosclerotic process and subsequent cardiac events. Surprisingly, many athletes are unaware they have cardiovascular risk factors (i.e. hypertension, diabetes, or dyslipidemia). Therefore, use of pre-participation screening may provide the

greatest reductions in cardiovascular risk by assessing and reviewing one's own liability[32]. Additionally, with the diagnosis of a disease or a high-risk profile, appropriate treatment can be implemented to reduce the risk of SCD[52]. Finally, the pre-participation evaluation can be used as a vehicle to educate Masters athletes on the nature and significance of warning signs for CVD.

Even though there are numerous benefits to screening, concern exists that it might be a possible deterrent for participating in exercise. This may be viewed as a major drawback. Therefore, education is paramount demonstrating that exercise far outweighs the risks and should be continued, in a safe, progressive manner, complementary to the individual's level of fitness.

Emergency Response Procedures

Considering the imperfect sensitivity of determining all those at high risk and the increased risk of cardiac events in the sporting venue, it is essential to have emergency response procedures in place. Automatic external defibrillators (AEDs) have been shown and proven to increase survival rates from 41% to 74% if cardiopulmonary resuscitation (CPR) is provided and defibrillation occurs within three to five minutes of collapse[53].

With increasing sporting events in the Masters' athlete population, it is imperative to mandate implantation of these devices in visible areas. The presence and timely access of AEDs at sporting venues provides a means of successful resuscitation not only for the athletes, but allays the fears for spectators, coaches, event staff, and other attendees.

Conclusion

First and foremost the take home message is: the health and performance benefits of exercise far outweigh the risk associated with competitive sport. However, although the absolute risk of cardiac events is small, the relative risk is high and increases with age due to the increased prevalence of atherosclerotic disease in the Masters athlete population.

Backed by years of scientific evidence, this risk can be attenuated with habitual exercise of at least 150 minutes/week. Physical activity beyond the general recommended amount has been shown to slow the natural decline in aerobic, anaerobic and vascular health and this phenomenon has even been coined "successful ageing". Both endurance and interval training can optimise aerobic capacity, heart function, insulin sensitivity, glucose metabolism, lipid profiles, body composition, bone density, neuromuscular function, and tendinopathy.

Conversely, certain risks have been associated with vigorous exercise, especially in those unaccustomed to such exercise. Additionally, possible adverse cardiovascular structural and functional changes and subsequent potentially lethal heart arrhythmias have been associated with long-term intensive endurance training.

More evidence is needed to determine the catalyst of these adverse effects, as well as the best training strategy to mitigate them. In the meantime, strategies to reduce the risk need to be studied in this growing athletic population (Masters) and enforced. Possible strategies include screening individuals before participation in exercise, appropriately modifying exercise for high-risk individuals, promptly reporting and evaluating new and unusual symptoms, ensuring emergency procedures are in place, placement of AEDs in all sporting venues, and educating athletes on safe exercise participation.

The primary and current area of controversy on strategies to mitigate the risk lies in pre-participation screening. Screening in the young competitive athlete (12-35 years-old), where heredity abnormalities are the main cause of sudden cardiac death (SCD), and are exceedingly rare, has been extensively studied. Conversely, in the Masters athlete, where atherosclerotic disease is the primary cause of SCD, and is far more common than disease found in the younger athlete, data is lacking. Therefore, limited recommendations exist to guide clinicians and health professionals. In response to

these concerns, the European Association of Cardiovascular Prevention and Rehabilitation (EACPR) and the American heart association (AHA) have outlined protocols, but have yet to be systematically evaluated extensively.

We do know that in the Masters population the protocol should specifically address identification of CAD and potential life threatening arrhythmias. Thus far, evaluation of traditional risk factors (i.e. SCORE or FRS) and family history of coronary artery disease (CAD) have been widely accepted as an essential first step in the assessment of all athletes. Proposed screening methods include personal symptoms and family history questionnaires, physical examinations, cardiovascular risk scores, resting ECG, ETT, autonomic nervous system assessment, biomarker assessment, and cardiac imaging (i.e. echo, CCT/CACS, CMR). Although consensus groups agree that screening should include a family history and personal symptoms questionnaire and a physical examination, controversy lies around whether or not, routine ECG, ETT and/or cardiac imaging and biomarkers should be included[5]. Studies to date show that the ECG and ETT can play a critical role in detecting CVD, with the ETT providing additional prognostic information by assessing the individual's exercise capacity.

Beyond detecting disease that could potentially lead to SCD, the pre-participation screen can provide additional information on the Masters athlete's health. This has the benefit of detecting and mitigating the development of cardiovascular risk factors, slowing the age-related progression of atherosclerotic disease and ultimately preventing the cardiac tragedies.

Key Points and Recommendations

1. First and foremost – regular physical activity is a potent non-pharmacological means for reducing risk of disease and death.
2. Habitual exercise (moderate to vigorous activity, ≥ 2-3 times a week for at least 30 minutes) provides the greatest protection against SCD.
3. Physical activity beyond the recommended weekly amount may promote successful aging, optimising health across an individual's lifespan, however the ideal dose re-mains unclear. Physical activity should be pursued in a safe and progressive manner, and training should be adapted (i.e. proper recovery) if excessive fatigue is experienced (i.e. symptoms of over-training).

4. Don't ignore symptoms! Promptly evaluate and report to a physician new/unusual symptoms (i.e. chest pain, palpitations during exercise, excessive breathlessness, unusual fatigue, indigestion/heartburn/gastrointestinal symptoms, ear or neck pain, vague malaise, upper respiratory tract infection, dizziness, severe headache).

5. Masters athletes should be screened, especially those at moderate to high risk before embarking on a vigorous exercise programme (> 6 METs). Although debate exists on the best method, evidence suggests that screening can detect individuals at risk and reduce cardiac events. At minimum, individuals beginning a vigorous exercise programme should answer a pre-participation questionnaire (i.e. AHA, PAR-Q+), and regularly evaluate their cardiovascular risk score (i.e. FRS, SCORE).

6. If an individual's risk is moderate to high, they should seek the counsel of a physician before engaging in vigorous activity. A health care professional should also be involved so that appropriate exercise modifications can be made. Light activity can be continued in the meantime.

7. Activity levels should be modified according to exercise capacity, habitual activity level and environmental conditions (i.e. hot, humid environments, high altitudes).

8. Allow adequate recovery after competitions. In older and less fit individuals, longer rest is advised.

9. Exercise does not make one exempt from cardiovascular disease and although not included in current risk factor scores, an unhealthy lifestyle (i.e. poor dietary habits, high psychological stress, and excessive alcohol intake) makes one susceptible to development of atherosclerosis and increases all-cause mortality, despite a regular exercise routine.

10. Appropriate training (warm-up, cool down, training complimentary to exercise capacity) is important in preventing cardiac events, especially in those at risk of underlying disease.

11. Fitness personnel, coaches, and athletic trainers should be trained (i.e. cardiopulmonary resuscitation) for cardiovascular emergencies.

12. Automatic external defibrillators (AEDs) should be placed in sporting arenas and events.

Sports Cardiology B.C. will be conducting a prospective research study in Masters athletes (> 35 years old) without known CAD, involved in recreational or high performance sport. Their aim is to evaluate the cardiovascular risk and the best screening method for detecting cardiovascular disease in Canadian Masters athletes in order to prevent adverse cardiovascular events and/or SCD. This study is expected to commence in March 2015.

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