special attention to classifications of mortality, from sudden cardiac arrest, from intractable cardiac arrhythmia, and from cardiac death of all other causes. Without clearly defining causes of death in this population of people, it will be impossible to evaluate the efficacy of AICD-like technology.

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# Exercise and sudden cardiac death

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Studies have shown that regular exercise is associated with a substantially lower over-all risk for sudden death and cardiovascular mortality.[1-3] However, performing exercise is associated with a small but statistically significant increase in the risk for sudden death.[3,4] Siscovick et al.[3] showed that exercise is associated with varying degrees of both short-term risk and long-term benefit, with the magnitude of risk and benefit dependent upon the degree of habitual vigorous exercise performed.

Obstructive coronary artery disease secondary to atherosclerosis is the most frequent cause of exercise-related sudden death in patients over the age of 35,[4-10] whereas hypertrophic cardiomyopathy and congenital coronary artery disease are the major etiologies underlying exercise-related mortality in younger patients.[6,7,11] The goal of the clinician is to determine the presence and extent of underlying disease that may predispose to morbidity and mortality, and to make a recommendation regarding how to maximize benefit and minimize risk from exercise.

**Hypertrophic cardiomyopathy.** Maron et al.[11] evaluated the cause for sudden death in 29 highly conditioned, competitive athletes, ranging in age from 13 to 30 years. Autopsy revealed that 50% of the patients had hypertrophic cardiomyopathy. Waller's series[6] showed that 21% of athletes dying during or shortly after exercise had hypertrophic cardiomyopathy.

Patients with hypertrophic cardiomyopathy may seek medical advice because of chest pain, dyspnea on exertion, or syncope. However, many with hypertrophic cardiomyopathy have no functonal limitation or symptoms, and sudden death may be the first manifestion of disease.[12-15] A profile of 71 patients with hypertrophic cardiomyopathy who died suddenly showed that 46% were asymptomatic or had rare transient symptoms, while another 31% had only mild symptoms.[16] Of note, 40% of subjects in this series had been engaged in moderate to severe exertion at the time of cardiac arrest.[16]

Physical examination may reveal characteristic abnormalities of carotid upstroke and apical impulse, and a murmur that changes with diagnostic maneuvers, but classic findings are frequently not present. An abnormal ECG is found in a majority of patients with hypertrophic cardiomyopathy,[12,16,17] so that a normal ECG is useful for excluding disease.[18] It should be noted that the most frequent ECG abnormalities are not specific for hypertrophic cardiomyopathy and may be seen in athletes without cardiac disease.[19] The diagnosis of hypertrophic cardiomyopathy is best made by echocardiography.

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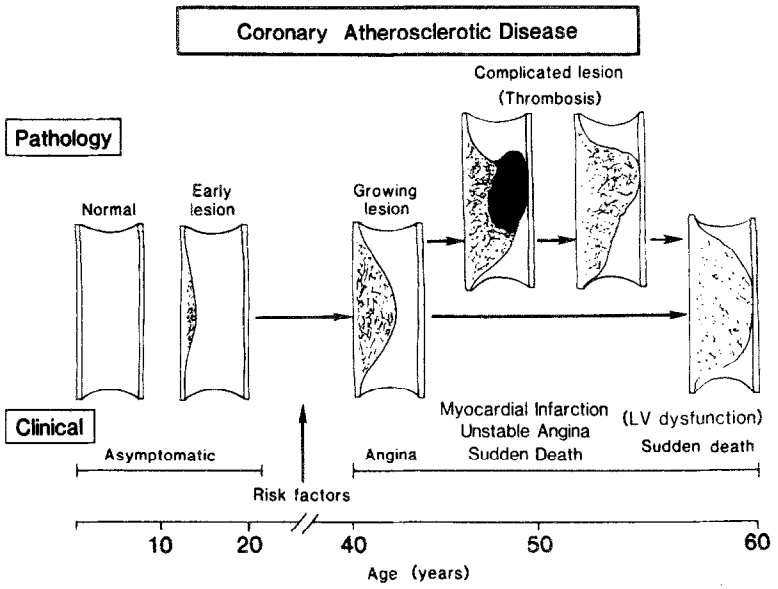


**Fig. 1.** Evolution of coronary atherosclerotic disease. A patient with an "early lesion" may have a negative exercise test because the obstruction is not hemodynamically significant. Development of a "complicated lesion" at a later point could lead to coronary occlusion, with resultant ventricular arrhythmia and sudden death. (From Fuster V, et al. J Am Coll Cardiol 1985;5:175B-184B. Reproduced by permission.)

