Recommendations for Preparticipation Screening and the Assessment of Cardiovascular Disease in Masters Athletes : An Advisory for Healthcare Professionals From the Working Groups of the World Heart Federation, the International Federation of Sports Medicine, and the American Heart Association Committee on Exercise, Cardiac Rehabilitation, and Prevention

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In recent years, there has been a major international focus on both the risks and benefits of exercise, as well as the relationship between physical activity and cardiovascular health at all ages.[1–8] Based on more than 40 observational epidemiological studies, physical inactivity and sedentary lifestyle are recognized as major risk factors for the development of coronary heart disease, adverse cardiovascular events, and mortality.[9–28] Regular aerobic exercise (occupational or leisure time) confers many health benefits, may reduce the risk for fatal and nonfatal myocardial infarction and other coronary events,[1–28] and has been promoted as a national public health agenda in several countries for both primary and secondary prevention of cardiovascular disease.[3–7,22,24,29,30]

Conversely, acute vigorous physical exertion may trigger sudden death or myocardial infarction[31–52] in the presence of underlying heart disease,[48] particularly in individuals not accustomed to such activity or to regular exercise.[31–33,35,41,51,52,52a,52b] As sedentary individuals begin (or reinitiate) an exercise program, there is a period of increased risk during which exertion can provoke a cardiac event, whereas such risk associated with habitual exercise in active individuals is relatively low.[31] Also, it is generally assumed that particularly strenuous exertion or sports competition may predispose athletes to greater cardiac risk than nonstrenuous physical activity. Nevertheless, there is widespread agreement that the overall benefits of exercise usually outweigh associated risks.[1–30,35] Mechanisms by which exercise may protect patients from coronary events have also been proposed.[53,54]

Prior expert consensus documents have addressed the risk for sudden death associated with sports participation in competitive or recreational athletes with cardiovascular disease, including the criteria for disqualification and eligibility,[49–51,55,56] and the screening of general populations for detection of cardiac abnormalities.[47,50,51] These previous recommendations[49,50,55–59] have focused largely on young competitive athletes or older athletes in recreational sports settings such as health and fitness facilities.[51]

Consequently, in the present scientific statement, the panel addressed issues relevant to competitive sports participants at the masters level primarily in midlife and beyond, cognizant of the unique psychological and physiological stresses that competition places on such athletes, particularly those with cardiovascular disease.[22,52,60–74] It is believed that by offering prudent insights and advice to physicians concerning the screening and detection of cardiac disease, as well as recommendations for sports clearance and eligibility in such older athletes, this document may contribute to the prevention of cardiac catastrophes and events during exercise.

Scope and Definition of Masters Sports Competition

The designation of a competitive athlete refers to one who participates in an organized team or individual sport that requires regular competition against others, places a high premium on excellence and achievement, and requires systematic training.[49] It is understood that this definition is somewhat arbitrary, because many individuals participate in casual sporting activities in a competitive fashion, and in some organized sports the distinctions between truly competitive and recreational physical activity may be ambiguous.

Masters sports are organized forms of competition specifically designed for older athletes. The stimuli for continuing in sports or beginning involvement at relatively advanced ages are wide-ranging but include an awareness that physical inactivity is a risk factor for cardiovascular disease and

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associated events, as well as emotional and motivational issues such as self-image and prior success in sports.

Masters sports participation is rapidly growing and consists of a rather loose and unofficial conglomeration of many independent organizations representing a wide variety of athletic disciplines at the international, national, and local levels of competition. Presently, more than 50 countries sponsor masters sporting events, with the most participation in the United States, Canada, Western Europe, and parts of Asia and South America. Although difficult to tabulate precisely, participation rates are substantial worldwide, involving many thousands who aspire to organized competition and recognition in a variety of team and individual sports on a regular basis. Organized masters programs include ≈50 sports, many characterized by intense competition, and most commonly swimming (including aquatic sports such as diving and water polo), track and field, soccer, rowing, basketball, tennis and other racquet sports, weight lifting, orienteering, rugby, ice hockey, cycling, and skiing. In addition, there are substantial numbers of participants in endurance sports such as marathons, long-distance road racing, and triathlons, and more recently, competition in so-called extreme sports.

Masters participants range widely in age but are typically older than 35 years, with many more than 40 to 50 years of age, and they are predominantly male. Certain masters sports competition may begin earlier (eg, at >25 years of age in some swimming associations). Despite the wide range in age of masters athletes, the primary focus of the present document is on participants >40 years of age. Masters athletes may include conditioned, experienced competitive athletes who continue to compete after their formal careers end but also include "walk-up" competitors (sometimes referred to as "weekend warriors") with only sporadic training regimens, as well as those who resume competition after long periods of physical inactivity.

Aging and Cardiovascular Function

Aerobic cardiovascular function declines with age,[60,64,68,69,73,74] as evidenced by the progressive decrease in maximal oxygen consumption ($\dot{V}O_2$max) at an average rate of ≈10% per decade after age 25. Whether vigorous endurance exercise training blunts this rate of decline in $\dot{V}O_2$max remains controversial.[63,67,69,71] Nevertheless, older endurance athletes usually retain greater aerobic capacity than their sedentary counterparts of the same age and sex.[60,62,63,71–73] The possibility that the normal age-associated decline in cardiovascular performance might be attenuated by exercise training in older individuals suggests a potential benefit for participation in organized masters sports.[66,67]

Cardiovascular Risks of Masters Sports

Causes of Sudden Death or Morbidity

Masters sports programs primarily comprise apparently normal and healthy individuals from the general population. However, unlike most organized sports for young people, some participating masters athletes compete with known, documented cardiovascular disease. By far, atherosclerotic coronary artery disease is the most common form of heart disease relevant to the masters population as a cause of morbidity or sudden death. Occasionally, such athletes may harbor other congenital or acquired heart diseases with the potential to cause sudden death, such as hypertrophic cardiomyopathy, aortic stenosis, dilated cardiomyopathy, mitral valve prolapse, and myocarditis. Masters athletes, typical of their age group, could also be expected to be susceptible to particular arrhythmias and conduction abnormalities[75–77] (eg, atrial fibrillation and flutter, sick sinus syndrome, and atrioventricular block).

