

Task Force 8: Classification of Sports

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This classification of sports has been developed to allow a fundamental question to be addressed: whether it is reasonably safe to recommend that an athlete with a specific cardiovascular abnormality be eligible for a particular competitive sport (1,2). We recognize that cardiovascular disease assessments are imprecise and may change over time and be influenced by exercise training. Furthermore, there are potentially life-threatening aspects to the nature of the risk involved. We have attempted to incorporate these realities into the classification system.

Sports can be classified according to the type and intensity of exercise performed and also with regard to the danger of bodily injury from collision, as well as the consequences of syncope. Exercise can be divided into two broad types: dynamic (isotonic) and static (isometric) (3-6).

Dynamic exercise involves changes in muscle length and joint movement with rhythmic contractions that develop a relatively small intramuscular force; static exercise involves development of a relatively large intramuscular force with little or no change in muscle length or joint movement. These two types of exercise should be thought of as the two opposite poles of a continuum, with most physical activities involving both static and dynamic components. For example, distance running has low static and high dynamic demands, water skiing has principally high static and low dynamic demands, and rowing has both high static and dynamic demands.

The terms *dynamic* and *static* exercise characterize activity on the basis of the mechanical action of the muscles involved and are different from the terms *aerobic* and *anaerobic* exercise. The latter characterize activity on the basis of the type of muscle metabolism. Most high-intensity static exercise is performed anaerobically, whereas high-intensity dynamic exercise lasting for more than several minutes is performed aerobically. However, some dynamic exercises, such as sprinting or jumping, are performed primarily anaerobically. Thus, many sports are placed in the high dynamic category, including such diverse activities as skiing (cross country), running (distance), soccer, and squash. Because the cardiovascular demands of very high resistance dynamic exercise are similar to sustained static exercise, those sports that have either a sustained static component or a very high resistance dynamic component are classified together as high-intensity static exercise (e.g., weightlifting, gymnastics, and field events [throwing]).

The two primary factors determining the cardiovascular risk of competitive sports are, clearly, the athlete's abnormality and the stress under which it is placed by the sport. This involves: 1) the specific cardiovascular diagnosis and its

pathophysiological consequences; and 2) the cardiovascular response to the demands of the sport during both competition and training, which a competitive athlete in a sport may typically or reasonably be expected to undertake. The stress of the sport involves both static and dynamic components that determine the cardiovascular demands of the sport. Thus, for athletes with cardiovascular abnormalities, recommendations regarding eligibility for competition will recognize these factors as well as the attendant psychological stresses that invariably accompany competitive athletics. The cardiovascular demands produced by training or competition in a particular sport involve the type, intensity, and duration of the activity, with both peak intensity and total work performed as well as attendant neurohumoral effects and environmental factors.

RESPONSE AND ADAPTATION TO EXERCISE

The acute responses of the cardiovascular system to dynamic and static exercise are summarized in Figure 1 (5,6). Dynamic exercise performed with a large muscle mass causes a marked increase in oxygen consumption (Fig. 1, Panel A). There is a substantial increase in cardiac output, heart rate, stroke volume, and systolic blood pressure; a moderate increase in mean arterial pressure; and a decrease in diastolic blood pressure. Also, there is a marked decrease in total peripheral resistance. Static exercise, in contrast, causes a small increase in oxygen consumption, cardiac output, and heart rate, and no change in stroke volume (Fig. 1, Panel B). Moreover, there is a marked increase in systolic, diastolic, and mean arterial pressure and no appreciable change in total peripheral resistance. Thus, dynamic exercise primarily causes a volume load on the left ventricle, whereas static exercise causes a pressure load. The cardiovascular responses during dynamic exercise of a small muscle mass at low resistance or during dynamic exercise of a large muscle mass at high resistance are similar to the responses during static exercise.

The acute response to both dynamic and static exercise changes several factors that are important in determining myocardial oxygen demand: heart rate, wall tension, and contractile state of the left ventricle (LV) (7,8). Wall tension is affected by pressure development and ventricular volume. In 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) and a decrease in end-systolic volume (increased contractile state). In high-intensity static exercise, a smaller increase occurs in heart rate and little change occurs in end- and end-systolic volumes of the LV.

