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AHA/ACC SCIENTIFIC STATEMENT

Eligibility and Disqualification Recommendations for Competitive Athletes With Cardiovascular Abnormalities: Task Force 8: Coronary Artery Disease

A Scientific Statement from the American Heart Association and American College of Cardiology

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Atherosclerotic coronary artery disease (ASCAD) is the leading cause of sudden cardiac death (SCD) and acute myocardial infarction (AMI) in adult athletes, variously defined as people older than age 30, 35, or 40 years (1). ASCAD can occur in younger athletes who have inherited hyperlipidemia. For many adults, SCD or AMI is the first manifestation of ASCAD, because most of these acute events are caused by coronary plaque disruption and acute coronary thrombosis in plaques that were previously not sufficiently narrowed to have caused ischemia, even during intense exercise (1). There is universal agreement that vigorous exercise, such as athletic competition, acutely, albeit transiently, increases the risk of SCD and AMI in previously healthy people (1). Vigorous exercise also transiently increases the risk for SCD and AMI in people with diagnosed ASCAD. These events may be caused by plaque disruption, but SCD in these patients may also be produced by malignant arrhythmias caused by demand ischemia or originating in areas of myocardial scar (1). In addition to ASCAD, other coronary conditions such as coronary

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vasospasm, myocardial bridging, and coronary dissection, as well as infection such as Kawasaki disease, vasculitis, and cardiac transplant vasculopathy, may also cause acute cardiac events during exercise. The present section makes recommendations on how to evaluate patients with disease of the coronary arteries and make appropriate recommendations for athletic competition. Anomalous coronary arteries are considered in the Task Force 4 report (2).

We searched PubMed for English language articles reporting exercise-related issues related to coronary diseases. This search produced no clinical trials examining how competitive athletes with coronary artery diseases should be advised regarding vigorous exercise in general or athletic competition in particular. Consequently, the following recommendations are based on case series, case reports, and consensus among the committee members.

ATHEROSCLEROTIC CORONARY ARTERY DISEASE

Patients with ASCAD can be divided into clinically manifest or symptomatic and clinically concealed or asymptomatic subgroups. The former have either experienced an acute cardiac event or have symptoms consistent with inducible myocardial ischemia, or they have findings of ischemia identified by a diagnostic testing modality such as exercise testing with or without adjunctive nuclear or echocardiographic imaging. This group includes those with "silent ischemia" who have no symptoms but have ischemia documented by provocative testing. Patients with clinically concealed ASCAD are presently and previously asymptomatic and are diagnosed as having ASCAD by the presence of coronary artery calcification on computerized tomography or by the presence of noncalcified plaque by coronary computed tomography angiography but do not have evidence of ischemia on provocative testing.

Evaluation and recommendations for patients with ASCAD are based on the following assumptions: 1) The risk of an acute exertion-related cardiac event is greater in those who have had a previous acute cardiac syndrome and lower in those whose ASCAD is clinically silent and was diagnosed by such techniques as coronary artery calcification scanning or computed tomography angiography. 2) The risk of an acute exertion-related cardiac event increases with increasing extent of coronary artery disease, reduced left ventricular systolic function, the presence and extent of ischemia, and increased electrical instability. Unstable or "vulnerable" plaques are often lipid rich (3), so it is also likely that the risk of an exertion-related plaque disruption can be reduced by aggressive lipid-lowering treatment, which has been shown to reduce the lipid content of atherosclerotic plaques (4). 3) Patients with clinically manifest ASCAD should strongly consider deferring their possible return to athletic competition to permit lesion regression and regression of lipid from the plaque. The length of this delay is not defined, but some have suggested 2 years, because substantial lesion regression has been documented to occur within 2 years of aggressive lipid management (5).

Recommendations

1. **Athletes with ASCAD should undergo maximal exercise testing to evaluate exercise tolerance, the presence of inducible ischemia, and the presence of exercise-induced electrical instability. Testing should be performed on the subject's standard medical regimen, including β-adrenergic blocking medications *(Class I; Level of Evidence C)*.**
2. **Athletes with ASCAD should undergo an evaluation of left ventricular function *(Class I; Level of Evidence C)*.**
3. **Once informed of the results of the evaluations contained in recommendations 1 and 2, adult patients with ASCAD should participate in the decision as to whether the health and psychological benefits of exercise for them outweigh the risk *(Class I; Level of Evidence C)*.**
4. **Athletes with ASCAD should undergo aggressive risk factor reduction with high-intensity statin therapy to reduce the chance of plaque disruption (6) *(Class I; Level of Evidence A)*.**
5. **It is reasonable for athletes with clinically concealed ASCAD to participate in all competitive activities if their resting left ventricular ejection fraction is >50% and they have no inducible ischemia or electrical instability *(Class IIb; Level of Evidence C)*.**
6. **It is reasonable for patients with clinically manifest ASCAD to participate in all competitive activities if their resting left ventricular ejection fraction is >50%, they are asymptomatic, and they have no inducible ischemia or electrical instability *(Class IIb; Level of Evidence C)*.**
7. **It is reasonable to restrict patients with clinically manifest ASCAD that does not fulfill the criteria in recommendation 6 to sports with low dynamic and low to moderate static demands *(Class IIb; Level of Evidence C)*.**
8. **It is reasonable to prohibit patients with clinically manifest ASCAD from competitive sport participation:**
   a. **For at least 3 months after an AMI or coronary revascularization procedure *(Class IIb; Level of Evidence C)*;**
   b. **If they have increasing frequency or worsening symptoms of myocardial ischemia *(Class IIb; Level of Evidence C)*.**