Athletic training may be associated with cardiac changes that mimic those of hypertrophic cardiomyopathy.[20] The two can usually be distinguished, but there is a grey zone where differentiation may be difficult.[20]

Young patients with hypertrophic cardiomyopathy and patients with history of syncope or a family history of sudden death appear to be at increased risk for sudden death.[12, 13, 16, 21] Ambulatory electrocardiography has identified ventricular tachycardia as an indicator of increased risk for sudden death.[22, 23] Hemodynamic variables have no predictive value with respect to risk for sudden death.[12, 16]

Screening for hypertrophic cardiomyopathy is controversial. As the young appear to be at increased risk for sudden death, it is important to identify patients with this disorder as early as possible. Although not specific, the ECG is sensitive for identifying those with hypertrophic cardiomyopathy (particularly in patients with hypertrophic cardiomyopathy and sudden death) and is a useful screening test. Echocardiography should be performed on high-risk patients (i.e., history of syncope, family history of hypertrophic cardiomyopathy or sudden death, suggestive physical examination or ECG). Patients with hypertrophic cardiomyopathy must be advised regarding exercise.[24]

**Congenital coronary anomaly.** Congenital anomalies of the coronary arteries are the second frequent cause of exercise-related sudden cardiac death in young athletes.[6, 25-28] In a combined series, Waller[6] found that 35% of athletes less than 30 years old who died during or shortly after exercise had congenital coronary anomaly as the cause of death. Origin of the left main coronary artery from the right sinus of Valsalva, with passage of the artery between the aorta and pulmonary artery,[6, 26] is the most common coronary artery anomaly in autopsy studies following exercise-related sudden death. The proposed mechanism for death in this entity is closure of the orifice of the left main artery secondary to expansion of the great vessels during exercise.[26] Less usual coronary anomalies implicated in exercise-related sudden death include origin of the right coronary artery from the left sinus of Valsalva (with the artery coursing between the aorta and pulmonary artery), coronary artery hypoplasia, and origin of the left main coronary artery from the pulmonary artery.[27, 28] An intramural coronary artery, or myocardial bridge, is of questionable importance for sudden cardiac death.[29, 30]

With the exception of a left main coronary artery arising from the pulmonary artery, a coronary artery anomaly is rarely suspected during life. Liberthson et al.[31] reviewed 20 cases of exercise-related death in patients with the left coronary artery originating from the right sinus of Valsalva, and found that 20% had exertional chest pain, syncope, or ventricular arrhythmia before death. Roberts[25] noted that the