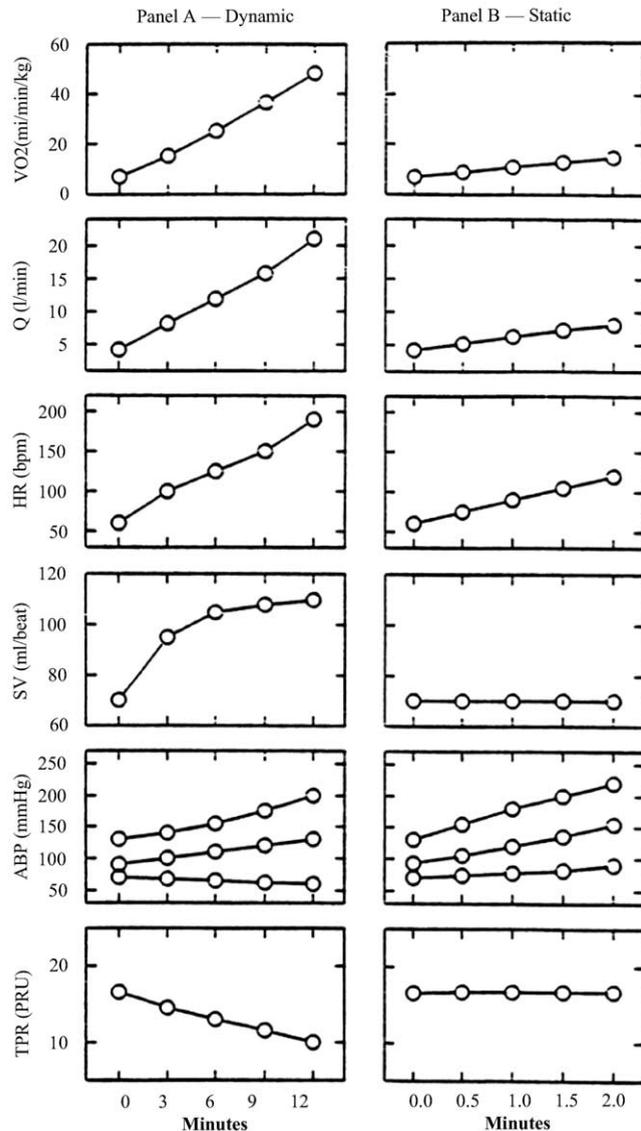


Figure 1. Cardiovascular response to exercise. (A) Response to dynamic exercise of progressively increasing workload to maximal oxygen consumption. (B) Response to a static handgrip contraction at 30% maximal voluntary contraction. ABP (mm Hg) = systolic, mean and diastolic arterial blood pressures; HR (bpm) = heart rate (beats/min); Q (l/min) = cardiac output (liters/min); SV (ml/beat) = stroke volume; TPR (PRU) = total peripheral resistance in peripheral resistance units; VO₂ (ml/min/kg) = oxygen consumption (ml/min × body weight in kg). Reprinted with permission from Mitchell JH, Raven PB. Cardiovascular adaptation to physical activity. In: Bouchard C, Shephard RJ, Stephens T, editors. Physical Activity, Fitness and Health: International Proceedings and Consensus Statement (Fig. 17.2). Champaign, IL: Human Kinetics Publishers. Copyright 1994 by Human Kinetics Publishers, Inc.

However, arterial pressure and contractile state of the ventricle are increased. Thus, both dynamic and static exercise increase factors that are important in determining myocardial oxygen demand.

The chronic adaptation of the cardiovascular system to repeated bouts of dynamic exercise (training) results in an increase in maximal oxygen uptake (5,6). This is due to an increased maximal stroke volume and arteriovenous oxygen difference. Athletes who train in sports with a high dynamic

component have a large absolute LV mass and chamber size (eccentric hypertrophy) (6,9,10). This eccentric hypertrophy develops gradually and correlates with a high maximal stroke volume and high maximal oxygen uptake. Also, the skeletal muscles involved in the dynamic exercise training become more oxidative and less glycolytic with an increase in the number and size of the mitochondria and an increased number of capillaries. These changes contribute to the larger maximal arteriovenous oxygen difference seen in endurance athletes.