Prevalence

The frequency with which sudden cardiac death (due primarily to coronary artery disease) occurs during organized masters competition or training is not known with precision. Nevertheless, it is possible to extrapolate from available information in the older general athletic population of marathon racers,[37] apparently healthy male athletes and sports participants,[31,36] and joggers.[36,39] These data suggest a risk for sudden cardiac death in the range of 1:15 000 joggers per year or 1:50 000 participants in marathons, with a marked predominance of deaths in men. On the other hand, the risk in young high school and college-aged athletes has been calculated to be ≈1:200 000 to 1:300 000 per academic year.[46,78]

Other observations also suggest that death and cardiac events such as exercise-related myocardial infarction are not rare in older athletic populations.[32,33,36–45] The aforementioned prevalence figures were obtained in US populations, and the incidence of coronary artery disease and sudden cardiac death related to exercise may well differ in other countries and regions of the world. Nevertheless, as participation in masters sports accelerates throughout the world, greater numbers of athletes will be enrolled in intensive training programs or competition abruptly after long periods away from strenuous training and the competitive sports arena, often without preparticipation medical evaluations. Therefore, the prevalence of cardiac events among these athletes may increase further, and it is this subset of athletes that is a major focus of this document.

General Objectives of Preparticipation Screening

As a prudent and beneficial measure, preparticipation medical evaluations are recommended for athletes before entry into masters sports training programs. At present, such examinations are limited, inconsistent, or nonexistent. A major objective of this process would be to identify (or raise the suspicion of) occult cardiovascular disease that has the potential to cause sudden cardiac death, nonfatal myocardial infarction, stroke, angina, acute coronary syndromes, or heart failure (when triggered by intense athletic activity or abrupt burst exertion); this warrants evaluation and recommendations regarding future participation in competitive athletics. In athletes with known cardiovascular disease, the purpose of this preparticipation evaluation is to determine whether continued sports participation is judicious and consistent with the severity and status of their disease.

Therefore, such screening evaluations represent the opportunity to formulate recommendations for the initiation or resumption of participation in competitive sports, as well as

AHA Consensus Panel Recommendations for Preparticipation Screening[50]

Family history
1. Premature sudden death
2. Heart disease in surviving relatives
Personal history
3. Heart murmur
4. Systemic hypertension
5. Fatigability
6. Syncope
7. Exertional dyspnea
8. Exertional chest pain
Physical examination
9. Heart murmur*
10. Femoral pulses
11. Stigmata of Marfan syndrome
12. Blood pressure measurement

*Precordial auscultation is recommended in both supine/sitting and standing positions to identify heart murmurs consistent with dynamic left ventricular outflow tract obstruction.

the mechanism by which reentry into a training regimen might best be effected. Cardiovascular evaluation should be repeated if symptoms intervene or perceived risk increases, or for other clinical reasons based on the judgment of the personal physician. However, in general, asymptomatic status does not confer immunity from cardiac events, and subsequent, periodic clinical examinations may be prudent.

The proposed medical evaluation and its objectives may differ with regard to national origin or culture. One example is Italy, where, under a national governmental program, competitive athletes at all levels undergo compulsory annual medical clearance, including evaluation for cardiovascular disease by certified sports medicine physicians.[58] This mandatory screening protocol is extended for any athlete, regardless of age, who is engaged in official organized sports activities, including many masters competitions. In addition to a standard history and physical examination, noninvasive tests such as the 12-lead ECG and submaximal exercise test are performed, as well as 2-dimensional echocardiography or maximal exercise testing (treadmill or bicycle ergometer) if judged appropriate at the discretion of the examining sports medicine physician.

Specific Recommendations for the Preparticipation Cardiovascular Evaluation

History and Physical Examination

The preparticipation evaluation should emphasize the detection of previously undiagnosed coronary artery disease. It is proposed that the personal and family history and physical examination for masters athletes adopt the 12 relevant points of the American Heart Association preparticipation screening recommendations[50] (Table).

ECG Exercise Testing

Symptom-limited maximal ECG exercise testing (with treadmill or cycle ergometer) is a widely used and well-established test for the detection of coronary artery disease that may also indirectly predict patient outcome.[23,79–88] The exercise ECG is a relatively effective and inexpensive test for myocardial ischemia due to coronary artery disease in a moderate- to high-risk asymptomatic population. Interpretation of the test includes exercise capacity, as well as clinical, hemodynamic, and electrocardiographic responses.[23,79–81] An important ECG finding is >1 mm (0.10 mV) of horizontal or downsloping ST-segment depression for >80 ms during and/or in the first minutes of the recovery period.[23] Additional risk stratification is possible by considering the degree of ST-segment depression, hypotensive blood pressure response to maximal exercise (relative to baseline), complex ventricular ectopy, and reduced exercise capacity, which are frequently associated with severe coronary artery disease, ischemic myocardial dysfunction, and poor prognosis.[23,79–91]

It is recognized that the absolute risk of a major event during physical activity is small in asymptomatic patients without known cardiac disease. Nevertheless, it is recommended that those masters athletes having a moderate-to-high cardiovascular risk profile for coronary artery disease,[23,91–93] and who desire to enter vigorous competitive situations, undergo exercise testing. Specifically, this risk profile would include men more than 40 to 45 years old or women more than 50 to 55 years old (or postmenopausal) with 1 or more independent coronary risk factors. These include the following: hypercholesterolemia or dyslipidemia (total cholesterol >200 mg/dL; elevated low-density lipoprotein [LDL] cholesterol [>130 mg/dL]; low high-density lipoprotein [HDL] cholesterol [<35 mg/dL for men; <45 mg/dL for women]); systemic hypertension (systolic blood pressure >140 mm Hg or diastolic pressure >90 mm Hg); current or recent cigarette smoking; diabetes mellitus (fasting plasma glucose ≥126 mg/dL or treatment with insulin or oral hypoglycemics); or history of myocardial infarction or sudden cardiac death in a first-degree relative <60 years old. In addition, an exercise test is recommended for those masters athletes of any age with symptoms suggestive of underlying coronary disease and for those ≥65 years old even in the absence of risk factors and symptoms.

