of championship performance who are found to have evidence of cardiac hypertrophy and enlargement, a slow resting heart rate, systolic ejection murmur and, occasionally, a variety of arrhythmias and conduction disturbances. This condition has been thought adaptive, benign and reversible, and restrictions on physical activity were not thought to be indicated. Nevertheless, occasional reports of sudden death in marathon runners with patent coronary arteries and the knowledge that only a history of athletic prowess deters a diagnosis of cardiomyopathy in a patient with unexplained cardiac enlargement, calls into question the presumed benign nature of this condition. Moreover, the adaptive response of a normal heart to isotonic and isometric exercise differs, and it is not known under what circumstances (if any) the response may become pathologic.

Just as a patient with the athletic heart syndrome is asymptomatic at diagnosis, so too may be persons from a kindred destined to manifest familial cardiomyopathy, who often have a subsequent malignant course characterized by congestive heart failure or sudden death. If these patients were initially capable of competitive physical exercise, it is possible that a spurious diagnosis of athletic heart syndrome would be made and the person encouraged to pursue limitless physical exertion, perhaps at some hazard.

In this paper we report the case of a young national champion tennis player who carried the diagnosis of athletic heart syndrome for six years following a complete cardiac evaluation in 1972. In this woman and several members of her family, all athletically active, concentric left ventricular hypertrophy was shown by echocardiography. The woman collapsed while playing tennis and died within 24 hours. On autopsy there was evidence of acute myocardial infarction, patent coronary arteries and diffuse cardiomyopathy.

**Reports of Cases**

**Case 1.** At the time of initial evaluation in 1972, the then 23-year-old patient had a negative past medical history and had participated competitively in a variety of sports including tennis, field hockey, basketball, volleyball, swimming and track. Her best time in the 100 yard dash was 11.9 seconds, in the 440 yard dash 61.1 seconds and in the two mile run 12 minutes 36 seconds. She was a recent singles runner-up in the All-Navy Tennis Tournament. The patient...
desired to be a naval aviator and was referred to the Cardiology Department, Naval Regional Medical Center, San Diego, California, after a routine x-ray study of the chest and an electrocardiogram showed left ventricular prominence and hypertrophy (Figures 1 and 2A).

Physical examination disclosed a resting pulse of 50, blood pressure 120/75/55 mm of mercury, a grade I/VI systolic ejection murmur at the left sternal border, and a third heart sound. A treadmill test done according to the protocol by Bruce was stopped after 18 minutes because the patient became fatigued, a maximum heart rate of 200 beats per minute having been achieved. No chest pain occurred; the postexercise tracing is shown in Figure 2B. An echocardiogram (Figure 3 and Table 1) disclosed increased thickness of the ventricular septum and posterior wall of the left ventricle with a normal ratio and normal left ventricular function. Findings at cardiac catheterization included a symmetrically thickened ventricle, mildly elevated right and left ventricular end-diastolic pressures (11 and 17 mm of mercury), an ejection fraction of 83 percent, normal coronary arteries, and no valvular disease or gradients. Hypertrophic subaortic stenosis (IHSS) was excluded by postpremature ventricular contraction pressure changes and isoproterenol infusion. A diagnosis of athletic heart syndrome was made.

Several months later the patient embarked on a professional tennis career, playing competitively until 1976. At that time, she experienced the onset of frequent 15- to 30-minute episodes of crushing substernal chest pains associated with shortness of breath, nausea and diaphoresis, occurring at rest. The frequency of discomfort remained stable at every other day, and the patient continued to play tennis and jog 1 to 3 miles a day.

In February 1978 (at age 28), the patient collapsed shortly after beginning a tennis match; she was resuscitated, but died within 24 hours. Her brief time in hospital was complicated by clinical, electrocardiographic and enzymatic evidence of acute anteroseptal myocardial infarction, adult respiratory distress syndrome and malignant ventricular arrhythmias.

Autopsy Findings

At necropsy, the heart weighed 500 grams and showed left ventricular prominence. The atria were mildly dilated. Transverse sections of the heart showed bilateral, diffuse, concentric hypertrophy

**Figures 1 and 2.**—Roentgenogram of the chest and electrocardiogram in an outstanding 23-year-old female athlete showing left ventricular hypertrophy. Item B in Figure 2 is the postexercise electrocardiogram.
which was more pronounced in the left ventricle. Although the right ventricle had a normal chamber size, the pronounced left ventricular hypertrophy significantly reduced left ventricular chamber size. The anterior, posterior and lateral walls of the left ventricle and interventricular septum were equally involved, averaging about 2.8 cm in thickness (Figure 4). The interventricular septum/posterior wall thickness ratio of the left ventricle was 1.0. At its point of maximum thickness the right ventricular free wall measured 0.9 cm. The coronary arteries and valves were normal.

At the left ventricular apex extensive regions of circumferential subendocardial infarct were noted. More cephalad, there were multifocal infarcts involving all regions of the ventricular wall, predominantly in the inner half. Several regions of old infarct were present in the posterior apical wall of the left ventricle.

