Causes of Sudden Death in Competitive Athletes

BARRY J. MARON, MD, FACC, STEPHEN E. EPSTEIN, MD, FACC, WILLIAM C. ROBERTS, MD, FACC

Bethesda, Maryland

Cardiovascular diseases responsible for sudden unexpected death in highly conditioned athletes are largely related to the age of the patient. In most young competitive athletes (<35 years of age) sudden death is due to congenital cardiovascular disease. Hypertrophic cardiomyopathy appears to be the most common cause of such deaths, accounting for about half of the sudden deaths in young athletes. Other cardiovascular abnormalities that appear to be less frequent but important causes of sudden death in young athletes include congenital coronary artery anomalies, ruptured aorta (due to cystic medial necrosis), idiopathic left ventricular hypertrophy and coronary artery atherosclerosis. Diseases that appear to be very uncommon causes of sudden death include myocarditis, mitral valve prolapse, aortic valve stenosis and sarcoidosis. Cardiovascular disease in young athletes is usually unsuspected during life, and most athletes who die suddenly have experienced no cardiac symptoms. In only about 25% of those competitive athletes who die suddenly is underlying cardiovascular disease detected or suspected before participation and rarely is the correct clinical diagnosis made. In contrast, in older athletes (≥35 years of age) sudden death is usually due to coronary artery disease, and rarely results from congenital heart disease.

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The highly conditioned competitive athlete epitomizes the most healthy segment of our society. Nevertheless, both youthful and older competitive athletes may occasionally die suddenly and unexpectedly, and such events take on a particularly shocking and tragic tone. For many years, our ignorance of the causes of such catastrophes was clothed in esoteric sounding descriptions, such as “sudden death syndrome,” which merely masked the fact that the precise causes were unknown. Over the past several years, the cardiovascular diseases responsible for sudden death in highly trained athletes have been the subject of several reports (1–5). This communication reviews these published findings and updates our own observations on the causes of sudden death in young competitive athletes (1).

Definitions

In our modern society, a substantial proportion of the population may consider themselves to be “athletes.” However, for the purposes of this discussion, a competitive athlete is considered to be an individual who participates in an organized team or individual sport in which regular competition is a component, a high priority is placed on excellence and achievement and vigorous training in a systematic fashion is required (6). Such competitive participation may occur at any age and encompass sports at either the youth, interscholastic, intercollegiate, professional or master’s (>40 years old) level.

Sudden death is defined as a witnessed or unwitnessed natural death resulting from sudden cardiac arrest occurring unexpectedly within 6 hours of a previously witnessed usual normal state of health.

Causes of Sudden Death in Young Athletes (<35 years of age)

Considering the large number of youthful participants in competitive athletic programs in the United States, sudden death in such individuals must be considered a relatively uncommon event. The causes of such catastrophes differ considerably, but age has proved to be the most useful variable in predicting the cause of an unexpected catastrophe in an athlete (and, hence, in determining which cardiovascular lesions should be most vigorously pursued in the diagnostic evaluation of an athlete during life) (Fig. 1).

Young athletes who die suddenly are usually male, have

From the Cardiology and Pathology Branches, National Heart, Lung, and Blood Institute, National Institutes of Health, Bethesda, Maryland.

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Address for reprints: Barry J. Maron, MD, National Heart, Lung, and Blood Institute, Building 10, Room 7B-15, National Institutes of Health, Bethesda, Maryland 20892

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participated in a variety of sports (most frequently basketball and football) and most commonly are junior high school or high school athletes at death (1). Some sudden deaths, however, occur in young athletes who reach the collegiate or even professional level of competition. Usually such catastrophes occur during or just after exertion on the athletic field, and as a result death is often attributed to intense physical activity.

At necropsy, most youthful, highly trained athletes who die suddenly show structural congenital cardiovascular lesions that probably or unequivocably could be ascertained to be the cause of death. In a series (1) of 29 young competitive athletes who had died suddenly and prematurely, we found evidence of structural cardiovascular disease in 28; in only 1 athlete was the heart structurally normal (Fig. 2). We inferred that this individual may have died of a primary ventricular arrhythmia.