Because most exercise testing is performed in adults with symptoms of known or suspected ischemic heart disease, the panel acknowledges that the use of the exercise ECG to screen an asymptomatic general athletic cohort for coronary artery disease is a complex issue.[23] There are limitations in negative predictive value, as well as in the accuracy for positive identification of disease in such a population (which has not been conclusively defined because coronary arteriography is rarely performed in individuals with normal treadmill tests).[8,23,80] Therefore, nonselective use of the exercise ECG in low-risk populations is associated with poor positive predictive accuracy[23] and false-positive tests that can have negative psychological implications and result in unnecessary and costly medical testing, as well as adverse consequences in relation to employment and insurance. Consequently, the routine use of the exercise ECG in healthy asymptomatic athletes without major risk factors is not recommended.

Nevertheless, findings from the Multiple Risk Factor Intervention Trial (MRFIT) suggest that in asymptomatic

populations with coronary risk factors, development of a positive exercise ECG test for myocardial ischemia is associated with a higher incidence of future coronary events[6,81–83,86] such as angina pectoris, acute myocardial infarction, or sudden death by as much as 15 times in men and 5 times in women.[18,24,85,86] In the Seattle Heart Watch Study,[81] asymptomatic men over 40 years of age with >1 coronary risk factor and >2 abnormal features on exercise testing (including chest pain during maximal exertion, exercise duration <6 minutes on the Bruce protocol, failure to attain at least 90% of age-predicted maximal heart rate, or ischemic ST-segment depression) showed a 30-fold increment in 5-year cardiac risk; the greater the number of risk factors (ie, pretest probability), the more substantial is the potential value of screening.

Maximal graded exercise testing serves only as a preliminary evaluation to estimate the likelihood that coronary artery disease is present. A positive test necessitates further diagnostic evaluation to establish more definitively the presence and anatomic severity of the atherosclerotic arterial narrowing. On the other hand, a negative exercise test does not rule out the presence of coronary artery disease.

Other Testing

The standard 12-lead ECG is of limited diagnostic value for detecting coronary artery disease in an asymptomatic masters population, particularly given the variability of ECG patterns associated with athletic training.[94] Nevertheless, the ECG, when used as part of a preparticipation screening evaluation, may occasionally identify unexpected evidence of a healed myocardial infarction and can also be particularly helpful in detecting certain diseases less common in the masters population, such as hypertrophic cardiomyopathy[95,96]; long-QT, Brugada, and Wolff-Parkinson-White syndromes; and arrhythmogenic right ventricular cardiomyopathy.[97–99] For these reasons, a standard 12-lead ECG is recommended as part of a routine evaluation for all masters athletes (male and female) >40 years old.

Measurement of maximal oxygen consumption by ventilatory gas analysis is dependent on many factors (age, sex, body composition, and training),[61–64,67–69,74] has not been shown to add relevant clinical information over that of the standard exercise ECG for predicting coronary heart disease, and is, in fact, more costly. At present, directly measured maximal oxygen uptake and anaerobic threshold are more often used as measures of physical and cardiovascular fitness and to develop an index of aerobic capacity, as well as for training evaluations in athletic populations.

Diagnostic echocardiography is indicated when clinical, historical, or physical findings suggest the possibility of valvular heart disease (particularly aortic stenosis), hypertrophic cardiomyopathy with or without left ventricular outflow obstruction, arrhythmogenic right ventricular cardiomyopathy, or prior myocardial infarction.[50,57,89,95,96,99]

Education

Another objective of the preparticipation evaluation is the education of athletes regarding various aspects of self-monitoring and risk of cardiovascular events. Making the athlete aware of the nature and significance of warning signs or symptoms of cardiovascular disease (such as angina pectoris, anginal equivalents, or impaired consciousness), the risk of a cardiovascular event associated with the abrupt onset of intense athletic training in otherwise sedentary and untrained individuals, and the importance of modifying identified cardiovascular risk factors all represent critical educational issues.

Guidelines for Disqualification and Eligibility

In determining the criteria for cardiovascular disqualification from masters sports, the panel relied on a review of the available literature, personal experience, and the consensus recommendations of Bethesda Conference No. 26, designed to promote eligibility standards for competitive athletes.[55] The recommendations take into consideration a large number of cardiovascular conditions, the severity of these diseases, and profiles of sporting activity and training that estimate the intensity of physical exertion and its potential impact as a trigger for sudden cardiac death and/or disease progression. In general, organized sports requiring vigorous physical activity, such as basketball, track, or swimming, pose greater cardiovascular risk and are therefore less desirable than low-intensity competitive sports, such as golf and bowling, given the same cardiac disease of similar severity. There is particular concern that risk may be increased for those former trained athletes who enter masters competition and participate in short-distance or short-duration events involving maximal "burst"-type exertion (eg, certain swimming and cycling events, track sprinting, basketball, tennis, and soccer) with little or no recent formal training.

These recommendations should not create barriers to exercise and its varied physiological and psychological benefits for older athletes. Finally, these recommendations should not be regarded as mandatory or absolute but are offered only as guidelines and do not prohibit individualization by practitioners exercising their independent clinical judgment.

Atherosclerotic Coronary Artery Disease

For the purpose of this discussion, coronary artery disease is defined as narrowing of a major coronary artery (generally regarded as >50% luminal diameter narrowing), as documented by coronary angiography. Acute myocardial infarction also frequently occurs in association with atherosclerotic luminal narrowing of <50%.