Histologically, the zones of gross infarct showed characteristic changes including fiber disruption and cellular infiltrate consistent with an infarct of 24 to 48 hours duration. Sections of the coronary arteries were normal (Figures 5 and 6). Other myocardial lesions comprised diffuse interstitial broad band fibrosis and myocardial fiber hypertrophy which were present in sections from the walls of both ventricles. No changes of amyloidosis, sarcoidosis, storage disease or rheumatic heart disease were present.

Case histories of family members are given below.

Case 2. The father of the patient is a 60-year-old former semiprofessional athlete who was active in track, basketball, swimming and tennis. An excellent tennis player all his life, in recent years he has confined tennis to doubles because of eye surgical procedures. He has had no symptoms of cardiovascular disease, but gives a history of having “a large heart” all his life. During evaluation in 1972, he was found to have a resting pulse of 50, blood pressure 175/90/90 mm of mercury and a grade II/VI systolic ejection murmur at the left sternal border. An x-ray study of the chest showed left ventricular prominence and an electrocardiogram indicated left ventricular hypertrophy (Figures 7 and 8A). He exercised seven minutes on the treadmill and achieved a maximum heart rate of 135 beats per minute before the test was stopped due to shortness of breath. The postexercise tracing appears in Figure 8B. Echocardiographic results appear in Table 1.

Case 3. The 33-year-old brother of the patient in the index case is an aviator with an outstanding athletic record. In 1972 an x-ray study of the chest and an electrocardiogram showed left ventricular prominence. During evaluation in 1972 he completed 18 minutes on the treadmill without chest pain, arrhythmia, or significant postexercise ST segment changes developing. Echocardiographic results appear in Table 1.

Case 4. The 31-year-old married sister of

![Image](image_url)
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Figure 4.—Concentric hypertrophy, myocardial fibrosis and acute and old myocardial infarctions in a 28-year-old female champion athlete. Darkened areas show fresh subendocardial infarction.

Figure 5.—Microscopic section of anterior left ventricular myocardium using hematoxylin-eosin stain. Entire field shows infarction with an interstitial leukocytic infiltrate.

Figure 6.—Normal left anterior descending coronary artery representative of remaining coronary system.

Case 5. The 62-year-old mother has no history of competitive athletics or heart disease. Physical examination, electrocardiogram and x-ray study of the chest showed no abnormalities. Echocardiographic results appear in Table 1.

Discussion

The myocardial response to exercise has been studied extensively.14-19 While the resting left ventricular function of a sedentary person is comparable to that of a trained athlete,19 differences emerge during exercise. Increased oxygen needs, hence increased cardiac output, can be supplied by increases in heart rate, stroke volume, or both of these. In an untrained person this requirement is met chiefly by the heart rate increasing, while in an athlete both heart rate and stroke volume are utilized.15,16 The latter may be due partially to increased cardiac end-diastolic volume.17 Moreover, in an athlete there is an enhanced ability to extract oxygen peripherally.18

Both chronic isometric exercise and isotonic exercise result in increased left ventricular mass, while isotonics also produce increased ventricular volume.3,14,22 In an athlete these changes are brought about by an enlargement of myocardial fiber size rather than number, and occur without accompanying fibrosis (as in the volume stress of regurgitant valvular or primary myocardial disease; see Table 2).20-22 There has been doubt as to the universal adequacy of capillary flow to match an enlarging myocardial mass,22 such that stressed muscle fibers might ultimately suffer nutritional deficiency and hypoxia,24 or perhaps be prone to failure. In fact, elevated end-diastolic pressures have been recorded in young athletes,7

the patient was an outstanding athlete in high school, but subsequently injured her knee and retired from competitive sports. An x-ray study of the chest, electrocardiogram and echocardiogram are normal. Echocardiographic results appear in Table 1.
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...as well as sudden death with patent coronary vessels.8,9

Fortunately, from a clinical standpoint, the heart has never been shown to outgrow its own blood supply, with the possible exception of cases of congenital single coronary artery.25 Could familial cardiomyopathy be a second example, at least in the presence of exercise? This may have been the case in our patient.

The patient in the index case reported in this paper presented without symptoms in 1972, and evidence of left ventricular hypertrophy was noted. Secondary causes of ventricular hypertrophy such as hypertensive cardiovascular disease, aortic stenosis and idiopathic hypertrophic subaortic stenosis were excluded at catheterization and autopsy; nor were secondary causes of diffuse myocardial disease found to be present. A diagnosis of familial cardiomyopathy was entertained10-12 in light of the presence of several similarly affected family members, but this diagnosis was not established because of several considerations.