A variety of cardiovascular diseases were responsible for sudden death in the 29 trained athletes (Fig. 2). The most common cause was hypertrophic cardiomyopathy, which occurred in about half of the patients. It should be emphasized, however, that the precise prevalence of cardiovascular diseases in a group of athletes dying suddenly probably cannot be derived from our series because of unavoidable limitations in patient selection. We did not study a consecutive series of sudden deaths in athletes occurring in a well-defined geographic area. Instead, such deaths were identified from several locations in the United States in several ways: 1) through news media reports describing sudden death in athletes, or 2) referral through hospital-based pathology registries or medical examiner offices. Furthermore, we also required that the athlete’s heart be available for examination, a stipulation that excluded many instances of sudden death from our series. In this report, we will describe each cardiovascular lesion that is a proved cause of sudden death in the young competitive athlete on the basis of data in our original study group (1) and in additional cases that were identified subsequently.

**Hypertrophic Cardiomyopathy**

Hypertrophic cardiomyopathy is a primary disease of cardiac muscle that is usually genetically transmitted (7) and is characterized by a hypertrophied but nondilated left ventricle, in the absence of another cardiac or systemic disease that may produce left ventricular hypertrophy (8). This increase in left ventricular mass usually results in impaired ventricular filling and compliance (9–12).
Figure 3. Clinical and morphologic features of athletes with hypertrophic cardiomyopathy. A, Heart of a 13 year old male football and baseball player, showing disproportionate thickening of the ventricular septum (VS) with respect to the posterior left ventricular (LV) free wall. RV = right ventricular wall. B, Marked disorganization of cardiac muscle cells in the disproportionately thickened ventricular septum. Adjacent hypertrophied cardiac muscle cells are oriented at oblique and perpendicular angles. C, Four second period of ventricular tachycardia, which occurred in the second minute of recovery after a routine treadmill exercise test and terminated spontaneously, in a 23 year old collegiate basketball player with hypertrophic cardiomyopathy who died suddenly. D, Standard 12 lead electrocardiogram obtained under basal conditions, showing marked symmetric T wave inversion (as much as 15 mm in depth) as well as left ventricular hypertrophy from the same athlete shown in C.

Anatomic features. Competitive athletes in our series (1) who died suddenly of hypertrophic cardiomyopathy ranged in age from 13 to 30 years (mean 19). Heart weight was 360 to 630 g, and ventricular septal thickness ranged from 15 to 30 mm (mean 20). Ventricular wall thickening was usually asymmetric (13), with the septum disproportionately thicker than most of the left ventricular free wall (Fig. 3); the ratio of septal thickness to that of the posterior free wall was usually 1.3 or greater (mean 1.5) (14). In addition, the thickened left ventricular wall (particularly the ventricular septum) characteristically showed two other morphologic features that are of potential pathophysiologic significance: 1) bizarre cellular architecture in which there is a markedly disordered arrangement of cardiac muscle cells (Fig. 3B) (15), and 2) an increased number of abnormal intramural coronary arteries with thickened walls and narrowed lumen (16).

Clinical profile. Hypertrophic cardiomyopathy is known to be an important cause of sudden death in the young (17–20), usually occurring in individuals without significant prior symptoms, including many in whom sudden death was the first manifestation of their disease (21). A clinical profile of 78 patients with hypertrophic cardiomyopathy who died suddenly (19) showed that 70% died before 30 years of age, 55% experienced no functional limitation before death and about 40% were actively engaged in moderate to severe
physical activity just before death. That many patients with hypertrophic cardiomyopathy died during or just after vigorous activity is particularly impressive, especially when one considers the relatively small proportion of the day during which most individuals perform moderate to severe exertion.

**Mechanism of sudden death.** The mechanism by which sudden death occurs in patients with hypertrophic cardiomyopathy is unclear. It is probable, however, that the structural and functional abnormalities present in hypertrophic cardiomyopathy predispose certain susceptible individuals to a malignant ventricular arrhythmia (22, 23) (which may be primary, or conceivably preceded by supraventricular tachycardia or bradyarrhythmia). Alternatively, it is possible that abrupt and marked hemodynamic changes associated with a reduction in left ventricular volume (in the absence of arrhythmia) may precipitate syncope or sudden death in selected patients with hypertrophic cardiomyopathy (24).

It is remarkable that even though this cardiac malformation (or the predisposition to it) is probably present at or near the time of birth, individuals so afflicted can compete for many years without either being aware of their disease or experiencing significant symptoms. For example, one of our athletes who died of hypertrophic cardiomyopathy was a professional tennis player at the time of her death, having competed intensely in that sport for more than 15 years (1).