For athletes with documented ischemic heart disease (whether or not symptoms are present and regardless of whether prior myocardial infarction or complete revascularization has occurred), it is not advisable to participate in high-intensity competitive masters sports, and such individuals should be confined to competitive sports judged to be of low intensity. The low-intensity class of sports, as defined by Bethesda Conference No. 26, is principally represented by competition in sports such as golf and bowling.[55]

This restriction on high-intensity sports (or any physical activity that triggers ischemic symptoms) is particularly pertinent for masters athletes, because the emotional impact of competitive situations may mask ischemic symptoms and augment risk for precipitating unstable angina, myocardial

infarction, or sudden cardiac death. These guidelines should not, however, be construed as a prohibition against regular (recreational and noncompetitive) exercise, which is generally advisable for patients with coronary artery disease owing to the cardiovascular benefits derived from such physical activity.[1–30,52a,52b]

The probability of an exercise-induced cardiac event is generally higher in the presence of more severe coronary atherosclerosis and heart disease. Consequently, the aforementioned general restrictions on high-intensity competitive sports should be most closely followed in patients with any of the following criteria:

1. impaired left ventricular systolic function under resting conditions (ejection fraction <50%);
2. evidence of exercise-induced myocardial ischemia, including angina, positive ECG, or positive imaging findings with exercise testing;
3. evidence of exercise-induced frequent or complex supraventricular or ventricular arrhythmias, including nonsustained ventricular tachycardia;
4. exercise-induced systolic hypotension.

Some masters athletes with mild atherosclerotic disease, normal left ventricular function, and no inducible ischemia or arrhythmias may be allowed to participate in more vigorous competitive sports, but this clearance should be provided on a case-by-case basis and with full understanding by both the patient and physician that such vigorous exercise probably increases the risk of an acute cardiac event.

Systemic Hypertension

Effort should be made to normalize blood pressure by drugs or lifestyle before training or competition is initiated. The presence of systemic hypertension of a mild degree (stage 1, 140 to 159 mm Hg systolic or 90 to 99 mm Hg diastolic blood pressure) in the absence of target organ damage or concomitant cardiac dysfunction should not restrict eligibility from most competitive masters sports.[100,101] In general, because of the documented benefits of low- to moderate-intensity exercise in lowering blood pressure and improving other coronary risk factors, the engagement in regular forms of exercise may be of benefit to hypertensive subjects, although competitive resistance training should be discouraged for athletes with moderate to severe hypertension. After beginning a regular athletic training program, hypertensive athletes should have their blood pressure monitored at least every 2 months to assess the impact of exercise.

Athletes with moderate to severe systemic hypertension (stage 2, 160 to 179 mm Hg systolic or 100 to 109 mm Hg diastolic blood pressure; or stage 3, ≥180 mm Hg systolic or ≥110 mm Hg diastolic blood pressure)[100,101] should be restricted, particularly from highly static competitive sports (such as weight lifting or gymnastics), until their blood pressure is controlled.[101] These restrictions regarding hypertension seem appropriate, because competitive athletics involves more intense physical exertion than is required for routine cardiovascular conditioning. The present recommendations are offered, although at present there are no persuasive data available that definitively link strenuous exercise and the risk for sudden cardiac death or disease progression among hypertensive subjects. When hypertension coexists with coronary artery disease, eligibility for participation in masters competition will usually be based on the nature and severity of the coronary disease.

Congenital and Valvular Heart Disease Conditions

Hypertrophic cardiomyopathy is a heterogeneous and unpredictable primary cardiac disease for which there is increased risk for sudden cardiac death in some patients.[95,96,102] For these reasons, and because of the lack of precision that prevails in stratifying risk for sudden death in hypertrophic cardiomyopathy[59,95,96,103] (particularly in competitive athletes), we believe that older individuals with an unequivocal diagnosis of this condition should be advised against participation in intense masters sports and confine their competitive athletic activity to only low-intensity sports. Patients with documented dilated cardiomyopathy or arrhythmogenic right ventricular cardiomyopathy[99] should be excluded from all competitive sports.[59]

The vast majority of athletes with mitral valve prolapse do not have complicating factors and will be able to participate fully in sports. Asymptomatic athletes with mitral valve prolapse and any of the following criteria should be restricted to low-intensity competitive sports (such as golf and bowling): (1) history of syncope, judged probably arrhythmogenic in origin; (2) family history of sudden death due to mitral valve prolapse; (3) repetitive supraventricular or complex ventricular tachyarrhythmias, particularly if exacerbated by exercise; (4) moderate to severe mitral regurgitation; and (5) prior embolic event. For other less common congenital and valvular heart diseases in the masters age group, the consensus guidelines of Bethesda Conference No. 26 should be followed.[55]

Myocarditis

Athletes judged to have myocarditis (with or without evidence of a dilated cardiomyopathy) may return to competition when there is no longer evidence of active infection, ie, ventricular function and cardiac dimensions have returned to normal, and clinically relevant arrhythmias (frequent or repetitive forms of ventricular ectopic activity or sustained supraventricular tachycardia) are absent on ambulatory ECG (Holter) recording or exercise testing. A prudent convalescent period of ≈6 months is recommended before these athletes return to competitive sports.

Chagas Disease

Chagas is an infectious but noncontagious disease caused by the parasite *Trypanosomal cruzi*, typically transmitted by a mosquito bite and confined to rural, undeveloped areas in the tropics or subtropics or South America.[104] In these endemic areas, Chagas disease still constitutes a major public health problem. For example, an estimated 5% of the population of Brazil is infected, which may include older individuals participating in sports. Diagnosis can be made by direct parasite identification or positive serological reaction.

In its chronic phase, Chagas presents a well-defined clinical profile affecting organs with muscarinic receptors,

such as the heart (in the myocardium and conduction system), colon, and esophagus. About one third of affected individuals develop cardiac complications leading to heart failure or thromboembolism-related or sudden cardiac death, often much later in life. Cardiac involvement (often in the form of chronic myocarditis) can be suspected when atrial and ventricular arrhythmias or the characteristic ECG pattern of right bundle-branch block and left anterior hemiblock with or without echocardiographic demonstration of apical aneurysm are present.

In the presence of Chagas disease, sports competition can be unrestricted if no objective signs or symptoms of cardiac involvement are evident (and resting and exercise ECG or echocardiographic tests are normal). With evidence of cardiac involvement (with or without pacemaker implantation), all intense competitive sports activity is contraindicated. Fortunately, the incidence of new Chagas disease cases has decreased recently in some South American countries.