The chronic adaptation of the cardiovascular system to static exercise training results in little or no increase in maximal oxygen uptake. However, athletes who participate in sports with a high static component also have a large LV mass but no increase in chamber size (concentric hypertrophy) (6,9,10). In addition, the skeletal muscles involved in the static exercise training become more glycolytic and less oxidative, and there is an increase in skeletal muscle mass primarily by fiber hypertrophy with a small degree of hyperplasia via stem cell activation.

ATHLETE'S HEART

As previously mentioned, participation in sports with a high dynamic demand (endurance) or with a high static demand (power) causes an increased cardiac mass and structural remodeling in many athletes. This finding has been shown in detail over three decades in a multitude of echocardiographic studies and more recently by cardiac magnetic resonance imaging (6,9,10). The changes resulting from training include enlargement and increased volume of the right and LV chambers, sometimes accompanied by increased thickness of the LV wall, and increased size of the left atrium, with preservation of systolic and diastolic function. Extreme changes in cavity dimensions and LV wall thickness are most commonly associated with training in rowing, cross-country skiing, cycling, and swimming, but paradoxically are uncommon as a consequence of training in ultra-endurance sports (10). The increased cardiac dimensions associated with athletic training are related to body surface area or lean body mass and consequently are less pronounced in women (10).

Participation in sports with a high static demand (e.g., weightlifting or wrestling) is associated with LV wall thickness that is usually normal in absolute terms (less than 12 mm) but disproportionately increased in relation to cavity size. More substantial LV wall thickness (13 mm or more in men and 12 mm or more in women), occasionally encountered in competitive athletes, creates a differential diagnosis with hypertrophic cardiomyopathy (9,10) (also see Task Force 1).

CLASSIFICATION OF SPORTS

A classification of sports is provided in Figure 2, which relates individual competitive sports to the two general types of exercise: dynamic and static (3-6). Each sport is catego-

