JOURNAL OF THE AMERICAN COLLEGE OF CARDIOLOGY
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THE AMERICAN COLLEGE OF CARDIOLOGY FOUNDATION
PUBLISHED BY ELSEVIER INC.

VOL. 66, NO. 21, 2015
ISSN 0735-1097/$36.00
http://dx.doi.org/10.1016/j.jacc.2015.09.033

AHA/ACC SCIENTIFIC STATEMENT

Eligibility and Disqualification Recommendations for Competitive Athletes With Cardiovascular Abnormalities: Task Force 1: Classification of Sports: Dynamic, Static, and Impact

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

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The "classification of sports" section has been a part of each iteration of the recommendations for participation in sports and provides a framework by which athletes with heart disease can be prescribed or proscribed specific sports for participation (1–3). For the 36th Bethesda Conference, an earlier version of the **Figure** was constructed that characterized sports by their strength component, expressed as the relative intensity of static muscle contractions (percentage of a maximal voluntary contraction), and their endurance component, reflected by the relative intensity of dynamic exercise (regular contraction of large muscle groups) or percentage of maximal aerobic power ($\dot{V}o_{2max}$) (3). The rationale for a classification scheme applicable to the competitive athlete with cardiac disease is based on the well-described hemodynamics of each different type of exercise (static versus dynamic) (3,4), as well as the apparent cardiac adaptation of athletes who compete in these sports (5), which reflects the chronic load on the

*On behalf of the American Heart Association Electrocardiography and Arrhythmias Committee of the Council on Clinical Cardiology, Council on Cardiovascular Disease in the Young, Council on Cardiovascular and Stroke Nursing, Council on Functional Genomics and Translational Biology, and the American College of Cardiology.

The American Heart Association and the American College of Cardiology make every effort to avoid any actual or potential conflicts of interest that may arise as a result of an outside relationship or a personal, professional, or business interest of a member of the writing panel. Specifically, all members of the writing group are required to complete and submit a Disclosure Questionnaire showing all such relationships that might be perceived as real or potential conflicts of interest. The Preamble and other Task Force reports for these proceedings are available online at www.onlinejacc.org (J Am Coll Cardiol 2015;66:2343-9; 2356-61; 2362-71; 2372-84; 2385-92; 2393-7; 2398-405; 2406-11; 2412-23; 2424-8; 2429-33; 2434-8; 2439-43; 2444-6; and 2447-50).

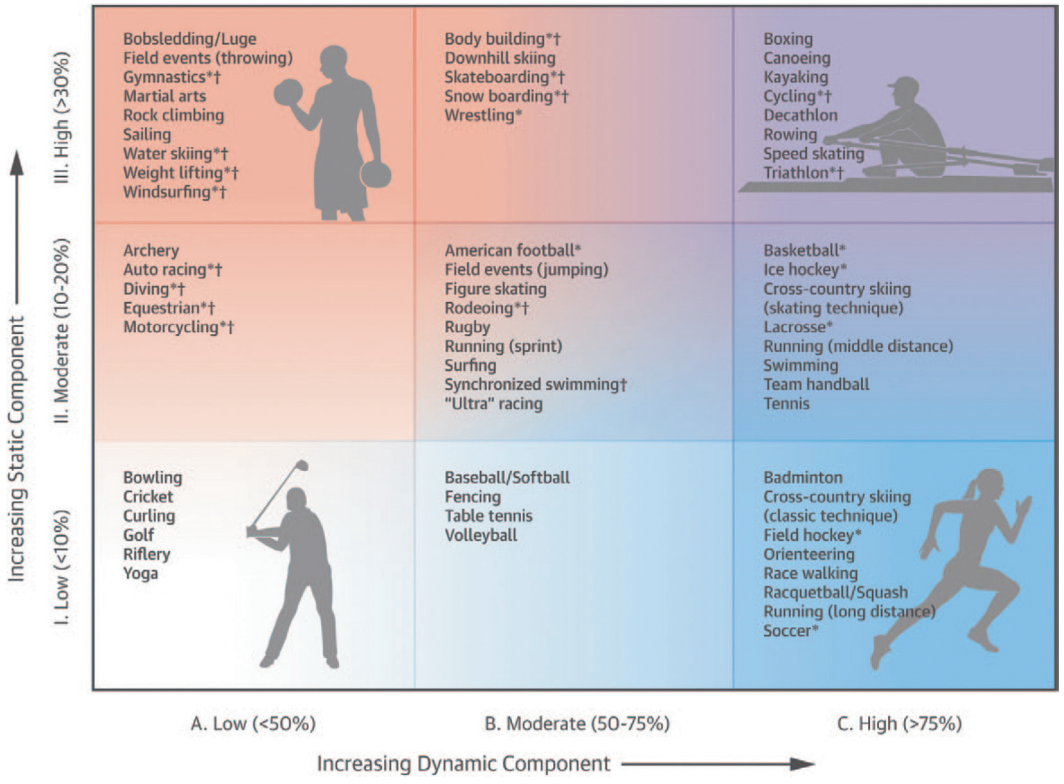
This statement was approved by the American Heart Association Science Advisory and Coordinating Committee on June 24, 2015, and the American Heart Association Executive Committee on July 22, 2015, and by the American College of Cardiology Board of Trustees and Executive Committee on June 3, 2015.