Cardioactive Drugs

Those masters athletes using cardioactive drugs should discuss their competitive sports interests and needs with their physician. It may or may not be possible to adjust drugs and dosages to achieve compatibility with this desired lifestyle. However, β-adrenergic blocking agents can generally be expected to impair performance during participation in intense competitive sports.[105]

Implementation of Screening and Eligibility Recommendations

In contrast to the common practice for high school and college-aged student athletes,[50] it is unrealistic to expect governing authorities or sponsors to provide medical evaluations for all (or even most) masters athletes because of the often loose affiliations between masters sports organizations and their athletes and a lack of resources to support organized systematic screening programs. These considerations are particularly relevant to certain individual sports such as road racing or marathon events, in which preparation and training for competition are usually medically unsupervised and left to the discretion of the athlete. The practical limitations of broad-based preparticipation screening for public running events is illustrated by the London Marathon, which has 40 000 participants annually.[106]

For all these reasons, it becomes largely unavoidable that the primary responsibility rests with the masters athlete to identify and initiate contact with an appropriate physician (primary care practitioner, subspecialist cardiologist, or an appropriately trained sports medicine specialist) for the purpose of a preparticipation cardiovascular evaluation to include blood pressure measurement and risk factor analysis (with exercise ECG testing, if indicated). Even in those circumstances in which important cardiovascular disease is excluded or regarded as unlikely, we encourage the entry (or reentry) into competitive athletics to occur with a gradual training program that progressively leads to more vigorous levels of physical exertion.

With regard to prevention of sudden death, it is recommended that both personnel trained in cardiopulmonary resuscitation and automatic external defibrillators be available at all sanctioned masters sports events.[107,108] The relatively advanced ages of many masters athletes and the anticipated frequency of cardiovascular events in this age group make these recommendations particularly reasonable and relevant, especially with the recent development of easy-to-use, low-cost defibrillators targeted for use at locations where large numbers of high-risk people congregate.[107,108]

Conclusions

The recommendations developed in this document provide general cardiovascular screening guidelines for older individuals entering, resuming, or continuing in organized sports competition. Adequate cardiovascular evaluations are important for identification of underlying cardiovascular disease (typically ischemic heart disease) before initiation of training programs at moderate to intense levels. This recommendation is underscored by the fact that masters athletes are unique, given the physiology associated with advancing age, the possibility of unsuspected coronary artery disease, and their often abrupt entry (or reentry) into competitive training programs. Specific recommendations are presented for the identification and evaluation of cardiovascular disease, as well as guidelines for clearance and disqualification. Also, a major impetus of this consensus document is to create a focus on the expanding medical issues concerning older individuals (potentially with underlying cardiovascular disease) who are involved in intense competitive sports. The achievement of these objectives will require the support of various athletic and professional medical organizations, the physician community, and insurance companies, as well as that of masters athletes and their families. Implementation of the screening approaches described here can aid in identification of high-risk athletes who can then be referred to effective risk-reduction programs, which ultimately will enhance the safety of sports participation for older athletes.

References

1. Fletcher GF, Balady GJ, Blair SN, et al. Statement on exercise: benefits and recommendations for physical activity programs for all Americans: a statement for health professionals by the Committee on Exercise and Cardiac Rehabilitation of the Council on Clinical Cardiology, American Heart Association. *Circulation*. 1996;94:857–862.
2. Pate RR, Pratt M, Blair SN, et al. Physical activity and public health: a recommendation from the Centers for Disease Control and Prevention and the American College of Sports Medicine. *JAMA*. 1995;273:402–407.
3. *Physical Activity and Health: A Report of the Surgeon General.* Atlanta, Ga: US Dept of Health and Human Services, Centers for Disease Control and Prevention; 1996.
4. Fletcher GF, Blair SN, Blumenthal J, et al. Statement on exercise: benefits and recommendations for physical activity programs for all Americans: a statement for health professionals by the Committee on Exercise and Cardiac Rehabilitation of the Council on Clinical Cardiology, American Heart Association. *Circulation*. 1992;86:340–344.
5. NIH Consensus Development Panel on Physical Activity and Cardiovascular Health. Physical activity and cardiovascular health. *JAMA*. 1996;276:241–246.
6. Williams C, ed. *Physical Activity and Cardiovascular Disease Prevention in the European Union*. Brussels, Belgium: European Heart Network; 1999.
7. Bijnen FC, Caspersen CJ, Mosterd WL. Physical inactivity as a risk factor for coronary heart disease: a WHO and International Society and