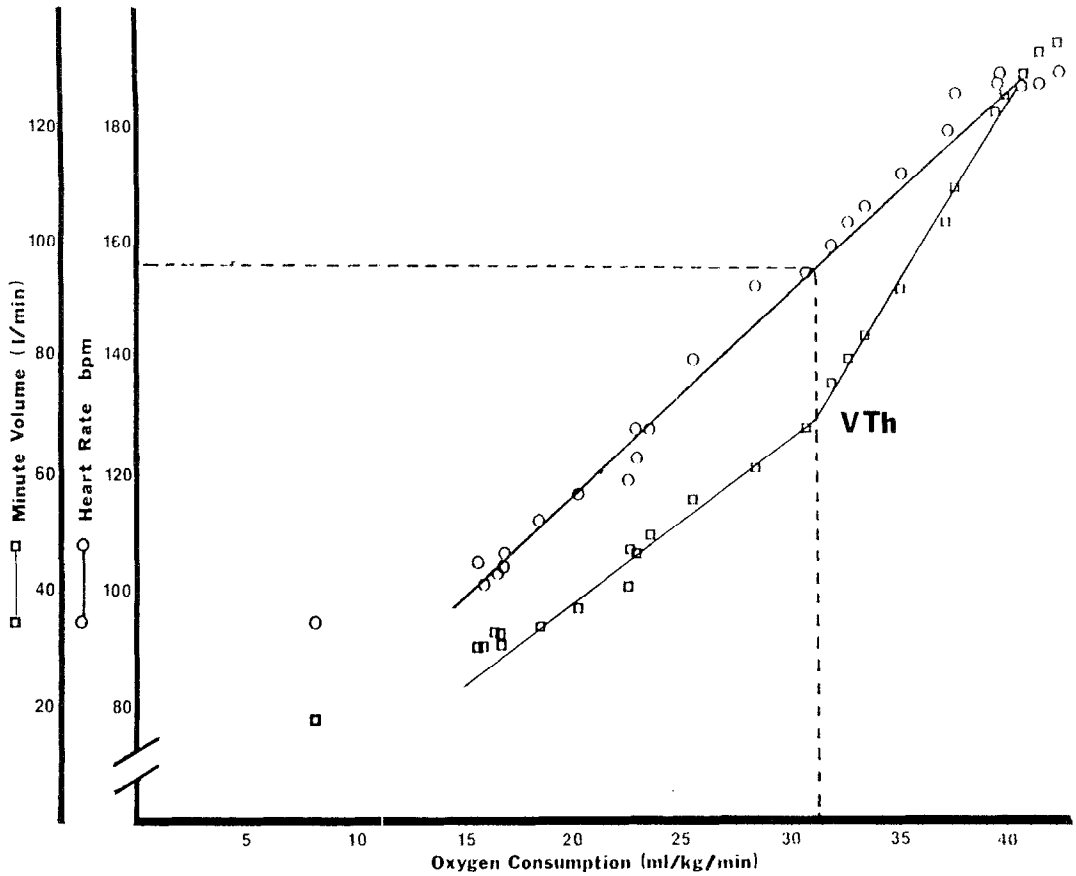


**Fig. 2.** Use of exercise ventilatory measurements to derive the heart rate at ventilatory threshold *(VTh)*. VTh is located where a nonlinear rise in minute ventilation with respect to oxygen consumption occurs during progressive exercise. The corresponding oxygen consumption and heart rate can be derived from this graph, and used to formulate an exercise prescription.

stress ECG, particularly when submaximal, is not a reliable test. The two-dimensional echocardiogram may be able to evaluate the origin of the left main coronary artery. Patients with a coronary artery configuration predisposing to sudden death should be restricted from exercise.[24]

**Atherosclerotic coronary artery disease.** Over 90% of conditioned (defined as exercise performed daily or several times per week) patients over the age of 30 who suffer exercise-related sudden death have atherosclerotic coronary artery disease.[6] Many adults with exercise-related mortality have symptoms or are known to have coronary artery disease prior to death.[10, 32, 33] However, Weaver et al.[33] studied 23 survivors of exertion-related sudden death, and found that 43% had no prior manifestion of cardiovascular disease.

The results of an ECG stress test may be valuable for diagnosis of coronary disease and assessment of prognosis.[34-38] However, limitations of both a positive test and a negative test for identifying asymptomatic patients at increased risk for sudden death have been recognized.[39-45] Asymptomatic patients have a 5% to 10% risk of having coronary disease, resulting in a low predictive value for a positive response.[41, 44-47] Furthermore, sudden death may occur despite a negative exercise test.[45] For example, McHenry et al.[48] noted only one case of sudden death over an 8-year period from among a group of 61 asymptomatic males with abnormal ST segment depression during exercise, while sudden death occurred in 7 of 833 patients who had no abnormal ST depression. There are several factors that may explain these results. Death may have been secondary to a noncardiac etiology or to an abnormalty not present at the time of testing (i.e., electrolyte disturbance, drug use, etc.), or exercise may have been inadequate to produce myocardial ischemia. The ECG, a relatively insensitive measure of myocardial ischemia, may have failed to reflect myocardial ischemia during the test.[49] Recent information regarding the pathophysiology of myocardial infarction and sudden death[50-54] provides another possible explanation for the failure of the exercise test to identify a patient at risk for

sudden death (Fig. 1). Davies and Thomas[51] found that 95 of 100 patients with death due to coronary disease had an acute arterial lesion (either thrombus or plaque fissuring); 26 of 74 thrombi were found at sites without preexisting high-grade stenosis. Thus, at the time of the exercise test there may be minimal disease, and as a result there is no abnormality detected during exercise. Sudden development of a complicated plaque and/or platelet deposition and thrombus formation could create a critically narrowed artery, resulting in myocardial ischemia and fatal arrhythmia.

