Exercise: A Risk for Sudden Death in Patients With Coronary Heart Disease

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Although sudden arrhythmic death is usually unrelated to exertion, there is more than anecdotal evidence that strenuous exercise in patients with coronary heart disease carries an additional risk for sudden death. When cardiac arrest has been observed after exercise stress testing or within seconds after collapse associated with exertion, ventricular fibrillation has usually been present.

In recent years, regular physical exercise has been widely advocated, popularized and commercialized, at least in part because of perceived health benefits. Indeed, a body of evidence (1–9) suggests that regular exercise may help to prevent coronary heart disease and its complications. However, the value of exercise in patients with known coronary heart disease is controversial (10–12). Many patients acquire unquestioned psychologic benefits from regular exercise, as well as measurable (and perhaps beneficial) alterations in coronary risk factors (13–15). Nevertheless, even ardent proponents of physical conditioning would probably agree that there are uncertainties about the influence of regular exercise in favorably altering the course of patients with known coronary heart disease. Although the data are limited, exercise conditioning has not been shown to have beneficial effects on extant coronary atherosclerosis or on the incidence of reinfarction, complex ventricular arrhythmias or mortality (16–20). Moreover, there is an uncomfortable but persistent awareness of an association between vigorous exertion and sudden arrhythmic death (21–23). The general recognition of this association is emphasized by the ever present availability of a defibrillator during exercise stress testing and during exercise sessions in cardiac rehabilitation programs.

In this report we have attempted to establish an estimate of the risk of sudden death associated with physical exertion, particularly in patients with coronary heart disease.

Who Is at Risk for Exertion-Related Cardiac Arrest?

The normal heart, even when subjected to vigorous forms of stress, is protected from lethal arrhythmias except in unusual conditions such as profound electrolyte derangement or adverse drug reactions. Victims of sudden death almost always have underlying heart disease, although it has sometimes not been previously recognized. Coronary artery disease is found in about 80% of victims of sudden cardiac death (24), whereas other abnormalities, such as cardiomyopathy, valvular heart disease or primary arrhythmic disorders, may also cause unexpected cardiac arrest.

Although exertion-related cardiac arrest appears to be confined to patients with structural heart disease or metabolic abnormalities, a third of these individuals may be asymptomatic and unaware of underlying disorders (25,26). Perhaps the legendary Pheidippides, who collapsed and died after running from Marathon to Athens, was free of cardiac disease but, more likely, such was not the case. It is probable that all individuals with cardiovascular disease, including a proportion of asymptomatic men during their middle and later years, are at relatively increased risk for sudden cardiac death.
Mechanisms Underlying Sudden Death in Cardiac Patients

Ventricular fibrillation. Ventricular fibrillation is the arrhythmia usually underlying the sudden cardiac death syndrome (27), particularly in cases of exertion-related events (28–30). In following up a large number of patients resuscitated from out-of-hospital ventricular fibrillation, we have recognized three major clinical settings in which ventricular fibrillation occurs: 1) as a complication of typical acute myocardial infarction; 2) as a manifestation of transient myocardial ischemia, especially during or after exertion; and 3) as an event apparently unassociated with evidence of ischemia and occurring during sedentary activities. In the latter setting, ventricular fibrillation most often occurs in patients with advanced coronary heart disease, as evidenced by prior myocardial infarction and impaired left ventricular function. The specific events precipitating ventricular fibrillation are elusive. Factors that might cause the arrhythmia include coronary vascular spasm (31), local release of potent circulating substances such as thromboxane (32) and arousal of the central and peripheral nervous systems (33). Any of these events could cause dispersion of electrical conduction and recovery, thereby providing a milieu for the emergence of ventricular fibrillation.

Myocardial ischemia. Transient myocardial ischemia is a plausible cause for most episodes of exertion-related cardiac arrest in patients with coronary heart disease. In assessing resuscitated patients who collapsed during or after moderate to heavy exertion, we found that, compared with persons with nonexertion-related cardiac arrest, these patients had fewer physical limitations and more often had no recognized heart disease preceding collapse. In addition, premonitory warning symptoms were noted in only about 25%, and less than one-third had new Q waves or enzymatic evidence of myocardial necrosis after resuscitation. Further evaluation has demonstrated that these patients have few episodes of ambiant ventricular arrhythmia during prolonged periods of electrocardiographic monitoring (34). Clearly, there is a distinct possibility that physical exertion might have provoked ischemia and cardiac arrest in these patients.

Incidence of Exertion-Related Cardiac Arrest

Although there has been no large, prospective assessment of the role of exertion in precipitating cardiac arrest, some relevant information is available. For example, in patients treated by our paramedic system in Seattle, 36 (11%) of 316 consecutive victims had collapsed during or immediately after exertion or stress. This incidence is similar to that of 17% of 150 patients reported in Miami (35). In autopsy registries, which have obvious limitations and may also include a greater proportion of unexplained deaths and younger victims than is present in a consecutive series, the incidence of exertion-related cardiac arrest was reported to be 10 to 30% of all sudden deaths (36,37). In studies of unexpected instantaneous death in younger persons (38–40), cardiac arrest was commonly found to be associated with physical activity or stress. Unfortunately, these reports fail to define the risk of physical exertion in precipitating cardiac arrest, principally because they provide no assessment of the total number of hours devoted to exertion by these victims.

A statewide survey in Rhode Island of cardiac arrest related to jogging (41) reported 12 deaths over 6 years, representing a rate of 13 deaths per 100,000 joggers per year, or 1 death per 396,000 hours of jogging. In contrast, the total death rate for nonvigorous activity, including both expected and unexpected deaths, was one death per 3 million person hours—a 10-fold difference. Although these observations are epidemiologically incomplete, they tend to point toward a causal relation between physical exertion and sudden death.