**Idiopathic Concentric Left Ventricular Hypertrophy**

We also examined at necropsy other young athletes with an unexplained increase in left ventricular mass (Fig. 4). Although each of these athletes had a hypertrophied, nondilated left ventricle (heart weight 420 to 530 g; maximal wall thickness 16 to 23 mm), we considered these athletes to have "idiopathic concentric left ventricular hypertrophy" rather than hypertrophic cardiomyopathy since they differed from the usual patient with hypertrophic cardiomyopathy in several important respects: 1) the pattern of left ventricular hypertrophy was symmetric (concentric); 2) disorganization of cardiac muscle cells was not present in left ventricular myocardium (15); and 3) there was no clinical or echocardiographic evidence of genetic transmission of hypertrophic cardiomyopathy in first degree relatives (7). Some athletes with idiopathic concentric left ventricular hypertrophy (as well as hypertrophic cardiomyopathy) may show distinctive abnormalities of the intramural arteries to the sinoatrial or atrioventricular nodes, consisting of fibromuscular hyperplasia with thickening of the vessel wall and marked narrowing of the lumen (Fig. 5). Of note, the magnitude of ventricular wall thickening that athletes with idiopathic left ventricular hypertrophy exhibited at necropsy greatly exceeded that which could be considered "physiologic" left ventricular hypertrophy induced by athletic conditioning (25–32). It is also possible that idiopathic left ventricular hypertrophy (as described in our athletes) represents a variant of hypertrophic cardiomyopathy, which is not characterized by genetic transmission or asymmetric left ventricular hypertrophy. Alternatively, it is conceivable that some of these athletes may have had undetected severe systemic hypertension as the cause of their left ventricular hypertrophy.

**Congenital Anomalies of Coronary Arteries**

**Left coronary artery arising from right sinus of Valsalva.** Congenital malformations of coronary artery anatomy and distribution have been incriminated as the cause of sudden death in several young athletes (4,33–40). These potentially lethal malformations are usually of two types. The most common is anomalous origin of the left main coronary artery from the right (anterior) sinus of Valsalva. Cheitlin et al. (33) were the first to point out that the origin
Figure 5. Small arteries in the conduction system of two athletes who died suddenly. a, b, and c. The artery to the sinoatrial node of a 24 year old basketball player with hypertrophic cardiomyopathy (hematoxylin-eosin stain). d and e. Arteries to the atrioventricular node of a 17 year old basketball player with idiopathic concentric left ventricular hypertrophy (elastic van Gieson stain). a. Artery to the sinoatrial node has a markedly narrowed lumen as a result of extreme thickening of the arterial wall. b. Same artery as in a, but at a different level and at a lower magnification, showing calcium deposits in adjacent myocardial fibers (outlined by broken line). c. Higher magnification view of the calcium-containing fiber shown in b. d. Low magnification view of the portion of atrioventricular node tissue that penetrates the central fibrous body. Three distinctly abnormal small arteries are evident (arrows). e. Higher magnification of one of the vessels shown in d, but at a different level. The lumen is markedly narrowed because of thickening of the arterial wall. Original magnification: a, ×95; b, ×50; c, ×400; d, ×40; e, ×130; all reduced by 25%.

of both the left main and the right coronary arteries from the right sinus (emanating from either a common ostium or two separate ostia), with passage of the left main coronary between the aorta and pulmonary trunk, was a clinically significant malformation (Fig. 6).

The exact mechanism by which this coronary anomaly may produce sudden death is not definitively known. However, it has been postulated (33,35) that the important anatomic feature is the acute takeoff angle of the left main coronary artery from the right sinus, which results in a narrowing of the coronary ostium. The increased stroke volume caused by physical activity probably causes dilation of the ascending aorta; this, in turn, leads to an even greater acute takeoff angle with the coronary ostium assuming an even more narrowed "slit-like" configuration, presumably resulting in diminished coronary blood flow and myocardial ischemia. It is also possible that dilation of the aorta and pulmonary trunk during exercise "squeezes" the left main
Figure 6. Diagrammatic representation of congenital coronary artery anomalies capable of causing sudden death in the young athlete. **Left**, Normal anatomy is shown for comparison. **Center**, Anomalous origin of the left coronary artery from the right (anterior) sinus of Valsalva. The left coronary artery may have a common (or separate) ostium with the right coronary artery, which also arises from the right (R) sinus of Valsalva. Note the acute leftward bend at its origin and the posterior course of the left coronary artery between the aorta and pulmonary trunk (PT). **Right**, The right coronary (RC) artery may arise from the left (L) coronary sinus and show a similar acute bend at its origin before it courses between the great arteries. LAD = left anterior descending coronary artery; LC = left circumflex coronary artery; P = posterior (noncoronary) cusp.

coronary artery as it passes between these two great arteries (38).