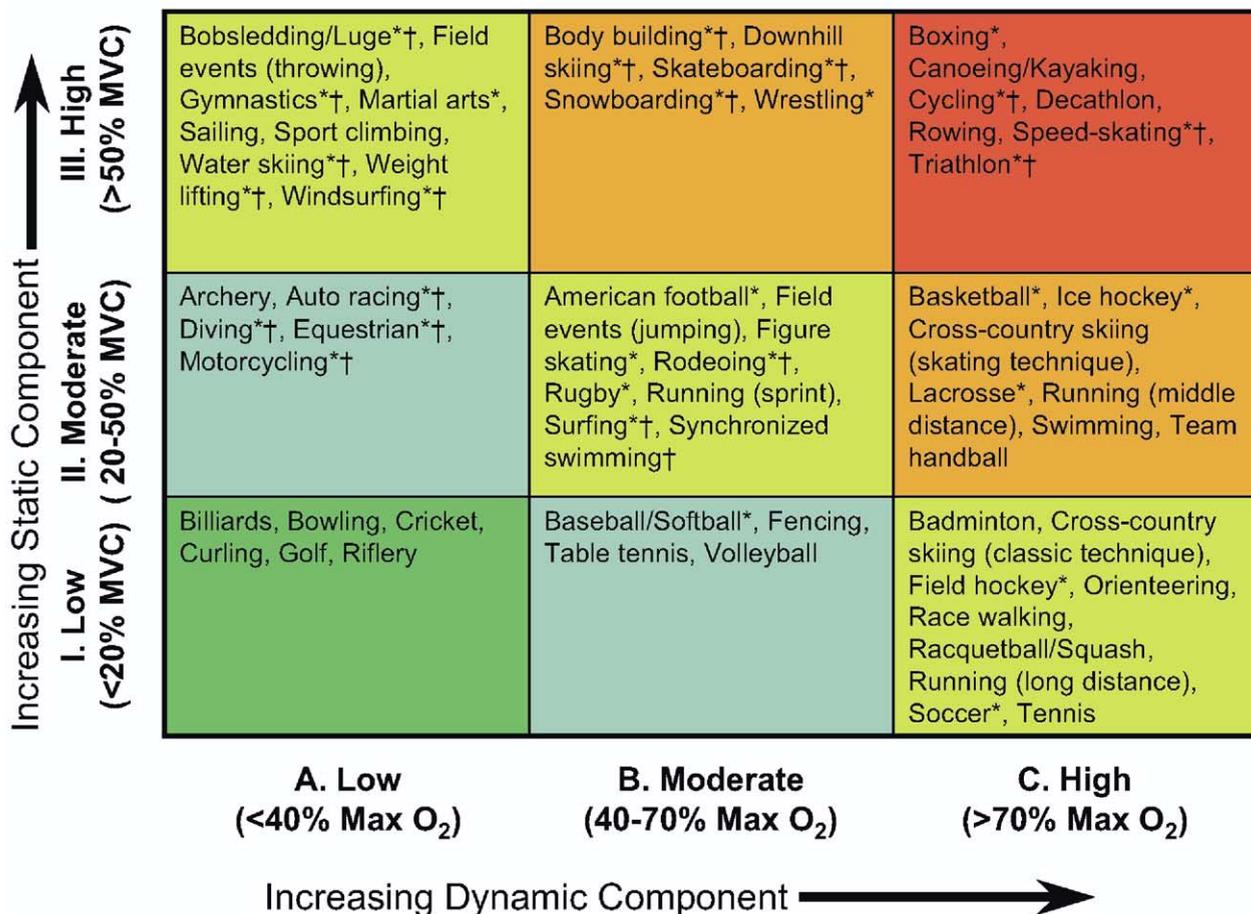


Figure 2. Classification of sports. This classification is based on peak static and dynamic components achieved during competition. It should be noted, however, that higher values may be reached during training. The increasing dynamic component is defined in terms of the estimated percent of maximal oxygen uptake (MaxO₂) achieved and results in an increasing cardiac output. The increasing static component is related to the estimated percent of maximal voluntary contraction (MVC) reached and results in an increasing blood pressure load. The lowest total cardiovascular demands (cardiac output and blood pressure) are shown in **green** and the highest in **red**. **Blue, yellow, and orange** depict low moderate, moderate, and high moderate total cardiovascular demands. *Danger of bodily collision. †Increased risk if syncope occurs.

ized by the level of intensity (low, medium, high) of dynamic or static exercise generally required to perform that sport during competition. It also recognizes those sports that pose significant risk due to bodily collision, either because of the probability of hard impact between competitors or between a competitor and an object, projectile, or the ground; as well as the degree of risk to the athlete or others if a sudden syncopal event occurs. Thus, in terms of their dynamic and static demands, sports can be classified (Fig. 2) as IIIC (high static, high dynamic), IIB (moderate static, moderate dynamic), IA (low static, low dynamic), and so forth. For example, an athlete with a cardiovascular abnormality that contraindicates a sport that produces a high pressure load on the LV may be advised to avoid sports classified as IIIA, IIIB, and IIIC. It should be emphasized that in terms of the classification of sports matrix presented in Figure 2, cardiovascular abnormalities designated as compatible with a high level of intensity in any particular category also (by definition) permit participation in levels of lesser intensity. For example, if class IC sports are appro-

priate (low static/high dynamic), then so are classes IA and IB (low static/low and moderate dynamic).

The sports matrix in Figure 2 should not be regarded as a rigid classification, but rather a spectrum in which some athletes in the same sport could possibly deserve placement in different categories. Furthermore, some sports involve heterogeneity with respect to static and dynamic cardiovascular demands in either different athletic disciplines—such as parallel bars and floor exercises in gymnastics or positions such as lineman and running back in football, or goalkeeper and mid-fielder in soccer. We have not formulated such distinctions in the matrix, but these should be taken into consideration when making clinical decisions regarding the eligibility and disqualification for competitive sports.

LIMITATIONS OF CLASSIFICATION

There are important limitations to the present classification of sports according to the type and intensity of exercise performed, as presented in Figure 2. For example, it does not consider the emotional stress that an athlete experiences

during a competitive event, the effects of environmental factors, electrolyte abnormalities, or the specific training regimen used by the athlete. Also, for team sports the classification is based on the highest cardiovascular demands that are experienced during competition and does not consider the different cardiovascular demands of specific positions.