The American College of Cardiology requests that this document be cited as follows: Levine BD; Baggish AL, Kovacs RJ, Link MS, Maron MS, Mitchell JH; on behalf of the American Heart Association Electrocardiography and Arrhythmias Committee of the Council on Clinical Cardiology, Council on Cardiovascular Disease in the Young, Council on Cardiovascular and Stroke Nursing, Council on Functional Genomics and Translational Biology, and the American College of Cardiology. Eligibility and disqualification recommendations for competitive athletes with cardiovascular abnormalities: Task Force 1: classification of sports: dynamic, static, and impact: a scientific statement from the American Heart Association and American College of Cardiology. J Am Coll Cardiol 2015;66:2350-5.

This article has been copublished in *Circulation*.

Copies: This document is available on the World Wide Web sites of the American Heart Association (http://my.americanheart.org) and the American College of Cardiology (www.acc.org). For copies of this document, please contact Elsevier Inc. Reprint Department via fax (212-633-3820) or e-mail (reprints@elsevier.com).

FIGURE Classification of Sports



This classification is based on peak static and dynamic components achieved during competition; however, higher values may be reached during training. The increasing dynamic component is defined in terms of the estimated percentage of maximal oxygen uptake ($\dot{V}o_{2max}$) achieved and results in an increasing cardiac output. The increasing static component is related to the estimated percentage of maximal voluntary contraction reached and results in an increasing blood pressure load. The lowest total cardiovascular demands (cardiac output and blood pressure) are shown in the palest color, with increasing dynamic load depicted by increasing blue intensity and increasing static load by increasing red intensity. Note the graded transition between categories, which should be individualized on the basis of player position and style of play. *Danger of bodily collision (see **Table** for more detail on collision risk). †Increased risk if syncope occurs. Modified from Mitchell et al. (3) with permission. Copyright © 2005, *Journal of the American College of Cardiology*.

cardiovascular system. The underlying principle is that specific cardiovascular conditions may be more or less susceptible to complications (primarily ischemia, heart failure, or vascular compromise) based on unique characteristics of each lesion and the load placed on the heart during athletic competition.

Static contractions stimulate mechanical and metabolic afferents in skeletal muscle, which leads to large, sustained changes in blood pressure via the exercise pressor reflex (6–8). The larger the muscle mass involved, the greater the intensity of contraction, and the greater the rise in blood pressure (9); incorporation of a Valsalva maneuver during contractions will acutely and transiently increase transmural arterial pressure markedly in blood vessels outside of the chest, although left ventricular (LV) afterload does not appear to increase (10) because of a balanced rise in intracardiac and intrathoracic pressure inside the chest. Dynamic exercise increases the demand for blood flow and cardiac output in proportion to the metabolic demand ($\dot{V}o_2$): for every 1 L/min increase in oxygen uptake, there is an obligate requirement for a 5 to 6 L/min increase in cardiac output (4,11) as a function of the Fick equation. This increase is independent of age, sex, or fitness (4,12,13).

Both dynamic and static exercise result in an increase in myocardial oxygen demand: heart rate, wall tension (before and after the contraction, which determines preload and afterload), and contractile state of the LV (14). During high-intensity dynamic exercise, there is a large increase in heart rate and an increase in stroke volume that is achieved by both an increase in end-diastolic volume (Frank-Starling mechanism) (15) and a decrease in end-systolic volume (increased contractile state); for athletes, the most important factor is the increase in