**Right coronary artery arising from left sinus of Valsalva.** Recently, Roberts et al. (34) emphasized that the "mirror image" coronary anomaly in which the right coronary artery arises from the left sinus of Valsalva (and courses between the aorta and the pulmonary trunk) may also convey a risk for sudden death in young conditioned individuals (Fig. 6). In this lesion, an anatomic and pathophysiologic situation exists at the right coronary ostium, which is analogous to that previously described for the left main coronary artery ostium (when that vessel originated from the right sinus). With origin of the right coronary artery from the aortic wall behind the left sinus, there is an acute rightward takeoff of the right coronary artery, which may become obstructed with exercise as the acute angle increases further as a result of increased stroke volume and aortic dilation.

**Other variants.** Other unusual variants of coronary artery anatomy may be occasional causes of exercise-related sudden deaths in young conditioned individuals (35). These include single coronary artery (in which the left coronary artery is absent), hypoplasia of the right and left circumflex coronary arteries (Fig. 7) or origin of the left main, left anterior descending or right coronary artery from the pulmonary trunk. Also, one of us (W.C.R.) examined the heart of a 19 year old collegiate football player who died unexpectedly and showed at necropsy an intussusception of a large ramus intermedius coronary artery branch off the left main coronary artery; this intussusception occluded the arterial lumen for 1 cm of its length. Atherosclerosis was absent from any portion of the coronary tree, and myocardial fibrosis was not evident.

**Myocardial bridging.** It has been suggested that "tunneled" major coronary arteries (myocardial "bridges") constitute a potential lethal anatomic variant and may cause sudden unexpected death in otherwise healthy young individuals (41). Such tunneled major coronary arteries (usually the left anterior descending artery) are completely surrounded by myocardium for at least a portion of their course, and it has been postulated that in certain susceptible individuals the artery may be critically constricted during systole and produce myocardial ischemia. Necropsy studies, however, have shown that tunneling of the left anterior descending coronary artery occurs in at least 20% of all hearts. We, therefore, believe that there is no convincing evidence that a tunneled coronary artery may be responsible for such sudden catastrophes in young athletes.

**Aortic Rupture**

Young athletes may die suddenly because of rupture of the aorta (Fig. 8). At necropsy, such individuals have a decreased number of elastic fibers in the aortic media, an abnormality that is presumably responsible for intrinsic weakening of the wall (commonly known as "cystic medial necrosis") (Fig. 8B). Certain individuals with cystic medial necrosis may participate successfully in strenuous competitive sports for many years without experiencing a catastrophic event. Some of these athletes also manifest aortic regurgitation or features of Marfan's syndrome (including mitral valve prolapse) that may facilitate their clinical recognition.
Coronary Heart Disease

Coronary heart disease is infrequently responsible for sudden death in young athletes (1,42). We studied three such athletes (24, 26, and 28 years of age, respectively) who had coronary artery atherosclerosis when they died. Two of these athletes had severe (>75%) cross-sectional area luminal narrowing by atherosclerotic plaque of each of the three major extramural coronary arteries. One of these two had total occlusion of the right coronary artery by an antemortem thrombus and hemorrhage into an atherosclerotic plaque. He was a 28 year old professional football player with type II hyperlipoproteinemia who, at necropsy, had a large healed posterior wall myocardial infarct (42). On retrospective analysis of his medical history, it was apparent that he had experienced symptoms compatible with myocardial infarction about 45 days before death, and also had had frequent episodes of chest pain with exertion thereafter. During life, however, these symptoms had not been considered to be due to heart disease, but rather as manifestations of a traumatic injury.

One other patient had approximately 50% cross-sectional area luminal narrowing of the left anterior descending coronary artery by atherosclerotic plaque and no, or only minimal, atherosclerosis in each of the other two major extramural coronary arteries. However, the left anterior descending coronary artery was completely occluded by an antemortem thrombus 2 cm from the bifurcation of the left main coronary artery.