CORONARY ARTERY SPASM

Focal coronary artery spasm, usually in the presence of various degrees of coronary atherosclerosis, is a defined but uncommon cause of life-threatening arrhythmias and SCD (7,8). It can also be identified in the absence of identifiable atherosclerotic lesions by provocation studies (9). Coronary artery spasm in its classic form usually occurs with little or minimally obstructive coronary artery lesions. Although exercise-induced spasm during stress testing has been documented, it is uncommon. In most instances, it was induced during pharmacological stress tests with either dobutamine or adenosine. Reports of cardiac arrest survivors in whom coronary artery spasm has been identified as the mechanism of cardiac arrest are limited (10), although the presence of coronary vasomotor spasm identifies people with a higher risk of sudden death than the general population (11). However, susceptibility to spasm is not constant over time, being dependent on the state of the endothelium. Finally, there are also few data to suggest a specific propensity to coronary artery spasm and consequent arrhythmias in competitive athletes. When coronary vasospasm is identified or strongly suspected during exercise, treatment should be initiated with calcium blockers and nitrates to reduce the possibility of spasm and to control symptoms.

Recommendations

1. **It is reasonable to restrict the small subset with silent ischemia caused by coronary artery spasm who have had documented life-threatening arrhythmias and in whom the absence of clinical pain impedes identification of an adequate response to therapy (12) to sports with low dynamic and low to moderate static demands *(Class IIa; Level of Evidence C)*.**
2. **It is reasonable that athletes whose symptoms and objective evidence of spasm can be controlled with medications be allowed to participate in all levels of competition *(Class IIb; Level of Evidence C)*.**

SPONTANEOUS CORONARY ARTERY DISSECTION

Spontaneous coronary artery dissection refers to dissection of the coronary arteries without underlying atherosclerosis (13). Spontaneous coronary artery dissection is associated with late pregnancy and the peripartum state, female hormonal therapy, Marfan syndrome, exercise, chest trauma (13), and fibromuscular dysplasia (14). It is a rare cause of exercise-related cardiac events but should be considered in any young person who develops an acute cardiac syndrome during vigorous exercise or after sports-related chest trauma.

Recommendation

1. **There are insufficient data to provide definitive recommendations for sports participation, but because spontaneous dissection can occur with exertion, it is reasonable that patients with prior spontaneous coronary artery dissection be restricted to participation in sports with low to moderate dynamic and low to moderate static demands *(Class IIa; Level of Evidence C)*.**

MYOCARDIAL BRIDGING

Myocardial bridging is diagnosed when a portion of a major epicardial coronary artery is completely covered by myocardium. Myocardial bridging is commonly observed by angiography as coronary artery compression during systole. It is usually asymptomatic and of no clinical consequence but has been rarely associated with exercise-induced ischemia and exercise-related acute cardiac events (15). Pathological studies suggest that vessels whose tunneled length is long and deeper than 3 mm beneath the epicardium create the greatest vulnerability for cardiac events.

Recommendations

1. **It is reasonable for athletes with myocardial bridging and no evidence of myocardial ischemia during adequate stress testing to participate in all competitive sports *(Class IIa; Level of Evidence C)*.**
2. **It is reasonable to restrict athletes with myocardial bridging of an epicardial coronary artery and objective evidence of myocardial ischemia or prior myocardial infarction to sports with low to moderate dynamic and low to moderate static demands *(Class IIa; Level of Evidence C)*.**
3. **It is reasonable to restrict athletes who have undergone surgical resection of the myocardial bridge or stenting of the bridge to low-intensity sports for 6 months after the procedure. If such athletes have no subsequent evidence of ischemia, they may participate in all competitive sports *(Class IIa; Level of Evidence C)*.**

KAWASAKI DISEASE

Kawasaki disease is an acute febrile illness of unknown pathogenesis that is among the leading causes of acquired heart disease in children. Kawasaki disease can produce coronary artery aneurysms that predispose to myocardial ischemia, myocardial infarction, and SCD. Aneurysms can be divided by their internal diameter into small (<1.5 times normal, or <5 mm), moderate (1.5 to 4 times normal, or 5 to 8 mm), and large (>4 times normal, or >8 mm) aneurysms (16). Prompt recognition and treatment of the acute phase of Kawasaki disease can reduce the cardiac

complications, but 20% of untreated people and 4% of those treated with aspirin and intravenous immunoglobulin still develop coronary artery aneurysms (17). Risk scores for prediction of coronary artery aneurysm development are imperfect, and the broad use of intravenous immunoglobulin is recommended. Ongoing surveillance of patients after the acute phase is recommended, including serial stress tests in those patients with manifest coronary artery disease (18). Treatment of coronary artery aneurysms with antiplatelet agents, anticoagulant agents, or myocardial revascularization must be considered in evaluating decisions about a patient's return to competition.