Federation of Cardiology position statement. *Bull World Health Organ*. 1994;72:1–4.
8. Fletcher GF, Balady G, Froelicher VF, et al. Exercise standards: a statement for healthcare professionals from the American Heart Association. *Circulation*. 1995;91:580–615.
9. Paffenbarger RS Jr, Hyde RT, Wing AL, et al. Physical activity, all-cause mortality, and longevity of college alumni. *N Engl J Med*. 1986;314:605–613.
10. Paffenbarger RS, Hyde RT, Wing AL, et al. The association of changes in physical activity level and other lifestyle characteristics with mortality among men. *N Engl J Med*. 1993;328:538–545.
11. Morris JN, Clayton DG, Everitt MG, et al. Exercise in leisure-time: coronary attack and death rates. *Br Heart J*. 1990;63:325–334.
12. Sandvik L, Erikssen J, Thaulow E, et al. Physical fitness as a predictor of mortality among healthy, middle-aged Norwegian men. *N Engl J Med*. 1993;328:533–537.
13. Blair SN, Kohl HW III, Paffenbarger RS Jr, et al. Physical fitness and all-cause mortality: a prospective study of healthy men and women. *JAMA*. 1989;262:2395–2401.
14. Powell KE, Thompson PD, Caspersen CJ, et al. Physical activity and the incidence of coronary heart disease. *Annu Rev Public Health*. 1987;8:253–287.
15. Peters RK, Cady LD Jr, Bischoff DP, et al. Physical fitness and subsequent myocardial infarction in healthy workers. *JAMA*. 1983;249:3052–3056.
16. Lee IM, Hsieh CC, Paffenbarger RS Jr. Exercise intensity and longevity in men: the Harvard Alumni Health Study. *JAMA*. 1995;273:1179–1184.
17. Kushi LH, Fee RM, Folsom AR, et al. Physical activity and mortality in postmenopausal women. *JAMA*. 1997;277:1287–1292.
18. Leon AS, Connett J, Jacobs DR Jr, et al. Leisure-time physical activity levels and risk of coronary heart disease and death: the Multiple Risk Factor Intervention Trial. *JAMA*. 1987;258:2388–2395.
19. Bijnen FC, Caspersen CJ, Feskens EJ, et al. Physical activity and 10-year mortality from cardiovascular diseases and all causes: the Zutphen Elderly Study. *Arch Intern Med*. 1998;158:1499–1505.
20. Erikssen G, Liestøl K, Bjørnholt J, et al. Changes in physical fitness and changes in mortality. *Lancet*. 1998;352:759–762.
21. Kujala UM, Kaprio J, Sarna S, et al. Relationship of leisure-time physical activity and mortality: the Finnish twin cohort. *JAMA*. 1998;279:440–444.
22. Morris JN, Everitt MG, Pollard R, et al. Vigorous exercise in leisure-time: protection against coronary heart disease. *Lancet*. 1980;2:1207–1210.
23. Gibbons RJ, Balady GJ, Beasley JW, et al. ACC/AHA guidelines for exercise testing: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Exercise Testing). *J Am Coll Cardiol*. 1997;30:260–311.
24. Leon AS, Connett J. Physical activity and 10.5 year mortality in the Multiple Risk Factor Intervention Trial (MRFIT). *Int J Epidemiol*. 1991;20:690–697.
25. Wannamethee SG, Shaper AG, Walker M. Changes in physical activity, mortality, and incidence of coronary heart disease in older men. *Lancet*. 1998;351:1603–1608.
26. Berlin JA, Colditz GA. A meta-analysis of physical activity in the prevention of coronary heart disease. *Am J Epidemiol*. 1990;132:612–628.
27. Villeneuve PJ, Morrison HI, Craig CL, et al. Physical activity, physical fitness, and risk of dying. *Epidemiology*. 1998;9:626–631.
28. Blair SN, Kohl HW III, Barlow CE, et al. Changes in physical fitness and all-cause mortality: a prospective study of healthy and unhealthy men. *JAMA*. 1995;273:1093–1098.
29. Snell PG, Mitchell JH. Physical inactivity: an easily modified risk factor? *Circulation*. 1999;100:2–4. Editorial.
30. Fletcher GF. How to implement physical activity in primary and secondary prevention: a statement for healthcare professionals from the American Heart Association. *Circulation*. 1997;96:355–357.
31. Siscovick DS, Weiss NS, Fletcher RH, et al. The incidence of primary cardiac arrest during vigorous exercise. *N Engl J Med*. 1984;311:874–877.
32. Mittleman MA, Maclure M, Tofler GH, et al. Triggering of acute myocardial infarction by heavy physical exertion: protection against triggering by regular exertion: Determinants of Myocardial Infarction Onset Study Investigators. *N Engl J Med*. 1993;329:1677–1683.
33. Willich SN, Lewis M, Löwel H, et al. Physical exertion as a trigger of acute myocardial infarction: Triggers and Mechanisms of Myocardial Infarction Study Group. *N Engl J Med*. 1993;329:1684–1690.
34. Gibbons LW, Cooper KH, Meyer BM, et al. The acute cardiac risk of strenuous exercise. *JAMA*. 1980;244:1799–1801.
35. Ilkka V. The cardiovascular risks of physical activity. *Acta Med Scand*. 1986;711:205–214.
36. Ragosta M, Crabtree J, Sturner WQ, et al. Death during recreational exercise in the State of Rhode Island. *Med Sci Sports Exerc*. 1984;16:339–342.
37. Maron BJ, Poliac LC, Roberts WO. Risk for sudden cardiac death associated with marathon running. *J Am Coll Cardiol*. 1996;28:428–431.
38. Virmani R, Robinowitz M, McAllister HA Jr. Nontraumatic death in joggers: a series of 30 patients at autopsy. *Am J Med*. 1982;72:874–882.
39. Thompson PD, Funk EJ, Carleton RA, et al. Incidence of death during jogging in Rhode Island from 1975 through 1980. *JAMA*. 1982;247:2535–2538.
40. Waller BF, Roberts WC. Sudden death while running in conditioned runners age 40 years or over. *Am J Cardiol*. 1980;45:1292–1300.
41. Thompson PD. The cardiovascular complications of vigorous physical activity. *Arch Intern Med*. 1996;156:2297–2302.
42. Northcote RJ, Flannigan C, Ballantyne D. Sudden death and vigorous exercise: a study of 60 deaths associated with squash. *Br Heart J*. 1986;55:198–203.
43. Thompson PD, Stern MP, Williams P, et al. Death during jogging or running: a study of 18 cases. *JAMA*. 1979;242:1265–1267.
44. Jackson RT, Beaglehole R, Sharpe N. Sudden death in runners. *N Z Med J*. 1983;96:289–292.
45. Noakes TO, Opie LH, Rose AG, et al. Autopsy-proved coronary atherosclerosis in marathon runners. *N Engl J Med*. 1979;301:86–89.
46. Van Camp SP, Bloor CM, Mueller FO, et al. Nontraumatic sports death in high school and college athletes. *Med Sci Sports Exerc*. 1995;27:641–647.
47. Maron BJ. Cardiovascular risks to young persons on the athletic field. *Ann Intern Med*. 1998;129:379–386.
48. Maron BJ, Shirani J, Poliac LC, et al. Sudden death in young competitive athletes: clinical, demographic and pathological profiles. *JAMA*. 1996;276:199–204.
49. Maron BJ, Mitchell JH. Revised eligibility recommendations for competitive athletes with cardiovascular abnormalities. *J Am Coll Cardiol*. 1994;24:848–850.
50. Maron BJ, Thompson PD, Puffer JC, et al. Cardiovascular preparticipation screening of competitive athletes: a statement for health professionals from the Sudden Death Committee (Clinical Cardiology) and Congenital Cardiac Defects Committee (Cardiovascular Disease in the Young), American Heart Association. *Circulation*. 1996;94:850–856.
51. Balady GJ, Chaitman B, Driscoll D, et al. Recommendations for cardiovascular screening, staffing, and emergency policies at health/fitness facilities. *Circulation*. 1998;97:2283–2293.
52. Kujala UM, Sarna S, Kaprio J, et al. Heart attacks and lower-limb function in master endurance athletes. *Med Sci Sports Exerc*. 1999;31:1041–1046.
52a. Albert CM, Mittleman MA, Chae CU, et al. Triggering of sudden death from cardiac causes by vigorous exertion. *N Engl J Med*. 2000;343:1355–1361.
52b. Maron BJ. The paradox of exercise. *N Engl J Med*. 2000;343:1409–1411.
53. Hambrecht R, Wolf A, Gielen S, et al. Effect of exercise on coronary endothelial function in patients with coronary artery disease. *N Engl J Med*. 2000;342:454–460.
54. Roberts WC. An agent with lipid-lowering, antihypertensive, positive inotropic, negative chronotropic, vasodilating, diuretic, anorexigenic, weight reducing, cathartic, hypoglycemic, tranquilizing, hypnotic and antidepressive qualities. *Am J Cardiol*. 1984;53:261–262. Editorial.
55. Maron BJ, Mitchell JH, eds. 26th Bethesda Conference: recommendations for determining eligibility for competition in athletes with cardiovascular abnormalities. *J Am Coll Cardiol*. 1994;24:845–899.
56. Thompson PD, Klocke FJ, Levine BD, et al. 26th Bethesda Conference: recommendations for determining eligibility for competition in athletes with cardiovascular abnormalities: Task Force 5: coronary artery disease. *J Am Coll Cardiol*. 1994;24:888–892.
57. Corrado D, Basso C, Schiavon M, et al. Screening for hypertrophic cardiomyopathy in young athletes. *N Engl J Med*. 1998;339:364–369.
58. Pelliccia A, Maron BJ. Preparticipation cardiovascular evaluation of the competitive athlete: perspectives from the 30 year Italian experience. *Am J Cardiol*. 1995;75:827–829.