Exertion-Related Cardiac Arrest in Persons Without Evident Heart Disease

From reports of deaths during physical activity, it is clear that many affected individuals had symptoms that in retrospect suggested the presence of heart disease. Often, these symptoms had not been recognized, and cardiac arrest, was the first recorded manifestation of underlying heart disease.

In a prospective 5 year survey (42) involving approximately 270,000 men (most without, but some with a history of coronary heart disease), 42% of the sudden coronary deaths occurred in persons without previously recognized coronary heart disease. About one-third of these deaths occurred within minutes of engaging in activities known to be associated with myocardial ischemia, or in the setting of suspected sympathetic nervous system stimulation. In another report (43) of exercise in a predominantly normal population of middle-aged persons, one cardiac arrest occurred in 375,000 person hours of exercise.

In the Framingham Study (44,45), in which community members have been followed up for 20 or more years, the incidence of sudden death was inversely related to the amount of daily exercise and physical fitness. However, there was a significant association between the mode of death and activity: sudden death occurred more often in the setting of physical activity.

A recent report (46) of 133 men who experienced cardiac arrest in Seattle estimated that the incidence of cardiac arrest was 5- to 56-fold greater during high intensity exercise than at other times. The persons considered in that study were aged 25 to 75 years and were without previously recognized cardiovascular disease. The estimated incidence of cardiac arrest during vigorous activity ranged from one case per 137,000 hours to one per 4.7 million hours at risk.

These studies serve to point out that physical exertion
may precipitate cardiac arrest in the "normal" population and that to date, prior recognition of susceptible individuals has not been possible.

**Cardiac Arrest Related to Exercise Stress Testing in Patients With Heart Disease**

There is a small but definite incidence of cardiac arrest associated with exercise testing of cardiac patients, particularly in the early minutes of recovery. A large multicenter survey (47) of complications of exercise testing showed a combined mortality and morbidity rate of four events per 10,000 tests. A wide variety of exercise protocols was employed and the patient group was heterogeneous.

In a retrospective review (48) of 10,751 symptom-limited exercise tests, five cardiac arrests were reported. All occurred in the first 4 minutes of recovery, and all five patients survived after defibrillation or cardioversion. That rate of one arrest per 2,000 tests is comparable with that of another, smaller series (49). From these observations, the relative risk of developing cardiac arrest with exercise testing (lasting approximately 15 minutes) can be roughly estimated to be one arrest per 538 hours of treadmill exercise, or 160 times greater than what might be expected to occur spontaneously (one death per 88,000 hours) (Table 1). This latter figure assumes a 10% yearly rate of sudden death in patients with presumed cardiovascular disease; if a lower (and probably more realistic) rate is used, the relative risk becomes correspondingly greater.

In interpreting estimates of incidence, it is important to recognize the characteristics of the populations being tested.
Therapeutic Implications

There is ample reason to concur with the view that exercise-induced cardiac arrest is a real phenomenon, particularly in patients with known heart disease. However, the majority of sudden deaths are temporally associated with routine activities of daily life and not with exercise (26,53). Therefore, the number of deaths due to strenuous physical exertion is relatively modest.

Role of exercise stress testing. Although it is commonly believed that exercise testing is helpful in prescribing limits for exercise programs, the available data do not support the contention that establishing such limits results in a safe conditioning program (28,29,34,42). Thus, in the CAPRI experience (50), each of the 25 exertion-related cardiac arrests occurred in patients whose exercise program had been prescribed on the basis of a symptom-limited exercise test. However, patients who developed exertion-related cardiac arrest tended to exceed target heart rates more often than those who did not experience cardiac arrest. Accordingly, it is possible that strict adherence to prescribed limits might decrease the likelihood of cardiac arrest. In reviewing exercise stress tests in patients resuscitated from cardiac arrest, we observed no distinguishing characteristics that separated those who had previously collapsed during exertion from those who had experienced cardiac arrest while sedentary (26). In addition, others (39,50) have reported that electrocardiograms during or after exercise were either normal or equivocal in a number of individuals who were tested before cardiac arrest. Aside from the obvious limitations of exercise testing in identifying patients at risk, there are also the questions of the practicality and usefulness of stress testing in asymptomatic persons. The cost of testing, following up and treating all individuals who wish to join an exercise program would be staggering (54). The incidence of "false positive" test responses in individuals with a low likelihood of coronary heart disease is another factor that further raises the cost/benefit ratio of this screening procedure (55).

Current practice in counseling cardiac patients regarding exertion is probably a middle of the road policy. Only rarely are patients advised to avoid all forms of physical exertion, whereas many are encouraged to participate in regular low level exercise, recognizing that the major hygienic benefit may be psychologic rather than physical.

Conclusions

Despite limitations, the observations summarized here may help to provide a basis for advising some patients with coronary heart disease to avoid vigorous and unsupervised exercise. In the final analysis, the risk of exertion-induced cardiac arrest should be weighed against the perceived benefits and pleasures of physical exertion. Although not well defined, there is almost certainly a relation between the intensity of exercise and the risk incurred for sudden death. We are unable to advocate firm guidelines other than that the patient and his doctor both be informed and together make a determination based on individual considerations. For many patients, participation in low to moderate non-competitive exercise is prudent. On the other hand, for persons with coronary heart disease, whether evident or not, we should carefully ponder whether vigorous exercise is worth the risk.

References


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