Recommendations

1. **Patients with ≥1 large coronary aneurysms should continue antiplatelet therapy and possibly anticoagulant therapy. It is also reasonable for annual stress tests to be performed and activity to be guided by results, similar to adults with ASCAD *(Class I; Level of Evidence C)*.**
2. **Patients with myocardial infarction or revascularization should follow the guidance for adults with ASCAD *(Class I; Level of Evidence A)*.**
3. **Collision sports should be avoided in patients undergoing antiplatelet therapy *(Class I; Level of Evidence C)*.**
4. **In the absence of exercise-induced ischemia or arrhythmias, it is reasonable for patients to participate in low- to moderate-intensity static and dynamic competitive sports. Patients with persistent small to medium-sized aneurysms in ≥1 coronary arteries should continue antiplatelet therapy and undergo ongoing surveillance *(Class IIa; Level of Evidence C)*.**
5. **Patients with no coronary aneurysms during the convalescent phase and with no exercise-induced ischemia or arrhythmias may be considered for participation in all sports starting 8 weeks after the illness has resolved *(Class IIb; Level of Evidence C)*.**
6. **Patients with transient coronary aneurysms and with no exercise-induced ischemia or arrhythmias may be considered for participation in all sports 8 weeks after illness resolution. Risk reassessment is recommended at 3- to 5-year intervals or according to current guidelines *(Class IIb; Level of Evidence C)*.**

CORONARY VASCULITIS

Coronary vasculitis attributable to causes other than Kawasaki disease may affect competitive athletes of any age but is rare. These diseases include polyarteritis nodosa, Takayasu arteritis, Buerger disease, and other specific and nonspecific forms of coronary arteritis (16). SCD has been reported in previously healthy young people with unsuspected coronary vasculitis at autopsy (17). In a series of 50 cases of SCD associated with nonatherosclerotic coronary pathology (12 of whom died during or immediately after physical exertion), 6 of the 50 (12%) had autopsy evidence of coronary vasculitis (18).

There is no evidence that athletes are predisposed to coronary vasculitis at rates higher than the general age-corrected population or that the course of these athletes' disease is any different from that of the general population. There is no information in the medical literature to suggest care of the athlete should differ.

Recommendations

1. **Athletes who have recovered from coronary vasculitis can participate in all sports without restriction *(Class I; Level of Evidence C)*.**
2. **Athletes with coronary vasculitis are likely at increased risk for acute cardiac events during training or competition. It is reasonable to restrict participation in sports until the vasculitis has resolved *(Class IIa; Level of Evidence C)*.**

CARDIAC TRANSPLANT CORONARY VASCULOPATHY

The coronary arteries of orthotopic transplanted hearts develop a diffuse vasculopathy that is the leading cause of death in transplant recipients. Because the transplanted heart is initially denervated, recipients require surveillance, because they may not experience classic symptoms of cardiac ischemia. Our literature search did not detect any reports of exercise-related cardiac events in cardiac transplant recipients either because transplant vasculopathy is not associated with the same increase in exercise events as classic atherosclerosis or because too few transplant patients have participated in competitive events to highlight this issue.

Recommendations

1. **The transplant cardiologist should make the final recommendations for athletic participation for cardiac transplant recipients *(Class I; Level of Evidence C)*.**
2. **It is reasonable for cardiac transplant recipients participating in competitive athletics to undergo yearly maximal exercise testing with echocardiography using a protocol designed to simulate the cardiac and metabolic demands of the competitive event and its training regimen *(Class IIa; Level of Evidence C)*.**
3. **It is reasonable for cardiac transplant recipients with an ejection fraction >50%, no evidence of cardiac ischemia, and no electrical instability to participate in all competitive activities commensurate with their exercise tolerance *(Class IIa; Level of Evidence C)*.**

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Writing Group Disclosures

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This table represents the relationships of writing group members that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all members of the writing group are required to complete and submit. A relationship is considered to be "significant" if (a) the person receives $10,000 or more during any 12-month period, or 5% or more of the person's gross income; or (b) the person owns 5% or more of the voting stock or share of the entity, or owns $10,000 or more of the fair market value of the entity. A relationship is considered to be "modest" if it is less than "significant" under the preceding definition.
*Modest.

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†Significant.

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