59. Maron BJ, Isner JM, McKenna WJ. 26th Bethesda Conference: recommendations for determining eligibility for competition in athletes with cardiovascular abnormalities: Task Force 3: hypertrophic cardiomyopathy, myocarditis and other myopericardial diseases, and mitral valve prolapse. *J Am Coll Cardiol*. 1994;24:880–885.
60. Heath GW, Hagberg JM, Ehsani AA, et al. A physiological comparison of young and older endurance athletes. *J Appl Physiol*. 1981;51:634–640.
61. Pollock ML, Foster C, Knapp D, et al. Effect of age and training on aerobic capacity and body composition of master athletes. *J Appl Physiol*. 1987;62:725–731.
62. Rivera AM, Pels AE III, Sady SP, et al. Physiological factors associated with the lower maximal oxygen consumption of master runners. *J Appl Physiol*. 1989;66:949–954.
63. Rogers MA, Hagberg JM, Martin WH III, et al. Decline in $VO_{2\ max}$ with aging in master athletes and sedentary men. *J Appl Physiol*. 1990;68:2195–2199.
64. Fleg JL, Schulman SP, O'Connor FC, et al. Cardiovascular responses to exhaustive upright cycle exercise in highly trained older men. *J Appl Physiol*. 1994;77:1500–1506.
65. Seals DR, Hagberg JM, Spina RJ, et al. Enhanced left ventricular performance in endurance trained older men. *Circulation*. 1994;89:198–205.
66. Trappe SW, Costill DL, Vukovich MD, et al. Aging among elite distance runners: a 22-year longitudinal study. *J Appl Physiol*. 1996;80:285–290.
67. Pollock ML, Mengelkoch LJ, Graves JE, et al. Twenty-year follow-up of aerobic power and body composition of older track athletes. *J Appl Physiol*. 1997;82:1508–1516.
68. Proctor DN, Joyner MJ. Skeletal muscle mass and the reduction of $VO_{2\ max}$ in trained older subjects. *J Appl Physiol*. 1997;82:1411–1415.
69. Tanaka H, DeSouza CA, Jones PP, et al. Greater rate of decline in maximal aerobic capacity with age in physically active vs. sedentary healthy women. *J Appl Physiol*. 1997;83:1947–1953.
70. Katzel LI, Fleg JL, Busby-Whitehead MJ, et al. Exercise-induced silent myocardial ischemia in master athletes. *Am J Cardiol*. 1998;81:261–265.
71. Hagberg JM, Goldberg AP, Lakatta L, et al. Expanded blood volumes contribute to the increased cardiovascular performance of endurance-trained older men. *J Appl Physiol*. 1998;85:484–489.
72. Forman DE, Manning WJ, Hauser R, et al. Enhanced left ventricular diastolic filling associated with long-term endurance training. *J Gerontol*. 1992;47:M56–M58.
73. Child JS, Barnard RJ, Taw RL. Cardiac hypertrophy and function in master endurance runners and sprinters. *J Appl Physiol*. 1984;57:176–181.
74. Fleg JL, Lakatta EG. Role of muscle loss in the age-associated reduction in $VO_{2\ MAX}$. *J Appl Physiol*. 1988;65:1147–1151.
75. Furlanello F, Bettini R, Cozzi F, et al. Ventricular arrhythmias and sudden death in athletes. *Ann N Y Acad Sci*. 1984;427:253–279.
76. Furlanello F, Bertoldi A, Bettini R, et al. Life-threatening tachyarrhythmias in athletes. *Pacing Clin Electrophysiol*. 1992;15:1403–1411.
77. Furlanello F, Bertoldi A, Dallago M, et al. Atrial fibrillation in elite athletes. *J Cardiovasc Electrophysiol*. 1998;9:S63–S68.
78. Maron BJ, Gohman TE, Aeppli D. Prevalence of sudden cardiac death during competitive sports activities in Minnesota high school athletes. *J Am Coll Cardiol*. 1998;32:1881–1884.
79. American College of Sports Medicine. *Guidelines for Exercise Testing and Prescription*. 6th ed. New York, NY: Lippincott Williams & Wilkins; 2000.
80. Fletcher GF, Flipse TR, Kligfield P, et al. Current status of ECG stress testing. *Curr Probl Cardiol*. 1998;23:353–423.
81. Bruce RA, DeRouen TA, Hossack KF. Value of maximal exercise tests in risk assessment of primary coronary heart disease events in healthy men: five years' experience of the Seattle Heart Watch Study. *Am J Cardiol*. 1980;46:371–378.
82. Giagnoni E, Secchi MB, Wu SC, et al. Prognostic value of exercise EKG testing in asymptomatic normotensive subjects: a prospective matched study. *N Engl J Med*. 1983;309:1085–1089.
83. Allen WH, Aronow WS, Goodman P, et al. Five-year follow-up of maximal treadmill stress test in asymptomatic men and women. *Circulation*. 1980;62:522–527.
84. Blumenthal DS, Weiss JL, Mellits ED, et al. The predictive value of a strongly positive stress test in patients with minimal symptoms. *Am J Med*. 1981;70:1005–1010.
85. Okin PM, Grandits G, Rautaharju PM, et al. Prognostic value of heart rate adjustment of exercise-induced ST segment depression in the multiple risk factor intervention trial. *J Am Coll Cardiol*. 1996;27:1437–1443.