Idiopathic causes of death. Occasionally young athletes die unexpectedly and, even after careful gross and histologic examination of the heart at necropsy (often including microscopic analysis of the conduction system), no structural abnormalities that could possibly have been the cause of death can be identified (1,43). Some of these patients may, however, show patchy areas of fibrosis in the left and right ventricular walls. Obviously, one can only speculate on the potential etiology of such deaths. It is possible, however, that some of these catastrophes resulted from a primary arrhythmia (1,44) or exercise-induced coronary spasm (45).

Clinical Features

Sudden death in young athletes is rarely preceded by symptoms. On retrospective questioning of surviving relatives, we found that only 8 of our 29 young athletes who died suddenly had experienced transient symptoms (for example, lightheadedness, syncope or chest discomfort) that could conceivably have been cardiovascular in origin (1). Only 2 of the 29 athletes had the correct diagnosis established during life; 1 with obvious clinical stigmata of Marfan syndrome and the other with hypertrophic cardiomyopathy, in whom there was a family history of sudden death due to that disease.

Five other athletes were suspected clinically of having cardiac disease, and all died of hypertrophic cardiomyopathy. Each of these five individuals had a distinctly abnormal electrocardiogram and one had an abnormal left ventricular angiogram, but the correct diagnosis was not made clinically in any of the five. During life, three of the five athletes were mistakenly considered to have "athlete's heart syndrome" (46,47), one was thought to have a small ventricular septal defect and one was given the diagnosis of Wolff-Parkinson-White syndrome because his electrocardiographic pattern was characteristic of that dis-
order. After examination for possible heart disease, these individuals were permitted by the examining physician to continue participating in competitive athletics and each subsequently died on the practice field or during an athletic contest.

Other Less Common or Potential Causes of Sudden Death

The cardiac conditions described in this section have all been documented to cause sudden death in patients and, therefore, are potential causes of sudden death in highly conditioned athletes. However, in our own experience and after review of the available published reports, we could identify either no instances or only an isolated occurrence of sudden death in a highly trained athlete due to the following diseases.

Myocarditis. Myocarditis (with either a viral, undefined or other origin) has been traditionally considered to be an important cause of otherwise unexplained death in young individuals (20,48). Although myocarditis may occur in this context (with either no or relatively innocent prodromal symptoms), its prevalence and importance as a cause of sudden death in the young have probably been exaggerated owing to "overinterpretation" at necropsy of relatively small numbers of interstitial round cells. Jokl (49) reported a 25 year old runner who died after a 12 mile road race and proved to have unsuspected myocarditis. We also identified one exercise-related sudden death due to myocarditis in a 25 year old soldier who was not, however, a competitive athlete.

Mitral valve prolapse. Despite its high prevalence (probably about 5%) in the general population (50), mitral valve prolapse is not a frequent cause of sudden death in competitive athletes. Only approximately 40 subjects (average age about 35 years) with mitral valve prolapse and sudden death have been reported (51–53); death was rarely related to exercise and no subject was a trained athlete. In our series of 29 athletes studied at necropsy (1), one showed mitral valve morphology consistent with "floppy" mitral valve prolapse syndrome (that is, thickened, gelatinous and mildly scalloped leaflets with excessive mucoid material histologically) and a family history of mitral valve prolapse. However, this patient also had a marked increase in left ventricular mass, a finding that cannot be explained by the mitral valve prolapse syndrome: hence, this particular athlete may have had more than one cardiac abnormality that contributed to his unexpected death on the athletic field.

Aortic valve stenosis. Aortic stenosis is another disease that has traditionally been considered to be a common cause of sudden death in children and young adults (20). It is perhaps surprising, however, that on the basis of available information, aortic stenosis does not appear to be a frequent cause of sudden death in young athletes. This is probably due to the likelihood that this lesion will be identified early in life because of a loud heart murmur, thereby leading to prompt disqualification from competitive athletics.

Sarcoidosis. Sarcoid heart disease may cause sudden unexpected death in previously asymptomatic individuals. In a review by Roberts et al. (54) of 26 patients with cardiac dysfunction resulting from sarcoid granulomatous infiltration of the heart, 17 patients were reported to have died suddenly, including 6 in whom death occurred during exertion and was the initial manifestation of sarcoidosis. We evaluated a 25 year old professional basketball player who had recurrent ventricular tachycardias (including symptomatic ventricular tachycardia) before dying suddenly, and proved to have sarcoid infiltration of the heart at necropsy.