Besides false negative and false positive tests, there are other factors to keep in mind when determining the utility of ECG stress testing. The value of the test can be increased by taking into consideration risk factors for coronary disease, using stringent ST criteria for a positive test, and by application of multiple exercise test variables (i.e., hemodynamic response to exercise, duration of exercise, etc.).[41, 45, 46, 55-63] Combining the stress ECG with another testing modality (e.g., exercise thallium or radionucleotide ventriculography) also improves the diagnostic and prognostic value of the stress test.[38, 45, 46, 49, 63, 64]

The results can also be used to derive an exercise prescription.[65-67] A review of 25 cases of cardiac arrest from among 2,464 patients enrolled in supervised exercise rehabilitation[68] showed that compliance with prescribed training heart rate range was lower in patients who sustained cardiac arrest. Another study[69] showed that 9 of 15 patients who had sudden death during cardiac rehabilitation were exercising in excess of 85% of maximum heart rate. Compliance with recommended exercise intensity derived from the individual's response to exertion in the exercise laboratory may reduce the risk associated with exercise.

With these considerations in mind, we have adopted the following approach. There are data suggesting that the person who has not been exercising and wants to begin regular exercise is at the highest risk for exercise-related mortality.[3] It is reasonable to test such patients, particularly if there are cardiac risk factors or clinical findings suggestive of coronary artery disease.[43] In addition, patients with chronic stable angina are tested to determine whether there are indications of severe disease.[60-62] Following evaluation, an exercise prescription is derived. Based on the physiologic response to exercise, we feel that measuring ventilatory gases or venous lactate during exercise, in addition to following the hemodynamic and ECG response, is the most effective means for establishing optimal exercise intensity guidelines.[70] Exercise above the lactate threshold, identified as the point where there is an increase in lactate during incremental exercise, is associated with hormonal and metabolic changes that may increase the risk of exercise (e.g., elevated catecholamines).[71-75] In addition, exercise above the lactate threshold is characterized by a reduced capacity for sustained work.[76, 77] Since there is little to gain from an overall fitness perspective by exercising above this intensity, it seems reasonable to recommend that exercise intensity not surpass the lactate threshold. Ventilatory measurements made during exercise can be used to closely approximate the lactate threshold (ventilatory threshold)[76-78] and derive a target heart rate for exercise prescription (Fig. 2).

We recommend that patients with exercise-related angina or ischemic ECG changes exercise below a heart rate at which evidence of myocardial ischemia occurs. The heart rate corresponding to lactate or ventilatory threshold should be determined and used as the upper limit of exercise for patients with negative tests or for patients with signs of myocardial ischemia at higher heart rates. This approach will improve compliance, and may reduce cardiovascular risk during exercise. Care should be taken when generalizing the exercise prescription to different types of exercise, with consideration of the different hemodynamic effects of predominantly arm vs leg exercise or static vs dynamic type exercise.[79-84]

**Summary.** There are other causes of exercise-related sudden cardiac death (including aortic rupture, trauma, myocarditis, dilated cardiomyopathy, congential prolonged QT syndrome, valvular heart disease, and possibly mitral valve prolapse),[5, 7, 19] but the disorders described above account for the majority of structural cardiovascular disorders underlying mortality associated with exercise. Clinical evaluation (i.e., history, physical examination, ECG, chest x-ray examination) will identify some patients at increased risk.[85] Echocardiography and ambulatory ECG monitoring in selected subjects provide additional important information. In particular, one should consider performing electrocardiography or echocardiography on young athletes in an effort at early detection of hypertrophic cardiomyopathy. The issue of exercise testing of asymptomatic patients as a screening device for coronary artery disease remains unsettled. It is important to remember the limitations of exercise testing as a screening procedure. The information may be useful for identifying some high-risk patients, particularly when multiple variables are considered. In addition, the exercise test can be used to derive an exercise

prescription that may aid in reducing exercise-related mortality.

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