86. Rautaharju PM, Prineas RJ, Eifler WJ, et al. Prognostic value of exercise electrocardiogram in men at high risk of future coronary heart disease: Multiple Risk Factor Intervention Trial experience. *J Am Coll Cardiol*. 1986;8:1–10.
87. Rywik TM, Zink RC, Gittings NS, et al. Independent prognostic significance of ischemic ST-segment response limited to recovery from treadmill exercise in asymptomatic subjects. *Circulation*. 1998;97:2117–2122.
88. Josephson RA, Shefrin E, Lakatta EG, et al. Can serial exercise testing improve the prediction of coronary events in asymptomatic individuals? *Circulation*. 1990;81:20–24.
89. Dubach P, Froelicher VF, Klein J, et al. Exercise-induced hypotension in a male population: criteria, causes and prognosis. *Circulation*. 1988;78:1380–1387.
90. Busby MJ, Shefrin EA, Fleg JL. Prevalence and long-term significance of exercise-induced frequent or repetitive ventricular ectopic beats in apparently healthy volunteers. *J Am Coll Cardiol*. 1989;14:1659–1665.
91. Fuster V, Gotto AM, Libby P, et al. 27th Bethesda Conference: matching the intensity of risk factor management with the hazard for coronary disease events: Task Force 1: pathogenesis of coronary disease: the biologic role of risk factors. *J Am Coll Cardiol*. 1996;27:964–976.
92. Anderson KM, Wilson PW, Odell PM, et al. An updated coronary risk profile: a statement for health professionals. *Circulation*. 1991;83:356–362.
93. Smith SC Jr, Amsterdam E, Balady GJ, et al. Prevention Conference V: beyond secondary prevention: identifying the high-risk patient for primary prevention: tests for silent and inducible ischemia: Writing Group II. *Circulation*. 2000;101:E12–E16.
94. Pelliccia A, Maron BJ, Culasso F, et al. Clinical significance of abnormal electrocardiographic patterns in trained athletes. *Circulation*. 2000;102:278–284.
95. Spirito P, Seidman CE, McKenna WJ, et al. The management of hypertrophic cardiomyopathy. *N Engl J Med*. 1997;336:775–785.
96. Maron BJ. Hypertrophic cardiomyopathy. *Lancet*. 1997;350:127–133.
97. Vincent GM, Timothy KW, Leppert M, et al. The spectrum of symptoms and QT intervals in carriers of the gene for the long-QT syndrome. *N Engl J Med*. 1992;327:846–852.
98. Brugada P, Brugada J. Right bundle-branch block, persistent ST segment elevation and sudden cardiac death: a distinct clinical and electrocardiographic syndrome: a multicenter report. *J Am Coll Cardiol*. 1992;20:1391–1396.
99. McKenna WJ, Thiene G, Nava A, et al. Diagnosis of arrhythmogenic right ventricular dysplasia/cardiomyopathy: Task Force of the Working Group on Myocardial and Pericardial Disease of the European Society of Cardiology and of the Scientific Council on Cardiomyopathies of the International Society and Federation of Cardiology. *Br Heart J*. 1994;1:215–218.
100. The Sixth Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. *Arch Intern Med*. 1997;157:2413–2446.
101. Kaplan NM, Deveraux RB, Miller HS Jr. 26th Bethesda Conference: recommendations for determining eligibility for competition in athletes with cardiovascular abnormalities: Task Force 4: systemic hypertension. *J Am Coll Cardiol*. 1994;24:885–888.
102. Maron BJ, Shen W-K, Link MS, et al. Efficacy of implantable cardioverter-defibrillators for the prevention of sudden death in patients with hypertrophic cardiomyopathy. *N Engl J Med*. 2000;342:365–373.
103. Maron BJ, Klues HG. Surviving competitive athletics with hypertrophic cardiomyopathy. *Am J Cardiol*. 1994;73:1098–1104.
104. Elizari MV, Chiale PA. Cardiac arrhythmias in Chagas' heart disease. *J Cardiovasc Electrophysiol*. 1993;4:596–608.
105. Maron BJ. Sudden death in young athletes: lessons from the Hank Gathers affair. *N Engl J Med*. 1993;329:55–57.
106. Pedoe DT. Sudden cardiac death in sport: spectre or preventable risk? *Br J Sports Med*. 2000;34:137–140.
107. Nichol G, Hallstrom AP, Kerber R, et al. American Heart Association Report on the Second Public Access Defibrillation Conference, April 17–19, 1997. *Circulation*. 1998;97:1309–1314.
108. Kerber RE, Becker LB, Bourland JD, et al. Automatic external defibrillators for public access defibrillation: recommendations for specifying and reporting arrhythmia analysis algorithm performance, incorporating new waveforms, and enhancing safety. *Circulation*. 1997;95:1677–1682.

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