First, a review of 11 families with 47 members showing familial cardiomyopathy disclosed a mean age at diagnosis of 24 years, with most cases showing symptoms by the fifth decade.10 Our patient’s asymptomatic father in his 60’s mitigated against such a process. Second, while familial cardiomyopathy may be asymptomatic at discovery,10 enhanced performance is not seen.11,12 To our knowledge, in fact, there are no well-documented reports in the literature showing championship performance in a patient destined to die because of familial cardiomyopathy. Third, the criteria of Gott and coworkers1 for the diagnosis of the athletic heart syndrome were fulfilled: namely, a history of athletic endurance, biventricular enlargement, systolic ejection murmur, third heart sound, resting sinus bradycardia and increased indices of cardiac function.

In retrospect, we believe that the patient in the index case probably had a variant of familial cardiomyopathy26 with some features of the athletic heart syndrome. Her principal forms of exercise were isotonic. Yet, her echocardiogram in 1972 showed left ventricular hypertrophy without dilatation as did her father’s and brother’s echocardiograms in 1978. Often there is echocardiographic evidence of both left ventricular hypertrophy and dilatation in an endurance-trained athlete.18,22 Her resting electrocardiogram showed repolarization changes consistent with left ventricular strain (pressure or systolic overload). In contrast, the usual resting electrocardiogram in a trained athlete shows early repolarization with or without tall T-waves (volume or diastolic overload).27 Following treadmill exercise testing there was ST segment flattening and depression of greater than 2 mm consistent with an "ischemic

![Roentgenogram of the chest and electrocardiogram showing left ventricular hypertrophy in a 60-year-old former champion athlete (case 2). Item B in Figure 8 is the postexercise electrocardiogram.](image-url)

Figures 7 and 8.—Roentgenogram of the chest and electrocardiogram showing left ventricular hypertrophy in a 60-year-old former champion athlete (case 2). Item B in Figure 8 is the postexercise electrocardiogram.
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response" in both the patient (case 1) and her father (case 2). Such postexercise electrocardiographic changes are rarely seen in the athletic heart syndrome.22

Postmortem examination in the index case showed striking evidence of left ventricular hypertrophy which presumably progressed over the preceding years. The echocardiogram in 1972 showed septal and posterior wall thickness of 14 mm during diastole in comparison to 28 mm at necropsy in 1978. Even if the heart had arrested at end-systole, there had been significant progression of hypertrophy. In addition to hypertrophy there was evidence of old and new myocardial infarcts in the presence of patent coronary arteries. Clearly, either myocardial perfusion was inadequate to meet the demands of the hypertrophied heart or myocardial flow was redistributed, making the subendocardial zones more vulnerable to ischemic damage.

Because most reviews and case reports of the athletic heart syndrome fail to provide long follow-up or to examine other family members, it is possible that some athletes so labelled do, in fact, have underlying heart disease that will become manifest at a future date. For any individual case it may be difficult to differentiate cardiomyopathy from the athletic heart syndrome. In addition to cardiac enlargement and hypertrophy, ventricular gallop and systolic ejection murmur, both conditions may show a variety of arrhythmias, conduction disturbance and repolarization changes.6,10,27

Sudden death may be the first clinical sign of cardiomyopathy28 and, therefore, it is obvious that a correct diagnosis in a young patient with an enlarged, hypertrophied heart is essential. To this end, we believe that in addition to the diagnostic maneuvers described above, a complete history with examination of family members should be carried out. If cardiomegaly on x-ray films of the chest, left ventricular hypertrophy on electrocardiograms and measurably increased wall thickness on echocardiograms are found in family members (especially if not athletic), great caution should be exercised before a diagnosis of athletic heart syndrome is applied. Even if, as in our case, affected family members have been athletic, due to the highly variable clinical expression of familial cardiomyopathy10 the patient and relatives should receive close follow-up.

Summary

A young female athlete capable of championship performance presented in 1972 with left ventricular prominence, left ventricular hypertrophy with strain and concentric left ventricular hypertrophy on x-ray studies of the chest, electrocardiogram and echocardiogram, respectively. Several family members, including her asymptomatic 60-year-old father, were also found to possess both athletic histories and large hearts. In view of the history and asymptomatic status of older family members, a diagnosis of athletic heart syndrome was made. Six years later the patient suffered a cardiac arrest during exercise, and an autopsy showed evidence compatible with familial cardiomyopathy. It is concluded that patients with familial cardiomyopathy may be capable of competitive physical endurance initially and that physicians should defer a diagnosis of athletic heart syndrome in athletic patients with large hearts until proper diagnostic tests and family studies are done.

REFERENCES
2. Grande F, Taylor HL: Adaptive changes in the heart, ves-

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↑ increased ↓ decreased ↑↑ greatly increased

IHSS = hypertrophic subaortic stenosis
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Clinical Depression and Suicide

There exists a powerful clinical bias not to understand depression as an illness, but rather to explain it away. A recent study of completed suicides makes two striking and uncomfortable observations: . . . the vast majority of these patients were under the recent care of a physician, and . . . a review of their records showed that clinicians had documented sufficient signs and symptoms to make a diagnosis of clinical depression . . . without having done so.

—HENRY R. BLEIER, MD, Philadelphia

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