Abnormalities in the cardiac conduction system. Some investigators have suggested that abnormalities in the cardiac conduction system may be responsible for sudden death in athletes without other structural cardiac disease. Thiene et al. (55) reported a 26 year old cyclist whose only cardiac abnormality was a fascicle of right atrial myocardium that bypassed the atrioventricular node and connected with the His bundle. James et al. (56) described abnormalities of the small intramural artery to the sinus node in which the vessel wall was thickened and the lumen narrowed. We (1) also identified similar abnormal intramural arteries to either the sinoatrial or atrioventricular nodes in some of the young athletes dying with hypertrophic cardiomyopathy, idiopathic left ventricular hypertrophy or a mitral valve morphologically compatible with mitral valve prolapse syndrome.

Other conditions. Extremely uncommon diseases, such as QT interval prolongation syndrome (57,58) and arrhythmogenic right ventricular dysplasia (59,60), are potential causes of sudden death because of their intrinsic propensity to produce serious arrhythmias or sudden death. Although there are no reports of competitive athletes dying with these conditions, this may be due largely to the infrequency with which these lesions occur. In addition, Green et al. (61) reported on a 44 year old marathon runner who collapsed after completing 24 miles of a race, and at necropsy proved to have transmural myocardial infarction in the absence of extramural coronary artery disease. Exercise-related sudden death also has been described in individuals with such rare conditions as isolated thinning of right ventricular myocardium (62) and lipomatous infiltration of the heart (63).

Sudden Death in Older Athletes (≥35 years of age)

Increasing numbers of middle-aged or older individuals (≥35 years of age) are now participating in organized competitive sports and in extremely vigorous physical condi-
tioning programs (for example, jogging and swimming). Superior physical fitness in this age group (or at any other age), however, does not guarantee protection against exercise-related death. As is the case with the young competitive athlete, older athletes also may harbor occult cardiovascular disease and die suddenly and unexpectedly while participating in athletic activities (2,3,5,35,64-71). Nevertheless, the prevalence of such catastrophes is probably quite low considering the large number of individuals in the older age groups participating in sports (66).

Causes of sudden death. Unlike the more youthful athletes previously discussed, the cause of death in older conditioned athletes is usually not a congenital malformation of the heart. On the basis of the available data from several published studies (2,3,5,64-71), the cause of sudden death in the vast majority of older athletes is coronary heart disease. At necropsy, such individuals usually have severe (>75%) narrowing of the cross-sectional area by atherosclerotic plaque of one, two or three major extramural coronary arteries (left anterior descending, left circumflex or right) (Fig. 9), but rarely of the left main coronary artery. The remaining deaths in older athletes appear to be due to a variety of noncoronary causes such as hypertrophic cardiomyopathy (or idiopathic left ventricular hypertrophy), mitral valve prolapse or acquired mitral or aortic valve heart disease (3,5,70); in a few athletes, the cause of death could not be established definitively at necropsy (3,5).

Coronary heart disease. To date, about 100 older athletes who died suddenly of coronary heart disease have been reported in eight separate necropsy investigations (2,3,5,65-67,70,71). These individuals comprise a heterogeneous group of athletes, including runners (some in training for competitive long distance races) and joggers, and others participating in sports such as rugby and squash. Unlike the young competitive athletes who died of congenital heart disease, approximately half of the older athletes with coronary heart disease had experienced prodromal cardiovascular symptoms before death or had a known medical history of coronary artery disease. In addition, these athletes are usually described as having “severe” coronary artery narrowing at necropsy, although only a few studies provide

Figure 9. Sections of coronary arteries from a 49 year old man who ran an average of about 170 km/week and successfully completed six marathons and seven 80 km races, but died suddenly of coronary heart disease. The right (R), left anterior descending (LAD) and left circumflex (LC) coronary arteries are shown at the sites of maximal narrowing by atherosclerosis, both in the proximal (upper panels) and distal (lower panels) halves of the respective arteries.
detailed or quantitative morphologic descriptions of the severity and distribution of arterial narrowing.

Sudden death due to coronary heart disease may also occur in highly conditioned older athletes who have trained for and completed particularly long competitive races, including the marathon distance (26 miles) (Fig. 9) (2,69,71). Hence, marathon running itself does not provide "immunity to atherosclerosis," as previously suggested (72).

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