During all athletic competitions, the athlete's emotional involvement can substantially increase sympathetic drive, and the resulting catecholamine concentrations can increase blood pressure, heart rate, and myocardial contractility, thereby increasing myocardial oxygen demand. Also, the increase in sympathetic tone can trigger arrhythmias and aggravate existing myocardial ischemia. Thus, even in competitive sports such as golf or riflery, which have low myocardial oxygen demands owing to the exercise required, substantial increases may occur because of emotional involvement during competition. This problem is difficult (if not impossible) to quantitate, but it needs to be considered in determining the eligibility for sports participation of athletes with existing cardiovascular abnormalities.

Environmental exposure during athletic competition or training also needs to be considered. Performance at high altitudes or under water may decrease oxygen availability, whereas excessively hot or cold temperatures and high humidity can increase myocardial workload for the same intensity of exercise. Another potentially relevant environmental factor is air pollution, such as elevated carbon monoxide levels in a sport such as auto racing.

With the modern application of exercise science to competitive sports, training for competition can, in fact, be more demanding on the cardiovascular system than the competition itself. Many training regimens now use heavy resistance weight training (high static and low dynamic demand) for increasing strength and power in sports that do not include heavy static demands during competition (e.g., tennis, basketball). This concept that both the dynamic and static demands of a sport may be greater during training than in competition must be seriously considered when the eligibility of an athlete in a given sport is being determined. Also, in some cases where it is found acceptable for the athlete to participate in the competitive aspect of a specific sport but the existing training program is considered too vigorous, it may be possible to modify the training regimen

so as to reduce the cardiovascular demands to an acceptable level.

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TASK FORCE 8 REFERENCES

1. Mitchell JH, Blomqvist CG, Haskell WL, et al. Classification of sports. 16th Bethesda Conference: cardiovascular abnormalities in the athlete: recommendations regarding eligibility for competition. *J Am Coll Cardiol* 1985;6:1198-9.
2. Mitchell JH, Haskell WL, Raven PB. Classification of sports. 26th Bethesda Conference: cardiovascular abnormalities in the athlete: recommendations for determining eligibility for competition in athletes with cardiovascular abnormalities. *J Am Coll Cardiol* 1994;24:864-6.
3. Asmussen E. Similarities and dissimilarities between static and dynamic exercise. *Circ Res* 1981;48 Suppl 1:I3-10.
4. Mitchell JH, Wildenthal K. Static (isometric) exercise and the heart: physiological and clinical considerations. *Annu Rev Med* 1974;25:369-81.
5. Mitchell JH, Raven PB. Cardiovascular adaptation to physical activity. In: Bouchard C, Shephard R, Stephen T, editors. *Physical Activity, Fitness, and Health: International Proceedings and Consensus Statement*. Champaign, IL: Human Kinetics, 1994:286-98.
6. Gallagher KM, Raven PB, Mitchell JH. Classification of sports and the athlete's heart. In: Williams RA, editor. *The Athlete and Heart Disease: Diagnosis, Evaluation and Management*. Philadelphia, PA: Lippincott Williams & Wilkins, 1999:9-21.
7. Sonnenblick EH, Ross JJ, Braunwald E. Oxygen consumption of the heart: newer concepts of its multifactorial determination. *Am J Cardiol* 1968;22:328-36.
8. Mitchell JH, Hefner LL, Monroe RG. Performance of the left ventricle. *Am J Med* 1972;53:481-94.
9. Pelliccia A, Maron BJ, Spataro A, Proschan MA, Spirito P. The upper limit of physiologic cardiac hypertrophy in highly trained elite athletes. *N Engl J Med* 1991;324:295-301.
10. Maron BJ. Sudden death in young athletes. *N Engl J Med* 2003;349:1064-75.

Appendix 1

Dr. Steven P. Van Camp declared that he served as an expert witness for the following court cases which may be related to the 36th Bethesda Conference: 2001—Defense, young athlete with cardiomyopathy dying suddenly; 2002—Defense, ephedra ingestion and exercise-related death; 2003—Defense, two cases of ephedra ingestion and exercise-related death; 2005—Defense, ephedra ingestion and exercise-related death; Defense, ephedra ingestion and myocardial infarction. The other authors of this report declared that they have no financial relationships with industry or others pertinent to this topic.